

Chapter 4

Social Disparities in the Evolution of an Epidemiological Profile: Transition Processes in Mortality Between 1971 and 2008 in an Industrialized Middle Income Country: The Case of Hungary

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Abstract The present paper seeks to understand the transformation of mortality patterns in Hungary, by which mortality inequalities by education began to appear in the early 1980s, continued to grow in the following 25 years, and now seem to be stabilising. The first part of this paper overviews the theoretical innovations of the last decades regarding the interpretation of cause-specific mortality dynamics, often referred to as epidemiological transitions theories, and their relevance for the analysis of mortality inequalities. The paper then analyses the cause-specific trends of mortality for two educational classes between 1971 and 2008. The trends were corrected for changes in the coding system and divided into linear (stagnating, increasing or decreasing) periods. Causes of death were grouped according to the relationship between the sequences of these periods for the two educational classes. The 57 causes of death were finally clustered into six groups. One group, which is dominated by nutrition-related and cardiovascular diseases, is largely responsible for the onset of mortality inequalities in 1980. The results imply that the quality of nutrition has diverged for the educational classes since 1980, and this fact has left its footprint on the pattern of mortality. The history of food production and availability seems to be in line with nutrition-related mortality, and it is argued that nutrition transition theory provides a very plausible explanatory framework for the growth of mortality inequalities.

Keywords Epidemiological transition · Hungary · Inequality · Historical development

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4.1 Introduction

Countries in Central and Eastern Europe (CEE) experienced rapid industrialisation under equalising state-controlled regimes, and entered into the globalising international economy two decades ago. Their transformation into service economies is still an on-going process. Over the past 20 years social inequalities have increased sharply, reaching medium-level income inequalities in an EU context, which is considered high by the citizens of these countries. In the present paper I look at the implications these changes have had on the level and distribution of mortality in Hungary, as an example of this group of countries.

All-cause mortality is considerably higher in CEE countries than in the rest of the European Union, but it was recently shown to correspond to the income level of these countries (Spijker and von Wissen 2010). On the other hand, inequalities in mortality by education have been found to be extremely high in all of these countries (Mackenbach et al. 2008). So far the explanation for these developments has only been provided within a larger context that applies to the whole of the Eastern European region, including not only CEE and Baltic countries but also countries like Russia, Belarus and Ukraine. One of main conclusions has been that they have not so far undergone the healthier life style changes that have occurred in Western Europe, and this has resulted in a “reversed epidemiological transition”, in which an elevated burden of cardiovascular diseases dominates the pattern of mortality (Vallin and Meslé 2004). Is this framework applicable to Central and Eastern Europe and does it explain the evolution of their cause of death pattern and high level of inequalities? If so, what role did income play in these processes and what are the specific social processes that triggered these developments?

In Hungary, inequalities in all-cause mortality were negligible during the 1970's and widened during the 1980's. The next one and a half decades brought a further, dramatic, increase in inequalities, which appear to have stabilised at this very high level for the past half a decade (Fig. 4.1). As regards broad groups of causes of death, the data suggest that the apparent similarities in all-cause mortality during the 1970s might be attributable to causes other than the lack of inequalities in living conditions between people with different education. This period was characterized by the over-mortality of the less educated from cardiovascular diseases and the over-mortality of the more educated from malignant tumours (Fig. 4.2 and 4.3). An explanation is called for which will look at the historical development of cause-specific mortality within the framework of the epidemiological transition. In this paper I shall review recent developments in epidemiological transition theory, and test the applicability of some of these theories to the evolution of cause- and education-specific mortality inequalities in Hungary between 1971 and 2008.

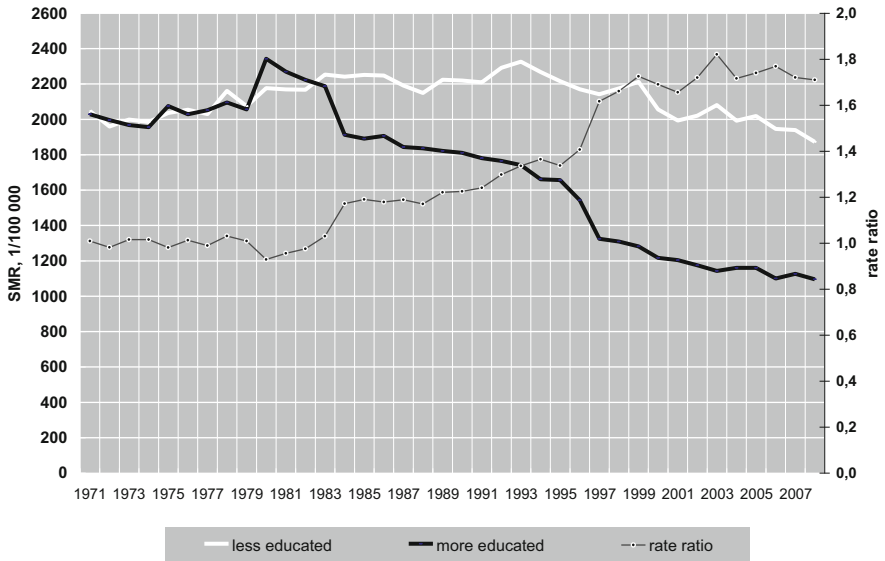


Fig. 4.1 Total mortality by education between 1971 and 2008, Hungarian population aged 30 and over

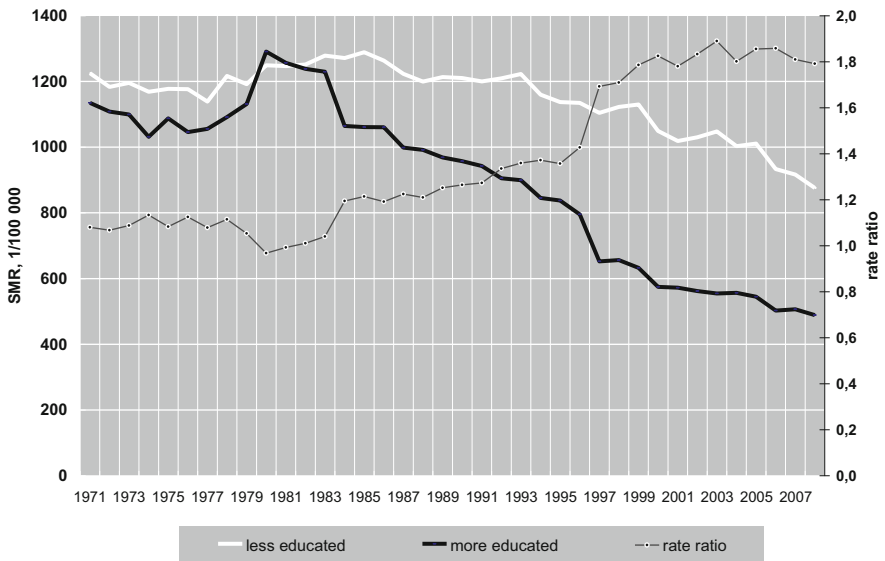


Fig. 4.2 Cardiovascular mortality by education between 1971 and 2008, Hungarian population aged 30 and over

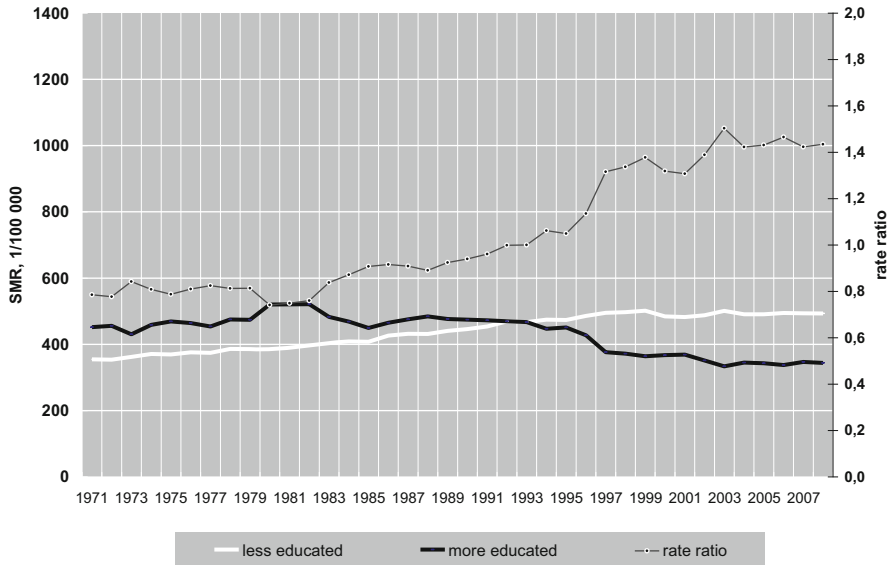


Fig. 4.3 Mortality due to cancer by education between 1971 and 2008, Hungarian population aged 30 and over

4.2 Understanding Changing Disease Patterns Over Time: The Epidemiological Transition Theory

Long-term mortality trends are commonly interpreted within the framework of epidemiological transition theory, outlined 40 years ago by Omran (1971). The original statements of the theory on mortality, fertility and population growth have already been tested, analysed, criticised and modified. By now, epidemiological transition theory and demographic transition theory have split: the first one has gradually shifted towards a focus on cause- and age-specific mortality patterns, while the second is now far more concerned with patterns of fertility and family formation.

The original postulates of Omran are, without doubt, of a heuristic nature: based on limited empirical basis (in its original form it was based on the long-term cause-specific mortality trends of just six countries), it provided a comprehensive picture of the evolution of cause-of-death patterns throughout the history of mankind. In a rather vague division of human history, three transitional phases were distinguished: the ‘age of pestilence and famine’, the ‘age of receding pandemics’, and the age of ‘degenerative and man-made diseases’ (Omran 1971). Stages were differentiated on the basis of average life expectancy, and age- and cause-specific mortality. During the first phase, which encompassed most of human history (the “pre-industrial period”, Omran 1998), mortality due to chronic malnutrition, endemic infectious diseases, and high prenatal and maternal mortality shaped the overall high level of mortality, which was further increased by epidemics, famine and wars in the “peak years”.

In the second stage, which started in the eighteenth or nineteenth century in Western societies, mortality declined considerably, mainly due to factors other than medical interventions: improved nutrition, improvement of personal cleanliness, ecological recession of certain diseases, better housing conditions and the start of using contraceptive methods. The cause-of-death pattern was less and less characterized by diseases caused by pandemics but communicable diseases—tuberculosis in particular—were still dominant. The third stage is characterised by the dominance of non-communicable diseases, such as diseases of the circulatory system and different types of cancer. From the perspective of the future development of the theory, the additional characteristics of the stages are less important, though Omran's approach, which has been modified several times by himself and others over the past 30 years, remained complex and aimed at explaining the whole of population dynamics.

The evidence which accumulated subsequent to Omran's original article, coming mainly from countries of the Americas, shows little correspondence with this original framework and offers an amazing variety of cause-specific mortality patterns and their changes over time (Albala and Vio 1995; Castillo-Salgado et al. 1999; Costello and Osrin 2005; Hill et al. 2007; Huicho et al. 2009; Marshall 1991; Vigneron 1989; Vigneron 1993). This evidence was incorporated into the original model as three models, the Classic, the Delayed and the Accelerated models (Omran 1983). Later on this was expanded to six models, the Classical Western Model, the Semi Western Model and four Non-Western Models: the Rapid, the Upper Intermediate, the Lower Intermediate and the Slow (Omran 1998). Other authors suggested a different classification of countries (Frenk et al. 1991) in order to incorporate new evidence that did not fit into the original sorting in the first form of the epidemiological theory. From the perspective of countries in Latin America, the concept of an epidemiological *transition* was in sharp contrast to the mortality experience of many countries of the region, which were characterized by a sharp divide between the mortality patterns of different population groups within one country. This experience questioned the choice of countries as the units of analysis, and even raised doubts about the usefulness of the whole concept of an epidemiological transition. Apart from total refutation, the experience of Latin American countries are best summarized as a "patchwork pattern" in which different social groups are often segregated geographically, and display diverse mortality patterns corresponding to different stages of the epidemiological transition. In other words, "different epidemiological worlds" live next to each other. For industrialised countries, on the other hand, a large collaborative study of WHO did confirm the previously proposed trend of age-specific death rates for two broad groups of causes of diseases in the last half of the twentieth century (Salomon and Murray 2002).

The more and more sophisticated classification, however, did not help to overcome one of the major theoretical drawbacks of the original theory. Despite the very complex, and somewhat apocalyptic, view of the future presented by Omran in his last article (1998), epidemiological transition theory presents a linear view of changes in mortality patterns, according to which more developed stages follow less developed ones, alongside with the course of 'modernisation'. This process may take place slowly or quickly, and with some variations, but it also follows a linear route. In

this respect, epidemiological transition theory does not differ from theories of modernisation propounded in the 1970's (Carolina and Gustavo 2003) and is very similar to the dominant view of demographic transition theories (Melegh and Őri 2003).

The theory of an epidemiological transition was attractive not only for public health researchers, it can also be viewed as a major contribution to the on-going debate of historians and historical demographers centred around the nature of mortality changes in the last centuries. In countries with a long history of collecting detailed mortality data, the distinctive phases of receding epidemics and the death toll of infectious diseases in general could be identified. Due to the great variability within the regions of one single country (for Sweden: Rogers and Nelson 1997), this further classification, unlike the contribution coming from the discipline of gerontology, has not become a commonly accepted modification of the original epidemiological transition theory. Olshansky and Ault (1986) carried out a detailed examination of the age-specific death rates of the US and pointed to the onset of an epidemiological phase that differs from the one specified as the third stage of the epidemiological transition in the original form of the theory. This fourth stage, they suggested, is characterized by the dominance of the same major causes of death as the third stage but with a continuing delay in mortality from some of these causes, leading to a further significant improvement in life expectancy. The new stage, called the 'age of delayed degenerative and man-made diseases' has become a standard part of the most commonly accepted form of the epidemiological transition theory.

Anthropology or 'evolutionary biology' has also made its contribution to refining epidemiological transition theory by adding a new transition stage, thus refining how the original theory divided up the other end of the historical time-scale (Armelagos et al. 2005). The addition of the "baseline" mortality pattern, called the 'Palaeolithic stage', however, is less relevant from the perspective of the current research. The approach of evolutionary medicine, which emphasises the links between the specific nature of human production, diet and other aspects of living conditions, and cause-specific mortality, can, however, be beneficial in interpreting contemporary mortality trends as well.

Another major discipline contributing to the further refinement of epidemiological transition theory was epidemiology itself. Recent trends in epidemiological research clearly exhibit some fragmentation. Epidemiology was first concerned with certain diseases in detail but recently more comprehensive approaches have emerged. Alongside the continuing research of the risk factors associated with specific diseases, trends in mortality due to major groups of diseases have also been studied and the results and hypotheses presented in the framework of "sub-transition models" such as cancer transition and cardiovascular transition (also known as the cardiovascular revolution).

The cancer transition is an extension of the classic transition theory that takes into account new discoveries on the role of infections in the development of certain types of cancers. The discovery of the presence of bacteria in the majority of stomach cancer cases promoted the recognition of how important infections are in cancer in general, though the infectious origin of certain other cancers (such as cancer of the cervix, testicular cancer and certain lymphomas) was already well-known. New discoveries triggered the formulation of "cancer transition theory" (Gerstein and

Wilmoth 2002), according to which there is a definitive restructuring process in operation within cancer mortality: those with an infectious origin lose their importance and other non-infection-related cancers emerge.

Most cancers, however, are known to be influenced by some major risk factors such as non-appropriate diet, smoking and excessive alcohol consumption. These well-known risk factors are now more and more closely linked to societal transformation processes, mostly of a global nature. From among these theoretical frameworks we note in particular the theory of a nutritional transition (Popkin 2006, Popkin and Mendez 2007). In this framework, major features of food production, distribution and several other characteristics of living conditions are connected to mortality patterns. Nutritional transition theory, just like epidemiological transition theory, divides human history into five distinctive patterns, out of which the fourth corresponds to the living conditions of contemporary CEE countries. The fifth profile describes the living conditions and dietary habits of the most health-conscious members of the most affluent countries. Labelling the phases not as stages but as profiles obscures the fact that these patterns are arranged in historical order so that they also represent some “developmental route”. The patterns, however, are connected with a large number of dimensions of actual living conditions. As regards the transition to the fourth (“obesity characterized”) profile, several processes, such as “supermarketisation”, are connected to several social processes like the demand for safer food, the changing opportunity cost of females’ time, technological changes, and changes in logistics and production systems. Altogether this transition is technically characterized by the growing importance of edible oil and animal products in human diet. Additionally sugar consumption is on the rise, often in the form of consuming sweetened beverages. The shift from high fibre intake to refined grains and additionally declining fruit and vegetable intake is also documented in many countries (Popkin 2006). Transition theories regarding other risk factors are less developed at the moment, but the term “smoking epidemic” is also in use and the influence of strong economic forces has already been recognised (Yach et al. 2007).

Regarding the other dominant groups of diseases, cardiovascular mortality has always been regarded as being strongly related to the epidemiological transition. Ischemic heart disease in particular often serves as a “marker disease” that indicates a country’s position in the phases of the epidemiological transition (Heuveline et al. 2002). Based on the observations of the contemporary occurrence and frequency of different cardiovascular diseases in different regions of the world, a complete framework for “cardiovascular transition” has gained popularity in the past few years. This framework provides a correspondence between particular cardiovascular diseases and stages of the epidemiological transition (Califf et al. 2010). The linearity of the occurrence of the stages is not stated but it is inherent in the logic of this scheme. The ‘pestilence and famine’ stage, with life expectancy around 35 years, is characterized by a modest share of CVDs in total mortality (5–10 %) and the dominant forms of cardiovascular mortality are rheumatic heart disease and other infection-related diseases of the circulatory system, cardiomyopathy in particular. The latter disease may also be connected to malnutrition. In the second stage the proportion of deaths caused by CVD grows to 15–35 % and cardiovascular mortality

is dominated by rheumatic valve disease, ischemic heart disease and haemorrhagic stroke. In this stage life expectancy reaches about 50 years. In the third stage, in which life expectancy reaches 60 years, the proportion of deaths due to cardiovascular diseases is greater than 50%. The dominant causes of death within CVDs are ischemic heart disease, and ischemic and haemorrhagic stroke. In the stage of ‘delayed degenerative diseases’ the proportion of CVDs among all deaths falls below 50% and life expectancy exceeds 70 years. Major cardiovascular causes of death are the same as in the previous stage, with the addition of congestive heart failure. Another important observation not exactly linked to stages is a major shift between stroke types: haemorrhagic stroke declines while ischemic stroke emerges (Lawlor et al. 2002).

A fourth stage also appears in some variations of the “cardiovascular transition” schemes. In some cases (Yusuf et al. 2001a) a stage of ‘health regression and social upheaval’ is visualised, characterised by the re-emergence of rheumatic heart disease and a new increase in ischemic heart disease due to increasing alcoholism. In the increasingly unregulated social environment, violence also becomes more common and hypertensive disease—which is otherwise characteristic of stage 2 according to these authors—also re-emerges. This visualisation, of course, relies heavily on recent Russian mortality trends. Other authors have predicted the emergence of heart failure as the main characteristic of a future scenario for CVD mortality (Bonnux et al. 1994; Gaziano et al. 2006).

Risk factors for cardiovascular diseases were found similar to the ones identified for cancers but the linkage between the single diseases and the exact role of single risk factors is less clear, with some exceptions. For two major different stroke types, for instance, different set of risk factors had already been identified (O’Donnell et al. 2010), though inappropriate diet, smoking and excessive intake of alcohol play an important role in the development of all cardiovascular diseases.

Predictions on the future trends of mortality and cause-specific mortality are not restricted to the field of cardiovascular diseases. In his last publication Omran (1998) also outlined a fifth stage, the ‘age of aspired quality of life with paradoxical longevity and persistent inequalities’. In this he expressed his hope for a future decrease of inequalities in survival, together with an expectation that there was a high probability of the re-emergence of infectious diseases. Nevertheless, the ‘invisible perils’ in the future of mankind are considered by Omran as well, such as the possibility of the evolution of an (airborne) virus with abilities similar to those of HIV; the potential misuse of atomic bombs; and high, uncontrolled population growth.

Omran’s view on the unpredictable role of infectious diseases is not unique. Several other authors foresaw the future emergence of new diseases and the re-emergence of “old” infectious diseases that were previously believed to have been controlled by medical interventions. Notable examples are the emergence of multi-drug resistant tuberculosis and avian flu. Following the emergence of the HIV/AIDS pandemic, the fear of new infectious diseases is spreading. Scientific examination reveals, however, that the majority of the emerging and newly recognised diseases are in fact not new but were known only in some peripheral regions of the world and have reached the consciousness of the wealthy only recently (Farmer 1996). A closer examination of

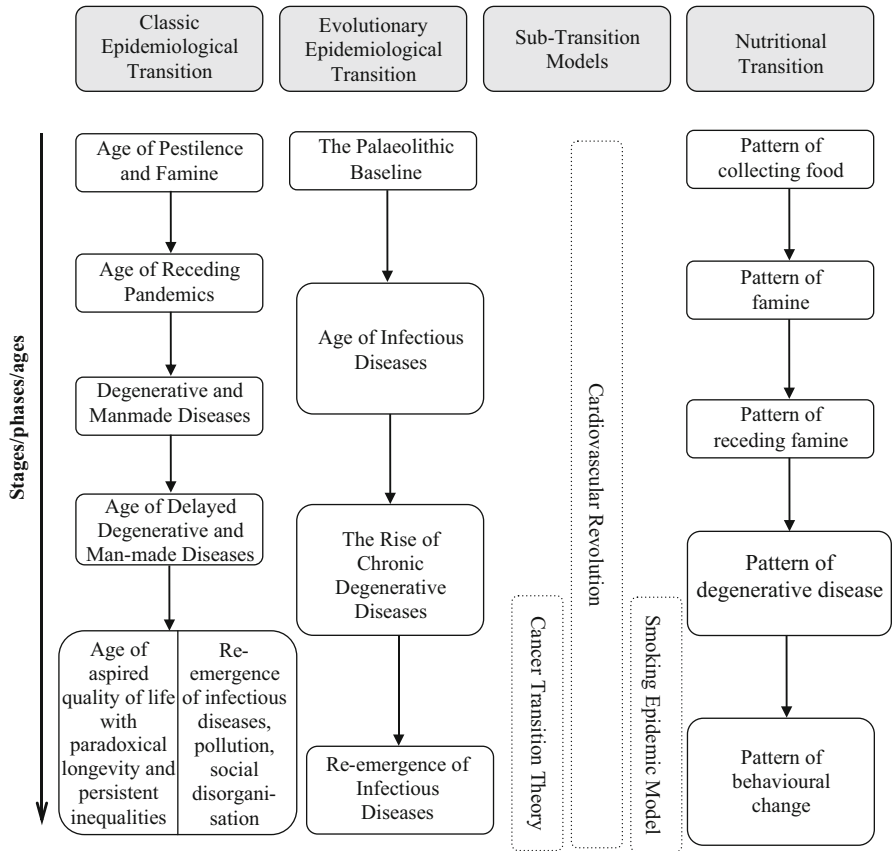


Fig. 4.4 Outline of the major variations of the epidemiological transition theory

the circumstances of the onset of 29 “newly identified” diseases during the 1990s pointed out that human activity played a triggering role in the majority of the cases.

In the integrated view of evolutionary medicine that divides human history only into three epidemiological transitions, the “third transition” is the new era of emerging and re-emerging infectious diseases (Harper and Armelagos 2010). The “end of the antibiotic era”, as this approach calls it, results mostly from the intensification of the globalisation process, especially that of the transportation system, which serves as a ‘virtual superhighway’ for pathogens.

Figure 4.4 outlines the theories providing a comprehensive explanation for changes in patterns of mortality and their phasing. While many epidemiological transition theories cover the whole of the history of mankind, others refer only to developments in the latest centuries, or even just decades. Most of them inherently treat the process of change in mortality patterns as “development”, i.e. as a linear, and in some respect hierarchical process. Possible reverses and uncertainties mostly appear regarding the latest stages—which is probably due to empirical observations being more numerous and diverse regarding the near past.

4.3 Understanding Social Disparities in Cause-of-Death Patterns

The issue of social disparities is present in nearly all approaches to the epidemiological transition. In most cases social inequalities in mortality or diverse mortality patterns that are characteristic of social classes, strata or groups are discussed in relation to major drivers (or causes) of the epidemiological transition. In some cases drivers or causes are stated only in general, like modernisation, industrialisation and urbanisation. In other cases propositions are well-formulated and corroborated by some empirical evidence. Omran, starting from his very first publication, continuously mentioned social disparities in mortality as well as the driving forces listed above but he did not provide a theoretical framework for the application of these in connection with particular mortality or disease patterns specific for single countries or population sub-groups.

McKeown (1976a, b, 2009; McKeown and Record 1962) studied the disappearance of infectious diseases in industrialising England and formulated his famous nutrition hypothesis. Detecting a time-lag between the almost complete disappearance of numerous infectious diseases, and a very notable drop in tuberculosis mortality, before the discovery of the appropriate treatment methods (mostly antibiotic drugs), he concluded that the major cause of decreasing mortality was the improvement in the living conditions and, in particular, the nutritional status of the population during the nineteenth century. The nutritional thesis provides an obvious explanation for social disparities in mortality, whose modified versions later appear in recently developed explanatory approaches.

Evolutionary medicine, with its anthropological orientation, considers the “Palaeolithic” baseline to have been free from social hierarchies in early human communities (Harper and Armelagos 2010). Notably they also focus on dietary habits. They suggested that there was a low mortality period before human communities settled down, as a result of their varied diet as well as small population size. Mortality started to grow when diet became heavily reliant on crops, which were unequally distributed across population strata. In parallel, the growth of average community size led to new, infectious, diseases becoming the leading causes of death. Based on this approach one can conclude that unequal access to food results in unequal resistance to diseases, thus inevitably leading to inequalities in mortality.

Historians and historical demographers, however, present a rather different picture of those centuries of human history which can be characterised by the dominance of infectious diseases. They suggest that some of the infectious diseases exhibit a “discriminative” nature: there is a long incubation period between the moment of infection and the development of the disease and the resistance of the host matters during the process of battling with these diseases. Other infections, by contrast, are “quick” enough not to allow time for the host (the human body) to develop resistance and they kill in a short time; consequently, they can be considered “non-discriminative”. Several infectious diseases, such as smallpox and mumps, have been observed to change over the centuries, as their originally “non-discriminative” nature turned into “discriminative”. It is still unclear if the changing nature of some formerly

fatal diseases is due to increased community-level resistance to those diseases or merely to the changing nature of the disease-scape. However, the disappearance of some infectious diseases, notably the plague, is still explained in several alternative ways (Slack 1981).

During early modern times, when infectious diseases dominated mortality, the excess mortality of those in disadvantaged social positions was likely to be more pronounced in those causes of death that were connected with epidemics and pandemics. According to historical demographers, excess deaths were indeed connected to the availability of food, though this relationship was largely influenced by the effectiveness of supportive networks (Bengtsson et al. 2004), which helped to mitigate the effect of economic hardships (e.g. famine). Regarding the plague outbreaks in London, it was observed that the locus of epidemics moved from the central, relatively wealthy parishes to the poorer suburban ones during the seventeenth century. Overall, it is likely that social disparities existed in the era of infectious diseases, though their importance might have changed over time, partly due to factors that operated independently of social organisation and human activity and partly due to greater awareness and ability to cope among the wealthy (Slack 1989; Hall 2008).

The early industrial era inevitably brought large mortality inequalities, which are well documented for some countries. Time series of mortality data by social groups, however, are not available for many countries. Studies using time series on income inequality for industrialised countries have suggested that mortality inequalities were narrowing from the first years of the twentieth century until about 1970, and widening afterwards. Detailed British data (Pamuk 1985) has reinforced this view. This process probably took place in varying ways in different regions: in Sweden no sign of the emergence of social inequalities in mortality was found till the 1950s (Bengtsson and Dribe 2011).

Theoretical explanations for modern inequalities have emerged in the fields of both epidemiology and sociology. In the epidemiology of cardiovascular diseases a particular “disease mobility” was observed first: in the beginning of the twentieth century myocardial infarction used to be the disease of the affluent in developed North American and European countries, but in the 1960s and the 1970s mortality rates due to infarction started to decline earlier and more rapidly among the better educated and the better off (Marmot et al. 1991; Kaplan and Keil 1993). These experiences led to the formulation of the social “following pattern” of diseases. Based on the concept of the diffusion of innovation, Pearson (2003) suggested an “adoption theory”: population groups with higher education and/or better income adopt new ideas, products and behavioural patterns more readily. Once a risk factor is recognised, it first becomes public knowledge among those with higher social status, mediated by health education or mass media. The messages reach the poorer and/or less educated groups of societies later.

The life course perspective for understanding the occurrence of chronic diseases also originates from cardiovascular epidemiology (Forsdahl 1978; Kuh and Ben-Shlomo 1997). Its scope, however, is much wider, identifying risk factors that act during the *in utero* period and early childhood, risk factors which are associated with the social position of the parents (Davey-Smith and Hart 2002). From the point of view of social sciences, these findings call for integrating intergenerational

mobility into epidemiological transition models that are used to understand the particular mortality patterns of single countries.

In the social sciences the “fundamental causes” concept was introduced in order to understand the relationship between socio-economic status and disease. These fundamental causes do not refer to causes of death but to dimensions of social position which are causally linked to resources that can be used to avoid risk or to minimize the consequences of diseases once they occur. Resources include money, knowledge, power, prestige and interpersonal relationships. Fundamental causes act, according to the proponents of this idea, when new diseases, new risk factors or new knowledge on risk factors emerge or new medical technologies are introduced (Link and Phelan 1995). In these cases living conditions and access to resources act directly to grant or restrict different groups’ access to, and application of, the new technology. Social position, therefore, is the fundamental cause of a disease (or death) and not a “proxy”, as it was previously treated in epidemiological research.

The concept of fundamental causes has only recently been applied to the analysis of cause-of-death patterns (Miech et al. 2011). The examination of education-specific mortality inequalities and their dynamics over the last decades of US history aimed at testing the fundamental cause hypothesis. A large number of causes of death (85) were included in this examination. In accordance with the concept of fundamental causes, the analysis found increasing inequalities for most “emerging” causes, e.g. those whose overall rate was in an increase.

Omran’s classic paper on the epidemiological transition (1971) positioned Hungary together with the rest of “Eastern Europe”, in the same model as Japan. Mortality developments have diverged significantly since then. The latest additions to the concept of epidemiological transition provide no direct guidance for understanding overall mortality trends and educational inequalities in mortality. Detailed knowledge has accumulated on the changes in mortality profiles in developed high-income countries. Mortality trends, especially the burden of infectious and non-communicable diseases, are widely discussed with regard to low income countries. Industrialized middle-income countries seem to be neglected in the discussion of the epidemiological transition. In order to fill this gap, first we examine the applicability of one of the previously outlined theories that focus primarily on other regions of the world: the plausible “following” hypothesis. The higher overall level of mortality as well as the cause of death patterns in Hungary (and other CEE countries), often referred to as “lagging behind” those of Western Europe, might be interpreted as the mortality pattern of a society in which large population segments who are “lagging behind” produce an overall “delayed” cause-of-death pattern and large mortality inequalities at the same time. If this proposal were true and meaningful, one would observe the same mortality dynamics for the more and the less advantaged segments of the population but with some time lag regarding the latter group. Existing data allow us to examine these processes by education only: I shall therefore compare the mortality development of the less and the more educated Hungarian adults. As a contrast I also examine the applicability of another popular branch of theories known as risk behavioural factor approaches, in particular, the possible role of nutrition in shaping cause-of-death pattern differences.

4.4 Data and Methodology

Mortality data for Hungary, by education, are available from 1971. For the period between 1971 and 2008 we calculated age-standardized cause-specific mortality rates by education for the population aged 30 and above. Cause-specific death rates were also calculated for the whole population and by education groups. Data on the number of deaths by education is provided by the mortality register of the Hungarian Central Statistical Office. Corresponding population estimates and forecasts were prepared by László Hablicsek, based on census data from 1970, 1980 and 1990 (Hablicsek and Kovács 2007). Underlying causes of death were included in the analysis. Education level was dichotomized: high (completed 12 years and passed the Matura exam) and low. These two groups will be referred to as the less and the more educated.

Selecting the relevant causes of death was a multi-stage process. First we selected causes cited in discussions of the epidemiological transition theory that linked their theoretical considerations to empirical analysis. The starting point, however, was the broad categorization into the two distinctive groups of causes of death which came out of the WHO Global Burden of Diseases study (Salomon and Murray 2002). Group 1 included the infectious diseases; diseases of the pulmonary system and several diseases connected to malnutrition and maternal mortality. Group 2 encompassed all other diseases, except the external causes: injuries, homicide and suicide. Looking at a large number of countries over shorter or longer observational periods (from 1950 to 2000) and taking into account total mortality and wealth (as measured by GDP), Salomon and Murray (2002) found no consistent relationship between external causes and total mortality or wealth, and we decided therefore to leave them out of the present analysis.

The next step in selecting the causes of death was based on those considerations which have been summarized in the introduction. Additional results from studies that analysed time trends for a number of diseases in specific countries with regard to the epidemiological transition were also included, particularly studies on the epidemiological transition in the Netherlands (Wolleswinkel-van den Bosch 1996; Wolleswinkel-van den Bosch et al. 2007) and in Canada (Lussier et al. 2008). For Group 1 causes, the identification of nutrition-related, pulmonary or maternal causes of death is not problematic. The large group of ‘infectious and parasitic diseases’, as the International Classification of Diseases calls it, was much more difficult to break down into smaller and meaningful causes of death, because if anything is clear from the literature, it is that infectious diseases are generally declining but they still vary significantly by country. Therefore we decided to select all those causes for which more than 100 cases were found for each year during the period between 1971 and 2008. This procedure resulted in a list of one disease: tuberculosis. We also added the “new diseases” such as HIV/AIDS and newly recognised and antibiotic-resistant infectious diseases. These categories turned out to be almost empty. In practice, the study also includes a number of infectious diseases which are traditionally classified under pulmonary diseases (such as influenza, pneumonia) or other major disease groups (peptic ulcer, appendicitis), or whose coding in some periods overlaps other broad cause-of-death groups (meningitis, enteritis).

Group 2 included different types of cancers and cardiovascular diseases, divided up according to those “sub-theories” of the epidemiological transition which we briefly introduced earlier. For cardiovascular diseases, the categorisation was based on the list of diseases that appear in different versions of the “cardiovascular transition”. Apart from these, some other distinctions were also made according to major coding categories such as chronic and acute ischemic heart diseases. Among cancers, we distinguished in particular all those cancer types which are connected with infections. A further distinction was made by major risk factors, including not only smoking, excessive drinking and obesity, but also environmental and occupational exposures (for a short summary see Table 4.1). This categorisation, however, does not lead to easy interpretation, due to the pervasive and complex nature of the everyday operation of risk factors. Some other diseases, specifically discussed by certain authors with respect to the epidemiological transition, such as Alzheimer and Parkinson’s Disease, were also added. The list of the causes of death that we selected for analysis is included in the Appendix, together with the coding used. Age-specific death rates by the selected causes of death (where possible) were used to create standardized mortality rates using the European standard population.

Mortality trends, resulting from the standardisation process, did not form continuous time series in most cases, as illustrated in Fig. 4.5. There were three different ICD coding versions in operation during the observed period, and in addition, “automatic coding” was introduced in 2005, which again affected the structure of the (underlying) causes of death, as if another new ICD version had been introduced. ICD-9 was introduced in 1979 and ICD-10 in 1996. First we fitted the different versions of ICD codes, often with the help of literature, in order to achieve the same content for each disease over time. When code-fitting was not obvious, we relied on code-fitting used by others (Wolleswinkel-van den Bosch et al. 1996; Wolleswinkel-van den Bosch et al. 1998; Hashibe et al. 2009; Lawlor et al. 2002). The resulting time series called “original values” still did not construct continuous curves in this study.

There are three known methods to deal with the changes of ICD coding system. The first one, the “double or bridge coding” would require coding death in a certain period according to both the outgoing and the new coding systems. This task was carried out only in 2005 for the Hungarian mortality data. The second method follows the exact matching of the disease categories by four-digit coding (Meslé and Vallin 1996). This method was partly used in this study but only for some specific causes of death. After establishing the coding we followed a third method of fitting the curves (Janssen and Kunst 2004) but applying a simpler method than they did. First the obvious outliers were excluded from the original time series, judged by visual observation. Then, based on standardized values presented in 2000, 2001, 2002, 2003 and 2004, a linear prediction for 2005 was compared with the actual value for each analysed cause of death. The ratio of these two values provided a coefficient with which we fitted the values for the period between 1996 and 2004 in order to have a continuous time trend. This procedure was repeated twice to fit the values taken in the period between 1971 and 1978 and between 1979 and 1995. The fitted curve can be rather different from the one based on the original values, as demonstrated by Fig. 4.5, and should be treated as an estimation for the period between 1971 and 2004.

Table 4.1 Major risk factors for selected cancer types. (Source: Parkin 2006; Dalton-Griffin and Kellam 2009; Calle and Kaaks 2004; Anand et al. 2008; Boffetta and Nyberg 2003)

Cancer type	Infections		Obesity		Smoking		Environmental factors				
	Strongly related to infections	Some connection to infections	High (≥ 2) relative risk for obese people	Moderate relative risk between 1 and 2	Strongly connected	Moderately connected	Alcohol	Asbestos	Air pollution	Drinking water with high arsenic content	Chlorate and nitrate in drinking water
Oral		X			X						
Oesophagus		X		X			X				
Stomach	X			X							
Colorectal	X		/X/	X		X				(x)	
Liver	X			X		X				(x)	
Gallbladder											
Pancreas			X			X					
Larynx			X		X			X			
Lung					X				X		
Melanoma											
Other skin											
Mesothelioma								X			
Breast				X							
cancer											
Cervix	X						X				
Uterus			X								
Ovary											
Kidney											
Bladder		X		X							
Non-Hodgkin disease		X									
Leukaemia		X									
											X

X Well established association, (x) Less established association, /x/ different risk profiles for men and women

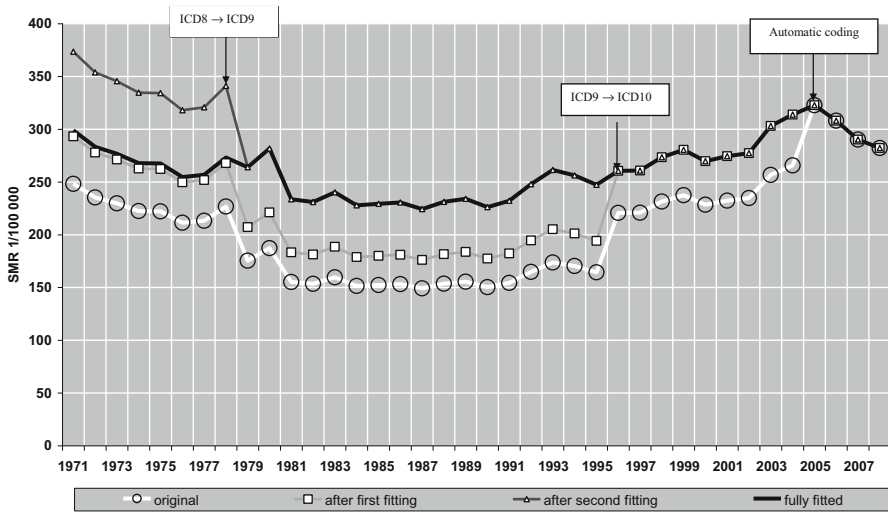


Fig. 4.5 Estimating the real value of mortality from chronic ischemic heart diseases: the fitting procedure

Fitting coefficients were calculated by causes of death, but always for the entire population. The same coefficients were used to fit curves for those with lower and higher educational background. The values of the coefficients, listed in the Appendix, provide an overview about the reliability of the estimated time series : the closer the coefficients are to 1, the higher the reliability. No fitting was applied in the case of those causes which were too small to calculate standardized rates or for those which showed outliers “too often”, such as influenza. Overall mortality trends were similarly not fitted (Fig. 4.1).

We now turn to consider the relation between the two estimated mortality time series for groups with lower and higher education. For certain causes of death, almost exclusively in those years when the annual number of deaths is very low, it was not possible to determine definitive relations since the low number of deaths did not allow for standardisation, so fitting was also not applied. Therefore the general trends of overall mortality due to these causes are difficult to establish. This fact is well illustrated in the case of obesity. From this cause less than 20 deaths were reported annually between 1979 and 2004, but about 200 in the following 4 years. As for inequalities, a clearer picture emerges from the distribution of the number of deaths: most of them appeared among the less educated. Deaths due to nutritional anaemia, malnutrition and obesity, as well as maternal death almost exclusively happened among those with lower education.

For other rare causes of death such as HIV/AIDS and “newly emerging infectious diseases”, however, no such pattern evolves. HIV/AIDS mortality was the highest in 1994, when 32 deaths were attributed to this disease. The number of cases declined afterwards and people with lower and higher educational attainment seem to be equally affected. Among the newly emerging diseases only 61 deaths were reported from 2009, again distributed proportionally between the educational classes.

After disregarding the above-mentioned causes of death, we categorized the remaining causes by the relationship between the two mortality time series displayed by the groups with lower and higher education. The classification of the relations rested on a simplified view of the time series. Given that we worked with estimated values in the classification, the dynamics represented by the time series were the focus. The time series were broken down to linear (growing, stagnating or declining) phases and the classification was based on the relationship between the sequences of these phases by causes of death, presented by the two mortality time series. Time series were broken down into phases using join-point regression analysis, with software provided by the National Cancer Institute of the United States.¹ This regression is for analysing trends and the software fits data in the simplest possible sequence of linear trends which are connected by the join-points. First a linear trend for the overall period is fitted, then trends with a growing number of joint-points are also fitted and their significances are tested against the Null-hypothesis (e.g. having 0 join-points). The tests of significance are based on a Monte Carlo permutation test. The breakdown of the time series was successful in most of the cases, though the method applied involves some uncertainties. The location of the join-points is provided together with confidence intervals, which were often very wide, covering even 8–10 years. In the following classification only those periodicities were considered when confidence intervals for the joint points were shorter than 8 years. Uncertainties were taken into account in all those cases when confidence intervals were wider than 3–4 years. The sequences of linear trends and the corresponding set of join-points by cause of death are not given here but are available from the author.

To examine the “follow-up” hypothesis, first one has to give a clear definition of a follow-up pattern of two curves. The method chosen for this analysis was not to construct a general definition but first to regard the estimated mortality time series for the two educational groups, then to classify them by their type of relation and then to examine the possible interpretations of their being “follow-up” by type.

4.5 Results

The application of this method resulted in six different groups of diseases, according to the relationship between the mortality trends estimated for the more and the less educated. This classification allows us to investigate the possibility of providing a proper definition of follow-up. In the case of diseases with strongly declining mortality (Type I) the definition of follow-up is not obvious at all. The dynamics of decline did not provide any meaningful definition of follow-up, since for the major diseases of this category (pulmonary tuberculosis, haemorrhagic stroke and cancer of the stomach) the timing of strongly declining and the less strongly declining periods, represented by the mortality of the less and more educated, mostly coincide (Fig. 4.6). The existence of sequences of declines with a different pace also means

¹ <http://surveillance.cancer.gov/joinpoint/>.

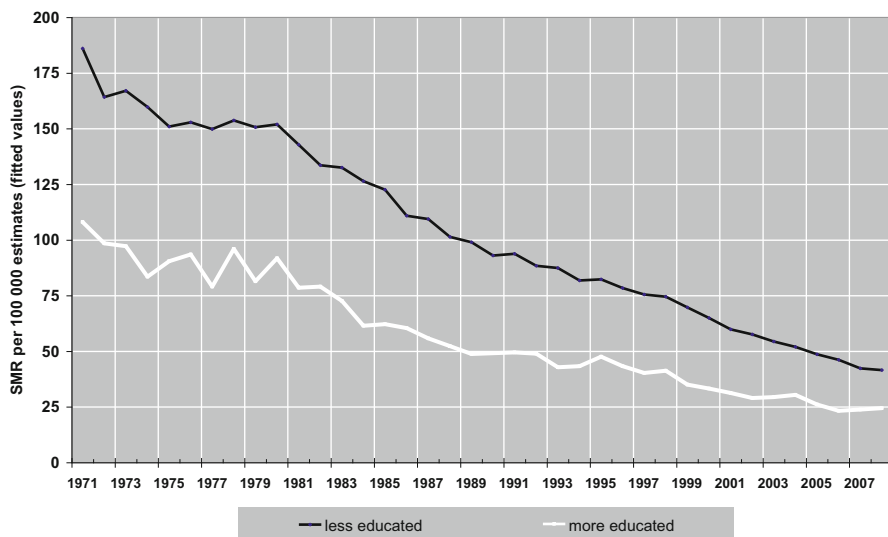


Fig. 4.6 Mortality due to haemorrhagic stroke by education 1971–2008, Hungarian population aged 30 and over, representing type I causes of death

that a definition based simply on when mortality of the less educated reached the mortality level of the better educated also gives no clear-cut answer: for instance, the value of tuberculosis mortality of the more educated in 1983 was reached 2 years later by the less educated, but the values for the more educated in 1988 or in 1996 were reached, by the less educated, only 12 or 9 years later, respectively. In the case of influenza, the level of fluctuation highly exceeds the level of inequalities. For rarer diseases that also belong to this class of causes of death, temporal but irregular high peaks of mortality among the better educated would make it difficult to define a follow-up pattern (Table 4.2).

In the case of some other diseases, mortality of the less and the more educated also shows similar sequences of periods of linear trends, but the overall trends are not declining (Type II, Fig. 4.7). Inequalities change little or not at all over time and the mortality of the more educated never (in “regular” cases such as the hypertensive diseases of the circulatory system or cervical cancer) or just in exceptional years (in the case of mesothelioma and epilepsy) reaches the level of the less educated. Providing any follow-up definition seems meaningless in these cases (Table 4.2). In a number of diseases, however, the sequences of the linear periods of different types are also similar for the less and the more educated, but the overall dynamics of the curves turn to be very different. For these causes of death mortality levels are quite similar at the beginning of the period considered here, but at a certain point of time mortality of the two groups starts to diverge quite distinctly (Type III, Fig. 4.8).

Regarding most diseases in the class of Type III mortality, negligible differences in mortality characterise the beginning of the observed period and then the same types of linear trends apply to both educational groups, but the levels of mortality

Table 4.2 Types of relation between the mortality of the less and more educated

Type	Sub-type	Cause of death	Possibilities to interpret the relation of the two mortality time series as follow up	Other relevant information regarding the relation of the two mortality time series
I. Diseases with strongly declining mortality in both educational groups	Continuously higher mortality for less educated but narrowing inequalities	Pulmonary tuberculosis	NOB	
		<i>Hemorrhagic stroke</i> (see Fig. 4.6)	NOB	
		Cancer of the stomach	NOB	
		Other tuberculosis	NOB	
II. Parallel sequences of growing, stagnating or declining periods of mortality from these diseases for more and less educated with stable mortality differences favouring the more educated	Slight irregularities in the declining pattern	Rheumatic heart disease	NOB	
	Fluctuating mortality	Influenza	NOB	
	The more educated had occasionally higher mortality	Appendicitis	NOB	
		Multiple sclerosis	NOB	
		Hodgkin disease	NOB	
		<i>Hypertensive diseases of the circulatory system</i> (see Fig. 4.7)	NAT	
	Regular			
		Cancer of cervix uteri	NAT	
		Mesothelioma	NAT	
		Irregular in terms of mortality differences: more educated have occasionally higher mortality	Epilepsy	NAT

Table 4.2 (continued)

Type	Sub-type	Cause of death	Possibilities to interpret the relation of the two mortality time series as follow up	Other relevant information regarding the relation of the two mortality time series
III. Parallel sequences of growing, stagnating or declining periods of mortality among more and less educated with little or no over-mortality of the less educated at the start, then inequalities widen dramatically to the benefit of the better educated	Confirmed by the joint-point analysis	Meningitis	NOB	1982 (W)
		Pneumonia	M	1985 (W)
		Chronic bronchitis	C	1980 (W)
		Emphysema	NOB	1980 (W)
		<i>Chronic ischemic heart disease</i> (see Fig. 4.8)	M	1980 (W)
		Cardiomyopathy	M	1980(W)
		Arrhythmias	M	1979–80 (W)
		Heart failure	M	1983 (W)
		Non-rheumatic valve disease	M	1990 (W)
		Atherosclerosis	NOB	1983 (W)
		Other disease of the veins	NOB	
		Cancer of the oesophagus	M	1986 (W)
		Gallbladder cancer	NOB	1982–92 (W)
		Melanoma	M	1981 (W)
		Other skin cancer	M	1982 (W)
		Diabetes	M	1983 (W)
		Cirrhosis of the liver	M	1977 (W)
		Cancer of uterus ^a	M	1980 (W)
		Mental disorders	M	1982 (W)
	With some irregularities	Cancer of larynx	M	1989(W) ^b

Table 4.2 (continued)

Type	Sub-type	Cause of death	Possibilities to interpret the relation of the two mortality time series as follow up	Other relevant information regarding the relation of the two mortality time series
IV. The two educational groups start with the same type (inclining, declining or stagnating) of trend and mortality from the disease is higher for the better educated. Mortality of the better educated changes its trend in a certain year for the better and finally huge over-mortality of the less educated is present	Confirmed only by visual inspection	Pulmonary heart diseases	M	1983 (W)
	Only mortality of the better educated shows major trend change during the period resulting in higher mortality among the less educated by the end of the period	Enteritis	C min 23y	1985 (T)
		Cancer of the colon	C min 15y	1993 (T)
		Cancer of rectum	C min 27y	1981 (T)
		Cancer of the liver ^c	M	1982 (T)
		Cancer of the pancreas	C min 18y	1990 (T)
		<i>Cancer of bronchus and the lung (see Fig. 4.9)</i>	C min 19y	1989 (T)
		Cancer of kidney	C min 19y	1989 (T)
		Cancer of the bladder	C min 14y	1994 (T)
		Ovarian cancer	C min 6y	2002 (T)
		Cancer of the brain	C min 29y	1979 (T)
		Leukaemia ^d	C min 28y	1980 (T)
		Diseases of the digestive system other than cirrhosis	C min 23y	1983 (T)

Table 4.2 (continued)

Type	Sub-type	Cause of death	Possibilities to interpret the relation of the two mortality time series as follow up	Other relevant information regarding the relation of the two mortality time series
V. Steady but profoundly different trends are shown by the two educational groups during the whole period starting with higher mortality of the better educated	Both educational mortality curves change trends significantly during the period	<i>Acute ischemic heart disease (see Fig. 4.10)</i>	Y, Follow-up time: 17y	1981 and 1998 (T)
	Mortality levels are similar at the end	Ischemic stroke Breast cancer Aortic aneurysm	Y, Follow-up time: 17y Y, Follow-up time: 7y M	1980 and in 1997 (T) 1992 and in 1999 (T)
VI. Not classified	Mortality is much higher for the less educated at the end	Cancer of the oral cavity Cancer of thyroid <i>Prostate cancer (see Fig. 4.11)</i> Non-Hodgkin disease Peptic ulcer	M M M M M	
		Parkinson disease Asthma		

NOB Not obvious, *MAT* Not at all, *C* Only with compromises, *Y* Yes, *M* Meaningless, *W* Calendar year from which mortality of less and more educated start to diverge, *T* Calendar year from which major changes in trend(s) appear—coinciding with a join-point follow-up time in years

^a Mortality of the less educated is higher at the start of the period, too

^b In some sense, 1982 can be also a turning point

^c Trend-change appeared in the mortality of less educated

^d Mortality reaches about the same level in both educational groups by the end of the period

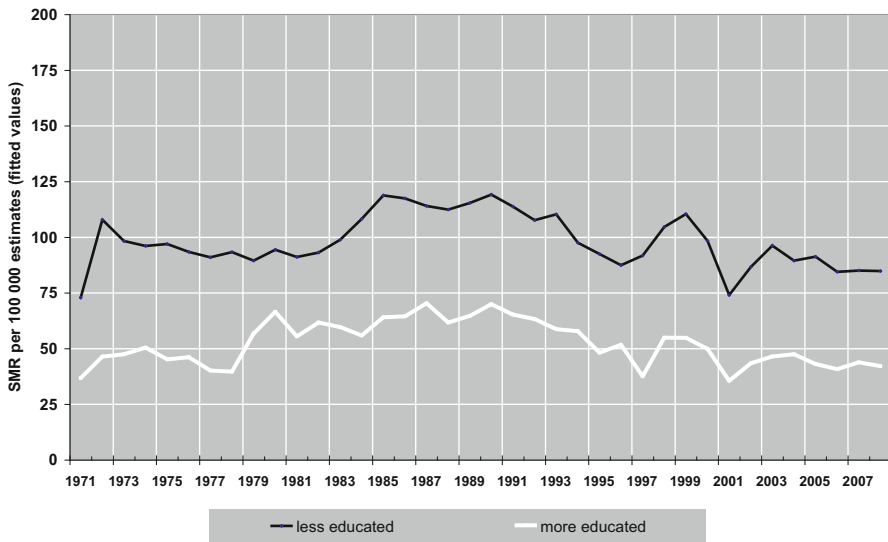


Fig. 4.7 Mortality due to hypertensive diseases of the circulatory system by education 1971–2008, Hungarian population aged 30 and over, representing type II causes of death

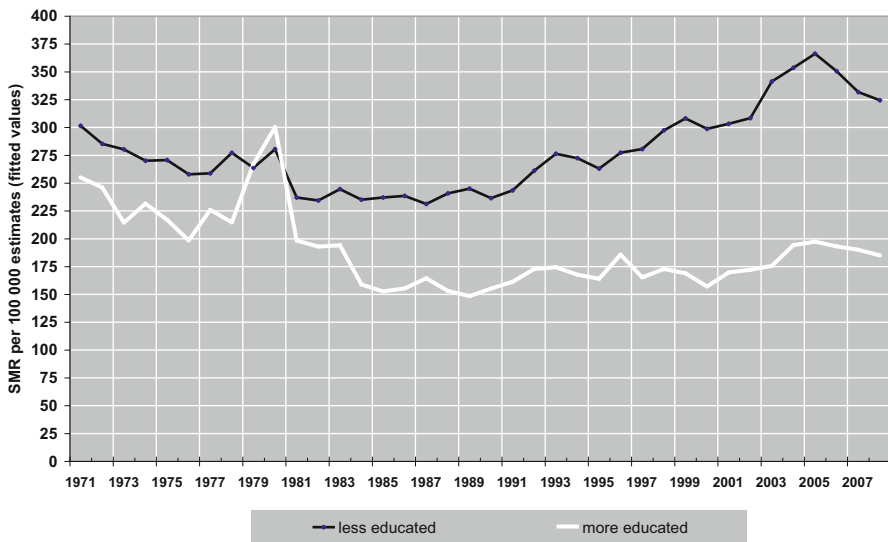


Fig. 4.8 Mortality due to chronic ischemic heart disease 1971–2008, Hungarian population aged 30 and over, representing type III causes of death

become more and more different. Some vague meaning can be given to a possible follow-up pattern only in those cases where the common trend is a decline (meningitis, emphysema, atherosclerosis, other diseases of the veins, and cancer of the

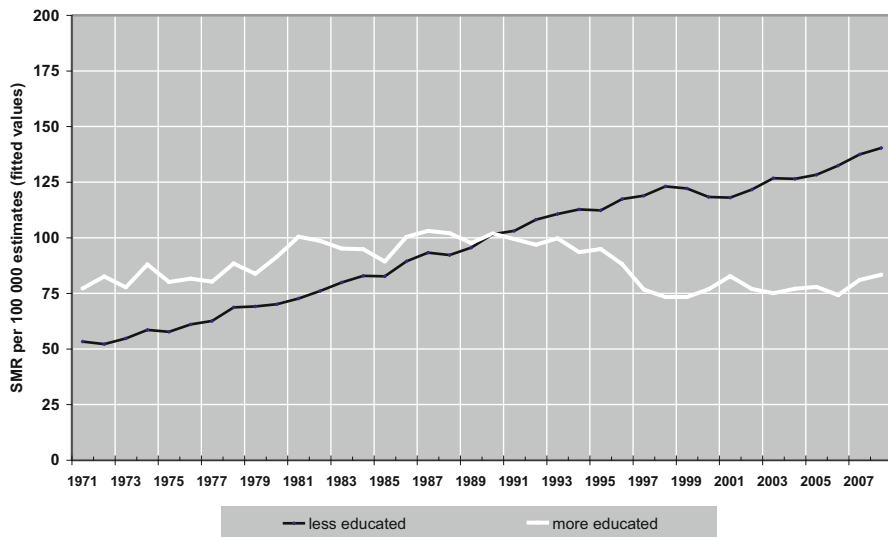


Fig. 4.9 Mortality due to cancer of the trachea, bronchus and the lung by education 1971–2008, Hungarian population aged 30 and over, representing type IV causes of death

gallbladder), similar to the one we give for Type I diseases. In other cases, however, when the mortality of both educational classes increases, there is no sign that mortality of the less educated would follow that of the more educated by any means. It is more plausible that “the same story is played out” for both of the educational groups concerning risk factors or general conditions of life but with very different risk levels.

For Type IV causes of death (Fig. 4.9), the less educated population is characterised by growing mortality, while the mortality of the better educated changed from a growing to a declining trend. Similar trend changes can be expected in the future for the mortality of the less educated, but this change will appear later than the end of our observation period. Approximate minimum time-lags for the onset of this change are given in Table 4.2. In practice these time lags can also be a bit longer, since we cannot be sure if the last couple of years of the observation period represented the beginning of a new type of trend or not.

Altogether, a clear follow-up pattern was detected only for three—though very important—causes of death (Fig. 4.10). As regards acute ischemic heart disease, ischemic stroke and breast cancer, the sequences of the rising and declining periods are similar for the less and the more educated with a time-lag, so the mortality of the less educated seems to follow the mortality of the more educated. Though it is impressive that the estimated follow-up time is the same for ischemic heart disease and ischemic stroke, it is important to mention that these “scenarios” are also “played out” at different mortality levels. At their maximum values, breast cancer and ischemic stroke mortality of the better educated is 20 % higher and that of acute ischemic heart disease is 40 % higher than those of the less educated, suggesting that follow-up type explanations need to be supplemented for a full understanding.

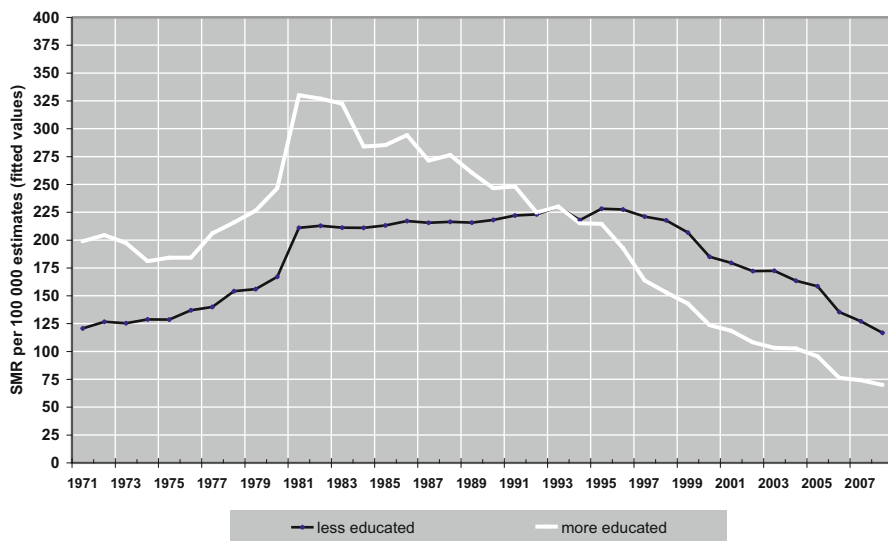


Fig. 4.10 Mortality due to acute ischemic heart disease by education 1971–2008, Hungarian population aged 30 and over, representing type IV causes of death and a follow-up pattern

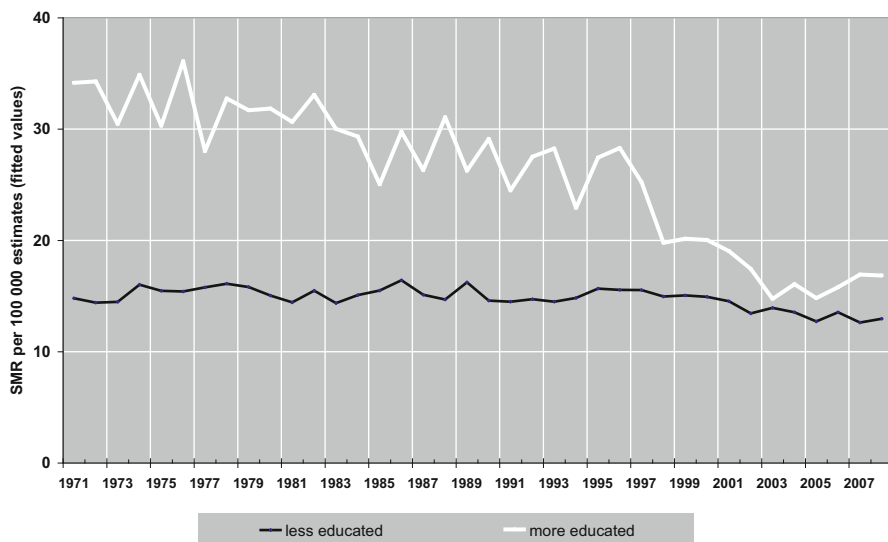


Fig. 4.11 Mortality due to prostate cancer by education 1971–2008, Hungarian population aged 30 and over, representing type V causes of death

Type V causes of death are characterized by different trends for the two educational groups for the whole of the period (see Fig. 4.11). For some, the mortality of the less educated increases and that of the more educated decreases (aneurysm); for

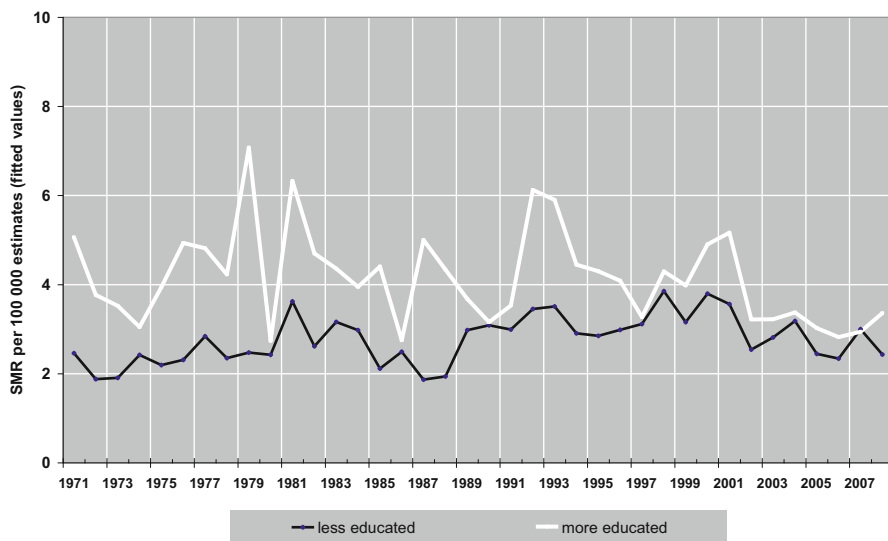


Fig. 4.12 Mortality due to Parkinson disease by education 1971–2008, Hungarian population aged 30 and over, representing type VI causes of death

others, both are in increasing but with different intensity (cancer of the oral cavity, non-Hodgkin disease) or the mortality of the better educated is declining while the mortality of the less educated is stagnating (cancer of thyroid, prostate cancer, peptic ulcer). The possibility that these diseases start to decline or strongly decline among the less educated can certainly be hoped for, but the follow-up time would be longer in these cases than our observation period. There is thus no point in laying down a definition for the purpose of this study. Some diseases, typically rare causes of death, could not be classified into the previous types and they are placed into Type VI, represented by Fig. 4.12.

Though a clear follow-up pattern was identified for only three causes of death, there is evidence of some kind of follow-up for a large number of diseases but it is not easily identified. Time lags are usually long, exceeding more than one or two decades, so that while follow-up may provide a vague and partial explanation for mortality developments and the development of inequalities for the chosen relatively short time period, it certainly does not provide a full picture. Taking a closer look at the onsets of trend changes, it is quite obvious that they cluster in time. Most of the changes occurred in the very first years of the 1980s and around 1990. Both these periods were important turning points, and the two clusters can thus be interpreted as indicators of two diverging trends in living conditions, in the widest possible sense of the term.

In the first cluster we find, surprisingly, a number of nutrition-related causes of death: diabetes, other endocrine diseases, and two strongly nutrition-related cancers (cancer of the uterus and gallbladder cancer). Trends of mortality by education diverge from about the same point of time for a number of causes of death related

to the circulatory system: chronic ischemic heart disease, arrhythmias, heart failure, atherosclerosis, other diseases of the veins and arteries and pulmonary heart disease. Causes of death which are possibly nutrition-related, such as cancer of the rectum, acute ischemic heart disease and ischemic stroke, also show signs of changing mortality relations by education between 1980 and 1983. Some other causes of death, which are clearly not nutrition-related, such as meningitis, cancer of the liver, cancer of the brain, melanoma, other skin cancer and leukaemia, join this cluster. The most likely interpretation of the existence of this cluster is that these changes reflect the widening inequalities in the quality of diet for the two social groups distinguished by educational level. Alternatively, within the risk-factor oriented explanatory framework, one can argue that all these changes are attributable to diverging trends of excessive alcohol consumption, noting that cirrhosis of the liver, the only cause of death which is clearly related to alcohol consumption, started to emerge a couple of years earlier. Alcohol-related changes are known to have an immediate mortality impact but some possible effects of the divergence in alcohol consumption cannot be ruled out. Altogether, divergence in nutrition seems to provide a more suitable explanatory framework.

The second cluster includes causes of death with important trend changes between 1989 and 1993. Smoking-related causes, such as cancer of the larynx and cancer of the trachea, bronchus and lung, clearly dominate this cluster, joined by some other diseases such as colon, pancreas, kidney and bladder cancer and valve diseases with other than rheumatic origin. Attributing the evolution of this cluster to the appearance of the divergence in smoking habits in the two educational classes, it is to be noted that this divergence point seems to be more diffused in time than the one related to the divergence in nutrition: trends of important smoking-related causes of death (cancer of the oesophagus) started to diverge a year earlier than 1989, though this cancer type is also influenced by nutrition.

4.6 Discussion

To explain the rise of mortality inequalities between the less and the more educated from the very beginning of the 1980s in Hungary, one might turn towards basic sociological approaches which would focus on the changing relations of education and income, assuming that the relationship between the two was non-existent in the 1970s and became gradually stronger over the period between 1981 and 2008. From a simplified point of view on the former state socialist states that assumes that these countries had no income inequalities at all, the onset of mortality inequalities during the 1980s must be a mystery.

In fact, income inequalities were already present and connected to educational levels during the 1970s in Hungary. Even after taking compensation in-kind into account—since a large share of incomes was undoubtedly distributed in this form—the income of those with higher education can be estimated as being twice as high as that of people without this qualification (Pető and Szakács 1985). During the

1980s the maturation of the “second economy” partly confused this relationship. In this period the state made some form of economic activities free from its direct control; therefore, in this sector (especially in agriculture) a limited market economy developed. Social status was distributed along two axes: in the formal economy, in which income and education were correlated, creating very mild income inequalities, and in the informal economy, in which education and income did not correlate strongly (Kolosi 1987). Since the emergence of the free market economy following 1990, the correlation of income and education has become stronger and stronger, just as in most European countries (Tóth 2005). This, coupled with the lack of a significant improvement in GDP, led to widening social inequalities and the extension of poverty.

The changing relation between income and education therefore plays a certain role in explaining widening mortality inequalities, but it cannot explain the negligible mortality inequalities which existed during the 1970s nor their revival during the 1980s. We should look, therefore, at nutrition-related risk factors.

The food supply in Hungary was mostly based on domestic production during the 1970s and 1980s. Limited exchange with other state socialist countries existed but imports were mainly limited to a small amount of tropical fruits. Domestic products, however, were satisfactory for domestic demand. Agriculture had developed into one of the leading ones in Europe and from the 1960’s there was no food shortage in Hungary. The distribution of food was rather even and quality differences by education hardly existed. During the 1980s, with the growth of the “second economy”, food provision varied and prices were already partly market-driven. The better-off could use their resources to purchase better quality food and these provisions were available to a large share of the population, but obviously not for everyone. Low food prices, together with energy prices which were still subsidized, made it possible for a larger proportion of the population to buy food of satisfactory quality. In the countryside, “around-the-house” agricultural activity was widespread, producing mostly for the household (occasionally producing for the market, too). During the 1980’s the proportion of food grown “around-the-house” was estimated at 40 % of the overall food consumed (KSH 2009).

From the 1990s the food supply and the price system of the country were placed into a global context. Open trade relations provided a great variety of available food, while domestic production, including around-the house output, started to decline. Food prices relative to income represented a greater and greater share of household expenditure and competed with rising energy costs. Around-the-house agricultural production, which had been characteristic for many households for decades, halved in less than a decade: its share in overall food consumption of 20 % in 2000 had shrunk to 10 % by 2008 (KSH 2009). As a result of these processes, the availability of quality food has been shrinking for an ever growing proportion of the population. Domestic agricultural production, however, started to recover in the last years of our observation period, as the states that joined the European Union in 2004 came to benefit from the unified European Agricultural Policy.

The history of food production and food availability seems to run in parallel to the inequalities in nutrition-related mortality, so this narrative provides a very plausible explanatory framework for our findings. If this framework is supported by similar

findings from other countries, then we can conclude that the mortality of the middle income industrialised countries, with moderate income inequalities, is still strongly determined by nutritional differences and by the lack of availability of quality food for large proportions of their populations.

Social differences in food intake have been described both in wealthy and poorer countries and are usually discussed in connection with obesity. Major changes in human nutrition have also been described, characterized by a growth in sugar and animal source food intake (Popkin 2006). In the context of wealthier countries, the poorer nutritional habits of the less educated is usually understood in the context of lack of knowledge, forced habits by tradition or lack of awareness due to putative or real economic interests. In the case of poor countries the phenomena is understood in the context of absolute deprivation and poverty. Several facts indicate that none of these scenarios are appropriate for middle income countries. Hungarian household surveys, for instance, indicate that the amount of sugar and sweetened beverages consumed is much lower in low income households than in households with higher income. The difference in this respect between the lowest and the highest income quintile households was fivefold in 2008². Some features of the differences in food consumption, however, run parallel with the pattern of the Western countries, such as the similar levels of pork consumption of households with different income and the large gaps in poultry, fruit and vegetable consumption. Relatively high pork intake is the only fact which would suggest that tradition also plays some role in forming nutrition patterns. Differences in fruit and vegetable consumption fluctuate and depend on yearly prices (Polgár 2005; KSH 2009) so there is good reason to attribute these differences to the decline of around the house production and the lack of financial resources. Food intake differences by education can largely be explained by rising poverty among the less educated and the changes in the system of food production and pricing.

As a generalisation of our findings, we note that the nutritional elements of living conditions are rarely measured in Europe and they are usually restricted to the poorest countries. In the first relevant Eurobarometer survey, however, less than 15 % of the West European population answered “yes” to the question if paying for food causes any (some or serious) problem, and the corresponding proportion was between 23 and 46 % for Central, Eastern and Baltic countries (not including the Czech Republic and Slovenia). These data refer to the years around 1990. Publicly available raw data of the second European Quality of Life Survey³ (2007) indicate that the question of food quality is still relevant in CEE and Baltic countries. For the only directly food-related question (“Can you afford a meal with meat, chicken or fish every second day if you want it?”) no more than 10 % of the population gave a negative answer in West European countries, whereas this proportion was around 25 % in most CEE and Baltic countries and in Greece, and even higher in some countries such as Slovakia,

² http://www.ksh.hu/docs/hun/xtabla/haztfogy/tahlhf10_05_04.html Az egy főre jutó éves kiadások részletezése COICOP-csoportosítás szerint, 2010 [Detailed annual household spending on food per capita by COICOP classification, by income quintiles, 2010]

³ <http://www.eurofound.europa.eu/areas/qualityoflife/eqls/2007/index.htm>.

Bulgaria and Hungary (31, 38 and 42 %, respectively)⁴. The same question was included in the same year in the European Statistics on Income and Living Condition survey and released results (Ward et al. 2009) suggest that that survey yielded a similar picture: no more than 12 % of the population in Western Europe was affected and 15 %–37 % in Central and Eastern Europe and in the Baltic countries (except for Estonia, Romania and Bulgaria).

Data indicate that even if starvation-related mortality is negligible in lower-middle income European countries, there are good reasons to assume that the quality of nutrition is still not satisfactory for large proportions of the populations in these countries, and leaves its footprint on their mortality pattern. As far as the history of the Hungarian food provision regime is concerned, some of its elements can be regarded as similar to other countries of the region, while some other elements are certainly different.

The above-mentioned developments in income inequalities and food provision in the 1970s are probably similar in all CEE countries, while the introduction of the second economy was unique to Hungary. The development of free market conditions from the 1990s and the degree of exposure to the global competition varied over time and between the countries, as did the role of around-the-house agricultural production. Rising income inequalities and the application of a global pricing system, however, seem to lead to similar levels of mortality inequalities in these countries, though the composition of over-mortality by cause differs (Leinsalu et al. 2009). CEE and Baltic countries, therefore, probably share more common features than differences in this respect. The generalization of the findings for the whole region of “Eastern Europe”, however, seems less fruitful, allowing for the fact that the CEE and Baltic countries have had consistently lower income inequalities than countries of the Former Soviet Union other than the Baltic countries. Several other aspects of household economy, such as the overwhelming role of energy expenditure in CEE countries, are not present in the same way.

4.7 Limitations and Shortcomings of the Study

The analysis of cause-specific mortality is a challenging task. These studies typically go beyond the time periods of consistent registration systems of causes of death and creating credible time series is demanding. The solution chosen in this paper can be criticized and other alternatives of code bridging should be considered in further research. The classification of causes of death by their relation to mortality developments between the more and the less educated can also be questioned and other alternatives should also be regarded. The method followed by this paper was to decompose the overall time series to sequences of linear trends and there is no doubt that other than linear approximate trends could also have been considered. Moreover, the linear approximation itself was carried out with a high level of uncertainty: the

⁴ http://www.eurofound.europa.eu/areas/qualityoflife/eqls/eqls2007/2eqls_07_05.htm.

exact point of time when trends changed was hard to establish, which introduces some uncertainties about the findings.

The changing composition of the population over time is an inherent problem of studies examining long term developments. In our case the share of the population aged 30 years or more with less than secondary school graduation was 87 % in 1971 and 63 % in 2008. A more detailed educational classification of the population would have been desirable but was impossible to carry out with consistency due to major changes in the schooling system during the observed period.

In our discussion we deliberately avoided some important issues which might naturally be regarded as good candidates for explaining mortality inequalities, such as health care provision and differences in health care utilization. The reason for this neglect was the lack of space to cover all elements of cause-specific mortality inequalities in one paper. Instead, we aimed at identifying some general driving forces contributing to widening inequalities. Setting up an accurate statistical record of the different health services, which would have been necessary to evaluate their role, was beyond the possibilities of this study. Similarly, we had to disregard other, similarly important elements of welfare policy, except for some aspects of income distribution.

Our discussion addresses only some of all the arguments raised in different theoretical approaches to the epidemiological transitions theory. We limited the scope of the paper to looking at the role of nutrition in the long term development of mortality and mortality inequalities. The intention of providing an explanation for the observed mortality trends in connection with the social processes of Hungary in the last four decades has left little space for discussing the applicability of other, similarly attractive, explanatory frameworks that undoubtedly have high potential.

Appendix

Cause of death	ICD 8 coding (1971–1978)	ICD 9 coding (1979–1995)	ICD 10 coding (1996–2008)	Fitting coefficients for 1978, 1996 and 2005
Diarrhoea, dysentery, enteritis	006–009, 561–563, 004, 532	006–009, 555–558, 004, 532, 562	A06–A09, K50–K55, A03, A04, K26, K65, K67	1.17; 0.85; 0.84
Septicaemia caused by other agents than streptococcus or staphylococcus	38	38	A41.40–A41.90	Small case number
Tuberculosis	010–018	010–018	A15–A19, B90	1.00; 0.91; 1.00
Pulmonary tuberculosis	010–012	010–012	A15–A16	0.94; 0.79; 0.89
Other tuberculosis	013–018	013–018	A17–A19, B90	1.23; 1.55; 1.42
HIV/AIDS	–	042–044, 279.5	B20–B24	Small case number
SARS and other new diseases with unknown aetiology and disease caused by antibiotic resistant bacteria	–	–	U00–U99	Small case number
Viral hepatitis	070	070	B15–B19	Small case number
Syphilis	090–097	090–097	A50–A53	Small case number
Encephalitis, meningitis	045–046 062–066 036 320–324	036 046–049 062–064 320–326	G22, I98.00 A85–A89 G00–G06 G37.30–G37.40 A17.00, A39, A81, A83, A94	1.10, 1.27, 1.17
Influenza	470–474	487	G08, G09, G92, G36.10 J10–J11	Not fitted
Other acute upper respiratory infections	460–466, 034.0	460–465, 034.0	J00–J06	Small case number
Pneumonia	480–486	480–486	J12–J18	0.91, 0.76, 0.76
Chronic bronchitis	490, 491	490.00 491, 494.00	J40–J42, J47	0.82, 1.23, 2.16
Emphysema	492	492	J43	1.05, 1.12, 1.19
Asthma	493	493	J45	0.89, 0.58; 0.94

Cause of death	ICD 8 coding (1971–1978)	ICD 9 coding (1979–1995)	ICD 10 coding (1996–2008)	Fitting coefficients for 1978, 1996 and 2005
Other diseases of the pulmonary system	460–519	460–519	J20–J30, I44, J46–J99	Small case number
Appendicitis	540–543	540–543	K35–K38	1.22; 1.27; 1.17
Pepic ulcer	533–534, 531	531, 533–534	K25, K27–K28	0.68; 0.76; 0.90
Nutritional anomyies	280–281	280–281	D50–D53	Small case number
Diabetes	250	250	E10–E14	1.24; 1.38; 1.40
Malnutrition, nutritional deficiencies	260–269	260–268	E40–E64	Small case number
Obesity	277–278	278	E65–E68	Small case number
Diseases of the endocrine system other than diabetes, malnutrition or obesity	240–246, 251–259, 270–273, 275–279	240–246, 251–259, 270–273, 275–277	E00–E07 E15–E35, E70–E90	Small case number
Cirrhosis of the liver	571	571	K70, K71.30–K71.80, K72.10, K73, K74, K76	0.71; 0.65; 0.95
Diseases of the digestive system other than cirrhosis	520–570, 572–577	520–570, 572–579	K00–K67, K71.00–K71.20, K72.00, K72.30–K72.90, K75, K77–K93	0.84; 0.91; 0.95
Parkinson disease	342	332.00	G20	1.20; 1.46; 1.43
Alzheimer disease	290	290.1, 331	F00–F03, G30	Small case number
Multiply sclerosis	340	340	G35	1.09; 0.81; 0.92
Epilepsy	345	345	G40, G41.00–G41.10	1.57; 1.31; 1.32
Maternal mortality	630–678	630–679	O00–O99	Small case number
<i>Maternal disorders</i>	290–315	287.80–319	F00–F99	2.01; 1.23; 2.14
Cancer of the oral cavity	140–149	140–149	C00–C14	1.14; 0.88; 0.97
Cancer of oesophagus	150	150	C15	0.91; 0.85; 0.89
Cancer of the stomach	151	151	C16	0.84; 0.85; 0.91
Cancer of the colon	153	153	C18	0.87; 0.93; 0.99

Cause of death	ICD 8 coding (1971–1978)	ICD 9 coding (1979–1995)	ICD 10 coding (1996–2008)	Fitting coefficients for 1978, 1996 and 2005
Cancer of the rectum, rectosigmoid junction, anus and anal canal	154	154	C19–C21	0.84; 0.79; 0.85
Cancer of the liver	155	155	C22	0.84; 0.79; 0.85
Cancer of the gallbladder	156	156	C23–C24	0.89; 0.91; 0.85
Cancer of pancreas	157	157	C25	1.00; 0.93; 0.93
Cancer of larynx	161, 146.80	161, 146.50	C32	0.88; 0.91; 0.98
Cancer of trachea, bronchus and the lung	162	162	C33–C34	0.92; 0.92; 0.91
Melanoma	172	172	C43	0.92; 0.91; 0.91
Other skin cancer	173	173	C44	0.68; 1.00; 0.76
<i>Mesothelioma</i>	158.90, 163.00, 163.10	163.00–163.90, 158.80–164.10, 171.40–171.60	C45	0.22; 0.21; 0.60
Breast cancer	174	174	C50	0.89; 0.90; 0.91
Cancer of the vulva	184	184	C51–C52	Small case number
Cancer of the cervix	180	180	C53	1.00; 0.87; 0.86
<i>Cancer of uterus</i>	182	182, 179	C54, C55	0.60; 0.60; 0.66
Cancer of ovary	181, 183	181, 183	C56, C57, C58	0.97; 0.90; 0.90
Cancer of penis	187.0	187.00–187.41	C60	Small case number
Cancer of prostate	185	185	C61	0.96; 0.90; 0.90
Cancer of testicle	186	186	C62	Small case number
Cancer of kidney	189.00–189.10	189.00–189.10	C64, C65	0.93; 1.12; 0.98
Cancer of bladder	188, 189.20–189.90	188, 189.20–189.90	C66, C67, C68	0.98; 0.90; 0.91
Cancer of the brain and other part of the nervous system	190.00–192.30	190.00–192.30	C69–C72	1.03; 0.90; 0.93
Cancer of thyroid	193	193	C73	1.16; 1.18; 1.35
Hodgkin disease	201	201	C81	1.72; 0.68; 0.79
Non-Hodgkin disease	200, 202	200, 202	C81–C82	0.65; 0.83; 0.89
Leukaemia	203–209	203–209	C90–C97	0.98; 0.86; 0.85
<i>Rheumatic heart disease</i>	390–398	390–398	I00–I09	0.90; 0.31; 0.31
Acute rheumatic heart disease	390–392	390–392	I00–I02	Small case number

Cause of death	ICD 8 coding (1971-1978)	ICD 9 coding (1979-1995)	ICD 10 coding (1996-2008)	Fitting coefficients for 1978, 1996 and 2005
<i>Chronic rheumatic heart disease</i>	393-398	393-398	I05-I09	0.79, 0.28; 0.31
<i>Rheumatic valve disease</i>	394.0, 395.0, 396.0	394.0-397.0	I05-I08	1.03, 0.15, 0.25
Hypertensive diseases	400-404	401-405	I10-I15	0.84; 1.34; 1.42
Ischemic heart disease	410-414	410-414	I20-I25	0.89; 1.11; 1.12
Chronic ischemic heart disease	412	414	I25	1.20; 1.50; 1.18
Acute ischemic heart disease	410-411, 413-414	410-413	I20-I24	0.64; 0.80; 0.99
Pulmonary heart diseases	426, 450	415-417	I26-I28	1.20; 1.28; 1.45
Other heart diseases	420-425, 427-428	420-429	I30-I52	1.97; 1.08; 1.03
<i>Valve heart disorders with no rheumatic origin</i>	424.0-424.1	424.00-424.30	I34-I37	0.42; 1.00; 0.88
Acute endocarditis	421	421	I33, I39.8	Small case number
Acute myocarditis	422	422	I40-41	Small case number
Cardiomyopathy	425	425	I42-I43	3.35; 1.11; 0.88
<i>Arrhythmias</i>	427	427	I46-I49	0.34; 1.12; 0.78
<i>Heart failure</i>	427-428	428	I50	0.36; 5.08; 4.38
Cerebrovascular diseases	430-438	430-438	I60-I69	1.01; 0.91; 0.88
Hemorrhagic stroke	430-431	430-432	I60-I62	0.96; 0.88; 0.90
Ischemic stroke ^a	432-435, 437	433, 434, 436	I63-I66	0.64; 0.79; 0.75
<i>Diseases of the arteries and the veins</i>	432-448	440-444, 447-448	I70-I79	0.23; 0.81; 0.84
Atherosclerosis	440	440	I70	0.76; 0.79; 0.86
Aortic aneurysm	441	441	I71, I72.00	0.77; 1.15; 1.13
Other disease of the arteries and the veins	442-448	450-458	I80-I89	1.04, 0.73; 0.68

Cursive Uncertain estimates

^a Several variations of coding this disease are in use

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