

BIRTH IS THE MESSENGER OF DEATH –
BUT POLICY MAY HELP TO POSTPONE
THE BAD NEWS

NEW EVIDENCE ON THE IMPORTANCE
OF CONDITIONS EARLY IN LIFE FOR
HEALTH AND MORTALITY AT
ADVANCED AGES



The background of the page is white with a series of thin, black, curved lines that sweep across the page from the top left towards the bottom right, creating a sense of movement and depth.

Gerard van den Berg and Maarten Lindeboom

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of death – but policy may
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bad news**

PANEL PAPER 4



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Colophon

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PREFACE

Netspar stimulates debate and fundamental research into the implications of the aging of the population, the sustainability of pensions and government policy. The aging of the population is front-page news, as many baby boomers are now moving into retirement. More generally, people live healthier and longer while at the same time families choose to have fewer children. Although the aging of the population often gets negative attention, with bleak pictures painted of the doubling of the ratio of the number of people aged 65 and older to the number of the working population during the next decades, it must, at the same time, be a boon to society that so many people are living longer and healthier lives. Can the falling number of working young afford to pay the pensions for a growing number of pensioners? Do people have to work a longer working week and postpone retirement? Or should the pensions be cut or the pension premium paid by the working population be raised to afford social security for a growing group of pensioners? Should people be encouraged to take more responsibility for their own pension? What is the changing role of the social partners in the organization of pensions? Can and are people prepared to undertake investment for themselves for their pension, or are they happy to leave this to the pension funds? Who takes responsibility for the pension funds? How can a transparent and level playing field for pension funds and insurance companies be ensured? How should an acceptable trade-off between social goals such as solidarity between young and old, or rich and poor, and individual freedom be struck? But most important of all: how can the benefits of living longer and healthier be harnessed for a happier and more prosperous society?

The Netspar Panel Papers aim to meet the demand for understanding the ever-expanding academic literature on the consequences of aging populations. They also aim to help give a better scientific underpinning of policy advice. They attempt to provide a survey of the latest and most relevant research, try to explain this in a non-technical manner and suggest some directions for policy-relevant research and, if possible,

offer some policy conclusions. Let there be no mistake. In many ways, formulating such a position paper is a tougher task than writing an academic paper or an op-ed piece. The authors have benefited from the comments of the Editorial Board on various drafts and also from the discussions during the presentation of their paper at a Netspar Panel. This is important, since it concern topics of immense importance for society.

Rick van der Ploeg

Chairman of the Editorial Board of the Netspar NEA Papers and Panel Papers

EXECUTIVE SUMMARY

This paper reviews the existing knowledge about the relationship between health and *SocioEconomic Status* (SES) over the life course. It is well known that both life and health expectancies vary substantially between socioeconomic cohorts as well as between countries. The central question is what causes these differences.

Health and SES are for obvious reasons interrelated. One of the key questions is whether there exists a causal effect of SES on health (the other way around), or a mutual dependency. There is still quite a lot of uncertainty about the relative importance of different factors that ultimately determine health and mortality at advanced ages. This paper surveys the evidence. More and more attention is also given to the importance of occurrences early in life on health and mortality later in life. Both the epidemiological and economic literature are discussed and extended. Moreover, the paper discusses the implications of the findings in the literature for public policies aimed at improving health.

The association between health and SES is strongest for the group between 35 and 60 years of age, and weakest for those 85 years old and over. Much of the SES-related health inequalities are determined early in life. Education provides an example of a way in which it can be compensated. One way to analyze the importance of early-life conditions is to compare health and mortality among elderly individuals who faced different living conditions early in life. But one must be careful in drawing conclusions in this respect, since observed associations do not necessarily imply the presence of causal effects of early-life conditions. Detection of causal effects requires observed exogenous variation in early-life conditions.

One can distinguish two main views regarding the way in which these conditions can affect later-life morbidity and mortality: a *direct* effect and an *indirect* effect. Examples of the first kind (adverse conditions during prenatal and post-neonatal period, from 0 to 12 months) are malnutrition and infectious diseases. Indirect effects are possible if poor

early-life conditions lead to poor health early in life and later in childhood, which, in turn, affect educational outcomes and subsequently social status and health in adulthood.

The paper discusses several indirect effects of early childhood to later-life morbidity and mortality by way of education. It turns out that the education of the mother does improve the health of their offspring, and that it causes a larger effect than the father's schooling. The evidence in the literature suggests that there is only a small role for the effect of income in explaining children's health in the developed world, and that its main effect is post-natal. Furthermore, the income elasticities are often found to be very small. This suggests that one should not have high expectations regarding the extent to which income and tax policies can influence educational choices.

There is a strong association between child health or nutrition and (later) educational attainment. The effect of education on later health is also investigated. It turns out that the better educated have the better jobs and higher incomes, which may lead to better health and lower mortality rates at later ages. Since education is one of the strongest determinants of later health and mortality later in life, public policy should focus on education.

BIRTH IS THE MESSENGER OF DEATH

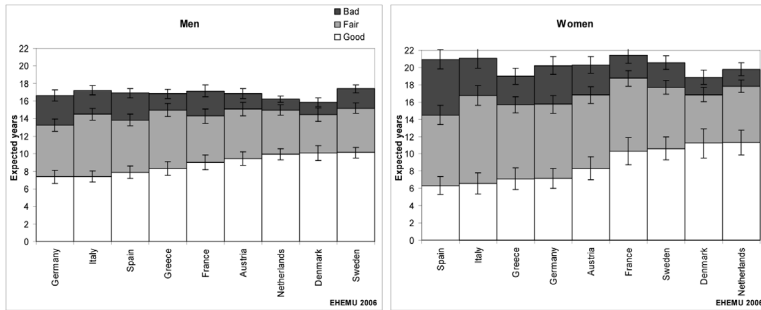
1. Introduction

This paper is concerned with health and mortality at advanced ages. Specifically, we examine how this depends on conditions early in life. We review the existing knowledge about the relationship between health and socioeconomic status (SES) over the life course, examine strategies to identify causal effects and discuss the implications of the findings for public policies aimed at improving the health of the population.

Two observations in particular motivated us to look at health at advanced ages and at the role of conditions early in life as a determinant. First, the larger part of healthcare that individuals consume during their life course is concentrated in the final few years of their life. Some argue that proximity to death is the driving factor of these costs (Zweifel et al., 2004), whereas others (e.g. Seshamani and Gray, 2004) argue that age has an additional effect on healthcare spending. The latter view implies that in the context of the trend towards aging, increases in healthcare costs are to be expected. It also implies that healthcare costs across cohorts will vary if mortality and morbidity rates differ across age cohorts. A second observation is that health is known to be very unevenly distributed at advanced ages. Calculations (Manton and Stallard, 1991; Portrait, Lindeboom and Deeg, 2001; van den Berg, Lindeboom and Portrait, 2006; EHEMU, European Health Expectancy Monitoring Unit, 2006) show that beyond the age of 65 a substantial part of life is spent in bad health.

Figure 1 below is taken from the EHEMU 2006 report, "Estimations of Health Expectancies in Europe in 2004". It shows that health expectancies (defined by the expected years of life in a state of good health) vary across countries and that females spend a greater share of their life beyond age 65 in bad health. Of course, there is much heterogeneity in residual life and health expectancies within countries. Some individuals at age 65 are perfectly healthy and spend only a small fraction of their remaining life in bad health, whereas others combine severe physical

limitations with cognitive impairments already at relatively young ages. What is causing these differences is an important research question.



Sources: Eurostat and SHARE 2004

Figure 1 Life expectancy and expected years of good, fair and poor health at age 65, by country and gender.

Figure taken from EHEMU (2006)

Currently, in many countries, disability trends among the old are falling (Cutler, 2001; Manton, Stallard and Corder, 1997; Portrait et al. 2006). The literature suggests that healthcare costs will decline as a result of these downward disability trends. However, recent work by Bhattacharya et al. (2006) and Lakdawalla et al. (2001) indicated that healthcare costs are expected to rise after 2020 as a result of increasing disability trends among the current young. This suggests that health differences across cohorts can be substantial, and that health does not necessarily improve over successive cohorts. This finding also suggests that the health of the old may to a large extent be determined by events and conditions earlier in life— and that adverse events in younger years may have irreversible long-term consequences.

There is a strong connection all over the industrialized world between an individual's current socioeconomic status (SES) and current health (the association between income and health is commonly denoted as the "the gradient"). This is sometimes taken as evidence of the importance of SES for health (thus, as evidence of a causal effect of SES on health). The magnitude of this gradient differs across countries. Moreover, SES-related inequality in health has increased over the last decades (Mackenbach et al. 2003; Banks et al., 2006). Clearly, the statistical relation between SES and health can also be explained by a reverse causality from

health to SES, or by a mutual dependence of SES and health on common determinants such as genetic characteristics, education or conditions early in life. This naturally leads to a dynamic view in which causal pathways between various factors may lead to observed associations between SES and health. There is still a lot of uncertainty about the relative importance of different factors that may ultimately determine health and mortality at advanced ages (see the survey by Cutler, Deaton and Lleras Muney, 2006). Not surprisingly, more and more attention is being given to occurrences early in life. For example, height at age 18, or the level of education, may be important predictors or determinants of health later in life. But height itself, or the level of education, may be affected by conditions in the first few years of life and by inheritable characteristics.

Section 2 of this paper examines recent evidence on the association between health and socioeconomic status at middle age and old age. The conclusion: much of the association between SES and health during middle age (the prime of life) and old age is driven by a causal effect of health on SES, rather than the other way around. Indeed, we provide simple statistics across different countries showing that already at relatively young ages (people in their twenties, thirties and forties) a substantial share of workers has a long-standing illness that limits them in their daily activities and/or work. These disabilities are strongly associated with outflow from the labor market (well before official retirement ages). All of this suggests that if economic conditions do indeed have any major effect on health later in life, then these economic conditions must originate earlier in life.

The role of conditions early in childhood is therefore the focus of section 3 of this panel paper. We review the epidemiological and economic literature in this field, present evidence of the importance of early childhood conditions for later life outcomes, discuss the methodological problems in this area when researchers have to rely on observational data, and propose appropriate research designs that allow one to assess the causal effect of early childhood conditions on health and mortality later in life. Section 4 discusses mechanisms that may underlie the causal effect of early childhood conditions and addresses the policy implications, while section 5 concludes.

It is important to emphasize that even if early-life conditions have a small overall effect on the per-period morbidity or mortality rate later in life, it may nevertheless be very important from a policy point of view to

intervene in the lives of individuals with a bad starting position. After all, the benefits of such interventions will be reaped over a very long time period, and intervention is facilitated by the fact that there is a time interval in between a particular cause and the moment its effect materializes. This is quite different from the instantaneous effects of current events on the health of elderly individuals, like a summer with unusually high temperatures. Such instantaneous effects may be large, but they may be relevant only over a short period, and policymakers would have to react very quickly to prevent the negative health implications.

2 The association of health and socioeconomic status among adults and the elderly

Figure 2, below, is taken from Smith (2005) and illustrates the relation between income and health across the different stages of the life course for US workers.

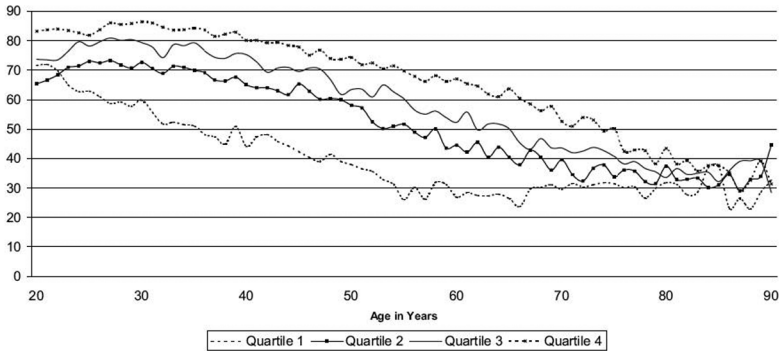


Figure 2 Percent reporting excellent or very good health by age and income quartile (from Smith 2005)

The figure seems to indicate a strong association between health and SES; individuals from the lower income quartiles are generally in poorer health. The figure also suggests that the “gradient” (the association between income and health) is strongest for those aged between 35 and 60. Beyond age 85 the association becomes weaker. This may be driven by a selection effect (van den Berg and Deaton, 2007). Although many studies have established the strong association between income (SES) and health, it has also been acknowledged that assessing causal mechanisms in this area is notoriously difficult. There may be direct causal mechanisms running from health to SES, or the other way around. For instance, health can have a direct effect on an individual’s capacity to work— and therefore on work outcomes, income and wealth. Alternatively, work, or aspects of work (income, working conditions, stress and so forth) may directly affect the health of an individual. Empirical analyses based on observational data are further plagued by the presence of unobserved factors that are related to both health and socioeconomic status. Independent variation in either health or SES is required to assess

the causal effect of one on the other. With observational data this condition is rarely satisfied. A few studies have nevertheless tried to assess the causal mechanisms underlying the Health – SES association.

Adams et al. (2003) suggested the use of Granger causality tests for panel data models to test for the absence of causal links from SES to health innovations and mortality and from health conditions to innovations in wealth. The tests examine whether lagged SES (health) has an effect on current Health (SES), once lagged health (SES) is included. The tests are applied to the AHEAD survey, a US panel of older persons (70+). The study concluded that SES has no effect on future health, once prior health is included, and that there generally seems to be an effect of health on future wealth, even after controlling for previous wealth. Their main conclusion is therefore that the larger part of association between health and SES at advanced ages is driven by a mechanism running from health to SES, rather than the other way around. This is a plausible finding for the older population studied in this paper. However, Mealli and Rubin (2003) (among others) have criticized the methodology used.¹ They deem it preferable to analyze individual data while taking account of joint unobserved determinants of SES and health.

Other papers have obtained results similar to those of Adams et al. (2003) for younger populations. Smith (1999, 2003) used the onset of chronic conditions as a measure for a health shock, and examined the effect of these conditions on the probability of work, household income and wealth. He found negative financial consequences of health shocks for a sample of individuals between 50 and 60 years old. Møller-Danø (2005) used road accidents as a measure for health shocks and found long-lasting effects on income and employment. Lindahl (2005) used lottery winners to assess the effect of a sudden change in income on health later in life. He reported small effects of large increases in income on health later in life— at least among the admittedly special population of lottery participants. Smith (2005) used the Panel Survey of Income Dynamics (PSID) to examine the impact of SES on health over the life course. He concluded that current- and lagged financial measures of SES have no effect on future health, although a non-financial measure like

1 In essence, Mealli and Rubin argue that those experiencing health (wealth) innovations differ with respect to unobserved characteristics and that assignment into the treatment (experiencing a health or a wealth innovation) is hence not exogenous to the outcome of interest.

education does. This result is persistent over the life course— meaning that this also holds for younger (younger than 40) and middle-aged (40 to 60 years) workers. The finding that education is determined primarily at younger ages suggests that much of the SES-related health inequalities are determined earlier in life. Case and Deaton (2003) found that people in manual occupations have worse self-reported health, and that the rate of health declines is larger in these occupations. They argued that much of the differences in health across the income distribution are driven by health-related absence from the labor force. This again suggests that the effect of health on SES is probably more important than the other way around.

The table below is taken from Kapteyn, Smith and van Soest (2007). It shows that disability rates are already high at relatively young ages. The table also shows that the Dutch disability rates are much higher than US disability rates. About 50% of this difference is due to differential health reporting (Kapteyn et al., 2007). Burkhauser et al. (1999) noted that work accounts for the larger part of the income of men aged 51–61 in the US, while for the Netherlands a large percentage of the men in this age category have already withdrawn from the labor market. They argued that differences between Dutch and US institutions, rather than health differences, could explain this disparity.

	25-34	35-44	45-56	55-64	65+
US	7.4	11.3	17.6	25.9	38.8
Netherlands	17.2	23.6	38.7	37.4	38.8

Table 1 Percentage with work disability by age US-Netherlands (from Kapteyn et al., 2007)

Disability rates similar to those of US workers were also found for UK workers (Lindeboom, Llana-Nozal and van der Klaauw, 2007). The latter study also found that a disability leads to substantially lower employment rates. This confirms the finding of Case and Deaton (2003) that much of the difference in health across the income distribution is driven by health-related absence of the labor force. These findings imply that well before the official retirement ages a substantial share of the labor force has already left the labor market. The study by Lindeboom,

Llena-Nozal and van der Klaauw also found that there is much heterogeneity in these rates: those with lower socioeconomic status have generally much higher disability rates, and reductions in employment rates are larger for these groups. Case and Deaton (2003) noted that men aged 20 who were at the bottom of the income distribution reported worse health than men aged 50 who were at the top of the income distribution.

The important conclusion from the literature cited above and the numbers in the table suggest that the determinants of health and SES-related differences in health originate earlier in life. Recent work by Heckman, Stixrud and Urzua (2006) has shown that "early intervention programs targeted to disadvantaged children have had their biggest effect on non-cognitive skills: motivation, self-control and time preference," and that these non-cognitive skills are powerful predictors of educational attainment, lifestyle and health behaviors. Their work also shows that for severely disadvantaged children early childhood interventions are important and can have a long-lasting effect on cognitive and non-cognitive functioning.

3 Causal effects of early-life conditions

3.1 Empirical approaches and empirical findings

A natural starting point to analyze whether early-life conditions are important is to compare health and mortality outcomes among elderly individuals who faced different living conditions early in life. Empirical studies have shown that adverse socioeconomic conditions early in life are associated with susceptibility to a wide range of health problems later in life (see e.g. Case, Fertig and Paxson, 2003; Hayward and Gorman, 2004, and references therein).² Similarly, medical studies have shown that individuals with a low birth weight (suitably adjusted for gestation time) are more likely to suffer from health problems later in life (see e.g. Rasmussen, 2001, and Davey Smith, 2005, for overviews).

Observed associations do not necessarily imply the presence of causal effects of early-life conditions. Individual socioeconomic and medical conditions during early childhood and health outcomes later in life may be jointly affected by unobserved heterogeneity. For example, certain genes may simultaneously influence the average level of the parents' income, the birth weight, and the health outcomes later in life. To be able to detect causal effects, one needs to observe exogenous variation in the early-life conditions, and relate this to outcomes later in life. In all fairness, it should be noted that even if descriptive studies do not capture causal effects, they are still useful from an intervention point of view. Markers for unfavorable future health outcomes can be used as a flag for monitoring or initiating interventions to mitigate such outcomes.

A new approach has recently become popular to detect causal effects, by using data on indicators Z of individual conditions X early in life with the following property: the only way in which the indicator Z can plausibly affect high-age morbidity or mortality Y is by way of the individual early-life conditions X . (An extreme example is where Z is the outcome of

2 Socioeconomic background may also be important for long-run outcomes because it also determines the rank or social position in the social distributions. The social position may be important, as it relates to the circumstances in which people live and work and to how much control they have over their lives (Marmot, 2003). It has been argued that people with a low position in the social distribution are more subject to the demands of others, which puts them at higher risk for cardiovascular diseases (Marmot, 2003; Cutler and Lleras Muney, 2007). This suggests that the pre-natal and post-neonatal environment of children may be affected via the family situation.

a lottery in which individuals with a baby may win some money. Below we give more common examples.) By analogy to the econometrics literature, such indicators Z may be called instrumental variables. Typically, these are not unique characteristics of the newborn individual or his family or household, but rather temporary characteristics of the macro environment into which the child is born. Indicators Z with the above "exclusion restriction" property do not give rise to endogeneity and simultaneity biases, because they are exogenous from the individual's point of view. If one observes an association between such an indicator Z and the health outcome Y later in life, then one can conclude that there is a causal effect of early-life conditions X on that health outcome Y .

We may distinguish between three types of such "instrumental variables" Z . First is the season of birth. The main reference is the overview in Doblhammer (2004) of publications using month of birth to study the effects of nutrition and disease exposure on mortality later in life. The idea is that the month of birth has no other effect on health outcomes later in life than by way of the early-life conditions of the child. Note that this requires that the composition of newborns should not be systematically different across seasons in terms of unobserved characteristics of the newborns. The literature has typically found significant effects of the season of birth on the mortality rate later in life, with an order of magnitude of a few months of extra lifetime if one is born in the fall, as compared to the late spring. In the southern hemisphere, these effects are mirror-imaged, in the sense that the effect of a month of birth is similar to the effect of the month half a year earlier or later in the other hemisphere. In equatorial areas, seasonal effects are in accordance to what constitutes the rainy (monsoon) and the dry season (see e.g. Moore et al., 1997).

A second type of exogenous variation is provided by epidemics, wars, famines and other disastrous events. For a recent example, see van den Berg, Lindeboom and Portait (2007), who examined whether exposure to nutritional shocks early in life affects later-life mortality. They used historical data that include the period of 1845–48, which includes the Dutch potato famine. During this period, all potato and grain crops in Europe failed due to the Potato Blight disease and bad weather conditions. They found that men who were exposed to severe famine at least four months before birth and directly thereafter had a residual life expectancy at age 50 that was significantly lower (a few years) than otherwise, but that the

mortality rate at earlier ages was not affected. They found no evidence for any long-run effects for men exposed at ages 0–2 or for women. Studies based on the Dutch “hunger winter” under German occupation at the end of World War II (Ravelli et al., 1998; Roseboom et al., 2001) and on China’s great famine (Meng and Qian, 2006; Chen and Zhou, 2007) indicated significant long-run effects on adult morbidity, but not on adult mortality (Painter et al., 2005). These studies confirmed that malnutrition has a separate effect on adult morbidity (and sometimes) mortality. Experimental animal research has also provided support for the theory that there are long-run effects of malnutrition during pregnancy (Ozanne and Hales, 2004).

Almond (2002) examined individuals born around the time of the 1918 influenza epidemic. He, together with most of the literature exploiting epidemics, found significant effects on the mortality rate later in life. Similar to many other studies falling under this second approach, Almond investigated primarily the sign and significance of the mortality-rate differences between birth cohorts, and not the exact size of the effect. This is because the interest ultimately is not in the size of the effect of the indicator Z on the mortality rate, but in the issue of whether there is a causal effect from early-life conditions X on the mortality rate. Long-run effects may, of course, be non-linear in terms of early-life conditions. In that case, the relevance of long-run effects of disastrous conditions may be limited, and may not lead to a full understanding of the effects of less spectacular variation in early-life conditions.

A third approach was pioneered by Bengtsson and Lindström (2000). They used the transitory component (or deviation) in the price of rye around the time of birth as an indicator of food accessibility early in life— and any observed relation between this indicator and the mortality rate later in life signified the existence of a long-run causal effect of food accessibility on mortality later in life. Similarly, the transitory component in the local infant mortality rate was used as an indicator of exposure to diseases early in life (see also subsequent applications such as Bengtsson and Lindström, 2003). These studies used data from a relatively small area in Sweden from the 18th and 19th centuries. The results indicate that individuals born in years with epidemics lived on average a few years less than otherwise, conditional on surviving the epidemic itself. Van den Berg, Lindeboom and Portrait (2006) used the state of the business cycle at early ages as a determinant of individual mortality. Cyclical

macroeconomic conditions during the pregnancy of the mother and childhood might affect mortality later in life because they are unanticipated and affect household income. In a recession, provision of sufficient nutrients and good living conditions for children and pregnant women may be hampered. Van den Berg, Lindeboom and Portrait (2006) found that the average lifetime duration in the Netherlands in the 19th century was reduced by about one- to three years if the individual was born in a recession, as compared to being born in a boom (under otherwise identical conditions during life, and conditional on surviving early childhood).

3.2 Detailed methodological issues

To explain the methodology in some more detail, consider the macroeconomic situation at birth. For this, we need historical time-series data on e.g. the national annual per-capita gross domestic product (GDP) in constant prices. To capture the long-run effects of conditions early in life, one might want to ignore business cycles and instead simply compare an individual born in a good era to an otherwise identical individual born in a bad era, following the line of thought that a high GDP goes along with better individual economic conditions for many individuals. This approach, however, is uninformative regarding the effects of individual early-life conditions, due to the steady secular improvements in life conditions over time. After all, a prolonged era with a high GDP also leads to innovation and investment in hygiene and healthcare, which decreases mortality and health problems later in life for those born in this era. A related practical complication is that GDP displays a strong positive trend over time. A high GDP level at birth implies a high GDP level throughout life. An empirical analysis that tries to take this into account by allowing the mortality rate at a given age to depend on current and past GDP levels yields estimates that are potentially very sensitive to small model misspecifications. For example, if the postulated relation is log-linear in the mortality rate and current GDP, and the true relation is slightly different, then this may show up as a significant effect of GDP earlier in life.

Now consider the effects of short-term cyclical movements in GDP. Contrary to the effect of the trend value or the current level of GDP, cyclical effects do not reflect secular improvements. At the individual level, cyclical changes often involve unexpected income shocks. So as an alter-

native approach one could try to compare cohorts born in booms to those born in recessions (with otherwise identical circumstances throughout life). Still, due to the secular improvements over time, being born in different stages of the cycle entails that individuals lead their lives under somewhat different current conditions at each age. A conservative strategy might therefore involve comparing a cohort born in a boom to the cohort born in the subsequent recession, because the latter benefit more from secular developments than the former, so that, for example, an observed decrease of expected lifetimes can be attributed to the cyclical effect.

Generally speaking, one may relate the mortality rate later in life to the state of the business cycle early in life for many different birth cohorts by estimating duration models. This offers the additional advantage of being able to take into account also the effects of the business cycle in later childhood years. Someone who is born in bad times is likely to experience good times during some childhood years, and vice versa, merely because good and bad times succeed each other with an average frequency of a few years. If conditions at birth as well as during childhood affect health and mortality later in life, then the effect of the bad times at birth may be mitigated by the effect of the good times during childhood. Duration models in which the individual mortality rate is allowed to depend simultaneously on conditions at birth and on conditions during childhood can deal with this. The estimation of these models exploits the variation in the timing of the stages of the business cycle across individuals, disentangling the long-run effects of conditions at birth and during childhood. Similar issues may arise with the use of the season of birth (e.g., the season at the 4th quarter may be important) and with the use of disastrous events (e.g., those who are born just before an epidemic may suffer from the epidemic in childhood years). Note that it is impossible to identify the separate long-run effects of seasons at different ages, because of the mechanical repetition of seasons over time.

One important requirement for the analysis of causal long-run effects of early-life conditions is that the individual data cover a sufficiently long time span. After all, the dates of birth and death (or high-age health) must be observed for a substantial number of individuals. An implication of this requirement is that the existing studies have necessarily considered cohorts of individuals who were born a long time ago.

In this sense, the most recent evidence comes from studies of individuals born in the Dutch hunger winter and from studies of more recent birth cohorts from developing countries. One way to circumvent this restriction would be to focus on adult health proxies such as adult height (see below).

3.3 Direct and indirect long-run effects

Subsection 3.1 listed studies that have used exogenous variation in the environment to show that there are causal effects from early childhood on later-life morbidity and mortality. The present subsection briefly sets out the main mechanisms underlying these long-term causal effects. Although there are many ways in which early-life conditions may affect outcomes later in life, we can distinguish roughly between two main views (Lundberg, 1997).

First, adverse prenatal and post-neonatal (from birth to 12 months) conditions can have a *direct effect* on later-life morbidity and mortality. The main idea is that the development of vital organs and the immune system is programmed when the body is exposed prenatally or just after birth to adverse conditions. According to the “programming” or “fetal origins” hypothesis (Barker, 1992, 1998), this may lead to increased vulnerability to chronic diseases in later life. The most commonly mentioned factors mentioned in the literature are malnutrition and exposure to infectious diseases. Other factors are increased stress in the household, and lower income to cover housing accommodation costs.

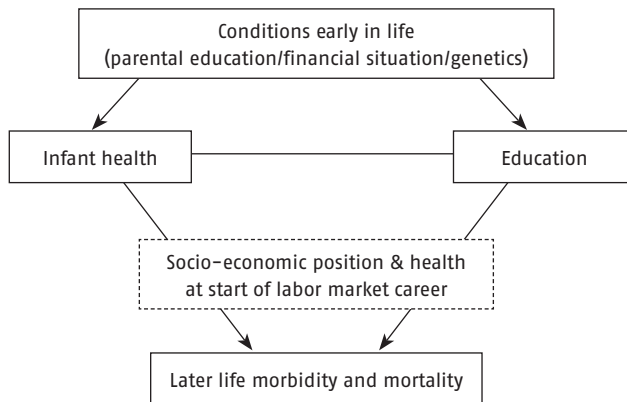


Figure 3 A graphic representation of the indirect effects of early childhood conditions

Most of the empirical studies mentioned in this section are consistent with a direct effect. As we have seen, in order to detect long-run effects, it is natural to focus on temporary shocks around the birth date. Any long-run effect found in this way could be a direct effect. Moreover, the estimated size of the mortality effects is usually moderate and in line with the medical evidence. The type of shock is informative regarding whether the effect concerns malnutrition, disease exposure, other adverse conditions, or just bad conditions in general.

Exposure to infectious diseases and malnutrition is likely to be less relevant for the developed world today than it was in the past. However, Bozzoli, Deaton and Quintana-Domeque (2007) recently examined the effect of income and disease exposure on adult height in populations. Adult height was used as a proxy for lifetime health. They used post-neonatal mortality as a measure for nutrition and disease load in early childhood, and examined their effect on height for cohorts born from 1950–1980 in the US and eleven European countries. They found a strong negative relationship between adult height and the burden of disease and malnutrition.

According to the second main view, adverse conditions early in life have *indirect effects* in that they may be the start of a causal chain of events or pathways during life that leads to worse health later in life. For instance, poor early-life conditions may lead to poor health early in life and later in childhood, which may affect educational outcomes and subsequently social status and health in adulthood. Or, more generally, a poor start may affect an individual's life career, which may ultimately lead to higher mortality rates. In section 4 we discuss this view and its policy implications in more detail.

Some authors have stressed that it is the interaction with social factors later in life that determines whether people who are exposed to adverse early childhood conditions will be more vulnerable to ill health in later life (Wadsworth, 1998). In a similar vein, McEwen (1998) argued that the body accommodates to stress, and that it is *repeated stress* that leads to higher risks of chronic diseases.

4 Indirect effects: causal pathways from early childhood by way of education to later life morbidity and mortality

Figure 3 shows the main causal pathways. To organize matters, we discuss the arrows depicted in the figure separately. Note that the discussion compared to the previous sections has been expanded: we do not restrict ourselves to pathways that can be tracked down to causes early in life, but consider also other possible determinants of later health that are realized before birth, such as genetic characteristics and educational attainment of parents (recall, however, the methodological concerns from subsection 3.1). The arrow that links infant health to later-life morbidity and mortality is not discussed explicitly because this link is represented by the epidemiological studies that link birth weight and other child health measures to later-life health outcomes (discussed already in Section 3).

Note that the methodological complications in the case of indirect effects are even larger than in the case of direct effects. In the former case, most studies typically have focused on just one of the arrows in the diagram, conditioning on the individual position at the starting point of the arrow. In general, this starting position can be endogenously affected by earlier events in the life of the individual or by unobserved determinants that also have a causal effect on the outcome.

4.1 Effects of parental income and education on child health

Studies have found that poor infant health persists into later childhood health and into adulthood, and contributes to the health-income gradient later in life (see Case, Fertig and Paxson, 2005, and the references cited therein). Of interest for this pathway is what factors determine infant health, whether their effect is causal, and whether policy interventions can be designed to reverse the adverse effects of poor infant health. The literature has focused on parental education, income and biological factors (genes).

Some recent studies have used schooling reforms as a source of exogenous variation to look directly at the effect of parental education on child health. This approach has become popular in labor economics, where most studies have focused on the causal impact of education on earnings (e.g. the survey by Card, 2001, and Meghir and Palme, 2005), or on the effect of parental education on the education of their children

(e.g. Black, Devereux and Salvanes, 2005). A few papers in health economics have used similar methods to examine the impact of education on health in later life (this will be discussed later; see Oreopoulos, 2006; Lleras-Muney, 2005; Arendt, 2005).

Currie and Moretti (2003) examined the impact of college openings on the educational attainment of women and the health of their infants. They found that maternal education does improve the health of the offspring. McCrary and Royer (2006) exploited discontinuities in school entry policies in California and Texas to assess the effect of education on fertility and infant health outcomes. They discovered that education does not affect observable inputs to infant health and has only small effects on infant health. Doyle, Harmon and Walker (2005) used an educational reform in the UK and found weak effects of parental education³. Finally, Lindeboom, Llena Nozal and Van der Klaauw (2007b) found for the UK that an increase in the age at which parents left school by one year had little effect on the health of their offspring. Schooling did, however, improve economic opportunities by reducing financial difficulties among households. They suggest that the effects of parental income on child health are at most modest.

Reforms of the educational system are helpful in assessing the causal effect of education on children's health. Still, it needs to be confirmed which factors drive a 'schooling effect'. There are different channels through which parental education can affect their children's health. Education can have a direct impact on child health because it increases the ability of the parents to acquire and process information. This helps parents to make better health investments for themselves and their children. Furthermore, attending school for a longer time could lead to a change in preferences by either lowering the discount rate or increasing risk-aversion (Cutler and Lleras-Muney, 2006). This could influence the health habits of the parents (drinking, smoking and exercise) and thus could influence child health outcomes. Currie and Moretti (2003) maintained that part of the educational effect that they found in their study was due to the increased use of prenatal care and reduced smoking.

Behrman (1997) provided a comprehensive survey of the literature on the effect of parental education on child education. The starting point of his survey was the observed strong association between the mother's

3 They also found that parental income has no effect on the health of offspring

schooling and the schooling of the child. Moreover, the effect of the mother's schooling was found to be much larger than the effect of the father's schooling. Most of the reviewed literature, however, did not control for usually unobserved factors such as abilities and preferences that may influence the mother's schooling, but that may also affect directly the child's education. More able women have more education, but also more able children, who also get more schooling. Furthermore, more educated women also marry more educated and more able men (assortative mating), which may lead to an additional bias over and above the ability bias induced by the mother (Behrman and Rosenzweig, 2002). Behrman and Rosenzweig (2002) tried to control for this by using data from identical twins. (The idea is that these children have the same genetic background; by differencing the twin outcome one can thus control for inherited ability.) They found that an increase in mother's schooling does not increase the child's schooling, and may even lower it. An increase in the father's schooling has a small and positive effect on the child's schooling. Since the mother's time in the labor market seems to increase with education, Behrman and Rosenzweig argued that the results are consistent with the assumption that a woman's time in the home is a crucial determinant of the human capital of her children. Plug (2004) used data on adoptees and confirmed the finding of Behrman and Rosenzweig (2002). Plug also suggested that inherited abilities and assortative mating play an important role in the intergenerational transmission of schooling. Bjorklund, Jantti and Solon (2007) investigated the association between the socioeconomic outcomes of sons and daughters and those of their biological and rearing parents. They used a unique Swedish dataset that contains exact information on both the biological and the nurturing parents. Their results suggest a substantial role for both pre-birth (including genetics) and post-birth environmental factors. Black, Devereux and Salvanes (2005) used a reform of the educational system that was implemented in different municipalities at different times. They also found little evidence for a causal effect of parental education on child health, and suggested that the high correlations between these variables in the raw data might be due to family characteristics and inherited ability.

An increased level of education may open the door to more skilled work with higher earnings. Income may therefore also have an independent (of education) effect on child health. A few papers have looked at

this in more detail. Case, Lubotsky and Paxson (2002) found a strong relationship between income and child health, and determined that this "gradient" becomes stronger as the children age. They suggested that the relationship could in part be explained by the fact that children in higher-income households experience fewer chronic conditions, and that their parents manage those conditions better. Currie and Stabile (2003) pursued this analysis further using panel data, and found that the gradient occurs because children of low socioeconomic status receive more shocks. Currie, Shields and Wheatley Price (2004) replicated the analysis with pooled data from the United Kingdom, a country with a universal healthcare system. They found a smaller health-income gradient— one that was not found to increase with child age. Their contention: an important part of the total variation in child health could be explained by unobserved family effects, such as parenting skills, health-related behavior and biological factors. Burgess, Propper and Rigg (2005) used cohort data from the United Kingdom and found that (controlling for maternal health and parental choice of health inputs in early childhood) there is almost no effect of income on child health. Their results also suggest that the transmission mechanism from income to child health operates through maternal health (mental health, in particular) rather than through health-related behaviors. Lindeboom, Llena Nozal and Van der Klaauw (2007a) found that there is a high degree of persistence in children's health (measured by height and the presence of serious conditions), and that this relation does not vary with parental socioeconomic status. Parental health (measured by body-mass index and chronic conditions) remains of prime importance in explaining the health of their children.

The above-cited studies indicate that there may be a small role for the effect of income in explaining child health in the developed world. The effect of income may be different in developing countries, however, where malnutrition, exposure to infectious diseases, a less-developed public healthcare system and unequal access to care characterize the child's environment. Chen and Li (2007) used data on adoptees to examine the effect of family income on child health. (The causal effect of income can be assessed if the assignment of adoptees to adoptive families is random.) They found that income is an important factor for the health of adopted children. Moreover, the effect of income on child health is similar in size for own-births. This suggests that the main effect

of income on child health is post-natal. Duflo (2000) exploited the rapid increase in the coverage and benefits of the South African Old-Age Pension that took effect at the end of the Apartheid era. She found that the extension of the pension program led to an improvement of the health and nutrition of children, and that this effect was stronger for girls. Interestingly, this effect was due entirely to pensions received by women. Case (2001) examined other data from South Africa that cover the same period. She found that the expansion of the pension program protected the health of all household members in families that pooled income. This could be ascribed to improved nutrition, better living conditions and less stress associated with day-to-day life. Combining this with the evidence for developed countries on the importance of parental health, one might conclude that income effects in these studies may persist across generations.

Many empirical studies have determined that family income is an important factor in explaining the school success of children (Becker and Tomes, 1985; Taubman, 1989; Haveman and Wolfe, 1995; Duncan and Brooks-Gunn, 1997). More recently, economists have become aware that the results found in these studies might be confounded due to ability bias. As with the literature on the effect of parental education on child education, the strong correlation of family income and educational attainment might be caused by (mostly unobserved) ability. Recent examples of studies that have attempted to distinguish between association and causation are Blau (1999), Cameron and Heckman (1998, 2001), Cameron and Taber (2001), Mayer (1997), Shea (2000) and Plug and Vijverberg (2005). The general conclusion that emerges from this literature: family income matters, but income elasticities are often found to be very small. We should, perhaps, not expect too much from income and tax policies to manipulate educational choices.

4.2 The effect of child health on educational attainment

Quite a few studies in the development literature study the effect of child health or child nutrition on schooling outcomes. The picture that emerges is that OLS estimation will generally give a strong association between child health or nutrition and educational attainment. Several studies have tried to assess the causal effect of child health via Instrumental Variable approaches (e.g. Alderman, Behrman, Lavy and Menon, 2001) and sibling fixed-effect approaches (e.g. Glewwe, Jacoby and King,

2001). These seem to confirm the naïve OLS estimates, but the size of the effect is generally larger. Miguel and Kremer (2004) used a randomized experiment to evaluate a program of a school-based treatment with a de-worming drug in Kenya. The study was based on a sample of 75 primary schools with a total enrolment of nearly 30,000 children. The sampled schools were drawn from areas with a high prevalence of intestinal parasites among children (92%). Miguel and Kremer found that absenteeism in treatment schools was substantially lower (25%) than in comparison schools, and that de-worming increased schooling by 0.14 years per pupil treated.

The literature for developed countries is rather small. Case, Fertig and Paxson (2005) used British data from the Child Development Study to look at (among other things) the effect of childhood health on educational attainment. They found a strong association between childhood health and later educational attainment. It appears that the presence of chronic conditions at age seven has a stronger impact on educational attainment than does health at age 16. Their conclusion: the negative effect of bad health is cumulative in its effect on education. These results are based on observational data that follow a single cohort, which makes it difficult to make causal statements. Case and Paxson (2006) used adult height as a measure for childhood conditions and childhood health, and found that the height premium in adulthood (that is, better labor market outcomes for taller people) could be explained by childhood scores on cognitive tests and by the fact that taller children selected into occupations that have higher cognitive skill requirements. Currie and Stabile (2007) examined the relationship between several common health disorders, such as ADHD, depression, anxiety and aggression, on future educational outcomes. They conclude that early childhood mental health problems affect educational outcomes and that there is little evidence that income protects against the negative effects of mental health. A recent and innovative approach of Ding, Lehrer, Rosenquist and Audrain-McGovern (2007) focused on a specific set of conditions (ADHD, depression and obesity), and used genetic markers that strongly predict these conditions as instruments. They argued that these genetic markers are valid instruments; using these genes as instruments, they found strong effects of these health conditions on student GPAs. The larger part of this effect seemed to be driven by the effect for females: females were strongly negatively affected by negative physical and

mental health conditions (for males they found no effect). They also found that it was important to take into account co-morbid health disorders as well as health enhancing- or health-deteriorating behaviors. This latter point is of importance for the design of health policies.

4.3 The effect of education on later health and mortality

Since Cutler and Lleras Muney (2007) recently provided an excellent review of the literature on education and health, there is no need to fully review the papers discussed in their study. Below, we draw frequently from their findings. Cutler and Lleras Muney performed some analyses of their own that confirm the strong association between education and (later-life) health. There is evidence for a causal effect of education on health. The most convincing evidence comes from studies that used changes in minimum schooling laws (Oreopoulos, 2003; Arendt, 2005; Lleras Muney, 2005). This implies that one can make statements about the effect of additional schooling only regarding those who are at the bottom of the schooling distribution. Identifying which mechanisms generate these causal impacts remains speculative. The better educated have the better jobs and higher incomes, which may lead to better health and lower mortality rates at later ages. Indeed, the study by Lindahl (2005) found that increases in income could lead to health improvements— but these effects concerned the response to a very large increase in income, for a special group of people (lottery players). Moreover, as argued in section 2, studies by Smith (2005) and Case and Deaton (2003) have concluded that the larger part of the association between SES and health is driven by the effect of health on SES, rather than the other way around. Case and Deaton (2003) found that people in manual occupations have worse self-reported health, and that there is a greater rate of health declines in these occupations. Their argument: much of the differences in health are driven by health-related absence from the labor force. Smith (2005) found that current- and lagged financial measures of SES have no effect on future health, but that education does. This holds for older and for younger workers, thereby suggesting a potential role for factors such as the rank in the social distribution (reflected in the local (psycho-social) environment), the ability to process information and health behaviors.

The Whitehall studies of British civil servants showed that morbidity and mortality fall with increases in social class. Marmot et al. (1997) used

three other datasets from the US and the UK, and found that this relationship also applied to non-civil servants.⁴ The idea is that a low position in the social distribution leads to low control and high (job) demands, which in turn lead to stress, which puts workers at risk for cardiovascular disease (Marmot, 2003). Indeed, Marmot et al. (1997) found a strong relation between a measure for control and cardiovascular disease risk— but this is merely an association.⁵ Cutler and Lleras Muney (2007) argued that social position could not be the main determinant of SES-related health differences. Life expectancy has increased in the developed world over the past three decades, although income inequality and crime have increased and social networks generally have become smaller. Also, some studies have shown that there are gradients in diseases that are not related to stress (Link and Phelan, 1995).

Schooling provides individuals with skills that help them acquire and process information, which helps them make better decisions. This may result in health differences. Kenkel (1990) examined whether consumer health information increases the demand for medical services. He found that more information increases the probability of care use, but that conditional on care use, the quantity of care use is not related to information. Some of his results suggest that poorly informed consumers tend to underestimate the productivity of medical care in treating disease. However, Meara (2001) found that differences in knowledge by SES create only modest differences in health behaviors by SES. She concluded that the most important determinants of differential health behavior are unobserved factors that simultaneously determine health habits and SES. Indeed, as noted by Cutler and Lleras Muney (2007), although both educated and uneducated people today are well aware of the dangers involved with smoking, smoking is still more prevalent among the uneducated.

- 4 Note that the difference in the healthcare system of the two countries indicates that access to medical care cannot be the main determinant of the SES-related health differences.
- 5 Social position may affect adult health via behaviors and via psychosocial pathways that affect the neuro-endocrine system more directly. Accumulations of stressors exert their effect. The body shows biological deviations due to cumulative burdens. The concept of allostatic load plays a role here. Those with high levels will have a breakdown in functioning and a higher risk of disease. (Seeman et al., 2001).

It has long been known that smoking is an important determinant of lung cancer and cardiovascular disease risk. We also recognize that there is a strong negative association between smoking and education; those who are more educated smoke less.⁶ Of interest is whether this association between smoking and schooling is causal, and if so, what mechanisms drive this effect. Two recent papers addressed this issue using Vietnam draft-avoidance behavior as an instrument for college attendance (De Walque, 2007; Grimard and Parent, 2007). The cohort of males born between 1945 and 1950 could avoid the Vietnam draft by enrolling into college, and this could be used as an instrument for college enrollment. The female cohort born between 1945 and 1950 were used as a control group. De Walque (2007) found that level of education did causally affect smoking, and that those who had initiated smoking were more likely to stop once they started their college studies. Grimard and Parent (2007) used a different dataset and confirmed De Walque's finding on smoking initiation, but found imprecise estimates for the smoking cessation decision. Grimard and Parent took a further look into the mechanisms that may drive the causal effect of health. They suggested that either peer effects or endogenous time preferences are likely to be important determinants. Improved information-processing capabilities due to increased schooling did not seem to be important. Khwaja, Silverman and Sloan (2007) examined the relationship between time discounting, other sources of time preference and choices about smoking. They found that subjective time discount rates are not related to smoking, but that more general measures of time preference and self-control, such as impulsivity and financial planning, are related to smoking. Khwaja et al. (2007) thus suggested a potentially important role for preference parameters. However, Cutler and Lleras Muney (2007) suggested that changes in preferences could not be the main reason why education affects health.

6 Prices and income are other probable determinants of smoking, but their effect seems to be limited (see the extensive survey by Chaloupka and Werner, 2000).

5. Summary and implications for health policy

The literature suggests that long-run effects of early childhood conditions are important for morbidity and mortality later in life. There are roughly two channels: direct long-run effects due to “programming,” and indirect effects via education, health and socioeconomic status at different points in the life course.

Direct effects are likely to be quantitatively relevant for developing countries, where exposure to extreme conditions is more common, and where behavior later in life may be less successful in mitigating early-life effects. There are however, some other studies that point towards the relevance of environmental insults, disease exposure and malnutrition for cohorts born in the 20th century in developed countries. Of importance for healthcare policy is that this suggests that one can expect mortality differentials across different cohorts and that the younger cohorts do not necessarily live longer in better health. Also, policies focused on vulnerable families (those living in poor circumstances, exposed to stress and employing bad health behaviors) can be effective in improving the health of the next generation.

Childhood conditions may affect child health, and this may persist into adulthood. The evidence on the effect of family income is mixed, at least for developed countries— although any effect that might be found is expected to be modest. Most studies point at a potentially strong role for the family-specific environment. This includes parenting skills, health behaviors and maternal- and paternal health. Maternal health is probably the most important determinant for child health. This does not mean that there is no role for health policies. Policies aimed at improving the health of young adolescents can be effective in improving the health of the next generation. These interventions may reverse the impact of a poor start early in life and improve health in adolescence and beyond.

Education is undoubtedly one of the strongest determinants of health in later life. Education increases income and labor market opportunities and positively affects health-enhancing behavior. The effect of education on health behavior is causal and likely to be of core importance for health later in life. Policies should focus on educational outcomes. Such policies should intervene at early ages. Recent work Heckman Stixrud and Urzua (2006) shows that early intervention programs targeted to disadvantaged children have their biggest impact on non-cognitive skills

such as motivation, self – control and time preference. Studies cited in section 4.3 showed the importance of these factors for health behaviors. Heckman, Stixrud and Urzua (2006) showed that these non cognitive skills strongly influence schooling decisions and later wages.

In sum, with new cohorts one should focus on early health and education interventions. It would be useful to screen babies and young children at their household circumstances, to determine whether nutrition, heating, stress levels and other indicators are at acceptable levels. Programs targeted to children of disadvantaged households should be implemented at an early age. Among existing cohorts, it is useful to screen individuals born in particularly adverse conditions, to verify whether they are susceptible to cardiovascular disease and other diseases thought to be programmed early in life.

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SUMMARY OF DISCUSSION

By Karen van der Wiel

Birth is the Messenger of Death – But Policy May Help to Postpone the Bad News. New Evidence on the Importance of Conditions Early in Life for Health and Mortality at Advanced Ages

By Gerard van den Berg and Maarten Lindeboom

Chairman: Rick van der Ploeg

Discussants: Dorly Deeg (VU University Medical Centre) and Katherine Carman (Tilburg University)

Netspar Panel: October 18, 2007

Dorly Deeg, professor of the epidemiology of aging at the VU University Medical Centre, opened the discussion. Her first comment involved the title of Gerard van den Berg and Maarten Lindeboom's paper. She would have preferred the subtitle 'New evidence' to the catchy main title because she felt the words 'But policy' put too much emphasis on the role policies can play in determining individual health. Besides, she said, the evidence presented was worthwhile, even without considering the direct policy implications.

Deeg then continued to highlight the life-cycle concept and life-cycle research focus of her own academic discipline, the epidemiology of aging. This background enabled her to discuss the paper from a different perspective. Her first specific comment on Van den Berg and Lindeboom's paper involved the use of societal information such as economic conditions in trying to establish causal health differences among individuals. She was concerned that this method might confuse developments in society with individual health differences. Deeg was also critical of the use of self-assessed health indicators because the responses have been known to differ across, for example, historical periods. Furthermore, she stressed that research results in one country do not necessarily carryover to others. The paper presents US evidence that at high ages the socio-

economic status (SES) health gradient diminishes. Research she has done on Norway, for example, has shown no such decline over age.

Additionally, Deeg provided some explanations for her findings. Van den Berg and Lindeboom reported that there is no age health gradient in the Netherlands with respect to disability. Deeg wondered whether this has to do with the self-assessed nature of the data. She furthermore suggested that the reason behind the lack of effects found when analyzing the impact of the Dutch 'hongerwinter' (1944 famine) on mortality might be found in the psychological resilience of the survivors of the famine.

Deeg raised two other issues. First, she regretted that in many of the studies discussed in the paper there is a lack of very old respondents. Second, she thought that smoking, as a determinant of the SES health gradient, was over-emphasized in the paper. Deeg's own research suggests that only 25% of the health difference between different socio-economic groups can be explained by the greater prevalence of smokers in the lower income groups.

Concerning the policy recommendations in the paper, Deeg doubted whether interventions should be targeted only at those at young ages and at those born in adverse conditions. First, she questioned why Van den Berg and Lindeboom focused on establishing causal instead of associative relationships between socioeconomic conditions and health. Her standpoint was that only when associations are known can you give policy advice. Certain risk groups qualify for policy interventions, irrespective of whether there is a causal link between their income or education and health status. Policies can be designed to improve health per se. Furthermore, she considered it to be rather harsh to target policies only at the current young generation, as this implies that the current old(er) generation is considered somehow to be lost.

Deeg also questioned the policy recommendations implied by the finding that education is an important determinant of health. Perhaps we should not stimulate higher education indefinitely, as there will always be a demand for low-skilled workers. Perhaps the education effect, moreover, is relative, thereby rendering the universal encouragement of education useless. Deeg also questioned the size of the observed effects, and how these change for different risk groups. Then she stressed that in the epidemiological literature there is often a large discrepancy between findings in observational and intervention studies. Drawing

policy conclusions on observational studies might over- or underestimate effects. Finally, Deeg argued for more and better Dutch health data.

The second discussant of Van den Berg and Lindeboom's paper was **Katherine Carman**, assistant professor of health economics at Tilburg University. Carman strongly agreed with the conclusion of the paper that the causal relationship between health and SES runs mostly from health to socioeconomic status. She especially liked the set-up of the reviewed paper by Van den Berg, Lindeboom and Portrait (2006), which uses cyclical macroeconomic conditions at birth as an indication of early-life health inputs, and measures the impact of these conditions on mortality. Carman stated that using exogenous early-life variables such as these is instrumental in determining the causal relationship between SES and health.

Carman then summarized some of the findings in the health economics literature on the relationship discussed. Irrespective of the definition used for both health status and socioeconomic status, a positive correlation between the two is a robust finding in the literature. People are generally either well off or not well off in both respects. A telling example is the fact that Academy Award- and Nobel Prize winners live longer than nominees.

The relationship between SES and health is complex, however, and involves more than early-life conditions alone. Carman presented several additional links between the two. One of these is that higher educated individuals have better knowledge of health and health-altering behaviors. Educated individuals, furthermore, are faster and better at adopting new health-improving technologies. A third additional difference between lower and higher socioeconomic groups involves malnutrition. She pointed out that poor nutrition is, sadly, prevalent also outside developing countries. Poorer inhabitants of the city of Detroit, for example, have a hard time buying vegetables, as only convenience stores are located in the area. Carman then continued to explain the issue of the cumulative effect of stress, when one is poor, on health indicators. This additional link between SES and health is missing from Van den Berg and Lindeboom's paper. Poverty leads to a sense of isolation and a lack of control over one's circumstances, and this accumulated stress is then associated with health problems. There is evidence from primate research

that changes in the hierarchy of a primate group diminish the health of animals that fall in status.

Furthermore, Carman emphasized the importance of third factors, such as risk aversion and discount factors, that influence both socioeconomic status and health. Someone that has an extremely high discount factor (i.e. appreciates the present much more than the future) is less likely to invest in education (leading to a lower socioeconomic status), and may be less likely to invest in their health by exercising, eating right and using preventive care (leading to worse health).

Carman concluded that while early-life factors are very important in explaining the SES health gradient, other factors are also influential. She then wrapped up her discussion with the remark that policy interventions targeted at the disadvantaged young are important, but cannot prevent every health problem later in life.

The chairman continued the discussion by collecting some questions from the audience. **Lans Bovenberg** questioned whether trying to intervene in the educational level of children might not be too late, as evidence suggests that non-cognitive skills are developed even before the start of formal education.

Henk Don asked what might be causing the increasing disability levels among young adults, other than measurement issues in the new WAJONG disability definition.

Peter Kooreman, finally, expressed interest in the magnitude of the effects of suggested interventions on health.

Gerard van den Berg then answered the questions raised. He began by thanking the discussants for pointing out additional aspects of the SES-health relationship. He then addressed Deeg's comment on the added value of establishing causal rather than associative relationships between SES and health. First, he believed that this remark reflects cultural differences between different academic fields. Second, he agreed with Deeg that it is not always necessary to identify exact causal relationships in order to formulate policy advice. He referred, as an example, to the strong association found in the medical literature between low birth weights and cardiovascular diseases later in life. Although a strong causal relationship has thus far not been established, there is enough evidence suggesting that a low birth weight should be treated as an

indicator for bad early-life conditions. According to Van den Berg, policy targeted at individuals with low birth weights will thus be beneficial.

Third, in many studies, associations are tacitly interpreted as displaying causal effects—even though the studies condition on outcomes earlier in life, and there may be unobserved confounders affecting those outcomes as well as the outcomes of interest. Van den Berg mentioned that so-called life-cycle studies that investigate health effects at different life stages are particularly vulnerable to this. The same confounding problem exists, for example, in the economic literature on the effects of education on earnings. Policy measures targeted at increasing education levels could be useless if the effects of education on earnings are restricted to the smart individuals who take up education anyway. Distinguishing a causal link between education and earnings has been crucial for determining the effectiveness of intervention policies.

Van den Berg then answered Deeg's comment on confusing societal developments for individual differences in health. He emphasized that in the current economic literature short-term cyclical developments such as GDP growth and season of birth are used to capture early-life conditions and that as such, the problem doesn't exist. This would be different if one used long-term trends. He pointed to studies of the 1944 Leningrad famine, which coincided with many other societal changes in Russia, thereby rendering it difficult to distinguish the impact on health of the famine alone. Van den Berg then suggested some reasons for the absence of effects of the 1944 Amsterdam famine on mortality: the relatively short period that has since passed and the small sample size.

Van den Berg agreed with Deeg on the need for health-altering policies for the current old(er) generations. He meant only that the effectiveness of policies targeted to children will be larger, since these will also impact the fertility of the children's children, for example. Van den Berg also agreed with Deeg that the reported studies depend both too much on self-reported measures of health and too little on very old respondents. In his own research, he is preventing this bias.

Van den Berg then touched upon the additional links between SES and health and the importance of the third factors mentioned by Carman. Some recent studies have indeed found an association between stress levels and current economic conditions. Carman's references to the recent evidence concerning the importance of allostatic loads are gratefully acknowledged.

Then, answering the question raised by Kooreman, Van den Berg explained the quantitative impact of early-life conditions. With both Dutch and Danish historical data he found a one-year shorter life of an individual born in a recession, conditional on surviving the recession. The negative causal effect on life expectancy of being born in spring in the Northern hemisphere is smaller, namely three months. It should be pointed out that the causal effect of early-life conditions on mortality could be large, even though the current instruments have small overall effects.

Van den Berg answered Bovenberg's question on education by saying that even though interventions are necessary as early as possible, it is still important to stimulate education because this will in due course influence the opportunities of the children of the targeted population. Don's question about disability of current young generations was answered by Deeg. She explained that obesity and inactivity are rapidly becoming more prevalent in the current population. Henk Don and **Maarten Lindeboom** added that psychiatric problems are also causing disability levels of the young to rise. **Lou Spoor**, furthermore, suggested that contemporary nutrition could be to blame for future health problems, but that 'we don't know the unknowns'.

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