Chapter 1

Introduction

Some observed trends in health and the life expectancy of cohorts are relevant for this thesis. Mortality in developed countries has decreased substantially in the recent decades. For example, Leon (2011) finds an increase in life expectancy in Western European countries by six to eight years between 1970 and 2009. This implies that the younger cohorts are expected to live longer than the older ones. Furthermore, Cutler and Landrum (2011) study the evolution of health of elderly people in the U.S. in the period 1991-2007. They find improvements in health and show in Figure 1.1 that the disability rates, which are indicated by at least one impairment in Activities of Daily Living and Instrumental Activities of Daily Living, have fallen. Their analysis of the possible underlying mechanisms indicates that the aging process of elderly individuals is currently associated with a less rapid deterioration in health then before.

Figure 1.1: Disability among the elderly (source: Cutler and Landrum, 2011)

This positive tendency does, however, not automatically imply that the younger co-
horts perform better in all aspects of health (García-Gómez et al., 2010). For instance, Lakdawalla et al. (2004) show that disability rates have risen for younger Americans in a study based on 1984-2000 National Health Interview Surveys (NHIS). They mention the rising obesity rates as one of the potentially underlying causes for this increase in disability.

Large inequalities in health associated with socioeconomic status (SES) are observed within countries. This association is commonly referred to as the ‘socioeconomic gradient’ in health (see for instance Cutler et al., 2008). Figure 1.2 reports the fraction of U.S. individuals assessing their self-reported health levels as excellent or very good by income quartile. This figure indicates that self-reported health is increasing in income. Furthermore, Cutler et al. (2010) indicate that 25 year old college-educated individuals had a life expectancy that is 7 years longer than their lower educated peers in the U.S. in 2000. Figure 1.3 shows for the U.S. and six European countries the increase in mortality risk within a year associated with the difference between less than upper-secondary education and at least upper-secondary education. The hazard ratios imply that individuals with lower than upper-secondary education are at least 20 percent more likely to die in a given year compared to higher educated individuals. Moreover, disparities in mortality are present in all investigated countries regardless of the health care system. The magnitude of the SES-related health inequalities appears to have been rising in the recent decades. Cutler et al. (2010) show that the observed decline in mortality in the US between 1960 and 2000 was much larger among the better than among the less educated individuals. For instance, the life expectancy of those individuals attending college rose by 1.6 years in the final decade of the 20th century, while there was no change among those who did not attend college. Mackenbach et al. (2003) similarly report that the socioeconomic inequalities in health are growing in Western European countries. Education levels and occupational types are used as indicators of socioeconomic class. Their findings indicate that changes in cardiovascular mortality are an important contributor to this widening relative mortality gap. They argue that health-related behaviors, like smoking, are important contributors to this trend.

Still a lot can be learned about the mechanisms underlying the strong statistical relation between SES and health (Fuchs, 2004). The health inequalities are already present at young ages and grow until age 50-55 (see Figure 1.2). The differences at early ages may result from inequalities in genetic endowment or early-life conditions. The health disparities decrease after age 70. This reduction at old ages is presumably driven by a selection effect. This selection implies that unhealthy individuals are less likely to be included in the sample due to poor health outcomes and mortality and these unhealthy individuals are more likely to belong to lower income groups. This selection is present at all ages but becomes more apparent later in life due to the age-related deterioration of health.

Health at later ages is the result of a large number of variables. Important corre-
Figure 1.2: Percent reporting excellent or very good health status by age-specific household income quartiles (source: Smith, 2005)

Figure 1.3: Education and mortality: the increase in annual mortality risk among adults over 40 associated with having less than upper-secondary education compared with at least upper-secondary education (source: Cutler et al., 2008)
lates are education, income, genetics, conditions early in life, lifestyle, access to medical technology and shocks later in life. See, for instance, Fuchs (2004). This thesis focuses on a subset of these determinants, i.e. the conditions very early in life. I focus on the early-life environment for the following reasons. The previously discussed findings indicate that later-life inequalities in health originate early in life. Recently, this idea has been picked up by several authors. Almond and Currie (2011) and Lumey et al. (2011) provide, for instance, surveys of this growing literature. These long-run effects also have important implications for the benefits of social policies. The benefits of interventions at early-ages accumulate over the life course and are reaped over a long period of time, whereas interventions at later ages may be less efficient.

Research on long-run causal effects of conditions early in life is hampered due to two main empirical issues. First, the nature of early-life conditions is endogenous implying that unobserved variables jointly affect early-life conditions and health later in life. For instance, genes may have an impact on both parental characteristics, like their education, income and parenting style, and later-life health. Second, there is a lack of data that covers both conditions early in life and later-life outcomes due to the required window of observation (Van den Berg and Lindeboom, 2012).

A way to deal with the endogeneity of early-life conditions is by looking for variation in the environment that has an impact on later-life outcomes but only through the effects of conditions early in life. Such variation in the environment early in life can then be used as an instrumental variable to consistently estimate the effects on later-life outcomes. Examples of such instruments are famines (Lindeboom et al., 2010, Neelsen and Stratmann, 2011 and Meng and Qian, 2009), maternal fasting induced by the Ramadan (Almond and Mazumder, 2011), epidemics (Almond, 2006), the season of birth (Doblhammer, 2004), business cycles (Van den Berg et al., 2006) and unexpected productivity shocks (Banerjee et al., 2010). The mentioned studies connect historic information of these events to outcome variables later in life.

Early-life conditions may have an impact on long-run outcomes in various ways. A commonly made distinction is between direct and indirect effects. The direct effects imply a direct biological link. They are often related to the ‘fetal origins’ hypothesis arguing that the in utero environment ‘programs’ the fetus to have particular metabolic characteristics that may affect health (Barker, 1990). The development of vital organs of the fetus may be hampered when pregnant women are exposed to adverse conditions and this increases, in turn, the risk for chronic diseases later in life. This view radically differs from the initial view held by epidemiologists in the late 1950’s that “the fetus was a perfect parasite, battening on maternal provender while it was afforded protection from nutritional damage that might be inflicted on the mother”(Susser and Stein, 1994). In the past decade, economists have started to study this hypothesis in the context of socioeconomic outcomes. Almond and Currie (2011) provide an overview of this literature and they report effects of adverse in utero conditions on human capital, labor market outcomes, marital status,
welfare dependency and neighborhood characteristics in adulthood.

Indirect effects are frequently related to ‘accumulation of risk’ models stating that risks to health gradually accumulate over the life course. Examples of such risk factors are smoking and stressful events. There is cumulated damage on health as the number, duration and the severity of exposure to risk factors grows over time. Factors acting at sensitive developmental periods, like conditions very early in life, may have a magnified impact. Exposure to risk factors may be clustered. For example, a birth in a low socioeconomic class household decreases health at birth and educational opportunities and leads to an increase in the probability of exposure to family stress and adverse health behaviors, like smoking, an unhealthy diet and alcohol consumption (Kuh and Ben-Shlomo, 2004). Wadsworth (1997) states in a closely related model that biological programming may deterministically affect the range of adult health parameters but the social and family environment in childhood are the start of pathways that will be protective to health or increase vulnerability to ill health.

Figure 1.4 shows a possible indirect pathway between early-life conditions and later-life health. The conditions early in life may have effects on health during infancy and together they could have a joint impact on educational outcomes. These infant health and educational outcomes, in turn, influence the socioeconomic position and health at the start of the labor market career. This position may subsequently affect morbidity and mortality later in life via, for instance, income, lifestyle and access to health care. Hence, health later in life is determined by endogenous variables, like socioeconomic status and behavior. These variables are influenced by abilities, which are, in turn, affected by early-life conditions.

Education appears to be a particularly important intermediary variable between early-life conditions and health later in life. There is a large and persistent association between education and health and there also is evidence of causal influences. Possible mechanisms underlying causal effects of education on health are changes in income, the type of jobs, the value attached to the future, information, cognitive skills, preferences, the rank in society and social networks (Cutler and Lleras-Muney, 2008). A substantial part of the research on these effects uses changes in laws of minimum school leaving ages as instrument for quantity of education (see Oreopoulos, 2007, Lleras-Muney, 2005 and Arendt, 2005). There also is a relation between conditions early in life and educational attainment. For instance, Case et al. (2005), Currie and Stabile (2007) and Ding et al. (2009) find that poor early-life health affects education outcomes.

During the life course individuals may be exposed to major adverse events. For instance, an individual may divorce, face the onset of chronic health conditions or fall into poverty. At later ages, bereavement is one of the most relevant shocks that individuals are exposed to. Such major adverse events during the life course may also be important in the indirect effects of early-life conditions on health later in life. Particular shocks, like the death of a spouse, are shown to be important sources of psychosocial stress and they
Chapter 1. Introduction

Figure 1.4: Indirect effects of conditions early in life on later-life morbidity and mortality (source: Van den Berg and Lindeboom, 2007)

are subsequently related to morbidity and mortality. For example, Stroebe et al. (2007) and Espinosa and Evans (2008) find an increase in mortality after the death of a spouse. Possibly, individuals growing up in adverse conditions are more likely to encounter such adverse events. This mechanism is in agreement with the accumulation of risk models that are mentioned above (Kuh and Ben-Shlomo, 2004). These models indicate that individuals who are exposed to adverse conditions early in life are more likely to face risk factors to health at later ages. Another possibility is that the effects of such events on health are exacerbated after exposure to adverse early-life conditions. A possible reason for this may be that the ones who are born in an adverse environment already have a lower health stock either via a biological mechanism or via indirect pathways. In this thesis I will explore this issue further. In a related study, Van den Berg et al. (2010) show that the effect of a stroke on cognitive decline is magnified for those born in a recession. Case et al. (2002) find that those individuals from lower-income households with chronic health conditions have worse health than those individuals with similar conditions from higher-income households. Philips et al (2001) indicate that prenatal growth restriction may have an impact on life expectancy through marital outcomes over the life course. This thesis addresses, among other things, questions that follow from these studies. First, is there also an interplay between early-life conditions and other major adverse life events, like the death of a spouse, the death of other family members or divorces, as joint determinants of health? Moreover, do various indicators of early-life conditions, like the economic and the nutritional environment, all lead to a differential impact of major events on health outcomes later in life? For this, we use exogenous variation generated by the business cycle at birth and the well-known Dutch famine of 1944-1945.
In particular, this thesis consists of two parts. The first part contributes to the existing knowledge on the long-run effects of adverse conditions very early in life on health and labor market outcomes (Chapter 2). The second part examines whether major adverse life events occur more often and have larger effects on health for those individuals who were exposed to adverse conditions early in life (Chapters 3 and 4). Thereby, this second part studies a specific pathway that may underlie part of the long-run effects of adverse environments early in life on health.

Chapter 2, jointly written with Gerard J. van den Berg and Maarten Lindeboom, analyzes the effects of gestation during the Dutch Hunger Winter famine of 1944-1945 on hospitalization and labor market outcomes. The study analyzes the long-run effects of in utero malnutrition and uses this famine as a source of independent variation in prenatal nutrition. The food shortages started towards the end of World War II and the subsequent famine is a unique natural experiment due to its severity, unanticipated nature, clear demarcation over time and space and its occurrence in a stable society with reliable data registration and adequate nutrition. Furthermore, the short duration of the famine enables us to distinguish between the trimesters of gestation and, therefore, to study so-called ‘critical periods’ in the fetal development. The analyses use variation in famine exposure resulting from both the date and place of birth. While this is the most studied famine in the epidemiological literature and the results consistently point to associations between prenatal famine and adult body size, diabetes and schizophrenia (see Lumey et al., 2011), the knowledge of long-run effects of in utero malnutrition on labor market outcomes is still relatively scarce. Related studies focus on famines that were less clearly defined in time and space and took place in more instable countries with less reliable data registration. Moreover, this chapter is the first study to use register data that cover the full Dutch population.

The second part of this thesis considers the interplay between adverse events later in life (or shocks) and early-life conditions as joint determinants of health. Chapter 3, jointly written with Gerard J. van den Berg, Dorly J.H. Deeg and Maarten Lindeboom, studies the impact of exposure to adverse economic conditions early in life on the effects of major shocks on later-life physical health. The major shocks include the onset or relay of chronic diseases, the death of a spouse, the death of a family member and the illness or accident of a partner. The number of functional limitations is adopted as the outcome variable. This is a commonly used measure for disability. We use the stage of the business cycle at birth as an exogenous determinant of household economic conditions. We use Dutch panel data surveys that follow a group of individuals born between 1908 and 1937 for a period of up to 15 years. The richness and the longitudinal dimension of our data are exploited to control for unobservables that may confound the results. The empirical analyses start with a check for the presence of long-run effects of adverse conditions early in life on physical health and the probability of facing adverse shocks. Next, we estimate fixed effects panel data models to assess whether the health impact of these shocks is
larger after exposure to adverse economic conditions early in life.

Chapter 4 examines whether the effects of major adverse events on health later in life are larger after in utero malnutrition. The events are the death of a spouse, a divorce and the death of a child. Hospitalization is adopted as the measure for health. I use, similar to Chapter 2, independent variation in nutrition early in life resulting from the Dutch Hunger Winter famine of 1944-1945. The analyses are based on longitudinal data that cover the entire Dutch population and record the precise date of the shocks and the hospitalizations. This detailed level of observation enables the exact determination of the order of the events and thereby limits the probability of an unobservable shock simultaneously causing both the adverse events and the hospitalizations. The empirical analyses start by examining whether the number of major adverse events is larger for the individuals who were prenatally exposed to the famine. Next, I analyze whether the impact of the shocks on health is exacerbated for those individuals.

Chapters 3 and 4 address closely related topics, but these chapters are complementary and jointly contribute to the literature. They are different in the following ways. On one hand, Chapter 4 is based on a larger set of data than Chapter 3. These data also contain more information on the timing of the events. This chapter uses a famine that is clearly defined in a small period of time. As a result, the birth window of the sample is small and this reduces the probability of cohort effects and time trends affecting the estimates. It also allows a focus on solely the in utero period. On the other hand, Chapter 3 is based on data that are richer in the number of observed variables that may confound the results. These data also contain more information on the background characteristics of the individuals. Moreover, the long-run effects of business cycles may nowadays be more relevant in developed countries than those effects of famines. Finally, the chapters examine different dimensions of conditions early in life, i.e. the nutritional or economic environment, and varying indicators of later-life health, i.e. the number of functional limitations and hospitalization rates.

Chapter 5 summarizes the results found in Chapters 2, 3 and 4. It also discusses the implications of these findings for public policy. The studies are, in general, relevant for the following reasons. First, the findings have implications for the demand for health care and labor market productivity of currently aging cohorts. They also contribute to the identification of factors that underly observed differences in health between cohorts. Second, knowledge of long-run effects of adverse conditions early in life is also important for the targeting of policy programs. Heckman and Masterov (2007) stress that investments in young children from disadvantaged backgrounds are a rare policy choice that does not involve an equity-efficiency tradeoff. These investments both reduce inequality associated with the incidence of birth and raise the productivity of society at large through, for instance, lower crime rates and higher labor market productivity. Finally, the chapters provide insight into the health effects of major life events and ease the identification of individuals who are most vulnerable to these shocks.