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THE RELATIONSHIP BETWEEN INTELLIGENCE AND HEALTH IN LUXEMBOURG

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“Für uns alle ist die Welt verwirrend, und wenn man es nüchtern betrachtet, besteht der Unterschied zwischen einem Gesunden und einem Kranken vor allem im Ausmaß der Fähigkeit, das Verwirrende an der Oberfläche zu kaschieren. Darunter tobt das Chaos.”

Arno Geiger

– to my family –

Abstract

Even though the general health and life expectancies of Western societies have been consistently rising throughout the 20th century, socioeconomic health inequalities continue to persist. Individuals from lower socioeconomic groups have substantially worse health and an increased mortality risk compared to individuals from higher socioeconomic groups. As external factors such as material resources cannot fully account for these health inequalities, personal factors such as intellectual abilities have been suggested as additional important explanatory factors. The research field concerned with the effects of intelligence on different health outcomes is called *cognitive epidemiology*. Results from this field of research have now established that childhood intelligence is an important predictor of different health outcomes in adulthood. Specifically, children with higher intelligence exhibit a lower mortality risk and enjoy better health in adulthood compared to children with lower childhood intelligence.

Despite these findings, several open research questions remain: (1) Almost all previous studies on the relation between childhood intelligence and adult health have been conducted in English-speaking or Scandinavian countries. Can these findings be generalized to countries with different cultural backgrounds, health-care systems, or levels of social mobility? Specifically, Luxembourg offers universal access to quality health care, which may compensate for some of the effects of individual differences in intelligence on health, and as a result, intelligence may lose its impact. (2) Physical health is a multidimensional concept with three distinct subdimensions: a physical subdimension (e.g., presence of diagnosed diseases, number of doctor visits in a certain time period), a subjective subdimension (e.g., satisfaction with one's own health), and a (social-)functional subdimension (e.g., unimpaired participation in social and occupational activities or performing household tasks). Most previous studies on the relation between intelligence and health outcomes have focused on

the physical health subdimension. Hence, considerably less is known about the effects of intelligence on the functional and subjective subdimensions. This issue is of particular importance as childhood intelligence may be differentially related to different aspects of adult health. (3) It remains unclear whether different facets of childhood intelligence (e.g., general, fluid, or crystallized intelligence) predict adult health equally well, as most studies on the topic have used only global measures of childhood intelligence to predict later health. However, investigating different facets of childhood intelligence as predictors of adult health would provide insights into which facets of intelligence are important in personal health management and could be targeted by interventions. (4) Despite considerable evidence that has emphasized the relevance of education and further indicators of subsequent socioeconomic status (SES) as potential mediators between childhood intelligence and later health outcomes, previous research has yielded inconsistent results regarding the extent to which these relations are mediated. Some studies have reported pronounced mediation via education and subsequent SES, yet others have reported little or no mediation. However, knowing and understanding which mediational mechanisms underlie the intelligence-health relation and the extent to which they mediate this relation are crucial for applying findings from cognitive epidemiology to public health. (5) As is the case for studies on intelligence and health outcomes, most studies on the relation between intelligence and mortality risk have been conducted in English-speaking or Scandinavian countries. Thus, it remains unclear whether the results of these studies can be generalized to Luxembourg. Further, there is controversy about whether the effect of intelligence on mortality exists across the entire range of intelligence scores or whether individuals at the lower end of the intelligence distribution constitute a risk group with a particularly high mortality risk. Identifying potential risk groups is crucial for determining which groups should be targeted by interventions to reduce inequalities in health and mortality risk.

The present Ph.D. thesis addressed these five research questions with three distinctive studies. Study I investigated whether childhood intelligence would predict adult physical, functional, and subjective health 40 years later even when controlling for the effects of childhood SES. Study I also investigated whether a global measure of childhood general intelligence or whether more specific facets such as fluid and crystallized intelligence would better predict adult health. Study II investigated whether and the extent to which educational attainment and SES in adulthood would mediate the effects of childhood intelligence on the three adult health dimensions. Study III investigated whether childhood intelligence would predict adult mortality risk when controlling for childhood SES and whether individuals at the lower end of the intelligence distribution would constitute a risk group with a particularly high mortality risk.

All three studies were embedded in the general framework of the Luxembourgish MAGRIP project. This large-scale longitudinal study comprised two waves of measurement over a 40-year period. In the first wave of measurement in 1968, detailed intelligence and socioeconomic data were collected on a randomly selected nationally representative sample comprising 2,824 students at the end of their primary education (M = 11.9 years; SD = 0.6 years; 50.1% male). In the second wave conducted 40 years later, 745 participants (M = 51.7 years, SD = 0.6 years; 46.7% male) provided data on their educational careers, adult SES, and functional, subjective, and physical health. In addition, the mortality rate was established for the participants in the first wave of MAGRIP: 166 participants (69.9% male) had died.

The results of the three studies demonstrated that childhood intelligence, particularly childhood fluid intelligence, showed a significant association with adult health: Lower childhood intelligence scores were associated with worse health outcomes on all three dimensions of physical, functional, and subjective health in adulthood, even when controlling for childhood SES. These effects were entirely mediated via educational attainment and adult

SES, with educational attainment playing a crucial role in these mediational processes. Further, childhood intelligence showed a significant association with adult mortality such that lower childhood intelligence scores were associated with an increased mortality risk. This effect was particularly strong among men at the lower end of the intelligence distribution. These results suggest that even high-quality public health care cannot fully offset the cumulative effects of childhood intelligence on adult health. Intelligence may thus be an important explanatory factor for socioeconomic inequalities in health. Promising means for reducing these socioeconomic health inequalities consist of interventions that are designed to improve childhood intelligence, to improve environments for childhood physical and intellectual development, and to make public health care and preventive measures or treatments accessible to adults with lower intellectual abilities.

Keywords: childhood intelligence, fluid intelligence, crystallized intelligence, childhood socioeconomic status, multidimensional adult health, premature mortality, mediation, educational attainment, adult socioeconomic status, socioeconomic health inequalities

List of Publications

This Ph.D. thesis is based on the following three manuscripts:

I.

Wrulich, M., Brunner, M., Stadler, G., Schalke, D., Keller, U., & Martin, R. (2012). Forty years on: Childhood intelligence predicts health in middle adulthood. *Health Psychology*, Advance online publication. doi:10.1037/a0030727

II.

Wrulich, M., Brunner, M., Stadler, G., Schalke, D., Keller, U., Chmiel, M., & Martin, R. (2013). Childhood intelligence and adult health: The mediating roles of education and socioeconomic status. *Intelligence*, 41(5), 490–500. doi:10.1016/j.intell.2013.06.015

III.

Wrulich, M., Stadler, G., Brunner, M., Keller, U., & Martin, R.. Childhood intelligence predicts premature mortality: Results from a 40-year population-based longitudinal study (*submitted*).

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Chapter I

General Introduction

1. Introduction

Health is one of the most important domains of human life. When asked, people rate health as more important than finances, standard of living, or housing (Bowling, 1995; Limb, 2011). Unfortunately, health is not equally distributed among us. Some people enjoy good health until old age. For instance, some seniors enjoy a good swim or riding their bikes well beyond the age of 80. However, some people suffer from ill health very early in life and even from premature death.

Why do these differences in health occur? Of course, we differ with respect to our genetic and physical makeup, which renders us more or less vulnerable to disease. But health is also related to our behavior. Smoking, heavy drinking, and eating fatty food are bad for our health, and exercise and eating plenty of fruits and vegetables are good for our health. Yet, to behave healthily, we also have to know more about which behaviors are healthy and which ones are not. Thus, next to behavior, education in general and health literacy in particular are related to health and health differences. Further, socioeconomic factors, such as a higher income, a prestigious occupation, or a safe working environment contribute to good health. And of course, social relations are related to our health—social isolation or a lack of social support can be very detrimental to our health.

Yet, all these important factors—genetic and physical makeup, behavior, education, socioeconomic status, social support—cannot entirely explain why we differ so much with respect to our health. Maybe there is something more, some kind of general capacity that lies within us that is essential to our success in all these crucial life domains—to health, healthy behavior, education, socioeconomic status, and social support. Could it be that a person's

intelligence as measured by a typical IQ test constitutes this general capacity? Could it be that how smart we are matters to our health? The present Ph.D. project was designed to shed light on this question. It investigated the importance of childhood intelligence for adult health and mortality in Luxembourg and considered the roles of education and socioeconomic factors.

In this chapter, I will review the main concepts that are at the heart of the present Ph.D. project. First, I will provide introductory sections on health and intelligence before bringing these two crucial concepts together to introduce the framework of cognitive epidemiology. After this general introduction, I will provide an outline of my Ph.D. thesis.

2. Health

2.1 Definition of health

As health is such a crucial aspect of our lives, the definitions of health and illness have preoccupied philosophers, physicians, policy makers, and others concerned with human health since ancient times. One health definition that influenced medicine for centuries was formulated by Hippocrates (c. 460 BC – c. 370 BC), the father of Western medicine (Grammaticos & Diamantis, 2008). He posited that health might be the result of a balance of four fluids: blood, yellow bile, black bile, and phlegm. Ill health, he believed, resulted from an imbalance of these fluids. This theory of "humoralism" or "humorism" influenced the view on health and the treatment of ill health in Western medicine well into the age of the Renaissance, when it was first criticized by Paracelsus (c. 1493-1541), and into the 19th century.

Today, many different and sometimes conflicting definitions of health exist (Shroufi, Chowdhury, Aston, Pashayan, & Franco, 2011). One of the most important definitions was formulated by the World Health Organization (WHO) over half a century ago, conceptualizing health as "a state of complete physical, mental and social well-being and not

merely the absence of disease or infirmity" (World Health Organization, 1958, p. 469). This definition is still in use and forms the first principle of the WHO's constitution (World Health Organization, 2006). However, it has been criticized for different reasons. First, many critics argue that the WHO definition is unrealistic and that including the term "complete" makes it highly unlikely that anyone would be healthy for a reasonable period of time (Brüssow, 2013; Smith, 2008). Thus, this definition may contribute to the "medicalization of society" (Huber et al., 2011, p. d4163), as the focus on complete well-being may lower thresholds for screening and treatment of "conditions" that were not previously defined as health problems. Second, some authors argue that this definition lacks operational value (Jadad & O'Grady, 2008), as the term "complete" may be difficult to operationalize and measure (Brüssow, 2013; Huber et al., 2011). Third, a further problem for the WHO definition may be the change in demographic and disease patterns. When the WHO definition was formulated, acute diseases presented the main burden of illness, and chronic disease led to an earlier death. Today, aging with chronic illness has become the norm. The WHO definition may thus be perceived as counterproductive as it declares people with chronic diseases and disabilities definitively ill (Huber et al., 2011). Fourth, other critics have argued that the WHO definition corresponds much more closely to happiness than to health (Saracci, 1997). Failure to distinguish happiness from health would thus imply that any disturbance in happiness, however minimal, may be perceived as a health problem.

These criticisms have led to further attempts to define health. For instance, Bircher (2005) defines health as "a dynamic state of well-being characterized by a physical and mental potential, which satisfies the demands of life commensurate with age, culture, and personal responsibility" (p. 336). This definition moves away from the more static definition of the WHO toward a more dynamic one, and it takes into account changing health needs, especially in relation to age, culture, and personal responsibility. Saracci (1997) suggested a

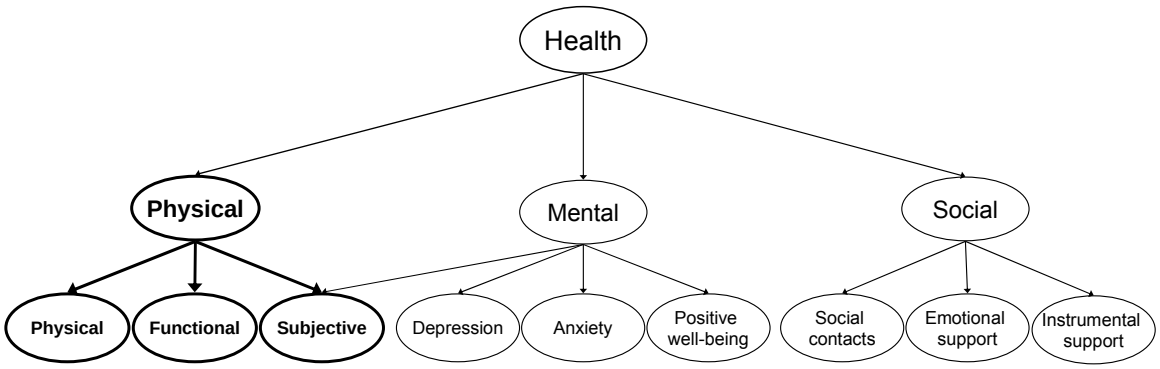
further descriptor of health as "a condition of well-being, free of disease or infirmity, and a basic and universal human right" (p. 1410). According to the author, this description of health does not contradict the WHO definition. Rather it provides an intermediate concept linking the WHO's ideal to the real world of health as measurable by means of appropriate indicators of mortality, morbidity, and quality of life. Finally, a consortium of international health experts suggested that health be framed as "the ability to adapt and self manage in the face of social, physical, and emotional challenges" (Huber, 2010; Huber et al., 2011, p. 1).

Despite these criticisms and attempts to reformulate the definition of health, the WHO version still remains the most widely accepted definition (McDowell, 2006). This is because the WHO's effort to define health was groundbreaking with respect to its breadth and ambition in different domains (Huber et al., 2011). First, it overcame the negative definition of health as the absence of disease. Second, and most importantly, it widened the view on health to a multidimensional perspective that includes the physical, mental, and social dimensions (Bircher, 2005; Saracci, 1997). Thus, it challenged political, academic, community, and professional organizations to pay attention to the social determinants of health (Jadad & O'Grady, 2008). In a more general sense, the WHO approach includes indispensable elements of a definition of health as it accounts for the bio-psycho-social nature of human existence (Engel, 1977). Even the more recent attempts to reformulate a definition of health take into account its multidimensional nature, comprising physical as well as social and mental aspects (Huber et al., 2011; Saracci, 1997). Also, the WHO itself has formulated an extension of its original definition in the Ottawa Charter (World Health Organization, 1986), which once more stresses the multidimensionality of health, stating that "health is a positive concept emphasizing social and personal resources, as well as physical capacities" (p. 1). Finally, the measurement techniques that were based on the WHO's formulation have further contributed to its wide acceptance (McDowell, 2006).

As the WHO's definition of health has been highly influential, several attempts have been made to address the criticism of its lack of operationalizability. One prominent and influential model that translates the WHO's three general dimensions of physical, mental, and social health into more specific terms was formulated by Liang and colleagues (Liang, 1986; Liang, Bennett, Whitelaw, & Maeda, 1991; Whitelaw & Liang, 1991). Figure I-1 depicts their hierarchical model. The global construct of health consists of three distinct yet interrelated dimensions on an intermediate level, namely, the physical, mental, and social dimensions, as suggested by the WHO. These three health dimensions can be defined in different ways, resulting in different subdimensions of each dimension on a lower level. For instance, even though the term "physical health" may seem unambiguous, the physical health dimension—which is at the heart of the present Ph.D. project—can be defined in three different nonexclusive ways (Liang, 1986): (a) According to the medical or physical definition, physical health refers to the presence or absence of disease. (b) According to the social or functional definition, physical health refers to conformity to norms or the capacity to adequately participate in social activities. Sickness in this sense refers to incapacity or nonconformity to norms due to health problems (Twaddle, 1974). (c) According to the psychological definition, physical health refers to an individual's subjective perception and evaluation of his or her overall physical health status. As a consequence of these three approaches to defining physical health, the physical health dimension in the model by Liang and colleagues comprises the subdimensions of *physical*, *functional*, and *subjective* health. The subjective subdimension of physical health is also part of the more general mental health dimension, which additionally comprises depression, anxiety, further mental health problems, and aspects of positive well-being. Finally, the social dimension of global health comprises, among other factors, social contacts and emotional and instrumental support (Whitelaw & Liang, 1991). Liang and colleagues use the term "physical health" for the physical dimension

of global health as well as for the physical subdimension of the physical health dimension. I followed this approach in this Ph.D. thesis. However, I clarified in each case whether the term "physical health" referred to one of the three dimensions of global health or to the subdimension of physical health.

Figure I-1. *Hierarchical model of health*



Note. This figure draws on the hierarchical model of health as formulated by Liang and colleagues. The physical health dimension and its subdimensions have been highlighted due to their central role in this Ph.D. thesis.

In a nutshell, the definition of health by the WHO (1958, 1986, 2006) together with the hierarchical model as specified by Liang and colleagues (Liang, 1986; Liang, et al., 1991; Whitelaw & Liang, 1991) are highly influential in all areas of health research and policy. Further, they have been shown to be highly useful as they successfully combine the physical, psychological, and social aspects of health (*Lancet*, 2009). Moreover, they provide a starting point for the measurement of multidimensional health. Thus, they were adopted for the present Ph.D. project. Specifically, the WHO definition and the model by Liang and colleagues were chosen as the basis for the measurement of health in this Ph.D. project's two studies that investigated the relation between childhood intelligence and adult health.

To provide further information on the measurement of health in general and in the present Ph.D. project, I will briefly review different approaches to health measurement in the following section.

2.2 Measurement of health

Societies in the 21st century show a progressively growing interest in the promotion, improvement and maintenance of adequate health. This requires appropriate health measures that incorporate the multidimensional and universal nature of the concept (Shroufi et al., 2011). Yet, there is no consensus on how best to measure health (McDowell, 2006; Shroufi et al., 2011). Instead, the choice of health indicators depends on the purpose of the measurement and the intended level of investigation. As there are a multitude of different health indicators, different approaches to their classification have been put forward (McDowell, 2006; McDowell, Spasoff, & Kristjansson, 2004). One approach makes a rather general distinction between population and individual health measures (McDowell, 2006). At the population level, health measures are mostly based on aggregated indicators of individual health and are used to summarize health in a particular population (McDowell et al., 2004). They may be used to compare the health status of different populations, to monitor changes in the health of a given population, or to investigate health inequalities within a population (Murray, Salomon, & Mathers, 2000). Frequent measures of population health consist of event counts and rates such as crude or standardized death rates, prevalence and incidence rates of a certain disease in a population, healthy life expectancy at birth (HALE, derived from the synonymous "health-adjusted life expectancy"), or years of life lost (YLL) due to a certain disease (Eurostat, 2009; Huber, 2010; Shroufi et al., 2011; WHO, 2010). Even though these event-based measures provide an objective, precise, and readily available estimate of health, they do not capture the multidimensional nature of the concept (Shroufi et al., 2011).

By contrast, the multidimensional assessment of health is easier to accomplish when health is measured at the individual level. Here, health measures are principally used to diagnose illness, to conduct cross-group comparisons between individuals, to investigate the same individual over time, to predict the need for care, or to evaluate treatment outcomes

(McDowell, 2006). To capture health on the individual level, several measurement methods are currently used (Smith & Goldman, 2010). One approach for classifying these methods is the general distinction between objective and subjective health measures (McDowell, 2006). Objective measures are based on laboratory or diagnostic tests, whereas subjective measures are based on the judgment of a person (e.g., a clinician, a patient). Objective health measures include diagnosed diseases, biomarkers associated with chronic disease and health conditions (e.g., Body Mass Index [BMI], systolic blood pressure, or high-density lipoprotein [HDL] cholesterol), physical functioning measures, and clinical measures based on medical exams (e.g., measured walking, grip strength, or forced expiratory volume [FEV]). These measures are suitable for capturing the physical subdimension of physical health (see Figure I-1). However, they are not able to capture the subjective or functional subdimensions of physical health, let alone the social and mental dimensions of global health. However, all these aspects are equally important for the complete state of health (Liang, 1986; Shroufi et al., 2011). Therefore, next to these more objective individual health measures, subjective health measures are widely used to capture different aspects of health. They may comprise judgments about an individual's health made by others (e.g., a physician rating the general health status of his or her patient), or they may be self-assessed health measures. Self-assessed health measures can be presented in interviews, questionnaires, or rating scales (McDowell, 2006). In these measures, the respondent makes a statement about certain aspects of his or her health status.

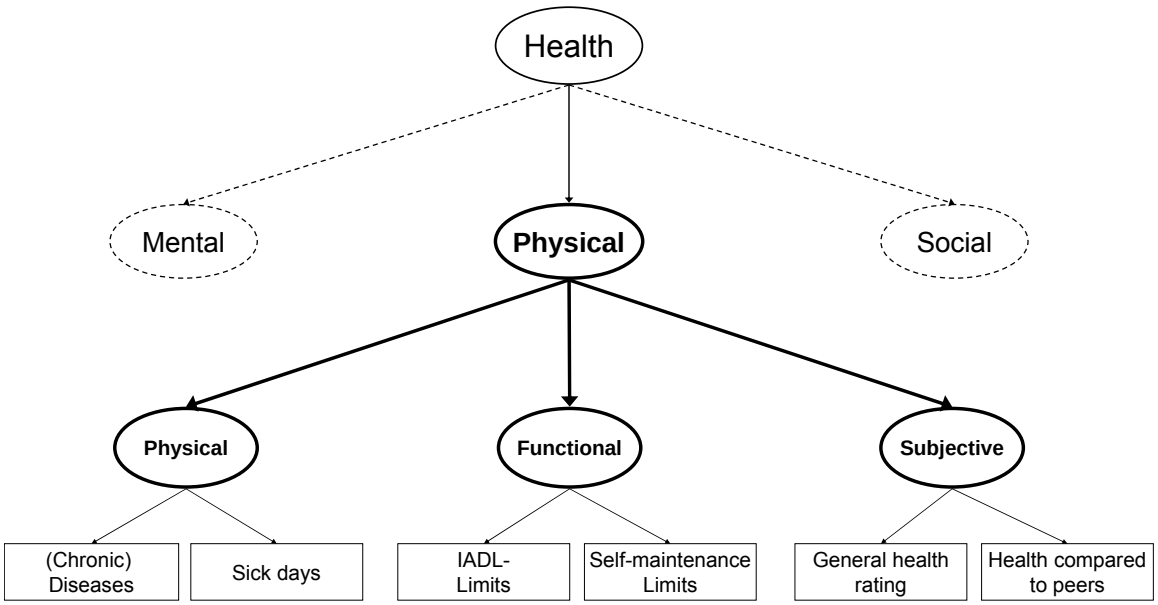
Self-assessed health measures may be used to capture all dimensions and subdimensions of health. To capture the physical subdimension of physical health, these measures may comprise self-reports on the presence or absence of diseases or symptoms, or health care usage. For instance, respondents may report whether they suffer from diabetes, whether they regularly take antihypertensive medication, or whether they were recently

admitted to a hospital (Smith & Goldman, 2010). To capture the subjective subdimension of physical health, self-assessed health measures often take the form of survey questions that ask respondents to rate their overall health, for instance on a 4- or 5-point scale that typically runs from *excellent* to *poor*. To capture the functional subdimension of physical health, respondents report the impact of health problems and diseases on various areas of everyday functioning. Assessments of this type are generally referred to as "functional disability indicators" (McDowell, 2006). Examples are the Barthel Index, which measures functional independence in personal care and mobility (Mahoney & Barthel, 1965), or the Instrumental Activities of Daily Living (IADL) scales, which cover impairment in activities needed for continued community residence (e.g., traveling out of the neighborhood, running errands, playing a game of skill; McDowell, 2006). One example of an IADL scale is the Functional Activities Questionnaire (Pfeffer, Kurosaki, Harrah Jr., Chance, & Filos, 1982). To capture the mental dimension of global health, general self-assessed health measures such as the Rand Mental Health Inventory (Veit & Ware, 1983) or disease-specific measures such as the Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) are used. To capture the social dimension of global health, different self-assessed health measures are used, depending on the subdimension of social health in question. These instruments may capture social or instrumental support, (satisfaction with) social contacts, or the ability to fulfill social roles (Liang, 1986; McDowell, 2006). Examples include the Rand Social Health Battery (Donald & Ware, 1984) or the Medical Outcomes Study Social Support Survey (Sherbourne & Stewart, 1991). Moreover, self-assessed health measures may be used to have respondents report their health behavior. Finally, some health measures combine the physical, mental and social dimensions of global health in one instrument. Among these general health status measures are the Medical Outcomes Study short-form surveys, namely, the SF-36 (Ware & Gandek, 1998) and SF-20 (Stewart, Hays, & Ware, 1988). The common ground of all these

self-assessed health measures is that they assign a numerical value to each response category (i.e., the frequency, severity, or quality judgment of a certain behavior, symptom, or disability). These values may then be used to derive a general numerical indicator of the degree of health or illness in the respective dimension or subdimension (McDowell, 2006).

As mentioned in Section 2.1 on the definition of health, the WHO health definition and the hierarchical model by Liang and colleagues were chosen to provide the basic conceptualization of health in the present Ph.D. thesis. Beyond translating the WHO definition into the three health dimensions and their respective subdimensions (see Figure I-1), Liang and colleagues also suggested indicators for measuring these dimensions. For instance, the Liang model provides a choice of specific indicators for each of the three physical health subdimensions, which are at the heart of the present Ph.D. thesis (Liang, 1986; Liang, et al., 1991; Whitelaw & Liang, 1991). Figure I-2 shows several indicators of the physical, functional, and subjective subdimensions of physical health.

Figure I-2. *Selected indicators of the physical, functional, and subjective subdimensions of physical health*



Note. This figure depicts indictors of each of the subdimensions of physical health, as suggested by Liang and colleagues.

In the Liang model, indicators of the physical subdimension are comprised of self-reports of different health problems and chronic diseases, such as respiratory or circular conditions. As an additional indicator of physical health, Liang and colleagues suggested using self-reports of the number of sick days in a certain time period. Such indicators may come so close to reflecting objective health or sickness that they are referred to as "proxies" for acute and chronic illness (Liang, 1986, p. 252 and p. 258). Indicators of the functional subdimension are comprised of limitations in Instrumental Activities of Daily Living (IADL) and in self-maintenance due to health problems. IADL limits include limitations in activities such as carrying out minor household repairs or going on a train or airplane trip. Self-maintenance includes activities such as climbing stairs or grooming. Indicators of the subjective health dimension include respondents' ratings of their general health and their health status compared to the health status of their peers on a 4-point rating scale ranging from *very good* to *poor*. These indicators of the physical, functional, and subjective subdimensions of physical health were used as a starting point for the measurement of health in the present Ph.D. thesis (see Chapters II to IV for more comprehensive descriptions of the measures employed).

One of the major fields in which the measurement of health is applied is the investigation of differences in health status. Population measures are used to investigate health differences between populations, and individual measures are used to investigate health differences between groups of individuals. A highly relevant finding from research on health differences is that individuals from different socioeconomic strata differ substantially with respect to their health. These *socioeconomic health inequalities* form an important general framework for the interpretation of effects of intelligence on health and are one of the motivating forces for the present Ph.D. project. Therefore, they will be introduced in the following section.

2.3 Socioeconomic health inequalities

Health is not equally distributed among us. Some individuals enjoy comparatively good health across their life course, whereas others suffer from ill health, sometimes very early in life. These health inequalities are socially stratified: Individuals from lower socioeconomic groups exhibit systematic disadvantages in health compared to individuals from higher socioeconomic groups (Cutler, Lleras-Muney, & Vogl, 2008; Mackenbach, 2012; Mackenbach et al., 2008). An extensive body of knowledge about socioeconomic health inequalities has been compiled in recent decades. The following sections will provide a brief overview with an emphasis on several important questions. Many of these questions concern generalizability: Are socioeconomic health inequalities generalizable across different diseases and health indicators? Are they generalizable across different times, places, and populations? Are they generalizable across different indicators of socioeconomic status (SES)? Other questions concern their origins: What explains socioeconomic health inequalities? What are the driving forces behind them? Could intelligence be one of these driving forces?

2.3.1 The generalizability of socioeconomic health inequalities

To provide the reader with the most important finding right away: Socioeconomic health inequalities are remarkably general (Link, Phelan, Miech, & Westin, 2008). For instance, they can be observed for the large majority of health indicators (Adler et al., 1994). Most importantly, they can be observed for many diseases that carry a heavy burden of morbidity and premature mortality (Adler & Ostrove, 1999). Thus, socioeconomic inequalities in life expectancy amount to a 5- to 10-year difference in average life expectancy at birth, and to a 10- to 20-year difference in disability-free life expectancy (Mackenbach, 2012). Moreover, socioeconomic health inequalities are substantial and consistent for diseases involving different organ systems and seemingly different etiologies, such as cardiovascular, gastrointestinal, musculoskeletal, renal, chronic respiratory, and psychiatric

diseases, diabetes, the metabolic syndrome, arthritis, tuberculosis, as well as for accidental and violent deaths (Adler & Ostrove, 1999; Cantwell, McKenna, McCray, & Onorato, 1998; Cunningham & Kelsey, 1984; Cutler et al., 2008; Gottfredson, 2004; Kaplan & Keil, 1993; Pincus, Callahan, & Burkhauser, 1987). One example of a rare disease for which higher incidence rates can be observed in higher socioeconomic groups is breast cancer. This finding has been related to delayed childbearing in women with a higher SES. However, once diagnosed, higher SES women show higher survival rates than lower SES women (Adler & Ostrove, 1999; Cutler et al., 2008). Beyond mortality and specific diseases, socioeconomic inequalities can be observed for important predictors of these health outcomes, namely, for health literacy, health knowledge, and health behavior. Health literacy refers to "the ability to apply the literacy skills needed to function fully and effectively as a patient" (Davis, Meldrum, Tippy, Weiss, & Williams, 1996, p. 94), and is thus a rather general ability to obtain and process health-related information, make judgments, and behave accordingly. One aspect of health literacy is health knowledge (i.e., knowledge about diseases, symptoms, and factors that influence health; e.g., symptoms of cardiovascular disease or diabetes, or the effects of smoking, alcohol use, and physical fitness on health; Cutler et al., 2008; Gottfredson, 2004). Health behavior refers to various specific behaviors that can be conducive or harmful to health, such as smoking, drinking, diet, exercise, use of illegal drugs, use of preventive medical care, or care for hypertension and diabetes (Cutler & Lleras-Muney, 2006). Both health literacy and knowledge are closely linked to health behavior, which in turn influences health. For instance, low literacy has been associated with low use of preventive care, poor comprehension of one's illness, and noncompliance and nonadherence to medical regimens (Gottfredson, 2004). Health literacy, knowledge, and behavior have consistently been shown to be more present in higher socioeconomic groups (Cutler & Lleras-Muney, 2006; Cutler et al., 2008; Gottfredson, 2004).

A further question regarding generalizability is whether socioeconomic health inequalities are generalizable across different SES indicators. SES on the individual level is usually measured by education, occupation, or income (e.g., Mackenbach et al., 2008). These indicators reflect different aspects of SES and are only moderately correlated with each other (Adler et al., 1994; Adler & Ostrove, 1999; Cutler et al., 2008). In general, however, health inequalities between different socioeconomic groups are found regardless of which indicator is used (Adler et al., 1994; Adler & Ostrove, 1999; Cutler et al., 2008; Gottfredson, 2004; Link et al., 2008), and the common component of these indicators explains a good portion, yet not all, of these inequalities (Cutler et al., 2008). Despite similar associations between SES indicators and health, the association between SES and health may also vary across indicators and across the phases of the life cycle. For income, higher levels are usually associated with better health (Fuchs, 2004). However, the direction of causality is not clear, and the influence of income on health varies considerably by age. In adulthood, income can improve access to health inputs (such as medical care and food), but health also improves one's ability to participate in the labor market and earn a decent wage. Moreover, evidence suggests that income does not have a very large causal impact on adult health. Additionally, third factors such as education may determine both financial resources and adult health status. However, for children, parental income has strong protective effects on health (Cutler et al., 2008). For occupations, health status seems to improve with increases in occupational status (Cutler et al., 2008), and riskier and unhealthier occupations that are related to worse health are often found in lower occupational ranks. However, some riskier occupations come along with higher wages, so the effects of income and occupations may be contrary (Fuchs, 2004). For education, however, the associations with health seem the most robust (Cutler & Lleras-Muney, 2006; Cutler et al., 2008; Fuchs, 2004; Gottfredson, 2004): More highly educated individuals show better health than less educated individuals. In fact, education is so strongly

related to health that Grossmann (2003) stated that "years of formal schooling completed is the most important correlate of good health" (p. 32). Income and other labor market outcomes mediate only some of the effects of education on health. The higher frequency of healthy behaviors among more highly educated individuals seems to play a larger role. Evidence suggests that better educated individuals behave more healthily due to more health knowledge but also due to their greater ability to process information regarding healthy behaviors, to learn and to self-manage health. Moreover, there is evidence for reverse causality in the sense that worse health affects educational success (Cutler & Lleras-Muney, 2006; Cutler et al., 2008). In sum, socioeconomic health inequalities can be observed for different SES indicators. In childhood, parental resources such as education and income have a potent effect on health. Once childhood health is set, the effect of economic resources on health diminishes. In most of adulthood, income and wealth no longer appear to have a large effect on health. Education, by contrast, continues to be a powerful determinant of health, but to a great extent because of its impact on behaviors rather than its association with resources (Cutler et al., 2008).

A final result from research on socioeconomic health inequalities is that these inequalities are highly generalizable across different populations, times, and countries (Gottfredson, 2004; Link et al., 2008). Prevalence rates and profiles of different diseases can differ by gender and race, for instance, or they may change over time in all demographic groups. Further, major causes of death and disease can differ considerably between countries. However, even though the strength of the association may vary, the pattern of better health in higher socioeconomic groups consistently pervades all other differences between times, places, and populations. Importantly, socioeconomic health inequalities exist regardless of the level of health care, overall wealth, and economic prosperity of a country. In developed and developing nations, in rich and poor societies, in countries with universal as well as

market-based health care systems, individuals from higher SES groups tend to enjoy better health (Adler & Ostrove, 1999; Cutler et al., 2008; Gottfredson, 2004; Mackenbach, 2012; Mackenbach et al., 2008). Moreover, comparative studies in Europe have shown that socioeconomic inequalities in mortality and morbidity are not smaller in countries with relatively universal and generous welfare policies (e.g. the Nordic countries) than they are in other countries (e.g. the United Kingdom with its more liberal welfare regime or Southern European countries with their more family-based welfare arrangements; Mackenbach, 2012).

Different theories for explaining socioeconomic health inequalities have been put forward. The following section will present a brief introduction to a selection of theories, some of which suggest that intelligence may be one important explanatory factor for health inequalities.

2.3.2 Theories explaining socioeconomic health inequalities

Any theory that attempts to explain socioeconomic health inequalities faces two major challenges. First, it has to explain the remarkable generalizability of these inequalities across health indicators, SES indicators, times, populations, and countries with different levels of human development, economic prosperity, and health care. Second, it has to explain a very puzzling finding, namely, that socioeconomic inequalities have actually been widening in recent decades for some health outcomes (Gottfredson, 2004). For instance, English data on mortality suggest that whereas inequalities in mortality narrowed until 1950 in England, they have since then widened substantially (Mackenbach, 2012; Marmot, Kogevinas, & Elston, 1987). A widening of inequalities in mortality during the last three or four decades has also been reported for many other Western European countries, and it seems to have continued into the 21st century (Mackenbach, 2012). This widening may be attributed to faster proportional mortality declines for cardiovascular diseases in higher SES groups as well as other explanatory factors. For instance, rising rates of mortality from lung cancer, breast

cancer, respiratory disease, gastrointestinal disease, and injuries among men and/or women in lower socioeconomic groups have been observed in several countries (Mackenbach, 2012; Mackenbach et al., 2003).

Several theories have been proposed to account for socioeconomic health inequalities. For many years, the so-called *poverty paradigm* has dominated thinking about why such inequalities exist (Gottfredson, 2004). Under this paradigm, the inequalities are presumed to result from differences in access to health care and other such resources (Hummer, Rogers, & Eberstein, 1998). The bottom line of this approach is "wealth secures health" (Gottfredson, 2004, p. 181). The poverty paradigm has foundered, however, on a growing number of contrary facts. Most importantly, the paradigm's key health resource—greater access to medical care—has surprisingly little relation to differences in health. As mentioned before, Great Britain and other countries that had expected to break the link between SES and health by providing universal health care were dismayed when the inequalities in health not only failed to shrink but even grew (Gottfredson, 2004; Mackenbach, 2012; Mackenbach et al., 2003). Furthermore, equalizing the availability of health care does not equalize its use. For instance, less educated and lower income individuals seek preventive health care (as distinct from curative care) less often than do better educated or higher income individuals, even when care is free (Adler, Boyce, Chesney, Folkman, & Syme, 1993; Gottfredson, 2004). Moreover, greater use of medical care does not necessarily improve health (Marmot et al., 1987). For instance, a randomized controlled experiment in the U.S. tested the effects of subsidizing health care costs at different levels in six American cities. Participants with free care used more medical care than those with only partly subsidized care, but their health was no better after 2 years. Participants with free care had indiscriminately increased their use of inappropriate as well as appropriate care (Gottfredson, 2004; Lohr et al., 1986). Thus, as the poverty paradigm failed, researchers began to look for other plausible explanations of the

remarkably general and sometimes even widening socioeconomic health inequalities. One prominent approach is the theory of *fundamental social causes* of health inequalities (Link et al., 2008; Link, Northridge, Phelan, & Ganz, 1998; Link & Phelan, 1995). According to this theory, it is not the relatively proximal risk factors for disease that cause socioeconomic health inequalities, even though they are socially stratified. Such proximal risk factors include smoking, diet, cholesterol level, psychosocial stress, and working conditions. Rather, it is the social forces that underlie social stratification that ultimately cause health inequalities. Specifically, a person's SES provides him or her with "flexible resources" that can be used in different places and at different times to avoid disease and death. These resources include knowledge, money, power, prestige, and beneficial social connections. They can be deployed at the individual level such as when people use them to construct a healthy lifestyle, or at a contextual level such as when people use resources to gain access to salutary contexts such as good neighborhoods, safe jobs, and robust social networks. According to their proponents, the fundamental-cause theory explains the generalizability of socioeconomic health inequalities because the flexible resources may be deployed to avoid whatever risks may exist and adopt whatever protective strategies may be available. Access or non-access to flexible social and economic resources thus creates and recreates associations between socioeconomic circumstances and risk and protective factors. This results in the occurrence of socioeconomic health inequalities for different health and SES indicators as well as different countries and times (Link et al., 2008; Link & Phelan, 1995; Mackenbach, 2012).

One strength of the fundamental-cause theory is that it focuses attention not only on the proximal causes of disease but also on fundamental aspects of social stratification. It thus creates the possibility of more fully explicated sociological explanations for health inequalities. The theory has been challenged, however. Specifically, some researchers argue that the fundamental-cause theory provides nothing more than a reformulation of the health

inequalities problem and that it does not identify the specific pathways that link SES and health. In this view, additional theories are needed to explain why health inequalities continue to persist and even widen even though some of the possible causes, such as restricted access to universal health care have been eliminated or at least attenuated in modern welfare states (Gottfredson, 2004; Mackenbach, 2012). These additional theories should consider the importance of social selection and, most importantly, personal characteristics. Social selection refers to the fact that intergenerational social mobility has increased systematically in most high-income countries in recent decades (Breen, 2004; Mackenbach, 2012), meaning that selection into higher SES groups depends less on one's socioeconomic family background, but rather on personal characteristics, such as intellectual ability or personality profiles. Crucially, these personal characteristics are also highly relevant for health because health in developed nations depends to a large extent on behavior change, and behavior in turn strongly depends on personal characteristics (Mackenbach, 2010, 2012). Social selection may thus have made the lower social groups more homogeneous with respect to such personal characteristics that increase the risks of ill-health (i.e., low intellectual ability and less favorable personality profiles; West, 1991). In this view, socioeconomic health inequalities may be the result of the social stratification of personal characteristics. This idea has been most sharply formulated in an influential paper by Linda Gottfredson in which she states that intelligence may be the "epidemiologists' elusive 'fundamental cause' of social class inequalities in health" (Gottfredson, 2004, p. 174). According to this approach, intelligence is critical for health and for explaining socioeconomic health inequalities because it is a highly general and context-independent resource that individuals do (or do not) actively and directly use to obtain beneficial health circumstances (Gottfredson, 2004; Link et al., 2008).

In a nutshell, health is socially stratified: Systematic and generalizable health inequalities between higher and lower socioeconomic groups continue to persist. Current theories from medical sociology and social epidemiology have offered explanations but cannot account for the full pattern, including the widening of health inequalities. Additional theories emphasize the importance of personal factors such as intelligence. These factors, just as health, are socially stratified. Could intelligence really be one explanatory factor for socioeconomic health inequalities? And what are the specific mechanisms by which intelligence may influence health? Before bringing intelligence and health together in Subchapter 4, the following subchapter will first provide an introduction to the concept of intelligence.

3. Intelligence

The word intelligence is derived from the Latin verb *intelligere*: "to understand" or "to choose between." In the present Ph.D. thesis, the terms *intelligence* and *cognitive ability* or *abilities* are applied interchangeably. This is due to the most prominent definition of intelligence as higher cognitive processes (see Section 3.1 below). The branch of psychology that is concerned with research on intelligence or cognitive abilities is called differential psychology. In general, differential psychology investigates the nature, origins, applications and consequences of individual differences in intelligence and personality (Anastasi, 2007; Deary & Batty, 2007; Modig Wennerstad, 2010). The research field of *cognitive epidemiology* investigates individual differences in intelligence in relation to individual differences in health. Before the research field of cognitive epidemiology is introduced, the following subchapter will briefly review approaches to the definition, structure, and measurement of intelligence as well as the relation of intelligence to key life outcomes other than health.

3.1 Definition of intelligence

The concept of intelligence has a long history; as early as 380 BC, Plato argued that the human soul had three elements, namely, intellect, emotion, and will. According to Plato, the intellect was the thinking portion within us, which perceives what is real and not merely apparent, judges what is true and false, and makes rational decisions (Eysenck, 1979). Since then, the concept has been developed and studied, and this has mainly been done by psychologists in the 20th century (Modig Wennerstad, 2010).

In the last century, several successful attempts were made to obtain a consensus among researches on what intelligence refers to (Gottfredson, 1997; Neisser et al., 1996). A similarity among these different attempts to define intelligence was that all definitions identified higher cognitive processes as the core aspect of intelligence. For instance, in a study conducted by Snyderman and Rothman (1987), more than 600 participating experts concluded that abstract thinking or reasoning, problem solving, and the capacity to acquire knowledge were core aspects of intelligence (Gottfredson, 1997). Another prominent definition was formulated in 1994 on a declaration in the Wall Street Journal. It was signed by 52 well-known intelligence researchers and conceptualized intelligence as follows:

"Intelligence is a very general mental capability that, among other things, involves the ability to reason, plan, solve problems, think abstractly, comprehend complex ideas, learn quickly and learn from experience. It is not merely book learning, a narrow academic skill, or test taking smarts. Rather, it reflects a broader and deeper capability for comprehending our surroundings – 'catching on', making sense of things or 'figuring out' what to do." (Gottfredson, 1997, p.13).

This definition of intelligence as higher cognitive processes was adapted for the present Ph.D. thesis. It stems from the psychometric tradition of intelligence research. This psychometric tradition is the dominant one because of its extensive use in research and its

wide application in practical settings (Neisser et al., 1996). In the psychometric research tradition, a person's intelligence level is typically identified by his or her score on an intelligence test, which summarizes performance across a broad range of cognitive tasks. Further, in this tradition, a vast amount of research on the structure of intelligence has been conducted, the results of which will be briefly reviewed in the next section.

3.2 The structure of intelligence

Intelligence tests usually comprise several quite different subtests (e.g., questions about general information, recalling digits, vocabulary, arithmetic problems, completing pictures with missing details, or arranging pictures in a logical sequence), and test takers usually tend to perform either well or poorly on all of them. This finding led to the conclusion that there may be some kind of a general intelligence, a so-called "*g*-factor" that underlies all the different subtests of an intelligence test. The first researcher to describe the *g*-factor was Charles Spearman in a famous paper in 1904. He examined school children's scores on different academic subjects and found that they were correlated. Spearman concluded that these correlations could best be explained by assuming that there was a single factor underlying them (Spearman, 1904). This early work formed the basis of factor analysis (Modig Wennerstad, 2010). Across the following decades, the assumption of a *g*-factor was investigated and questioned by many researchers, and it is now known that more factors are needed to describe a person's intelligence. In 1993, John Carroll published his famous seminal treatise "Human cognitive abilities: A survey of factor analytic studies" (Carroll, 1993). He collected as many studies as possible on intelligence that he considered to be of good quality and then re-analyzed these studies, which included over 460 sets of data. In doing so, he developed the *Three Stratum theory*. This theory was based on Spearman's *g* factor, but also on the Cattell and Horn theory of fluid and crystallized intelligence (Cattell, 1963; Horn & Cattell, 1966; McGrew, 2009). As fluid intelligence is often denoted *Gf* and

crystallized intelligence *Gc*, this theory is also called the *Cattell-Horn Gf-Gc model*. In the Three Stratum theory, intelligence is divided into three hierarchically ordered strata (i.e., levels; see Figure I-3). Stratum I—the lowest, most specific level—consists of 50 to 60 or more narrow abilities that are linearly independent of one another (that is, possibly intercorrelated but with clearly separated vectors in the factorial space). These Stratum I abilities are all grouped under the Stratum II factors. Stratum II—the broad level—comprises approximately 8 to 10 broader abilities, which are also linearly independent of one another. These abilities include: fluid ability, crystallized ability, general memory and learning, broad visual perception, broad auditory perception, broad retrieval ability, broad cognitive speediness, and processing speed. Finally, Stratum III —the highest most general level—comprises only a single ability which constitutes *g*. Carroll considered the *g*-factor to be general in the sense that it was likely to be present to some degree in nearly all measures of cognitive ability (Carroll, 1993). In fact, the *g*-factor often accounts for nearly half the variance when a broad battery of cognitive tests is administered to a representative sample of the adult population (Deary, 2012).

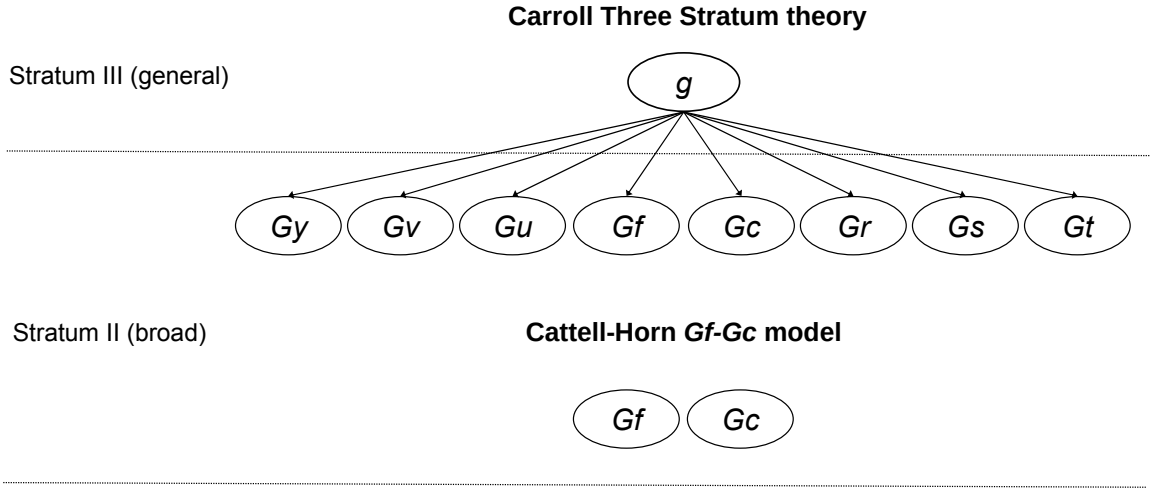
As was mentioned before, Carroll's influential Three Stratum theory of intelligence was to a large extent inspired by the Cattell-Horn model of fluid and crystallized intelligence (Cattell, 1963; Horn & Cattell, 1966). The concepts of fluid and crystallized intelligence are crucial for the first study of the present Ph.D. project. Cattell and Horn argued against the existence of the *g*-factor (the third stratum of Carroll's model). In their view, a single *g*-factor could not account for the patterns of variation seen among multiple intellectual abilities so that at least two broader factors were required to describe these variations. Their original theory separated around 100 abilities (Stratum I), which work together in various ways in different people, and which can be categorized into two different broader sets of abilities, namely, fluid intelligence (*Gf*) and crystallized intelligence (*Gc*) (Stratum II; see Figure I-3).

According to the original Cattell-Horn *Gf-Gc* model, there is no Stratum III (*g*), as fluid and crystallized intelligence already constitute the highest stratum of intelligence. Fluid intelligence reflects basic abilities in reasoning, thinking and acting quickly and abstractly, solving novel problems, and encoding short-term memories. It is grounded in physiological efficiency and was originally assumed to depend primarily on genetic processes, thus being independent of learning, experience, and education (Cattell, 1963; Horn & Cattell, 1966). However, more recent evidence has shown a substantial environmental determination also of fluid intelligence, as evidenced by the Flynn effect (i.e. the worldwide rise in the mean level of intelligence test performance over successive age cohorts throughout the 20th century; Dickens & Flynn, 2001), effects of schooling (Cliffordson & Gustafsson, 2008), and recent findings of the plasticity of the human brain, particularly in the early years (Blair, 2006). Tests of fluid intelligence include solving puzzles and coming up with problem-solving strategies (Horn & Cattell, 1966, 1967). Crystallized intelligence reflects general knowledge that comes from prior learning and past experience. Thus, it reflects the extent to which an individual has been able to learn and profit from education, culture, and experience. This ability is developed on the basis of one's personality and motivation in the course of education and depends to a lesser extent on the physiological influences that mainly affect fluid intelligence. However, it has been argued that the ability to take advantage of environmental learning opportunities that lead to the acquisition of crystallized intelligence also depends on fluid intelligence. In this view, fluid intelligence is invested into the acquisition of crystallized intelligence (Cattell, 1987; McArdle, Ferrer-Caja, Hamagami, & Woodcock, 2002). Tests of crystallized intelligence include reading comprehension, general knowledge questions, and vocabulary exams (Horn & Cattell, 1966, 1967). The distinction between fluid and crystallized intelligence mirrors the distinction between culture-fair and culture-bound intelligence tests (Eysenck, 1979; see Section 3.3). Fluid and crystallized

intelligence have different trajectories over the life course (Cattell, 1987; McArdle et al., 2002). Both cognitive abilities increase throughout childhood and adolescence. Yet, whereas fluid intelligence peaks in early adulthood and then declines, gradually at first and then more rapidly in older ages; crystallized intelligence continues to improve well into a person's 60s and 70s, as it improves with the accumulation of knowledge and understanding (Cattell, 1987; Horn, J. L., 1980; Horn & Cattell, 1967; McArdle et al., 2002). Both abilities are important in everyday life, and they are often complementary. For example, when taking a psychological exam, fluid intelligence helps a person to develop a strategy to solve the numerical questions, whereas crystallized intelligence helps the person to recall the necessary formulas for the calculations (Knox, 1977). Even if fluid and crystallized intelligences are correlated, a person can have a high fluid intelligence but a lower crystallized intelligence, depending on the environment, whereas a low fluid intelligence would make it difficult to score high on crystallized intelligence despite a favorable environment.

Carroll's Three Stratum theory and the Cattell-Horn *Gf-Gc* theory are the two most prominent theoretical models for capturing human intellectual abilities (McGrew, 2009). Despite several differences, such as the presence (Three Stratum theory) or absence (Cattell-Horn) of a *g*-factor at Stratum III, there are remarkable similarities between the two approaches. This is in part due to extensions of the original *Gf-Gc* theory, which led to the inclusion of additional broad abilities at Stratum II in the Cattell-Horn model (e.g., visual and auditory processing, cognitive processing speed, etc.; Horn & Noll, 1997; McGrew, 2009). The similarities between the two models became so substantial that a single umbrella term, the *Cattell-Horn-Carroll (CHC)* theory of intelligence was proposed (McGrew, 2009). Figure I-3 shows a simplified representation of Carroll's Three Stratum theory and the original Cattell-Horn *Gf-Gc* model.

Figure I-3. *Two approaches for capturing the structure of intelligence*



Note. This figure draws on the presentation of the Three Stratum and Cattell-Horn *Gf-Gc* models in McGrew (2009). As only general intelligence *g*, fluid intelligence (*Gf)*, and crystallized intelligence (*Gc)* are crucial concepts in the present Ph.D. thesis, additional factors at Stratum II that were added more recently to form the extended Cattell-Horn model have been omitted. Further, for reasons of clarity, all narrow Stratum I abilities have been omitted. Key: *g* = general intelligence; *Gf* = fluid intelligence; *Gc* = crystallized intelligence; *Gy* = general memory and learning; *Gv* = broad visual perception; *Gu* = broad auditory perception; *Gr* = broad retrieval ability; *Gs* = broad cognitive speediness; *Gt* = processing speed.

Some researchers believe that fluid intelligence is identical to *g*. The notion that *g* and fluid intelligence are equivalent corresponds to a third conceptualization of intelligence that combines the two former ones, namely, the Three Stratum theory and the Cattell-Horn theory of fluid and crystallized intelligence. In this third conceptualization, the third stratum factor *g* exists and is measurable. There are also the two second stratum factors of fluid and crystallized intelligence. Yet, in this theory, *g* is considered to be highly or even perfectly correlated with the second-stratum factor of fluid intelligence, but is considered to be linearly independent of the second-stratum factor of crystallized intelligence, or of any other possible second stratum factors (Gustafsson, 1984, 1989, 2001; Gustafsson & Balke, 1993; Gustafsson & Undheim, 1996).

Closely linked to the definition and structure of intelligence, especially in the psychometric tradition of intelligence research, is the measurement of intelligence, which will

be introduced in the next section.

3.3 Measurement of intelligence

Intelligence tests were developed and used for practical reasons, for example to select officers in the army, to select talented children for schools, or occupational selection in industry. There is a general distinction between culture-bound and culture-independent or culture-fair tests. Culture-bound tests measure the candidate's background knowledge and his/her ability to use his/her intelligence for the purpose of taking in information and to benefit from instructions. These tests will by definition vary by culture and over time. Culture- and time-independent tests should, in a perfect situation, be independent of environmental influences such as schooling and socioeconomic position. The distinction between culture-bound and culture-fair intelligence tests mirrors the distinction between crystallized and fluid intelligence (Eysenck, 1979; Modig Wennerstad, 2010) described previously in Section 3.2.

Alfred Binet, a French psychologist, developed the first modern intelligence test, the Binet-Simon intelligence scale, in 1905 (Binet & Simon, 1905). Its purpose was to distinguish mentally retarded children from those with behavioral problems (Neisser et al., 1996). Together with Theodore Simon, Binet published revisions of his intelligence scale in 1908 and 1911. In 1916, Terman published a refined version of the Binet scale, which he named the Stanford-Binet Intelligence Scale. It is still in use today in its modified form. The Binet test battery was the first widely used mental test of cognitive ability. There are many such tests today. Usually, these tests comprise subtests for different cognitive abilities and allow a single composite score to be derived as a measure of general intelligence g. Two of the most well-known and validated tests in English-speaking countries are the Wechsler Adult Intelligence Scale (WAIS; Wechsler, 2008) and Raven's Progressive Matrices (Raven, Raven, & Court, 1998). The WAIS is a comprehensive intelligence test that contains items

that measure verbal ability (e.g., general knowledge or vocabulary), perceptual reasoning ability (e.g., putting blocks together to match patterns on cards or picture completion), working memory (e.g., mentally manipulating mathematical problems), and processing speed (e.g., copying a coding pattern). Raven's Progressive Matrices measures reasoning ability or fluid intelligence. It contains 60 items with increasing difficulty. In each test item, the test taker is asked to identify the missing element that completes a pattern. In German-speaking countries, the German version of the Wechsler Adult Intelligence Scale—the Hamburg Wechsler Intelligenztest für Erwachsene (HAWIE-R; Tewes, 1991)—as well as the German version of Raven's Progressive Matrices are frequently used (Horn, R., 2009; Kratzmeier, 1979). Another widely used German intelligence test is the Leistungsprüfsystem (L-P-S [Performance Test System]; Horn, W., 1962, 1983). The L-P-S was also used in the three studies that constitute the present Ph.D. project (see Chapters II, III, and IV).

Within the tradition of intelligence measurement, the highly prominent IQ or "Intelligence Quotient" was developed. Originally, IQ referred to the ratio of mental age to chronological age. Even though the term IQ is still commonly used (also as an abbreviation for general intelligence *g*), the scoring of modern IQ tests is now based on a projection of the participant's measured rank on the Gaussian bell curve with a mean of 100 and a standard deviation (*SD*) of 15 (Modig Wennerstad, 2010). The intelligence test that was used in the present Ph.D. thesis and the subtests used in Study I were transformed to this scale

3.4 Intelligence and key socioeconomic life outcomes

General intelligence (*g*) has been shown to be a powerful predictor of crucial outcomes across the lifespan (Gottfredson, 2002; Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007; Strenze, 2007). In fact, intelligence has been shown to be the best single predictor of major socioeconomic outcomes, both favorable (good education, occupation, income, job performance) and unfavorable (adult poverty, incarceration, chronic welfare use; Gottfredson,

2002). The following section provides a brief overview of the influence of intelligence on key socioeconomic outcomes as these outcomes play a crucial role when it comes to investigating the predictive capacity of intelligence for health.

Performance on intelligence tests is highly predictive of different aspects of educational attainment, such as school and college grades (Deary & Johnson, 2010; Deary, Strand, Smith, & Fernandes, 2007; Kuncel, Hezlett, & Ones, 2004). These effects are so well established that they have been referred to as an "unquestioned fact" (Jensen, 1988, p. 277). Next to educational attainment, the effect of intelligence on socioeconomic outcomes as indexed by income, occupational success, and achieved socioeconomic status (SES) is well-established (Kuncel et al., 2004; Schmidt & Hunter, 1998, 2004). For instance, intelligence has been shown to exert a strong influence on performance in highly complex occupations (Gottfredson, 2002). Further, intelligence predicts measures of acquired knowledge and skills, which are consistently related to success in academic and job performance settings (Kuncel & Hezlett, 2010). These effects of intelligence are highly generalizable and valid across different jobs, situations, and settings (Gottfredson, 2002; Schmidt & Hunter, 1998). Moreover, performance on intelligence tests predicts future wages (Cawley, Heckman, & Vytlacil, 2001) and occupational status (Gottfredson, 2002). As intelligence predicts educational attainment and occupational success, its substantial effects on achieved SES do not seem surprising. These effects have been replicated in numerous studies (Judge, Higgins, Thoresen, & Barrick, 1999; Nettle, 2003; Strenze, 2007).

The microprocesses that account for the substantial effects of intelligence on educational and socioeconomic outcomes may be pictured as a "chain reaction" (Gottfredson, 2002, p. 369). Individual cognitive resources in terms of intelligence are transformed into educational attainment (Kuncel et al., 2004; Lubinski, Benbow, Webb, & Bleske-Rechek, 2006). Educational attainment is then transformed into SES (Deary et al., 2005; Gottfredson,

2002; Johnson, Brett, & Deary, 2010), as indicated by occupational status (Jencks & Riesman, 1968; Taubman & Wales, 1975) or income (Becker, 1975; Miller, 1960). Thus, educational attainment acts as a "gate keeper" that creates the potential to obtain higher SES.

Beyond these socioeconomic life outcomes, a comparatively new research field has begun to investigate intelligence as a predictor of another key life outcome, namely, health. This research field, which is crucial for the present Ph.D. project, has been coined *cognitive epidemiology* (Deary & Der, 2005). The next subchapter will review the body of evidence that has been compiled within cognitive epidemiology.

4. Intelligence and health outcomes – Cognitive epidemiology

4.1 Results on intelligence and health outcomes

The systematic investigation of intelligence as a predictor of different health outcomes just began in the last decade of the 20th century (Deary, 2012). Mortality was probably the first health outcome studied in relation to intelligence. In an early article on this topic, O'Toole and Stankov (1992) showed that in a sample of Australian Vietnam veterans, those with lower intelligence test scores upon entry into the armed services were more likely to have died by midlife. Nearly a decade later, Whalley and Deary (2001) showed that lower IQ scores at age 11 were significantly associated with an increased risk of mortality up to age 76. Since these first publications, the number of studies linking intelligence measured early in life to adult mortality and nonmortality outcomes—a research field labeled cognitive epidemiology (Deary & Der, 2005; *Intelligence*, 2009)—has increased steadily. Today, mortality remains the most widely studied outcome in cognitive epidemiology (Der, Batty, & Deary, 2009), and the link between intelligence and all-cause and cause-specific mortality is well-established (Calvin et al., 2011; Deary, Weiss, & Batty, 2010). Different intelligence tests and different follow-up periods have been used in the respective studies, and almost all

studies have shown that a lower measured intelligence predicts a greater risk of mortality. For instance, a recent meta-analysis showed that across the 16 studies included in the analysis, a one standard deviation advantage in intelligence test scores was associated with a 24% lower risk of death during a 17- to 69-year follow-up (Calvin et al., 2011). Moreover, intelligence is a sizeable predictor of mortality risk when compared to other risk factors for mortality. Batty and colleagues compared early and middle adulthood intelligence and further risk factors for all-cause mortality by transforming them to Relative Indices of Inequality (RII) scores. These scores provide an impression of the size of the risk associated with one specific risk factor. Specifically, the RII in the study by Batty and colleagues can be interpreted as the mortality hazard between the extreme ends of the risk factor distribution. Thus, an RII of 2.0 indicates that the mortality hazard between the extreme ends of the risk factor distribution is twice as high for the most disadvantaged (high risk) as for the most advantaged (low risk). The risk factor most strongly related to all-cause mortality was family income (RII = 7.46), second came adult intelligence (RII = 4.41), and then smoking, educational attainment, pulse rate, and intelligence in early adulthood (Batty, Shipley, Gale, Mortensen, & Deary, 2008).

In addition to mortality, intelligence in childhood and early adulthood has been linked to various physical diseases. For instance, several studies have replicated the relation between higher intelligence in early life and a lower risk of cardiovascular disease (CVD; Hart et al., 2004; Modig Wennerstad, Silventoinen, Tynelius, Bergman, & Rasmussen, 2009). For instance, Hart and colleagues showed that a one standard deviation disadvantage in intelligence at age 11 was related to an 11% increased risk of hospital admission or death due to cardiovascular disease across the adult life span. Moreover, higher intelligence in early life has been related to risk factors for cardiovascular and other diseases, such as a lower risk of hypertension (Batty, Deary, Schoon, & Gale, 2007a; Modig & Bergman, 2012; Starr et al., 2004) and obesity (Batty et al., 2007a; Chandola, Deary, Blane, & Batty, 2006; Gottfredson

& Deary, 2004; Modig & Bergman, 2012). Intelligence has also been found to be related to the metabolic syndrome, a cluster of risk factors for CVD and other diseases. For instance, in one study, higher premorbid intelligence was associated with a lower risk of four of the five individual components comprising the metabolic syndrome: hypertension, high BMI, high triglycerides and high blood glucose. Moreover, a one standard deviation increase in intelligence was associated with a 14% lower risk for the entire metabolic syndrome (Batty, Gale, et al., 2008). The metabolic syndrome partially mediated the association between intelligence and CVD. In another study, Batty and colleagues compared different risk factors for CVD by again transforming them to Relative Indices of Inequality (RII) scores. When CVD mortality was the outcome, cigarette smoking was the number one risk factor, and intelligence was second, even before income, systolic blood pressure, and physical activity (Batty, Deary, Benzeval, & Der, 2010). In addition to cardiovascular disease, early life intelligence has been found to predict additional diagnosed diseases (e.g., diabetes, chronic lung disease, or arthritis) and self-reported physical health problems (e.g., ulcers, chest pain, or sleeping troubles; Der et al., 2009) as well as mental health outcomes and psychiatric diseases (e.g., psychological distress, depression, PTSD, schizophrenia, or anxiety; Deary, 2010; Der et al., 2009; Gale et al., 2008; Gale, Hatch, Batty, & Deary, 2009). Further, intelligence has also been found to predict health-relevant behaviors such as smoking (Batty et al., 2007a), alcohol intake (Batty, Deary, & Macintyre, 2006; Batty, Deary, et al., 2008; Hatch et al., 2007), physical activity, and the preference for low-sugar and low-fat diet (Batty, Deary, Schoon, & Gale, 2007b; Gottfredson & Deary, 2004). Finally, intelligence has been found to predict intentional and non-intentional injury, such as suicide and accidents (Deary, 2010). Beyond the effects on the individual level, intelligence has been shown to be an important predictor of health at the national and international levels. For instance, Reeve and Basalik (2010) showed that the average state IQ in the 50 U.S. states was substantially

associated with a range of health indicators even after controlling for differences in health care expenditures and state wealth.

The relation between intelligence and morbidity and mortality has been investigated in different age groups. Whereas an association between higher intelligence and a lower risk for these outcomes has been consistently shown in populations followed until the age of 65, results for populations older than 65 have been somewhat mixed. For instance, one study reported a significant association between higher childhood intelligence and lower risk of CVD, coronary heart disease, and stroke only for participants up to the age of 65, but not over 65 (Hart et al., 2004). The authors hypothesized that higher risk people could have been removed from the at-risk population after the age of 65 as they were more likely to have had a medical event in the period up to age 65. They further suggested—without specification, however—that the mechanisms behind these events may differ before and after age 65 and that childhood intelligence plays a role only in the former. By contrast, another study that investigated the association between intelligence and mortality among individuals 70 years of age and older reported that higher intelligence still significantly decreased the risk for mortality in this study sample. Moreover, the results indicated a stronger association between fluid intelligence and mortality than between crystallized intelligence and mortality (Batterham, Christensen, & Mackinnon, 2009). However, in this study, intelligence had been measured in older age and not in childhood. To my knowledge, no studies to date have investigated the impact of different types of intelligence assessed in childhood on adult mortality or morbidity.

Different non-exclusive mechanisms have been suggested to account for the relation between intelligence and mortality and morbidity; these will be reviewed in the next section.

4.2 Mechanisms that account for the intelligence-health relation

Among the different mechanisms that could account for the consistent relation

between childhood intelligence and adult health outcomes are physiological (including genetic), socioeconomic, as well as behavioral and personal factors. As early as 1972, Riegel and Riegel proposed two main theories to account for the relation between cognitive decline in adulthood and mortality. The first was a biological theory in which physiological mechanisms related to cell aging were responsible for cognitive decline and death. The second was a sociological theory in which disadvantages associated with a lower socioeconomic status (SES), such as education, income, nutrition, and medical assistance, affected cognitive performance and survival rates (Batterham et al., 2009; Riegel & Riegel, 1972). These two early theories have been extended and applied to account for potential mechanisms by which early life intelligence may influence different health outcomes in adulthood (Batterham et al., 2009).

4.2.1 Biological mechanisms

Modern biological theories that account for the intelligence-health relation focus on potential genetic or other physiological factors. Specifically, it has been suggested that common physiological factors may affect both intelligence and health outcomes. For instance, the so-called *system-integrity* hypothesis posits that individual differences in the integrity of an underlying general physiological makeup, partly determined by genetic factors, may explain the association between intelligence and health outcomes (Deary et al., 2010). The idea is that intelligence reflects not only brain efficiency but also the neural aspect of a well-put-together body in general—a body that is well placed to respond to environmental challenges and to be able to return to equilibrium after allostatic load. This would imply that the genetic factors that determine brain function also determine other body functions. If this is the case, intelligence would be a proxy for those underlying genetic factors, meaning that the association between intelligence and health outcomes would be confounded by genetic factors. This is difficult to study other than in twin studies. However, other possible markers

of system integrity—measurable indicators of the efficiency of the brain and body—have been used to test this hypothesis. For instance, reaction-time tasks can be seen as crude measures of the brain's general efficiency in processing information (Deary et al., 2010). Compared to psychometric intelligence tests, these tasks should be less sensitive to environmental factors and could be viewed as a proxy for a genetic setting. Reaction time has been significantly associated with all-cause mortality such that faster reaction times are associated with a reduced mortality risk. Moreover, reaction time explained the association between IQ and mortality in one study (Deary & Der, 2005). This finding lends support to the system-integrity explanation of the associations between intelligence and health outcomes, if processing speed is an effective indicator of neurological integrity that reflects overall physiological integrity. However, in the study by Deary and Der (2005), reaction time was measured at the age of 56, when disease processes could have started to lower both intelligence and reaction time and increased mortality risk. Moreover, without a full understanding of why intelligence and reaction time are significantly correlated, the interpretation of mechanisms remains problematic. Further, the construct of system integrity needs to be explicated more fully (Deary et al., 2010).

Another instance of a biological theory conceptualizes both early intelligence and later health as an "archaeological record" of prior (e.g., perinatal and childhood) insults (Gottfredson & Deary, 2004; Whalley & Deary, 2001). These insults influence both intellectual and physiological development, thus acting as another common biological cause of the intelligence-health relation. A possible example of the theory of prior insults is that cognitive differences, as well as the risk of illnesses such as diabetes and cardiovascular disease later in life, are correlated with fetal development and birth weight (Gottfredson & Deary, 2004).

4.2.2 Socioeconomic mechanisms

In general, modern sociological theories that account for the intelligence-health relation assume that this relation is mainly the result of confounding and/or mediation through socioeconomic factors either in childhood or adulthood. Confounding could occur when childhood SES affects childhood intelligence, adult SES, and adult health outcomes. In this view, childhood SES would be the driving force behind the effects of intelligence on health. However, the confounding theory has been put into question. First, modern societies have to a certain degree become "meritocratic", meaning that it is not so much childhood SES but rather personal factors, such as intelligence, talent, and effort that are the crucial factors that determine individual life trajectories (Mackenbach, 2012). Second, in most cognitive epidemiology studies that adjusted for childhood SES as measured by parental occupation or income, the effects of intelligence on later mortality and morbidity were barely affected. Thus, the intelligence-health relation does not seem to be driven to a large extent by childhood SES (Calvin et al., 2011; Deary, 2010). Rather, childhood intelligence and SES seem to have distinguishable effects on adult health. For instance, Osler and colleagues (2003) investigated the association between childhood SES and adult mortality and adjusted for intelligence at the age of 12. This association was attenuated, but not completely, suggesting a separate effect of childhood SES and intelligence on later health outcomes.

By contrast, mediation could occur when childhood intelligence influences adult health outcomes via adult SES. This view is an extension of Gottfredson's (2002) idea of a "chain reaction", which was mentioned in Section 3.4: Higher intelligence in early life leads to higher educational attainment, and educational attainment in turn facilitates access to a higher adult socioeconomic status, which includes a higher income and a safer working environment (Deary et al., 2007; Deary et al., 2005; Johnson et al., 2010). Higher adult SES is then related to good health (Deary, 2010; Mackenbach et al., 2008). Moreover, educational

attainment is positively related to good health over and above its effects on adult SES (Cutler & Llears-Muney, 2006; Cutler et al., 2008). On the other hand, disadvantages in early life intelligence lead to disadvantages in educational attainment and consequently to adverse working environments with high burdens (e.g., an increased burden of stress or occupational hazards). These may in turn be linked to poorer health outcomes. Furthermore, the adverse health effects of higher occupational burdens may be especially strong when perceived control over these burdens is low. According to the demand-control model (Karasek, 1979), higher occupational burdens interact with low levels of perceived control and end up causing outcomes such as depression and exhaustion, which adversely effect health outcomes (Siegrist & Marmot, 2004). Several studies have shown that educational and socioeconomic outcomes are important mediators of the intelligence-health relation. Their pivotal role is highlighted by the fact that statistical adjustment for these variables substantially attenuates and sometimes nullifies the relation between early intelligence and later health (Batty et al., 2007a; Deary, 2010; Deary et al., 2010). However, other studies have shown that, despite intelligence having a substantial impact on later educational, socioeconomic, and health outcomes, mediation via education and further socioeconomic outcomes cannot fully explain the association between intelligence and health. For instance, Hart and colleagues showed that lower childhood intelligence significantly predicted a higher risk for mortality and some specific causes of death and morbidity (e.g., cardiovascular and coronary heart disease) in adulthood. Adjustment for indicators of adult SES accounted for some, but not all, of this higher risk (Hart et al., 2003). Batty and colleagues reported a negative association between higher intelligence in early adulthood and the metabolic syndrome in middle adulthood, and this association was barely affected when they adjusted for education, income, and social prestige (Batty, Gale, et al., 2008). Johnson and colleagues reported that childhood intelligence significantly predicted body mass index (BMI), constraints on activities of daily

living, anxiety, and alcohol consumption in adulthood, even when they adjusted for several demographic variables including social class and education. Moreover, the effects of education and social class on all health and health behavior outcomes in their study were smaller when childhood intelligence was controlled for (Johnson, Corley, Starr, & Deary, 2011).

In sum, educational attainment and subsequent SES are important mediators of the intelligence-health relation. However, this mediation should be interpreted cautiously. It seems plausible that the chain reaction operates in such a way that high childhood intelligence leads to educational success, placement into a profession with a high social status, and increased income, which all confer protection against disease. However, it is possible that the often impressive attenuation of the intelligence-health associations found after adjusting for education and/or other indicators of SES could occur because SES indicators may be partial "surrogates" for intelligence (Batty & Deary, 2005, p. 1766). Specifically, the usual indicators of SES, namely, education, occupation, and income can be ranked according to their correlations with measures of general intelligence *g*. The number of years spent in education was found to be correlated $r = .68$ with intelligence, and occupation and income were still correlated $r = .50$ and $r = .35$ with intelligence in several large representative samples of men (Gottfredson, 2004). Deary and colleagues even found a correlation of $r = .81$ between a latent variable capturing general intelligence and a latent variable capturing educational attainment (Deary et al., 2007). Thus, variation in SES indicators may, to a large extent, reflect variation in earlier intelligence, and their inclusion when investigating intelligence-health relations may lead to over-adjustment (Calvin et al., 2011). However, the assumption that intelligence can be used as a surrogate for SES has been challenged. For instance, it has been demonstrated that intelligence and education are distinguishable concepts with differential relations to other life outcomes (Baumert, Lüdtke, Trautwein, &

Brunner, 2009; Brunner, 2008; see also Section 2.1 of Chapter V). Causally informative studies are required to disentangle such possibilities (Deary et al., 2010).

Altogether, the results to date suggest that the extended biological and socioeconomic theories that can be traced back to Riegel and Riegel (1972) probably do not cover the entirety of mechanisms that could account for the intelligence-health relation. Thus, modern approaches that attempt to account for this relation also consider additional factors, such as health behavior.

4.2.3 Behavioral mechanisms

Approaches for explaining the intelligence-health relation via behavioral factors assume that individuals with low intelligence do not behave as healthily as individuals with higher intelligence. There is growing evidence that this is the case, although the studies linking early life intelligence and health behaviors are often based on populations of individuals who are still too young to have accrued many deaths. Therefore, intelligence–health behavior associations have been found, but it is largely not yet known whether these behaviors mediate the associations between intelligence and later chronic illness and death (Deary, 2010). Several studies have explored the association between intelligence and specific health behaviors. For instance, the association between intelligence and smoking has been explored in some studies, and all of them have found inverse associations, with lower intelligence increasing the risk of smoking (Batty, Shipley, Mortensen et al., 2008; Batty et al., 2007a; Hemmingsson, Kriebel, Melin, Allebeck, & Lundberg, 2008). Often, these associations were attenuated or disappeared after adjustment for indicators of SES (Batty, Shipley, Mortensen et al., 2008; Batty et al., 2007a; Hemmingsson et al., 2008). A few studies have explored the association of intelligence with mortality and controlled for smoking. Kuh and colleagues found no evidence of smoking as a mediator of the association between intelligence and mortality (Kuh, Richards, Hardy, Butterworth, & Wadsworth, 2004),

whereas Batty and colleagues found that adjustment for smoking marginally attenuated the intelligence–mortality association (Batty, Shipley, Mortensen, et al., 2008). The conclusion is that there seems to be an inverse association between intelligence and smoking, but it remains unclear whether this can be explained by socioeconomic or other factors, and if so, to what extent. The evidence for smoking as an important mediator of the association between intelligence and mortality has been inconsistent.

Some studies have examined the association between intelligence and alcohol abuse. One study found that higher childhood intelligence predicted a lower risk for alcohol-induced hangovers in adulthood (Batty et al., 2006). However, two further studies found that higher childhood intelligence predicted a higher risk for some types of alcohol problems and higher alcohol intake in adulthood. According to the authors, further research is needed to examine possible psychosocial mechanisms that may be associated with both higher childhood intelligence and a greater risk for alcohol abuse (Batty, Deary, et al., 2008; Hatch et al., 2007). Other studies have demonstrated that higher intelligence predicts more physical activity and a healthier diet, for instance more fruit, vegetable, and whole wheat bread consumption (Batty et al., 2007b; Deary, 2010; Singh-Manoux et al., 2009) as well as complying in the long run with prescribed medications (Deary et al., 2009). Finally, lower intelligence is associated with a lower risk for intentional injury (e.g., attempted and completed suicide; Gunnell, Magnusson, & Rasmussen, 2005).

The mechanisms by which intelligence influences health behavior may be diverse and warrant further research. In some cases, lower intelligence may be a risk factor for unhealthy behavior because it goes along with a reduced capacity to understand and follow health information, such as medical prescriptions (e.g., persisting with medication; Deary et al., 2009). With respect to self-harming behavior (e.g., intentional injury), low intelligence may be a risk factor because low intelligence increases the risk for developing mental disorders.

Additionally, low intelligence may reduce an individual's capacity to solve problems while experiencing acute life crisis or suffering from a mental illness (Gunnell et al., 2005). In other cases, such as following a healthy diet, low intelligence may be a risk factor because the skills that are measured by intelligence tests, such as verbal comprehension and reasoning, may be important in the successful management of these behaviors (Batty et al., 2007b). In a more general sense, intelligence may predict health and health behavior because it is an indispensable global resource that is required to manage one's own health, remain healthy, prevent illness, and treat disease. This has led to the assumption that intelligence, rather than socioeconomic resources, may be the driving force behind socioeconomic health inequalities. The controversy between advocates of intelligence versus socioeconomic resources as driving forces behind socioeconomic health inequalities is captured by the *fundamental cause controversy*.

4.3 The fundamental cause controversy – a role for intelligence?

As mentioned in Section 2.3, systematic health inequalities between individuals with a higher and lower SES continue to persist and even widen. Some theories, such as the theory of fundamental social causes, attribute these inequalities to a set of flexible resources associated with a higher SES, for instance, knowledge, money, power, prestige, and beneficial social connections. If childhood intelligence affected adult health mainly or even merely through its influence on adult socioeconomic outcomes such as educational attainment, a higher income, or a safer working environment, one could argue that intelligence is not by itself the key resource. Rather, the flexible resources associated with adult SES would be more important for explaining socioeconomic health differences (Link et al., 2008). However, it has been suggested that intelligence itself may be "the 'fundamental cause' of social class inequalities in health" (Gottfredson, 2004, p. 174). In this view, health differences between different socioeconomic groups are actually the result of differences in

intellectual abilities between these groups. This provocative hypothesis is based on several important observations. First, intelligence is a content- and context-free ability. Thus, it is useful in different circumstances, times, and places, and therefore could be one explanatory factor for the highly generalizable socioeconomic health inequalities that occur (Gottfredson, 2004). Second, as summarized in the previous sections, higher childhood intelligence significantly predicts lower risks for adult mortality and morbidity as well as a higher incidence of health literacy, health knowledge, and healthy behavior. Third, intelligence is highly stable across the life course. Specifically, the rank order of individuals with respect to their intelligence scores does not change to a large extent—those who score low on intelligence tests in childhood tend to score low on intelligence scores in adulthood and vice-versa. For instance, Deary and colleagues reported that a measure of general intelligence *g* at age 11 was correlated $r = .63$ with a measure of *g* at age 77 (Deary, Whalley, Lemmon, Crawford, & Starr, 2000). Likewise, in a study based on the same longitudinal project as the present Ph.D. thesis, Schalke and colleagues showed that general intelligence *g* at age 12 was correlated $r = .85$ with *g* at age 52 years (Schalke et al., 2012). Thus, childhood intelligence predicts not only adult SES and adult health but adult intelligence as well. Therefore, childhood intelligence could influence adult health through its influence on intellectual ability in adulthood. Fourth, intelligence test scores are socially patterned, as children and adults from socially deprived backgrounds typically have worse results. Thus, low SES groups are more homogenously composed of individuals with low intelligence (Batty, Kivimaki, & Deary, 2010; Gottfredson, 2004; Mackenbach, 2012; Neisser et al., 1996). Fifth, and most crucially, health nowadays depends more than ever on private precaution and health lifestyle. For instance, seven of the 10 leading causes of death in the U.S. have aspects that can be modified by "doing the right thing" and by "making healthy choices" about one's own behavior (Gottfredson, 2004, p. 181). More generally speaking, we are our own primary

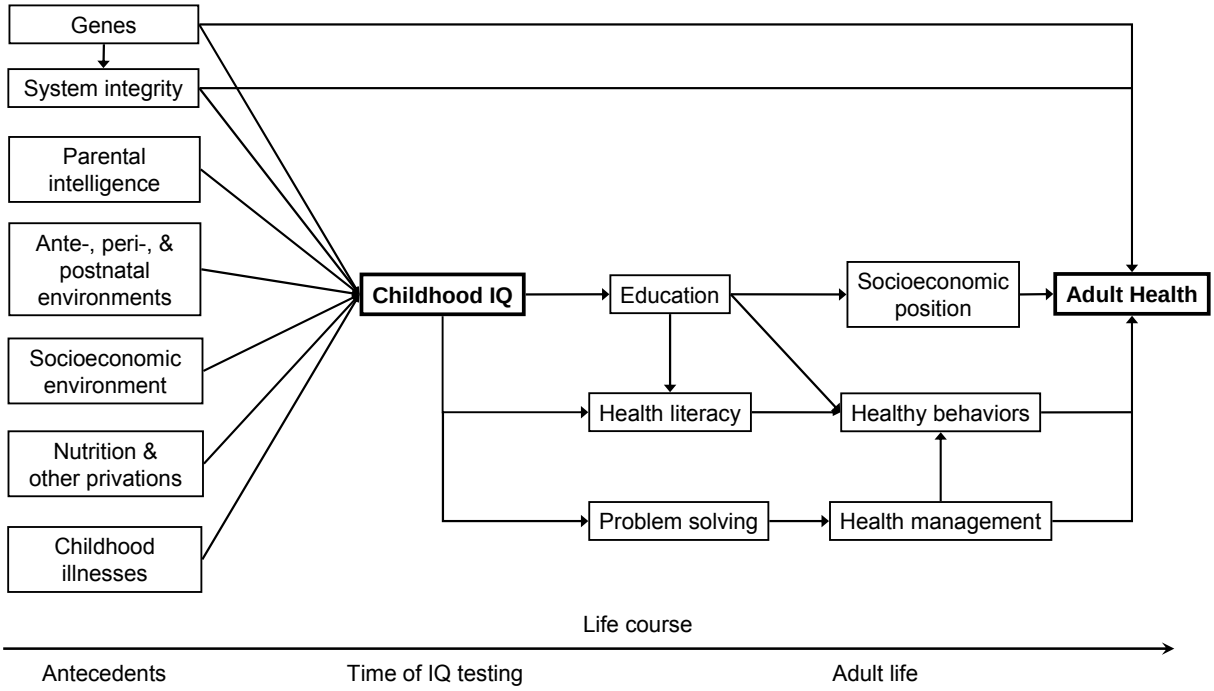
providers of health care. Thus, we have the "job" of being a patient, which includes managing our own health. However, health self-management demands that we deal with the novel, the ever-changing, and the complex (Gottfredson, 2004; Gottfredson & Deary, 2004). Preventive information proliferates, and new treatments often require regular self-monitoring and complicated self-medication. Good health depends as much on preventing as on ameliorating illness, injury, and disability, all of which are inherently cognitive tasks (Gottfredson, 2004). As just one instance, chronic conditions such as diabetes require close daily monitoring and adjustments in self-treatment. Moreover, such conditions keep changing so that the job of "disease management" cannot be routinized. Therefore, constant judgment in applying old knowledge and the need to spot and solve novel problems is required. Importantly, these are exactly the skills that are at the heart of general intelligence g. Recall that intelligence is manifested in generic thinking skills such as efficient learning, reasoning, problem solving, and abstract thinking. High intelligence is a useful tool in any life domain, but especially when tasks are novel, untutored, or complex and situations are ambiguous, changing, or unpredictable (Gottfredson, 1997). Crucially, maintaining health, protecting oneself against chronic disease and accidents, and adhering to complex treatment regimens can be construed as one of life's jobs, and success in this job might be associated with cognitive competences as measured by psychometric intelligence tests (Gottfredson & Deary, 2004).

In sum, the approach that conceptualizes intelligence as a fundamental cause assumes that childhood intelligence may predict adult health outcomes because childhood intelligence is an important predictor of adult intelligence. Intelligence encompasses cognitive skills that are crucial for managing one's own health and disease. As intelligence is a content- and context-free ability, it could explain the pervasive socioeconomic health inequalities better than traditional sociological theories such as the theory of fundamental social causes. Several findings have provided evidence for this hypothesis. First, the SES measures that best predict

health inequalities are also most highly correlated with intelligence (first education, then occupation, then income). Thus, instead of intelligence as a surrogate for SES in health matters, SES measures might operate primarily as rough surrogates for social-class differences in cognitive rather than material resources (Gottfredson & Deary, 2004). Moreover, some studies have shown that intelligence still predicted health outcomes even after adjusting for SES measures (Batty, Gale, et al., 2008; Hart et al., 2003). Second, there are results that have shown that next to SES measures, other important predictors of health, such as health knowledge, may be surrogates for intelligence. Recall that knowledge was one of the flexible resources that the theory of fundamental social causes held responsible for socioeconomic health inequalities. One study investigated the associations between intelligence and 10 different areas of widely available health knowledge (e.g., concerning reproduction, aging, nutrition, or safety) in two samples of college students and adults (Beier & Ackerman, 2003). The authors found that all areas of health knowledge formed one dominant factor, which in turn was correlated about $r = .90$ with a general intelligence *g*-factor that they derived from seven intelligence tests. Neither personality nor self-reported level of health knowledge had much relation to actual level of knowledge, and an education-income composite added nothing to the prediction of knowledge after *g* was controlled for. However, there are other studies that have challenged the idea of intelligence as a fundamental cause of socioeconomic health inequalities. For instance, Lager and colleagues investigated in a Swedish cohort whether mortality differences between individuals with higher and lower educational attainment could be explained by early intelligence and found that this was not the case (Lager, Bremberg, & Vagerö, 2009). Batty and colleagues investigated whether intelligence explained socioeconomic differences in total and CVD mortality in two different cohorts. The authors found that intelligence substantially attenuated the effects of adult SES on these outcomes and offered greater explanatory power than

traditional CVD risk factors (e.g., blood pressure and cholesterol). However, intelligence did not fully explain the socioeconomic CVD differences (Batty, Der, Macintyre, & Deary, 2006; Batty et al., 2009). Altogether, the results to date suggest that one factor alone is probably not sufficient to explain socioeconomic health inequalities (Batty, Kivimäki, et al., 2010). There may be a multitude of factors that lead to these inequalities, and intelligence may be an important one. However, further research on these factors and their relative importance for socioeconomic health inequalities is required. Figure I-4 provides an overview of potential mechanisms that link childhood intelligence to adult health outcomes.

Figure I-4. *Potential mechanisms linking childhood intelligence and adult health outcomes*



Note. This simplified figure is an extension of a presentation of several mechanisms linking intelligence and health and mortality in Batty et al. (2007). Key: IQ = intelligence.

In a nutshell, relations between early intelligence and health outcomes in adulthood are robust findings in the literature. Different non-exclusive mechanisms have been proposed to account for these relations. These mechanisms involve physiological (and maybe even

genetic), socioeconomic, behavioral, and personal factors. Evidence suggests that intelligence may be an important factor in explaining socioeconomic health inequalities. However, several important questions remain open with respect to the relation between intelligence and health. These questions set the stage for the three studies that constitute the present Ph.D. project and will be presented in the next subchapter.

5. The present Ph.D. thesis

5.1 Open research questions and aims of the present Ph.D. thesis

The present Ph.D. thesis investigated the relationship between intelligence and health in Luxembourg. It thereby addressed five important open questions regarding the relation between intelligence and health.

(1) Virtually all previous studies on this relation have been conducted in English-speaking or Scandinavian countries (Calvin et al., 2011; Deary, 2010). This is important for several reasons. First, from a general perspective, the universality of psychological processes can never be assumed in advance (Segall, Lonner, & Berry, 1998). This statement highlights the importance of extending findings gathered in one cultural setting to different settings to establish that they are indeed universal. Second, more specifically, one must ask whether the findings from previous studies can be generalized to countries with different levels of social mobility, different health care systems, or different levels of health care expenditure. Luxembourg has a level of social mobility below the OECD average (Organisation for Economic Co-operation and Development, 2010), indicating that—contrary to many modern societies (Mackenbach, 2010)—an individual's social and economic achievement depends largely on the socioeconomic position of the individual's family of origin. Previous research using the same longitudinal database as the present Ph.D. project (see Section 5.5) has also shown that Luxembourg is not or only to a very small extent a meritocratic society, as one's

socioeconomic family background seems at least as important in determining later educational and socioeconomic success as personal factors (Brunner & Martin, 2011). Therefore, the impact of intelligence as a personal factor on health could be smaller or even negligible in Luxembourg compared to more meritocratic societies, in which other crucial life outcomes such as socioeconomic success depend on personal factors to a greater degree. By contrast, the impact of childhood SES could be stronger in Luxembourg than in more meritocratic socially mobile societies. Further, Luxembourg provides universal access to quality health care. Specifically, the Luxembourgish health care system is ranked 16th among 190 worldwide health care systems (World Health Organization, 2000). The state covers the main part of health expenditures, and private "out-of-pocket" payments are low (Huber, 1999). Universal access to quality health care may compensate for some of the effects of individual differences in intelligence on health, and as a result, intelligence may lose its impact. Thus, both the low level of meritocracy, as well as the universal access to health care, could reduce or even offset the effects of intelligence on health in Luxembourg. On the other hand, if intelligence really is one of the fundamental causes of socioeconomic health inequalities, as advocates of this theory assume (see Section 4.3), its influence over and above childhood SES should be detectable even in Luxembourg with its universal health care system. In sum, a major goal of the present Ph.D. thesis was to examine whether the effects of intelligence on health found in other countries could be generalized to Luxembourg. Thus, this was the first project to investigate the intelligence-health relation in a Central European country.

(2) As introduced in Subchapter 2, physical health is a multidimensional concept with three distinct subdimensions (Liang, 1986; Liang et al., 1991; Whitelaw & Liang, 1991): a physical subdimension (e.g., presence of diagnosed diseases, number of doctor visits in a certain time period), a subjective subdimension (e.g., satisfaction with one's own health), and

a functional subdimension (e.g., unimpaired participation in social activities or performing household tasks). Most previous studies on the relation between intelligence and health outcomes have focused on the physical health subdimension (*Intelligence*, 2009). Hence, considerably less is known about the effects of intelligence on the functional and subjective health subdimensions. This issue is of particular importance as childhood intelligence may be differentially related to different aspects of adult health (Johnson et al., 2011). The present Ph.D. project therefore investigated intelligence in relation to all three subdimensions of physical health.

(3) As introduced in Subchapter 3, intelligence is a multifaceted hierarchically structured construct (McGrew, 2009): General intelligence *g* is located at the apex of the hierarchy; more specific facets, such as fluid intelligence (as measured by tasks that require reasoning processes or the solution of novel problems) and crystallized intelligence (as measured by tasks with high demands on verbal knowledge) are located at the next lower level of the hierarchy. Most previous studies in cognitive epidemiology have focused on general intelligence (Calvin et al., 2011); few have assessed fluid and crystallized intelligence separately. To my knowledge, no previous studies have investigated the potentially differential effects of general, fluid, and crystallized intelligence measured in childhood on adult health. However, this approach would provide insights into which aspects of intelligence are important in personal health management and could be targeted by interventions. Specifically, if crystallized intelligence were the more important predictor, then education in general, and interventions to foster health literacy in particular, could remediate deficits. If general or even fluid intelligence were more important, remedial interventions would be more complicated to implement, as these would have to target general reasoning, abstract thinking, and problem solving skills. The present Ph.D. project therefore investigated the predictive capacity of general intelligence and different facets of intelligence in childhood

on later health outcomes.

(4) Despite considerable evidence that has emphasized the relevance of education and socioeconomic status as potential mediators between childhood intelligence and later health outcomes, previous research has yielded inconsistent results regarding the extent of mediation in these relations. Some studies have reported pronounced mediation, yet others have reported little or no mediation (see Section 4.2.2). These inconsistent findings have been observed in studies that have investigated the effect of childhood intelligence on adult mortality risk, as well as in studies that have investigated the effect of childhood intelligence on other health outcomes, such as the metabolic syndrome, physical activity, or a healthy diet (Batty & Deary, 2005; Batty, Gale, et al., 2008; Batty et al., 2007b; Calvin et al., 2011; Deary et al., 2010). Establishing the amount of mediation via socioeconomic outcomes is crucial for solving the fundamental cause controversy. Specifically, such information helps to determine whether intelligence influences health mainly via a "chain reaction" ranging from childhood intelligence to education and socioeconomic success to health, and/or whether intelligence influences health because it encompasses generic thinking skills that are key in personal health management. Another aspect that warrants further research is the finding that the extent of mediation may depend on the time in life when intelligence is measured. For instance, one study that found complete mediation of the intelligence-mortality relation measured intelligence in early adulthood after participants had already completed their educations. Thus, the intelligence-mortality relation could have been confounded by education (Calvin et al., 2011). Measuring intelligence before participants have completed their educations may therefore be important for detecting unique effects of intelligence and education. The present Ph.D. project therefore investigated whether and the extent to which mediation via education and socioeconomic status occurs in the relation between childhood intelligence and adult health in Luxembourg.

(5) As is the case for studies investigating the relation between intelligence and health outcomes, virtually all studies on the relation between intelligence and mortality risk have been conducted in English-speaking or Scandinavian countries (Calvin et al., 2011; Deary, 2010). Thus, it remains unclear whether the results that have shown that intelligence predicts mortality risk can be generalized to Luxembourg. Further, there is a controversy in the literature about whether the effects of intelligence on mortality exist across the entire range of intelligence scores or whether individuals at the lower end of the intelligence distribution constitute a risk group with a particularly high mortality risk. Whereas some studies suggest an effect across the entire intelligence spectrum, including the especially gifted (Batty, Kivimäki, et al., 2010; Lager et al., 2009, Martin, & Kubzansky, 2005), other studies suggest that individuals at the lower end of the intelligence distribution exhibit a particularly increased mortality risk (Hart et al., 2003, 2005; Kuh et al., 2004). This controversy can also be expressed as a debate about whether there is an *incremental* or a *threshold* effect of intelligence on later mortality risk (Batty, Deary, & Gottfredson, 2007). The present Ph.D. project therefore investigated whether childhood intelligence would predict adult mortality risk in Luxembourg, and if so, whether the effects would operate in an incremental or threshold manner.

To answer the above-mentioned five research questions, we conducted three distinctive studies for the present Ph.D. project. Each of these studies drew on the same longitudinal database, comprising two waves of measurement across a 40-year time span in Luxembourg. The following sections will introduce each of the three studies and which of the five research questions it addressed. The last section of this chapter will then provide a description of the common longitudinal database of all three studies.

5.2 Study I – Forty years on: Childhood intelligence predicts health in middle adulthood

Study I (published in *Health Psychology*) is entitled "*Forty Years On: Childhood*

Intelligence Predicts Health in Middle Adulthood". Its major objectives were to address open research questions numbers. (1) to (3), as presented in Section 5.1.

Study I investigated (1) whether childhood intelligence would predict health across a 40-year time span in Luxembourg even when controlling for childhood socioeconomic circumstances. This was motivated by the fact that almost all previous studies on the relation between intelligence and health have been conducted in English-speaking or Scandinavian countries (Calvin et al., 2011). Thus, it remained unclear whether the effects of intelligence on health could be generalized to Luxembourg with its low level of social mobility but universal quality health care. If Study I could show that intelligence retained its predictive power for adult health even in Luxembourg and even when controlling for childhood SES, this would considerably support the notion of substantial and incremental effects of intelligence on health and provide further evidence that intelligence is one factor that could help socioeconomic health inequalities.

Further, Study I investigated (2) whether childhood intelligence would predict all subdimensions of physical health in adulthood equally well. Physical health comprises a physical subdimension, a subjective subdimension, and a functional subdimension (Liang, 1986; Liang et al., 1991; Whitelaw & Liang, 1991). Most previous studies on the relation between intelligence and health outcomes have focused on the physical subdimension (*Intelligence*, 2009). Hence, considerably less is known about the effects of intelligence on the functional and subjective subdimensions.

Finally, Study I investigated (3) whether different facets of childhood intelligence would predict health in adulthood equally well. Specifically, Study I investigated whether childhood general, fluid, and crystallized intelligence all predicted adult health equally. Most studies on the topic to date have used composite measures of childhood general intelligence *g* to predict later health outcomes (Calvin et al., 2011). However, knowing whether specific

facets are stronger predictors of health outcomes than others would provide insights into which facets should be targeted by interventions.

5.3 Study II – Childhood intelligence and adult health: The mediating roles of education and socioeconomic status

Study II (published in *Intelligence*) is entitled "*Childhood Intelligence and Adult Health: The Mediating Roles of Education and Socioeconomic Status*". Its major objective was to address research question number (4).

Specifically, Study II investigated how the impact of childhood intelligence on a variety of health outcomes measured in middle adulthood is mediated via later educational attainment and subsequent socioeconomic status. This investigation was motivated by the fact that previous research has yielded inconsistent results regarding the extent of mediation in the relations between childhood intelligence and later health. Moreover, the extent of mediation may depend on the time in life when intelligence is measured because when intelligence is measured after the completion of education, any effects of such a measure of intelligence on health may be confounded by education.

Importantly, the study of mediational mechanisms that underlie the intelligence-health relation has been identified as a "key priority" in cognitive epidemiology (Deary et al., 2010, p. 71). A better understanding of these meditational mechanisms is crucial for applying findings on the intelligence-health relation to public health (Deary, 2012). Further, it has been highlighted that studies investigating education and socioeconomic outcomes as mediators should ideally include intelligence measured early in life before differential effects of education have set in. Those studies should employ statistical techniques (such as structural equation modeling; SEM) that include several measures of each construct to alleviate the problem of measurement error (Deary & Johnson, 2010; Deary et al., 2010). Study II fulfilled these methodological requirements: It rigorously investigated the mediation via educational

attainment and subsequent socioeconomic status (SES) by employing SEM techniques. Moreover, it included intelligence measures that were obtained before the completion of primary education in Luxembourg.

5.4 Study III – Childhood intelligence predicts premature mortality: Results from a 40-year population-based longitudinal study

Study III is entitled "*Childhood Intelligence Predicts Premature Mortality: Results from a 40-Year Population-Based Longitudinal Study*". Its major objective was to address the last of the open research questions presented in Section 5.1, question number (5).

Specifically, Study II investigated whether childhood intelligence would predict adult mortality until the age of 52 in Luxembourg. As was the case with Study I, which investigated intelligence in relation to health in Luxembourg, if Study III could show that intelligence retained its predictive power for mortality even in Luxembourg and even when controlling for childhood SES, this would considerably support the notion of substantial and incremental effects of intelligence on a wide range of adult health outcomes.

Further, Study III was aimed at contributing to the controversy about whether the effect of intelligence on mortality exists across the entire range of intelligence scores or whether individuals at the lower end of the intelligence distribution constitute a risk group with a particularly high mortality risk. Further studies regarding the shape of the IQ-mortality relation are required (Batty et al., 2007), especially as results of these studies have potential policy implications for health care and preventive measures that are aimed at reducing premature mortality rates. If intelligence effects on mortality risk exist across the entire intelligence distribution, then health care interventions and preventive measures can be applied to anyone in a sort of an "indiscriminate all-round distribution". However, if there is a specific group at the lower end of the intelligence distribution that is at risk for premature mortality, this group of individuals should be the primary target of health care interventions,

preventive measures, and interventions to foster the intellectual abilities that are relevant to preventing ill health and premature mortality.

The last section of this chapter will now provide a description of the common longitudinal database of Studies I to III.

5.5 Participants and procedure – the MAGRIP project

All three studies of this Ph.D. thesis were conducted within the framework of the longitudinal MAGRIP project. The MAGRIP project was a longitudinal study initiated in 1968 by the Institut Pédagogique in Walferdange, Luxembourg (Bamberg, Dickes, & Schaber, 1977). It was originally designed to investigate the determinants of children's school careers in Luxembourg. In the 1960s, primary education in Luxembourg lasted 6 years, and secondary education lasted 7 years at the longest. The average number of years of schooling was 10 years. Eighty-three percent of the total student population completed primary education, and 47% enrolled in secondary education at that time (UNESCO Institute for Statistics, 2013).

MAGRIP is an acronym for the French expression "matière grise perdue" (i.e., lost grey matter). This expression reflects one main result of the first wave of the MAGRIP project, indicating that a child's academic achievements were linked directly to his or her social background rather than to his or her intelligence. Thus, children from socioeconomically disadvantaged families found themselves unable to tap their full cognitive potential under the school system of the time—in a way, their grey matter was lost. In 2008, a second wave of measurement of the MAGRIP project was initiated.

In the first wave of measurement in 1968, trained test administrators collected detailed information on a randomly selected nationally representative sample comprised of 2,824 students at the end of their primary education (M = 11.9 years; SD = 0.6 years; 50.1% male). Eighty-four percent of the students were in Grade 6 of primary school, 11% in Grade 5, and

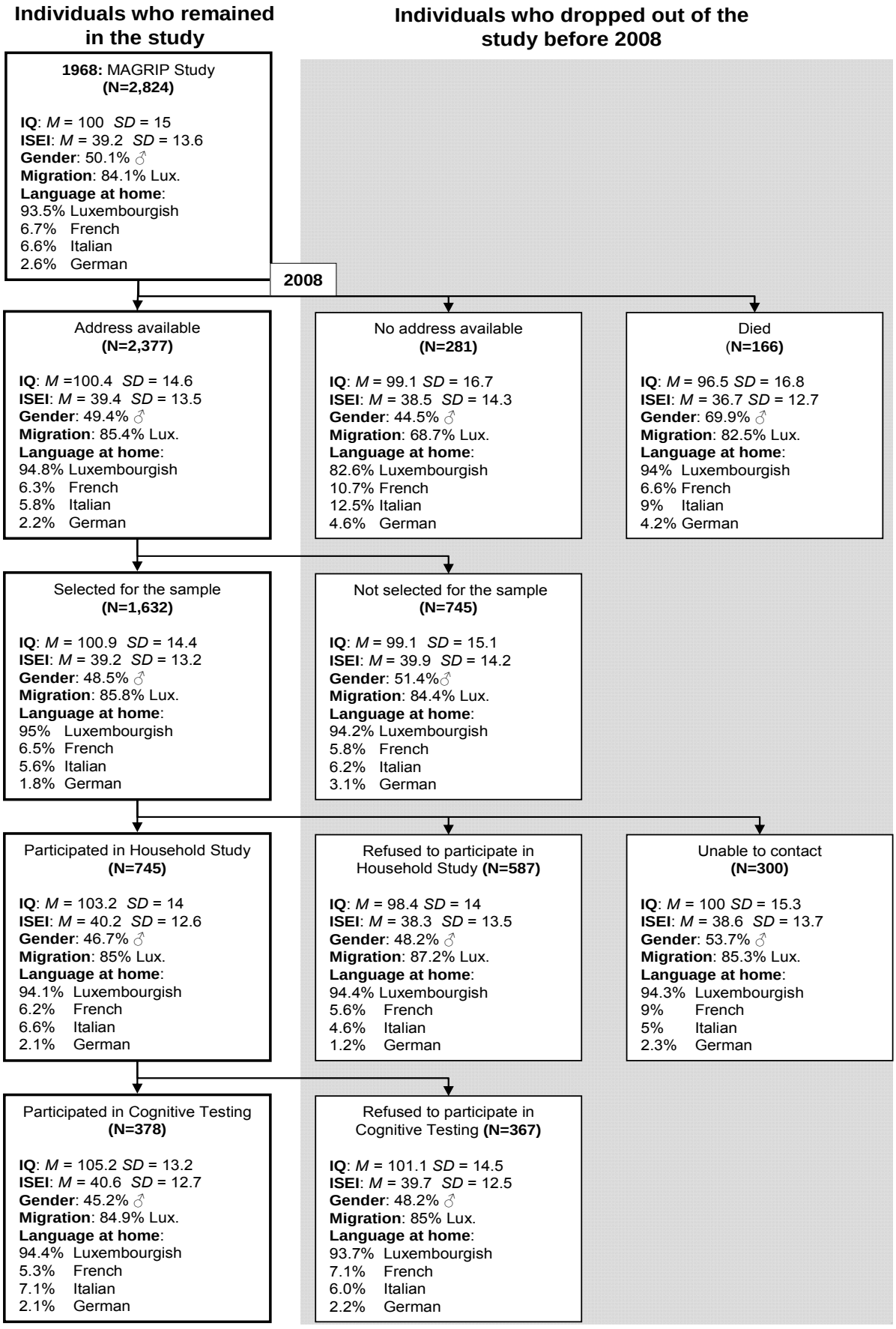
5% in Grade 4. Every Luxembourgish school participated in the data collection, and within each school, about half of the children who were 12 years old and/or attending the sixth grade were randomly selected for study participation. The students provided (among other kinds of information) data on their intellectual abilities and their socioeconomic family background.

The second wave of measurement was initiated in 2008. To collect data for this second wave, the current addresses of the original participants were identified using the database of the social security agency of Luxembourg (permission was granted by the Luxemburgish data protection committee "Commission Nationale Pour la Protection des Données"). Figure I-5 displays the multistage sampling procedure for the second wave of the MAGRIP project, including reasons why individuals remained in the MAGRIP project or why they dropped out, the final number of study participants in the second wave, and information on the representativeness of the samples. The current addresses of 2,377 (84%) surviving participants in the first wave could be identified by their social security ID numbers. A total of 166 (6%) participants had died by 2008. This group is the focus of Study III of the present Ph.D. project. Figure I-5 shows that relative to the total 1968 sample, the deceased individuals had lower mean childhood intelligence scores (Cohen's $d = 0.22$) and lower socioeconomic family background scores (as indexed by the acronym "ISEI"; Cohen's $d = 0.19$) in 1968. Furthermore, nearly 70% of the deceased were men. The remaining 281 (10%) former participants in the first wave of MAGRIP could not be found through their social security ID numbers and had probably left the country. A representative stratified random sample of 1,632 former participants was contacted and invited to participate in the second wave. Stratification criteria consisted of region of residence within Luxembourg in 1968 and gender. Out of these 1,632 individuals, 300 could not be contacted, and 587 refused to participate. The remaining 745 persons took part in the second wave of measurement. No financial incentive was offered.

The second wave of measurement was comprised of two phases: (a) a household study (lasting from November 2008 to February 2009) and (b) a cognitive testing session (March 2009 to August 2009). For the household study, trained interviewers visited the participants at home and conducted a structured interview about the participants' educational and occupational career paths as well as key socioeconomic variables (e.g., socioeconomic and marital status). After the interview, participants completed a comprehensive questionnaire to assess their health. Altogether, 745 participants took part in the household study (M = 51.7 years, SD = 0.6 years; 46.7% male). This is the sample that Studies I and II of the present Ph.D. project are based on. Figure I-5 shows that this sample was fairly representative of the original sample. Relative to the total 1968 sample, follow-up participants had slightly higher mean childhood intelligence (Cohen's d = 0.20) and SES (as indexed by the acronym "ISEI"; Cohen's d = 0.08). Furthermore, somewhat more women than men took part in the second wave, whereas the number of boys and girls was nearly balanced in 1968. About half of the 745 participants (N = 378) also took part in the cognitive testing. Here, participants completed the same intelligence test as in 1968 and provided additional information on their socioeconomic status. The percentage of native Luxembourgers as well as the percentage of persons who spoke Luxembourgish at home was approximately the same in the original 1968 sample, the household study sample, and the sample who participated in the cognitive testing session. In sum, the differences between the 1968 sample and the two 2008/2009 samples were relatively small. Hence, the 2008/2009 samples can be considered to be fairly representative of the student population of 1968 (as represented by the original representative 1968 MAGRIP sample).

Chapters II to IV will now present the three studies of the present Ph.D. project in greater detail.

Figure I-5: *Flow chart of the multistage sampling procedure of the MAGRIP project*[1]



[1]This flow chart draws on the flow chart presented in the final report on the MAGRIP project to the Luxembourg Fonds National de la Recherche (FNR), who funded both the overall MAGRIP project and the present Ph.D. thesis.

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Chapter II

Study I:

Forty Years On: Childhood Intelligence Predicts Health in Middle Adulthood

Abstract

Objective: To investigate whether childhood general intelligence, fluid intelligence (Gf), and crystallized intelligence (Gc) predict various health outcomes in middle adulthood. ***Method:*** This prospective longitudinal study followed a nationally representative sample of 717 Luxembourgers. Intelligence and socioeconomic status (SES) were measured at age 12; physical, functional, and subjective health were assessed at age 52. ***Results:*** Childhood general intelligence and fluid intelligence showed substantial positive effects on adult health outcomes, whereas the corresponding effects of crystallized intelligence were considerably smaller. ***Conclusion:*** Childhood intelligence incrementally predicts various dimensions of adult health across 40 years—even in a country in which all citizens are guaranteed access to high-quality health care.

Keywords: childhood intelligence, fluid intelligence, crystallized intelligence, adult health

Forty Years On: Childhood Intelligence Predicts Health in Middle Adulthood

1. Introduction

The identification of childhood characteristics that influence adult health is crucial for the development of effective health care policy and preventive measures with the potential to improve long-term individual health and well-being. Childhood intelligence has been identified as an important factor influencing adult health (Johnson, Corley, Starr, & Deary, 2011). Several nonexclusive causal processes have been proposed as possible explanations for this link. For instance, socioeconomic status, as reflected in education and occupation, is known to predict health and may mediate the relationship between childhood intelligence and adult health (Der, Batty, & Deary, 2009). Alternatively, health literacy, which is an important predictor of health knowledge and behavior, may serve as a surrogate for intelligence (Gottfredson, 2004).

Although theoretically plausible, it is only within the last decade that the newly emerging research field of *cognitive epidemiology* has begun to systematically and empirically test the core hypothesis that childhood intelligence predicts adult health (Der et al., 2009). In many studies, adult health has been operationalized by various indicators of physical health. Intelligent children have consistently been shown to live longer (Calvin et al., 2011). Further, intelligence has been found to affect various diseases, health-promoting behaviors, lifestyles, and risk factors (for an overview, see the special issue of *Intelligence*, 2009, on cognitive epidemiology).

2. The present study

In testing whether childhood intelligence positively predicts health across a 40-year period in Luxembourg, the present study makes several important contributions to this area of research: (1) Almost all previous studies in cognitive epidemiology have been conducted in

English-speaking or Scandinavian countries (Calvin et al., 2011). Can these findings be generalized to countries with different health care systems? Universal access to quality health care may compensate for the differential effect of intelligence on health, and as a result, intelligence may lose its impact. Thus, a major goal of this study was to examine whether the key findings of cognitive epidemiology generalize to Luxembourg—a country providing quality universal health care (World Health Organization, 2000). (2) Health is a multidimensional concept with three distinct dimensions (Liang, 1986): (a) a *physical* dimension (e.g., number of doctor visits), (b) a *functional* dimension (e.g., social activities), and (c) a *subjective* dimension (e.g., satisfaction with one's own health). Many previous studies in cognitive epidemiology have focused on the physical health dimension (*Intelligence*, 2009). However, considerably less is known about the effects of intelligence on the functional and subjective health dimensions. (3) It is widely acknowledged that intelligence is a multifaceted hierarchically structured construct (McGrew, 2009): General intelligence is located at the apex of the hierarchy; more specific facets, such as fluid intelligence (*Gf*, as measured by tasks that require reasoning processes or the solution of novel problems) and crystallized intelligence (*Gc*, as measured by tasks with high demands on verbal knowledge) are located at the next lower level of the hierarchy. Many previous studies in cognitive epidemiology have focused on general intelligence (Calvin et al., 2011); few have assessed Gc and Gf separately. In one study, Gf measured in adulthood was significantly related to mortality, whereas Gc was not (Batterham, Christensen, & Mackinnon, 2009). To our knowledge, no previous studies have investigated the potentially differential effects of general intelligence, Gf, and Gc measured in childhood on adult health. However, this approach would provide insights into which facets of intelligence (general intelligence, Gf, or Gc) are important in personal health management and could be targeted by interventions. (4) When studying the effects of childhood intelligence on health, it is crucial

to adjust for other early life circumstances, such as childhood socioeconomic status (SES; Johnson et al., 2011). In line with the most rigorous studies in cognitive epidemiology, we therefore investigated whether childhood intelligence predicts better adult health, with and without adjusting for the effects of childhood SES.

3. Method

3.1 Design

This study used a prospective epidemiological cohort design spanning 40 years. Childhood intelligence and SES were measured in a nationally representative sample of Luxembourg adolescents in 1968, and a random subsample reported adult health in 2008/2009.

3.2 Participants and procedure

In 1968, a nationally representative sample of N = 2,824 Luxembourg students (M = 11.9 years, SD = 0.6 years; 50.1% male) participated in a school-based data collection. Every school in Luxembourg participated, with data collected from at least one class. The study included a 50% random sample of all Luxembourg Grade 6 classes in 1968. Birth years ranged from 1953 to 1958, with the majority of participants (76%) born in 1957.

A random sample of 717 persons (M = 51.7 years, SD = 0.6 years; 46.3% male), stratified by region of residence in 1968 and gender, was selected to participate in the second wave in 2008/2009. Trained interviewers visited the participants at home and administered a health questionnaire. Analyses for selection bias showed that the follow-up sample was fairly representative of the original sample. Relative to the total 1968 sample, follow-up participants had only slightly higher mean childhood intelligence (Cohen's d = 0.20) and SES (d = 0.08).

3.3 Measures

Full descriptions of all measures and their psychometric properties are provided in the online supplemental materials.

Childhood intelligence. In 1968, participants were administered the Leistungsprüfsystem (L-P-S, [Performance Test System], Horn, 1962), a standardized, well-validated German intelligence test. Its 14 subtests provide a measure of general intelligence (total IQ score), as well as scores for more specific intellectual facets, such as fluid and crystallized intelligence. A correlation of .94 has been reported (Sturm & Büssing, 1982) between the L-P-S total score and the total score on the German version of the Wechsler Adult Intelligence Scale (WAIS). A total score for general intelligence *g* (z-standardized with $M = 0$ and $SD = 1$ for the full 1968 sample) was computed by summing the scores across all subtests ($\alpha = .86$). A z-standardized sum score for Gf was computed from two subtests measuring reasoning and logical thinking ($\alpha = .75$), and a z-standardized sum score for Gc was computed from three subtests with high demands on verbal knowledge ($\alpha = .73$).

Childhood SES. Children indicated their parents' occupations. The occupations were coded using the International Standard Classification of Education and Income (ISEI; Ganzeboom & Treiman, 1996). Higher ISEI values indicate a higher level of childhood SES. For the present analyses, the ISEI values were z-standardized ($M = 0$, $SD = 1$).

Adult health. Adult health was operationalized by well-validated and reliable measures that were adapted from previous large-scale surveys (ALLBUS; Terwey, 2000) and health research (Hultsch, Hertzog, Small, & Dixon, 1999). *Physical health* was assessed by three items measuring the number of doctor visits in the last 3 months, the number of sick leave days in the last 12 months, and the number of nights spent in the hospital in the last 12 months. Because of their differing response scales and severity, these three items were analyzed separately. *Functional health* was assessed by the following question: "Looking

back over the past two years, how much did your health status hinder the following activities?" Nine areas of activity were listed (e.g., work, household, mobility, social, intellectual, sports, and leisure activities). Responses were given on a 5-point rating scale. We computed the total sum score ($\alpha = .93$) across all items in terms of the Percent of Maximum Possible Score (POMP; Cohen, Cohen, Aiken, & West, 1999): 100% indicates the maximum level of functional health; 0% indicates the maximum possible impairment. *Subjective health* was assessed by a 5-point scale measuring participants' ratings of their overall health status and of their health status relative to peers and by a 7-point scale measuring their satisfaction with their health status. These items have been shown to measure distinct aspects of subjective health and have been used in influential studies (Liang, 1981) and large-scale surveys (ALLBUS; Terwey, 2000). We computed a total POMP score (that took the different response scales into account; see Cohen et al., 1999, p. 329) for these three items ($\alpha = .85$). A value of 100% indicates the highest possible level of subjective health.

3.4 Data analysis

Ordinal logistic regressions were used to predict each of the three indicators of physical health, and linear regressions were used to predict functional and subjective health. All regression models were run with and without adjusting for childhood SES and gender. All analyses were conducted with the Mplus 5.2 software (Muthén & Muthén, 1998–2007).

4. Results

Table 1 summarizes the results; see the online supplemental materials for a full description of the results, the regression models that were analyzed, and further effect size measures.

Table II-1. *Models analyzing the effects of childhood general intelligence (g), fluid intelligence (Gf), and crystallized intelligence (Gc) on three dimensions of adult health, unadjusted and adjusted for SES and gender*

Dependent variable	Model Set 1	Model Set 2	Model Set 3	Model Set 4	Model Set 5	
	Bivariate regression[a]	Multiple regression[b]	Bivariate regression[a]	Bivariate regression[a]	Multiple regression[b]	
	g	*g*	Gf	Gc	Gf	Gc
(1) Physical Health						
Doctor visits						
b	–0.27*	–0.26*	–0.35*	–0.17*	-0.35_c*	-0.02_d
95% CI	[–0.42, –0.11]	[–0.41, –0.10]	[–0.49, –0.20]	[–0.31, –0.02]	[–0.50, –0.19]	[–0.17, 0.13]
OR	0.77	0.77	0.71	0.85	0.71	0.98
ES	–.07	–.07	–.09	–.05	–.10	–.01
Sick leave days						
b	–0.16	–0.14	–0.25*	–0.15	-0.23_e*	-0.06_e
95% CI	[–0.34, 0.01]	[–0.32, 0.04]	[–0.42, –0.09]	[–0.32, 0.01]	[–0.40, –0.05]	[–0.23, 0.12]
OR	0.85	0.87	0.78	0.86	0.80	0.95
ES	–.05	–.04	–.07	–.04	–.06	–.02
Hospital nights						
b	–0.09	–0.08	–0.09	–0.19	0.00_f	-0.20_f
95% CI	[–0.30, 0.12]	[–0.30, 0.13]	[–0.30, 0.12]	[–0.39, 0.01]	[–0.24, 0.25]	[–0.43, 0.03]
OR	0.91	0.92	0.92	0.83	1.00	0.82
ES	–.03	–.02	–.02	–.05	.00	–.05
(2) Functional Health						
b	3.00*	2.74*	3.39*	1.74*	3.16_g*	0.18_h
95% CI	[1.33, 4.68]	[1.00, 4.48]	[1.69, 5.08]	[0.13, 3.35]	[1.38, 4.95]	[–1.51, 1.87]
ES	.14	.13	.15	.08	.14	.01
(3) Subjective Health						
b	1.84*	1.52	2.26*	0.29	2.23_i*	-0.72_i
95% CI	[0.07, 3.62]	[–0.30, 3.34]	[0.45, 4.07]	[–1.48, 2.06]	[0.25, 4.22]	[–2.65, 1.22]
ES	.08	.06	.09	.01	.09	–.03

Note. Regression coefficients in Model Set 5 that show different subscripts are statistically different ($p < .05$, two-sided testing), whereas those that share the same subscript are not. *b* = unstandardized regression coefficient; CI = confidence interval; *OR* = odds ratio; ES = effect size in correlation (Model Sets 1, 3, and 4) and partial correlation metric (Models Sets 2 and 5). See Table S1 in the online supplemental materials for a full description of the models applied and results, including effect sizes for SES and gender.

[a]Bivariate regression: Model Sets 1, 3, and 4 show the unadjusted effects of intelligence (*g*, Gf, or Gc).

[b]Multiple regression: Model Set 2 shows the effects of *g* adjusted for SES and gender, Model Set 5 shows the effects of Gf and Gc adjusted for SES and gender.

*$p < .05$, two-sided testing.

Model Set 1 analyzed the effects of childhood general intelligence on each adult health outcome separately. With respect to physical health, individuals with higher general intelligence reported significantly fewer doctor visits, but not sick leave days or hospital nights. The ordinal logistic regression coefficient of $b = –0.27$ indicates that each standard deviation increase in general intelligence was associated with a 23% decrease in the odds of reporting a higher number of doctor visits on the health questionnaire, rather than a lower number of doctor visits. Further, individuals with higher general intelligence experienced better functional and subjective health in adulthood. Each standard deviation increase in general intelligence was associated with an average increase of 3% in functional health and of 1.84% in subjective health in adulthood. Model Set 2 analyzed the effects of childhood general intelligence on each health outcome while adjusting for potential effects of SES and gender. The inclusion of SES and gender (effect sizes not shown in Table 1 but in Table S1 in the online supplemental materials) attenuated the effects of general intelligence (Model Set 2 in Table 1) to some degree. Nevertheless, individuals with higher childhood general intelligence still reported fewer doctor visits and better functional health in adulthood. Notably, in most cases, the effect sizes for intelligence were considerably higher than those for childhood SES.

Model Set 3 analyzed the effects of childhood Gf on each health outcome, and Model Set 4 analyzed the effects of childhood Gc on each health outcome. Individuals with higher Gf reported significantly fewer doctor visits and sick leave days, but not hospital nights. Further, they reported significantly better functional and subjective health. Effect sizes for Gf were slightly higher than those for general intelligence. The effects of Gc pointed in the same direction, yet most effect sizes were smaller than those obtained for Gf or general intelligence (but the number of hospital nights was an exception).

Model Set 5 analyzed the differential effects of childhood Gf and Gc while adjusting

for SES and gender (see Table S1 in the online supplemental materials for a full description of results). Importantly, Gf significantly predicted a lower number of doctor visits and sick leave days, as well as better functional and subjective health, above and beyond the effects of Gc, SES, and gender. By contrast, all effects of Gc that could be observed in Model Set 4 decreased substantially and did not reach statistical significance. A formal comparison showed that the effect of Gf on doctor visits and functional health was significantly higher than the effect of Gc. The difference of effects for the remaining health outcomes pointed in the same direction but did not reach statistical significance (see Table 1).

5. Discussion

This prospective cohort study capitalized on a hierarchical conceptualization of childhood intelligence and a multidimensional conceptualization of adult health. The three main findings were: (a) childhood intelligence (particularly fluid intelligence: Gf) predicted key dimensions of adult health over 40 years, (b) Gf seemed to be more important in predicting adult health than Gc, and (c) these effects persisted even when childhood SES and gender were adjusted for. Considering the time span of 40 years and the complexity of the phenomena under investigation, effect sizes were substantial (Meyer et al., 2001) and comparable to those for other factors that influence health (Johnson et al., 2011). These findings substantiate the broad generalizability of the core hypothesis of cognitive epidemiology: Childhood intelligence predicts better adult health, even in a country with universal access to quality health care, and even when adjusting for other important childhood variables such as SES (Calvin et al., 2011). Further, the effects of childhood intelligence on three dimensions of health were differentiated. Gf seemed to be the most important predictor of adult health. First, childhood Gf had a positive effect on key indicators of physical health: Higher Gf was associated with fewer doctor visits and sick leave days. This finding supports the idea that higher childhood intelligence fosters the development of

health literacy. Intelligent people may know when it is necessary to consult a doctor and be better at following medical instructions, which in turn decreases their overall number of doctor visits and sick leave days (Gottfredson, 2004). Note, however, that childhood intelligence did not significantly affect the number of nights in the hospital. This may be due to the comparatively young age of our study population. The effects of age-related, often chronic diseases that cause longer hospital stays may not yet be as pronounced at age 52 as they are in old age (Deary, Whiteman, Starr, Whalley, & Fox, 2004). Second, Gf had positive effects on functional health. More intelligent individuals may develop better coping strategies for dealing with functional impairment (Deary et al., 2004). Third, given that childhood intelligence, particularly Gf, was positively associated with functional health and vital indicators of physical health, it was not surprising that participants with high childhood intelligence subjectively evaluated their health in more positive terms. The finding that Gf seemed to be the most important aspect of childhood intelligence in predicting adult health may indicate that general problem solving and reasoning skills are key when dealing with health and health-related information (Gottfredson, 2004).

This study has several limitations. First, participants self-reported their health. Self-report measures may be subject to reporting biases. However, empirical research has confirmed that self-reported health ratings are reliable and valid measures of health (Hultsch et al., 1999; Liang, 1986). Nevertheless, for a more comprehensive assessment of health, additional data sources should be included (e.g., physician-diagnosed diseases). Second, we focused on the predictive power of childhood intelligence across a 40-year period. Future research should examine the mediating processes that link childhood intelligence and SES to adult health (e.g., health literacy, educational and occupational development). Third, this study was conducted in Luxembourg, where all citizens have access to quality health care. Hence, the positive effects of intelligence on health observed in the present study may be

lower-bound effect estimates rather than upper-bound effect estimates. Access to quality health care may compensate for some of the differential positive effects of intelligence on health. Further research in countries with different health care systems is needed to examine this hypothesis. Fourth, relative to other studies in cognitive epidemiology, the sample size was not large, although power calculations showed that it should be sufficient for detecting even small effects (Cohen, 1988).

In conclusion, our findings suggest that intelligence may be a potential cause of social class inequalities in health that cannot be fully offset even by excellent public health care. General reasoning and problem solving skills seem to be crucial in managing the "job of being a patient" (Gottfredson, 2004, p. 175). In light of these findings, early interventions fostering these intellectual abilities (e.g., the CARE or Abecederian Project; Campbell et al., 2008) and health literacy may help to reduce later health disparities. Further, interventions reducing risk factors to childhood intellectual and physical development (e.g., exposure to toxins; Evans, 2004) appear crucial, given the potential long-term detrimental effects on health. Finally, public health care and preventive measures, as well as patient contact with health care practitioners, should be adapted to the intellectual abilities of the recipients.

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Online Supplemental Materials

1. Appendix A: Comprehensive description of the measures

Childhood intelligence

Childhood intelligence was assessed by the Leistungsprüfsystem (L-P-S [Performance Test System]; Horn, 1962, 1983). The L-P-S is a standardized, objective, and comprehensive German intelligence test based on the model of primary mental abilities formulated by Thurstone (1938). Its 14 subtests provide a measure of general intelligence (total IQ score) as well as scores for more specific intellectual facets, such as crystallized intelligence and fluid intelligence (Neubauer, Fink, & Schrausser, 2000). The scores for crystallized intelligence are based on three subtests. Two subtests consist of misspelled six-letter words; participants have to identify the appropriate words as well as the spelling errors. The other subtest consists of anagrams (Borkenau & Liebler, 1993). The scores for fluid intelligence are based on two subtests inspired by Raven's Progressive Matrices (Horn, 1983). For both subtests, participants have to identify the inappropriate element in a series of eight elements, the elements of the first subtests being geometric figures and those of the second subtest being letters and digits (Borkenau & Liebler, 1993).

Split-half reliability of the overall test is .99, parallel-forms reliability is .94. Retest reliability across a time span of 32 months is .83 for the overall test score (Horn, 1983; Tent, 1969), .94 for the combined score for crystallized intelligence, and .78 for the combined score for fluid intelligence (Horn, 1983). There is ample evidence for the construct validity of the L-P-S. Specifically, the correlation of the L-P-S total score with the total score on the German version of the Wechsler Adult Intelligence Scale (WAIS)—the Hamburg Wechsler Intelligenztest für Erwachsene (HAWIE-R; Tewes, 1991)—is .94 (Sturm & Büssing, 1982). Furthermore, the correlation of the standardized L-P-S total score with the standardized total

score of the Intelligenz-Struktur-Test (IST; Liepmann, Beauducel, Brocke, & Amthauer, 2001) is .72. The IST is another well-validated and widely used German intelligence test that also correlates substantially with the HAWIE-R (Tewes, 1991). In a recent meta-analysis, Hülsheger, Maier, Stumpp, and Muck (2006) compared the predictive validity of the L-P-S and five other intelligence tests widely used in German-speaking countries, including the IST and Raven's Progressive Matrices (Kratzmeier, 1979), for the outcomes of vocational education. The authors found the L-P-S to be one of the instruments with the highest criterion-related validity. Further, the total and subtest scores of the L-P-S showed high correlations with grades in various school subjects (Horn, 1983). For instance, the total score showed a correlation of .55 with grade point average in Grade 4 of elementary school (Tent, 1965). The crystallized intelligence score showed a correlation of .47 with German grades, and the fluid intelligence score a correlation of .80 with mathematics grades (Horn, 1983). Given the strong empirical evidence for its reliability and validity, the L-P-S is widely employed in various areas of current research, such as research on gender differences in cognitive functions (Weiss, Kemmler, Deisenhammer, Fleischhacker, & Delazer, 2003) or clinical and neuropsychology (Kuelz, Hohagen, & Voderholzer, 2004).

Childhood socioeconomic status (SES)

Childhood SES was assessed by the widely used International Standard Classification of Education and Income (ISEI; Ganzeboom, de Graaf, Treiman, & de Leeuw, 1992; Ganzeboom & Treiman, 1996). The ISEI score used in the present study was based on students' reports of their parents' occupations, which were first mapped onto the categories of the International Standard Classification of Occupations (ISCO-88; Elias, 1997). Note that Ganzeboom and Treiman (1996) showed that the ISCO-88 scheme also applies to occupational data from 1968. These classifications were then transformed into the ISEI scale, which takes into account income and the educational level of occupations. The ISEI has

interval scale properties and a theoretical range from 16 (e.g., cleaners, unskilled agricultural laborers) to 90 (e.g., judges). With its grounding in international occupational classification schemes, the ISEI scale is internationally comparable; it has been demonstrated to be a reliable and valid indicator of socioeconomic status in many international large-scale assessments (e.g., PISA; Organisation for Economic Co-operation and Development, 2004). In the present study, we used the highest ISEI value in a family to indicate childhood SES. Interrater reliability of the ISEI coding was tested for two independent groups of raters and was satisfactory at .72.

Adult health

The conceptualization and measurement of adult health in the present study was based on the multidimensional model of health developed by Liang and colleagues (1986, 1991). In this model, health has a physical dimension, a (social-)functional dimension, and a subjective dimension. Liang (1986) based his model on the World Health Organization's definition of health as "[...] a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity" (World Health Organization, 1958, p. 469). This multidimensional approach to the measurement of health has been exceedingly influential in the social sciences. Liang's (1986) paper is, for instance, among the 20 most frequently cited social science articles ever published in the *Journal of Gerontology* (Ferraro & Schafer, 2008).

Physical health. Reliable and valid indicators of the physical dimension were adapted from the large-scale German ALLBUS survey (Haarmann, Scholz, Wasmer, Blohm, & Harkness, 2006; Terwey, 2000), which is similar in theoretical scope and methodology to the American General Social Survey (Davis, Mohler, & Smith, 1994). Specifically, participants reported: (a) the number of visits to the doctor in the last 3 months (0 = none, 1 = 1, 2 = 2, 3 = 3, 4 = 4, 5 = 5–6, 6 = 7–8, 7 = 9–10, 8 = 11–15, 9 ≥ 15), (b) the number of sick leave days in

the last 12 months (0 = none, 1 = 1–3, 2 = 4–7, 3 = 8–14, 4 = 15–30, 5 = 31–60, 6 ≥ 60), and (c) the number of nights spent in the hospital in the last 12 months (0 = none, 1 = 1–3, 2 = 4–7, 3 = 8–14, 4 = 15–30, 5 = 31–60, 6 ≥ 60). The response categories for this study were derived from data from the representative ALLBUS survey, in which these questions were presented in an open-answer format. The use of categories facilitated automated processing and scoring of responses in the present study.

*Functional health***.** The functional health measure was adapted from an influential study by Hultsch, Hertzog, Small, and Dixon (1999). Specifically, participants used a 5-point rating scale (1 = not at all, 2 = hardly, 3 = moderately, 4 = significantly, 5 = strongly, gave up the activity) to answer the question "Looking back over the past two years, how much did your health status hinder the following activities?" Nine activities suitable for indicating the functional health of adults aged 52 years were examined: (1) work activities, (2) household tasks (e.g., cleaning, doing laundry, carrying out repairs), (3) mobility (e.g., running errands, visits to the authorities), (4) maintaining relations (e.g., family reunions, meeting friends), (5) travel (e.g., holidays, excursions), (6) intellectual activities (e.g., playing chess, crossword puzzles), (7) sports activities (e.g., riding a bike, hiking), (8) leisure activities (e.g., going to the movies, to the theater), and (9) other hobbies (e.g., do it yourself, painting). Internal consistency for the total score was $\alpha = .93$.

Our measure of functional health shows certain similarities to other established scales, such as the SF-36 (Ware & Gandek, 1998). Specifically, the SF-36 physical functioning scale assesses health-related limitations in physical activities such as lifting heavy objects, sports, climbing stairs, kneeling, and walking various distances. Furthermore, it includes two questions assessing health-related limitations in social activities and one question concerning limitations in work activities. These aspects were also covered in the present study. Specifically, we assessed limitations in sports and mobility in a manner similar to the SF-36

physical functioning scale, limitations in the ability to maintain relations in a manner similar to the SF-36 social functioning scale, and limitations in work activities in a manner similar to the SF-36 question on work activities. However, the present study also went beyond aspects showing similarities to the SF-36 scales by assessing further aspects of functional health, such as traveling, intellectual activities, specific leisure activities, and hobbies.

Subjective health. Participants used a 5-point rating scale (1 = *very poor*, 2 = *poor*, 3= *satisfactory*, 4 = *good*, 5 = *very good*) to evaluate (a) their overall health status and (b) their health status relative to peers. The two questions were taken from Liang (1986) and were also employed by Hultsch et al. (1999) and in the ALLBUS survey. Participants used a 7-point rating scale (1 = *very dissatisfied*, 2 = *dissatisfied*, 3 = *somewhat dissatisfied*, 4 = *neutral*, 5 = *somewhat satisfied*, 6 = *satisfied*, 7 = *very satisfied*) to report (c) satisfaction with their health status. This question has been widely used in several international representative surveys (e.g., the European Values Study; Halman, 2001). Internal consistency of the total score was $\alpha = .85$.

2. Appendix B: Detailed description of the regression models

To test the effects of childhood intelligence and its facets on each of the five health outcomes, we ran five models for each of the five health outcomes (the three indicators of physical health—doctor visits, sick leave days, and hospital nights—as well as the measures for functional health and for subjective health), resulting in a total of 25 regressions (see Table 1 and Table S1). Model Sets 1 to 5 differed with respect to the predictors in the analyses.

Model Set 1 investigated the unadjusted effect of childhood general intelligence on each of the five health outcomes with five bivariate regressions. Equation 1 shows an exemplary model with a linear link function:

$$Health_{hi} = b_0 + b_1 g_i + e_i \quad (1)$$

The variable $Health_{hi}$ represents each one of the five health outcomes in Equation 1, with subscript *h* taking on 1 for doctor visits, 2 for sick leave days, 3 for hospital nights, 4 for functional health, and 5 for subjective health, and subscript *i* ranging from Individual 1 to Individual 717. Health is predicted by the grand mean in the sample, b_0; the effect of each individual's childhood general intelligence, b_1 g_i; and a residual error term for each individual, e_i.

Model Set 2 investigated the effect of childhood general intelligence on each of the five health outcomes, adjusted for childhood SES and gender, with five multiple regressions (see Equation 2).

$$Health_{hi} = b_0 + b_1 g_i + b_2 SES_i + b_3 gender_i + e_i \quad (2)$$

Model Set 3 investigated the unadjusted effect of childhood fluid intelligence (represented as Gf_i in Equation 3) on each of the five health outcomes with five bivariate regressions.

$$Health_{hi} = b_0 + b_1 Gf_i + e_i \quad (3)$$

Model Set 4 investigated the unadjusted effect of childhood crystallized intelligence (represented as Gc_i in Equation 4) on each of the five health outcomes with five bivariate regressions.

$$Health_{hi} = b_0 + b_1 Gc_i + e_i \quad (4)$$

Model Set 5 investigated the differential effects of childhood fluid and crystallized intelligence on each of the five health outcomes, adjusted for childhood SES and gender, with five multiple regressions (see Equation 5).

$$Health_{hi} = b_0 + b_1 Gf_i + b_2 Gc_i + b_3 SES_i + b_4 gender_i + e_i \quad (5)$$

To test whether the effects Gf_i and Gc_i on health outcomes were statistically different from each other, we applied the versatile methodological approach developed by Cheung (2009) where the difference in regression coefficients is represented as a new model parameter. To judge the statistical significance of the difference in regression coefficients, the Mplus software (Muthén & Muthén, 1998-2007) computes corresponding standard errors and *p* values for the new model parameter.

To account for the psychometric properties of the various measures of adult health, ordinal logistic regressions were used to predict each of the three indicators of physical health, and linear regressions were used to predict functional and subjective health. Irrespective of the type of link function applied in ordinal or linear regressions, models listed in the same column in Tables 1 and S1 share the common interpretation that higher values of the regression coefficients *b* indicate more influence of childhood intelligence on each health outcome.

3. Appendix C: Population Attributable Risk Percentage (PAR%) as an additional effect size measure

In the main article, the effects of childhood intellectual abilities on adult health were presented in terms of correlations (Model Sets 1, 3, and 4) and partial correlations (Model Sets 2 and 5). Correlational effect size measures are helpful in quantifying the strength of a relationship. However, they are of limited use in illustrating the public health significance of low childhood intellectual abilities as risk factors for poor adult health. As recommended by an anonymous reviewer, one measure that may be effectively used for this purpose is the *Population Attributable Risk Percentage* (PAR%; Levin, 1953; Northridge, 1995; Rothman, Greenland, & Lash, 2008; see also Deubner, Wilkinson, Helms, Tyroler, & Hames, 1980). In the present study, the computation of PAR% takes into account the frequency of the corresponding risk factor (i.e., low childhood intelligence) in the population, and the probability of developing poor health given a low level of childhood intelligence. In so doing, PAR% estimates the population percentage of all persons with poor adult health in which this outcome could be prevented if it were possible to raise children's intelligence such that they were no longer at risk (see Rothman et al., 2008). To this end, we defined childhood intellectual abilities (as measured in terms of the general IQ score as well as the Gf and Gc scores) to be at an at-risk level when children's test scores were one standard deviation below the corresponding population mean. As test scores were normally distributed, this implied that children whose scores were below the 16th percentile of the corresponding score distribution were considered to be at risk. Poor health levels in adulthood were defined as a number of doctor visits, sick leave days, or hospital nights in the highest quartile of the distribution. Likewise, a functional or subjective health score in the lowest quartile was considered to be indicative of poor health. Using these definitions, PAR% was computed as follows (Northridge, 1995):

$$PAR\% = 0.16*(RR-1)/[1+0.16*(RR-1)] \times 100 \quad (1)$$

where 0.16 is the prevalence of students with a low level of childhood intellectual abilities, be it general intelligence, Gf, or Gc. RR is the rate ratio—that is, the relative risk of developing poor health for children with a low level on a certain intellectual ability compared to children who did not demonstrate a low level on that ability.

Table S1 in this online supplement summarizes the results obtained for PAR%. The pattern of results largely corresponded to that observed for the correlational effect size measures. Importantly, the PAR% estimates indicate that increasing children's level of Gf has public health significance, particularly in view of the high costs associated with hospital stays, doctor visits, and sick leave days. For example, the PAR% estimates for fluid intelligence and the number of doctor visits suggest that an intervention that could raise the Gf of at-risk children above the threshold of one standard deviation below the population mean would reduce the number of later doctor visits by about 12%. In sum, the PAR% estimates in the present study further highlight the importance of early interventions targeting general reasoning and problem solving skills, and of adapting public health care and preventive measures to the intellectual abilities of the recipients

Table II-S1. *Prediction of three dimensions of adult health by childhood general (g), fluid (Gf), and crystallized (Gc) intelligence*

Dependent variable	Model Set 1	Model Set 2			Model Set 3	Model Set 4	Model Set 5			
	Bivariate regression[a]	Multiple regression[b]			Bivariate regression[a]	Bivariate regression[a]	Multiple regression[b]			
	g	*g*	SES	Gender	Gf	Gc	Gf	Gc	SES	Gender
(1) Physical Health										
Doctor visits										
b	–0.27*	–0.26*	–0.05	–0.29*	–0.35*	–0.17*	-0.35_c*	-0.02_d	–0.04	–0.33*
95% CI	[–0.40, –0.14]	[–0.39, –0.13]	[–0.17, 0.07]	[–0.51, –0.06]	[–0.47, –0.22]	[–0.29, –0.04]	[–0.48, –0.22]	[–0.15, 0.11]	[–0.16, 0.08]	[–0.56, –0.10]
OR	0.77	0.77	0.95	0.75	0.71	0.85	0.71	0.98	0.96	0.72
ES	–.07	–.07	–.01	–.08	–.09	–.05	–.10	–.01	–.01	–.09
PAR %[c]	11.61	--	--	--	11.99	7.73	--	--	--	--
Sick leave days										
b	–0.16*	–0.14	–0.11	–0.10	–0.25*	–0.15*	-0.23_e*	-0.06_e	–0.10	–0.12
95% CI	[–0.31, –0.02]	[–0.29, 0.01]	[–0.25, 0.04]	[–0.26, 0.17]	[–0.39, –0.11]	[–0.29, –0.01]	[–0.38, –0.08]	[–0.20, 0.09]	[–0.24, 0.05]	[–0.38, 0.15]
OR	0.85	0.87	0.90	0.91	0.78	0.86	0.80	0.95	0.91	0.89
ES	–.05	–.04	–.03	–.03	–.07	–.04	–.06	–.02	–.03	–.03
PAR %[d]	4.05	--	--	--	7.83	2.21	--	--	--	--
Hospital nights										
b	–0.09	–0.08	–0.04	0.08	–0.09	–0.19*	0.00_f	-0.20_f	–0.03	0.12
95% CI	[–0.27, 0.08]	[–0.26, 0.09]	[–0.22, 0.13]	[–0.26, 0.41]	[–0.26, 0.09]	[–0.36, –0.02]	[–0.20, 0.21]	[–0.39, 0.03]	[–0.20, 0.15]	[–0.22, 0.46]
OR	0.91	0.92	0.96	1.08	0.92	0.83	1.00	0.82	0.97	1.13
ES	–.03	–.02	–.01	.02	–.02	–.05	.00	–.05	–.01	.03
PAR %[c]	4.50	--	--	--	1.42	7.62	--	--	--	--
(2) Functional Health										
Intercept	82.366		81.589		82.212	82.701		81.204		
b	3.00*	2.74*	1.30*	1.46	3.39*	1.74*	3.16_g*	0.18_h	1.36*	1.85
95% CI	[1.65, 4.36]	[1.33, 4.15]	[0.01, 2.58]	[–1.09, 4.01]	[1.97, 4.80]	[0.42, 3.06]	[1.64, 4.68]	[–1.25, 1.60]	[0.09, 2.63]	[–0.74, 4.43]
ES	.14	.13	.06	.04	.15	.08	.14	.01	.06	.05
PAR %[c]	8.11	--	--	--	9.13	3.68	--	--	--	--
(3) Subjective Health										
Intercept	68.342		69.142		68.202	68.682		68.855		
b	1.84*	1.52	1.63*	–1.86	2.26*	0.29	2.24_i*	-0.72_i	1.73*	–1.42
95% CI	[0.36, 3.33]	[0.00, 3.04]	[0.15, 3.12]	[–4.64, 0.93]	[0.75, 3.78]	[–1.19, 1.77]	[0.57, 3.90]	[–2.34, 0.90]	[0.27, 3.19]	[–4.24, 1.41]
ES	.08	.06	.07	–.04	.09	.01	.09	–.03	.07	–.03
PAR %[c]	4.50	--	--	--	5.71	3.44	--	--	--	--

Note. Regression coefficients in Model Set 5 that show different subscripts are statistically different ($p < .05$, two-sided testing), whereas those that share the same subscript are not. *b* = unstandardized regression coefficient; CI = confidence interval; *OR* = odds ratio; ES = effect size in correlation (Model Sets 1, 3, and 4) and partial correlation metric (Model Sets 2 and 5). [a]Bivariate regression: Models 1, 3, and 4 show the unadjusted effects of intelligence (*g*, Gf, or Gc). [b]Multiple regression: Models 2 show the effects of *g* adjusted for SES and gender, Models 5 show the effects of Gf and Gc adjusted for SES and gender. [c]PAR % = Population Attributable Risk Percentage *$p < .05$, two-sided.

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Chapter III

Study II:

Childhood Intelligence and Adult Health: The Mediating Roles of Education and Socioeconomic Status

Abstract

The longitudinal relation between childhood intelligence and various health outcomes in adulthood is now well-established. One mediational model that accounts for this relation proposes that intelligence has cumulative indirect effects on adult health via subsequent educational attainment and adult socioeconomic status (SES). The aim of the present study was to examine whether and the extent to which educational attainment and SES mediate the impact of childhood intelligence on three dimensions of adult health in Luxembourg, a country with high-quality universal public health care. We used data from 745 participants in the Luxembourgish MAGRIP study. At the age of 12, participants completed a comprehensive intelligence test. At the age of 52, they reported their educational careers, SES, and functional, subjective, and physical health status. Using structural equation modeling, we investigated the direct and indirect effects (via educational attainment and adult SES) of childhood intelligence on adult health. We found that higher childhood intelligence predicted better functional, subjective, and physical health in adulthood. These effects were entirely mediated via educational attainment and SES. The mediational processes differed depending on the health dimension under investigation: Whereas SES was crucial in mediating the effect of intelligence on functional and subjective health, educational attainment was crucial in mediating the effect on physical health. These findings held up when considering adult intelligence and were similar for women and men. Our results suggest that even excellent public health care cannot fully offset the cumulative effects of childhood intelligence on adult health. Further studies are needed to investigate the relative importance of different mediators

in the intelligence-health relation while including a broader set of objective health measures.

Keywords: childhood intelligence, adult health, mediation, educational attainment, socioeconomic status

Note. For reasons of clarity, the text sections, tables, and figures in the supplementary materials section (pp. 154-184) have been arranged in the order in which they are mentioned in (a) the main manuscript (pp.121-153) and (b) the supplementary materials section.

Childhood Intelligence and Adult Health: The Mediating Roles of Education and Socioeconomic Status

1. Introduction

General intelligence (*g*) is a powerful predictor of important outcomes across the lifespan. For instance, intelligence predicts socioeconomic outcomes such as educational attainment, occupational success, and income (Deary & Johnson, 2010; Deary et al., 2005; Johnson, Brett, & Deary; 2010; Kuncel, Hezlett, & Ones, 2004; Schmidt & Hunter, 2004; Strenze, 2007). Further, intelligence predicts many health outcomes such as mortality, physician-diagnosed diseases, and health behaviors (Calvin et al., 2011; Deary, Weiss, & Batty 2010). To account for the positive effects of intelligence on health, several mediational models have been proposed. This paper focuses on one model that proposes that intelligence is the origin of a "chain reaction" (Gottfredson, 2002, p. 369) and has cumulative effects on subsequent health outcomes via socioeconomic outcomes. Several studies have investigated this proposed mediation model (Batty, Deary, et al., 2008; Batty, Deary, Schoon, & Gale, 2007a, 2007b; Batty, Gale, et al., 2008; Batty, Shipley, et al., 2008). However, studies investigating the "chain reaction" model with a prospective cohort design over several decades and with multiple health dimensions are still rare. Nevertheless, such studies are greatly needed as they (a) contribute to more substantive theories about the interplay between intelligence and key life outcomes and (b) pinpoint possible targets and stages for interventions to improve individuals' health. The major objective of the present study was therefore to examine whether and the extent to which education and socioeconomic status (SES) mediate the impact of childhood intelligence (at age 12) on adult health (at age 52).

1.1. Childhood intelligence and adult health

The systematic investigation of intelligence as a predictor of different health outcomes just began in the last decade of the 20th century (Deary, 2012). Mortality was probably the first health outcome studied in relation to intelligence. In an early article, Whalley and Deary (2001) showed that lower IQ scores at age 11 were significantly associated with an increased risk of mortality up to age 76. Since this publication, the number of studies linking intelligence measured early in life to adult mortality and nonmortality outcomes—a research field labeled *cognitive epidemiology* (*Intelligence*, 2009)—has increased steadily. Today, the link between intelligence and all-cause and cause-specific mortality is well-established (Calvin et al., 2011; Deary, et al., 2010). In addition to mortality, intelligence in childhood and early adulthood has been linked to various physical diseases. Several studies have replicated the relation between higher intelligence in early life and a lower risk of cardiovascular disease (Modig Wennerstad, Silventoinen, Tynelius, Bergman, & Rasmussen, 2009). Moreover, early intelligence has been related to risk factors for cardiovascular and other chronic diseases such as hypertension and obesity (Batty et al., 2007a). Intelligence also predicts health-relevant behaviors such as smoking (Batty et al., 2007a), alcohol intake (Batty, Deary, et al., 2008), physical activity, and diet (Batty et al., 2007b). Beyond cardiovascular disease, early intelligence predicts additional diagnosed diseases and self-reported physical health problems (Der, Batty, & Deary, 2009) as well as mental health outcomes and psychiatric diseases (Gale et al., 2008). Beyond the effects on the individual level, intelligence has been shown to be an important predictor of health at the national and international levels. For instance, Reeve and Basalik (2010) showed that the average state IQ in the 50 U.S. states was substantially associated with a range of health indicators even after controlling for differences in health care expenditures and state wealth.

1.2. Mechanisms that explain the link between childhood intelligence and adult health

Several nonexclusive mechanisms have been proposed to account for the relation of early intelligence with morbidity and mortality in adulthood. First, the link between early intelligence and adult health could be mediated by health behaviors (e.g., smoking, alcohol intake, dietary choices, or physical activity; Deary, 2010, 2012) that are tied to risk factors for diseases. Second, early intelligence could contribute to the acquisition of health-related knowledge. Higher knowledge could then lead to better health behaviors, and in the case of illness, to the ability to communicate effectively with medical staff and to understand medical instructions (Johnson, Corley, Starr, & Deary, 2011). Higher intelligence may thus help a person to successfully manage the "job of being a patient" (Gottfredson, 2004, p. 175).

A third possible mediational pathway—which is at the center of the present paper—links early life intelligence to adult health via favorable educational and socioeconomic outcomes (see Figure 1), as Gottfredson's (2002) "chain reaction" model suggests. In this model, higher intelligence in early life leads to higher educational attainment (Path 1). Educational attainment then facilitates access to higher adult socioeconomic status (SES; Path 3), which includes a higher income and a safer work environment (Deary, Strand, Smith, & Fernandes, 2007; Deary et al., 2005; Johnson et al., 2010). Higher adult SES is related to good health (Path 5; Deary, 2010). Statistical adjustment for socioeconomic outcomes substantially attenuates and sometimes nullifies the relation between early intelligence and later health (Batty et al., 2007a; Deary, 2010; Deary et al., 2010), highlighting the importance of socioeconomic variables as mediators. Thus, in the case of complete mediation, the direct effect of childhood intelligence on adult health (Path 6) would be expected to be zero.

Despite considerable evidence that has emphasized the relevance of education and socioeconomic status as potential mediators, previous research has yielded mixed results regarding the extent of mediation in the relations of early intelligence with adult mortality

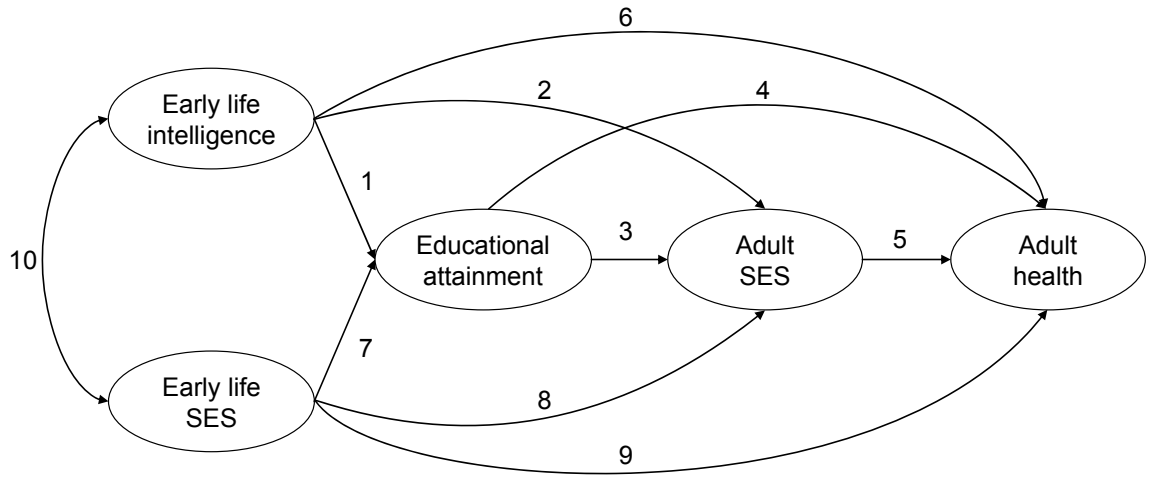
and morbidity. With respect to mortality, Batty and Deary (2005) reported that adjusting for educational attainment leads to inconsistent results, with some studies reporting pronounced mediation and others reporting little or no mediation. Likewise, in their meta-analysis, Calvin et al. (2011) reported varying degrees of mediation of the intelligence-mortality relation via educational attainment and subsequent SES. Moreover, the extent of mediation might depend on the time in life when intelligence is measured. For instance, one study that found complete mediation of the intelligence-mortality relation investigated intelligence measured in early adulthood after participants had already completed their educations. Thus, the intelligence-mortality relation could have been confounded by education (Calvin et al., 2011). Measuring intelligence before participants have completed their educations may therefore be important for detecting unique effects of intelligence and education. With respect to morbidity, results have also been inconsistent. For instance, Batty, Gale, et al. (2008) reported a negative association between higher intelligence in early adulthood and the metabolic syndrome in middle adulthood that was barely affected when adjusting for education, income, and social prestige. Further, Batty et al. (2007b) reported positive associations of higher childhood intelligence with a healthy diet and physical activity in adulthood. These associations were reduced when adjusting for indices of socioeconomic position in adulthood; however, they remained statistically significant in some analyses. In sum, studies investigating the extent to which educational attainment and SES mediate the relation between intelligence and health would add substantially to the literature.

1.3. The present study

The study of mediational mechanisms that underlie the intelligence-health relation has been identified as a "key priority" in cognitive epidemiology (Deary et al., 2010, p. 71). A better understanding of these meditational mechanisms will inform applications of findings from cognitive epidemiology to public health (Deary, 2012). Such studies will ideally (a)

investigate intelligence measured early in life before differential effects of education have set in and (b) employ statistical techniques (such as structural equation modeling; SEM) that include several measures of each construct to alleviate the problem of measurement error (Deary & Johnson, 2010; Deary et al., 2010). The present paper fulfills these methodological requirements: It rigorously investigates how the impact of childhood intelligence on a variety of health outcomes measured in middle adulthood is mediated via educational attainment and subsequent socioeconomic status (SES).

Figure III-1. *Mediation model illustrating how early life intelligence influences adult health via educational attainment and adult socioeconomic status (SES)*



Note. Latent factors are depicted as ellipses. For clarity of presentation, manifest indicators and variances of latent factors have been omitted. Double-headed arrows represent correlations.

Figure 1 shows the hypothesized model. In line with prior research (Batty et al., 2007a; Calvin et al., 2011; Deary, 2010; Deary et al., 2010), we predicted that the relation between childhood intelligence and adult health would be mediated via educational attainment and adult SES (Paths 1 to 5). Specifically, we proposed that childhood intelligence would be associated with higher educational attainment (Path 1) and better SES in adulthood (Path 2); further, that educational attainment would be associated with adult SES (Path 3); and that educational attainment (Path 4) and adult SES (Path 5) would in turn be associated with

better adult health. We also examined whether childhood intelligence would retain a direct effect (Path 6) on adult health (Deary, 2012; *Intelligence*, 2009) once the mediational effects via education and adult SES were taken into account. To adjust for potential effects of childhood SES (Paths 7 to 9), childhood intelligence and childhood SES were allowed to correlate (Parameter 10; cf. Deary et al., 2005). Treating childhood SES and intelligence as correlated constructs is a preferable theory-neutral position until more is known about their temporal order and causal relation. In a series of auxiliary analyses, we also explored whether the inclusion of adult intelligence as additional mediator (data available for a subgroup of our study sample) and gender would affect the results of the above-mentioned analyses (see online supplementary materials, Sections S2 and S3, respectively).

In sum, the present study examined whether and the extent to which education and socioeconomic status mediate the impact of childhood intelligence (at age 12) on adult health (at age 52). The present study thus makes several important contributions to the growing research area of cognitive epidemiology. First, almost all previous studies in cognitive epidemiology have been conducted in English-speaking or Scandinavian countries (Calvin et al., 2011). Can findings from these studies be replicated in countries with different levels of social mobility or different health care systems? For instance, universal access to quality health care may compensate for the differential effect of intelligence on health, and as a result, intelligence may lose its impact. Thus, a major goal of this study was to examine whether the key findings of cognitive epidemiology would generalize to Luxembourg—a country that has a level of social mobility below the OECD average (Organisation for Economic Co-operation and Development, 2010) but that provides universal access to quality health care. Specifically, the Luxembourgish health care system ranks 16th among 190 worldwide health care systems (World Health Organization, 2000). The state covers the main part of health expenditures, and private "out-of-pocket" payments are low (Huber, 1999). Second, health is a

multidimensional concept with three distinct dimensions (Liang, 1986): (a) a *functional* dimension (e.g., unimpaired participation in social activities or in carrying out household tasks), (b) a *subjective* dimension (e.g., satisfaction with one's own health), and (c) a *physical* dimension (e.g., presence of diagnosed diseases, number of doctor visits in a certain time period). Many previous studies in cognitive epidemiology have focused on the physical health dimension (*Intelligence*, 2009). Hence, considerably less is known about the effects of intelligence on the functional and subjective health dimensions. This issue is of particular importance as childhood intelligence and subsequent educational and socioeconomic outcomes may be differentially related to different aspects of adult health (Johnson et al., 2011).

2. Method

2.1 Participants and procedure

Participants were individuals enrolled in the MAGRIP study, a longitudinal study initiated in 1968 by the Institut Pédagogique in Walferdange, Luxembourg (Bamberg, Dickes, & Schaber, 1977). MAGRIP is an acronym for the French expression "matière grise perdue" [lost grey matter]. The MAGRIP study was designed to investigate the determinants of children's school careers in Luxembourg. In the 1960s, primary education in Luxembourg lasted 6 years, and secondary education 7 years at the longest. The average number of years of schooling was 10 years. Eighty-three percent of the total student population completed primary education, and 47% enrolled in secondary education at that time (UNESCO Institute for Statistics, 2013). In the first wave of the MAGRIP study in 1968, detailed information was collected by trained test administrators on a randomly selected nationally representative sample comprising 2,824 students who were 12 years old and/or at the end of their primary education at the time of testing (M = 11.9 years; SD = 7.2 months; 50.1% male). Eighty-four

percent of the study participants were in Grade 6 of primary school, 11% in Grade 5, and 5% in Grade 4. Every Luxembourgish school participated in the data collection. Students provided detailed information about their intellectual abilities, educational careers, and family backgrounds.

To collect data for the second wave of measurement, the current addresses of the original participants were identified using the database of the social security agency of Luxembourg (permission was granted by the national data protection agency). The addresses of 2,377 (84%) of the 2,824 former participants could be identified. A representative stratified random sample of 1,632 former participants was contacted and invited to participate in the second wave. Stratification criteria consisted of region of residence within Luxembourg in 1968 and gender. No financial incentive was offered.

The second wave of measurement had two phases: (a) a household study (lasting from November 2008 to February 2009) and (b) a cognitive testing session (March 2009 to August 2009). For the household study, trained interviewers visited the participants at home and conducted a structured interview about the participants' educational and occupational career paths as well as key socioeconomic variables (e.g., socioeconomic and marital status). After the interview, participants completed a comprehensive questionnaire to assess their health. Altogether, 745 participants took part in the household study (M = 51.7 years, SD = 0.6 years; 46.7% male). This is also the sample used in the present study. Analyses for selection bias showed that this sample was fairly representative of the original sample. Relative to the total 1968 sample, follow-up participants had slightly higher mean childhood intelligence (Cohen's d = 0.20) and SES (d = 0.08). About half of the 745 participants (N = 378) also took part in the cognitive testing. Here, participants provided additional information on their socioeconomic status used in this study. Further, they completed the same intelligence test that had been administered in 1968. About two thirds of the participants took this test in a

group setting; the remaining participants were once more visited at home to take the test individually. These intelligence data were used in the auxiliary analyses that included adult intelligence (see online supplementary materials, Section S2, and Figure 3 in Appendix A).

2.2 Measures

More detailed descriptions of the measures of childhood intelligence, childhood SES, and adult health, including their psychometric properties, are provided in the online supplementary materials (Section S1).

*2.2.1 Childhood intelligence***.** In 1968, participants were administered the Leistungsprüfsystem (L-P-S, [Performance Test System]; Horn, 1962, 1983) in classroom sessions. The L-P-S is a standardized, objective, and comprehensive German intelligence test. Its 14 subtests provide measures of general intelligence and more specific intellectual abilities. Each subtest contains 40 items that have to be completed within strict time constraints as specified in the test manual. For the purpose of the present study, performance on seven of the L-P-S subtests was summarized in terms of three scale scores: verbal ability (3 subtests; $\alpha = .73$), reasoning ability (2 subtests; $\alpha = .75$), and visual-spatial ability (2 subtests; $\alpha = .58$). These three scale scores were applied to measure a latent factor that captured general intelligence in childhood.

2.2.2 Childhood socioeconomic status (SES). Participants' childhood SES was measured by three indicators. First, in 1968, participants reported their parents' current occupations. These occupations were later mapped onto the widely used International Socio-Economic Index of occupational status (ISEI; Ganzeboom & Treiman, 1996). The ISEI scale takes into account the income and educational levels of occupations. It has interval scale properties and a theoretical range from 16 (e.g., cleaners, unskilled agricultural laborers) to 90 (e.g., judges). In the present study, we used the highest ISEI value in a family (usually the father's ISEI value) as a first indicator of childhood SES. Interrater reliability of this ISEI

coding was tested for two independent groups of raters and was satisfactory (.72).

In the household study in 2008, participants provided further information about their childhood SES. Specifically, they reported the occupation for which their father had been trained. These data were again mapped onto the ISEI scale and used as a second indicator of childhood SES. As a third indicator of childhood SES, participants reported their fathers' highest academic qualification, which was mapped onto the International Standard Classification of Education (ISCED) scale (UNESCO, 1997). ISCED scores range from 0 (preprimary education) to 6 (second stage of a tertiary education), with higher values indicating a higher educational level. Childhood SES was specified as a latent factor defined by these three indicators.

2.2.3 Educational attainment. Participants' educational attainment was measured by three indicators. In the household study, participants reported (a) the number of years they spent in secondary education, (b) the highest school they attended, and (c) the highest academic degree they obtained. The latter two variables were mapped onto the ISCED scale. Educational attainment was specified as a latent factor defined by these three indicators.

2.2.4 Adult socioeconomic status (SES). Participants' socioeconomic status in adulthood was captured as a latent factor that was defined by three indicators. In the household study, all 745 participants reported (a) their net monthly household income and (b) their current occupation, which was mapped onto the ISEI scale. Note that occupational data may be prone to measurement error (e.g., participants may be vague about defining their current occupation; or there may be disagreement between different raters about how to map an occupation onto the ISEI scale). We therefore also used (c) the information on participants' current occupations (in terms of the ISEI metric) as reported during cognitive testing. Although this last piece of information was available for only about half of the participants, using this indicator significantly improved the assessment of adult SES as a

latent factor.

2.2.5 Adult health. Participants' health status in adulthood was measured by well-validated and reliable indicators that have been applied in previous health research.

Functional health was measured by an instrument developed by Hultsch, Hertzog, Small, and Dixon (1999). Participants answered the following question: "Looking back over the past two years, how much did your health status interfere with the following activities?" Participants used a 5-point rating scale to indicate the extent of their functional impairment in nine everyday activities: (1) work activities, (2) household tasks (e.g., cleaning, making repairs), (3) mobility (e.g., running errands), (4) maintaining relations (e.g., family reunions, meeting friends), (5) travel (e.g., holidays, excursions), (6) intellectual activities (e.g., playing chess, crossword puzzles), (7) sports activities (e.g., riding a bike, hiking), (8) leisure activities (e.g., going to the movies, going to the theater), and (9) other hobbies (e.g., do it yourself, painting). We reverse-scored the nine items so that higher scores would reflect higher levels of functional health. The reliability of this nine-item scale was $\alpha = .93$.
For the purpose of the present paper, we computed three parcel scores (i.e., sum scores of subsets of items) to measure a latent factor representing functional health. Parcel scores have several psychometric advantages over individual item scores, including higher reliability and a smaller likelihood of distributional violations. First, we conducted preliminary confirmatory factor analyses to establish unidimensionality (i.e., that all nine indicators of functional health loaded on a common factor; cf. Little, Cunningham, Shahar, & Widaman, 2002). We then combined items that showed marked residual correlations into parcels (Hall, Snell, & Singer Foust, 1999). Specifically, items (1) to (3), items (4) to (6), and items (7) to (9) were combined into parcel scores, respectively. The residual term of each parcel score accounted for the specificity of the corresponding items.

Subjective health was specified as a latent factor that was measured by three

indicators. These indicators were taken from previous health research (Hultsch et al., 1999; Liang, 1986) and large-scale surveys such as the ALLBUS survey (Terwey, 2000), which is similar to the American General Social Survey (Davis, Mohler, & Smith, 1994). Participants used a 5-point rating scale to evaluate (a) their overall health status and (b) their health status relative to peers. Furthermore, participants used a 7-point rating scale to report (c) their satisfaction with their health status. The reliability of this three-item scale was $\alpha = .85$.

Physical health was specified as a latent factor that was measured by three indicators taken from previous studies (Hultsch et al., 1999; Liang, 1986) and large-scale surveys such as the German ALLBUS survey (Terwey, 2000). Participants reported: (a) the number of visits to a doctor in the last 3 months, (b) the number of sick-leave days in the last 12 months, and (c) the number of nights spent in the hospital in the last 12 months. For the present analyses, we reverse-scored these measures so that higher scores would reflect better physical health. The reliability of this three-item scale was $\alpha = .68$.

Table S1 in the online supplementary materials contains the intercorrelations and standard deviations of all study variables. Table S2 in the online supplementary materials additionally shows the frequency distributions for the item categories of all indicators of the three health dimensions (i.e., functional, subjective, and physical health) at age 52.

2.3 Statistical analysis

We used an SEM approach implemented in the software Mplus version 6 (Muthén & Muthén, 1998–2010). We ran three separate models, one for each health dimension (i.e., functional, subjective, and physical health; see Figure 2, Models 2a – 2c). In general, the proportion of missing data in the manifest indicators used in the SEM was low (6% on average). One exception was the assessment of participants' current occupation from the cognitive testing session that was used as one indicator of adult SES (54% missing data). However, the other two indicators of adult SES had far lower proportions of missing data.

Further, to account for missing data in all study variables, we employed full-information maximum likelihood estimation (FIML). FIML methods are considered to be among the best available methods for handling missing data (Jelicic, Phelps, & Lerner, 2009).

To assess the fit of our hypothesized models, we computed the χ^2 goodness-of-fit statistic and consulted three widely used descriptive fit indices with thresholds for judging model fit according to Hu and Bentler (1999). Specifically, we consulted the Comparative Fit Index (CFI; values above .95 indicate a good model fit), the Root Mean Square Error of Approximation (RMSEA; values below .05 indicate a good model fit), and the Standardized Root Mean Square Residual (SRMR; values below .08 indicate a good model fit).

In mediation analyses using SEM, the total effect of a latent predictor variable on a latent criterion variable can be decomposed into a direct and one or more specific indirect (i.e., mediated) effects. One specific indirect effect reflects one specific pathway by which the influence of the predictor is transmitted to the criterion via one or more mediators. The sum of all specific indirect effects of the predictor on the criterion equals the predictor's total indirect effect (i.e., the total mediation effect). The sum of the direct and total indirect effects equals the predictor's total effect. To assess the significance of the direct, specific indirect, total indirect, and total effects of childhood intelligence on adult health, we employed bias-corrected bootstrap confidence intervals for the model parameter estimates (MacKinnon, Lockwood, & Williams, 2004). This bootstrap method is considered to be one of the best approaches for accounting for the expected nonnormality of the sampling distribution of the mediation effect (Shrout & Bolger, 2002). Note that bias-corrected bootstrap confidence intervals are not necessarily symmetric around the parameter estimate (Muthén & Muthén, 1998–2010). To obtain an additional effect size measure of the total indirect effect of intelligence on health, we computed the proportion of the total effect of intelligence that was mediated by educational attainment and adult SES (percent mediated). Specifically, we

divided the total indirect effect by the total effect (cf. MacKinnon et al., 2001).

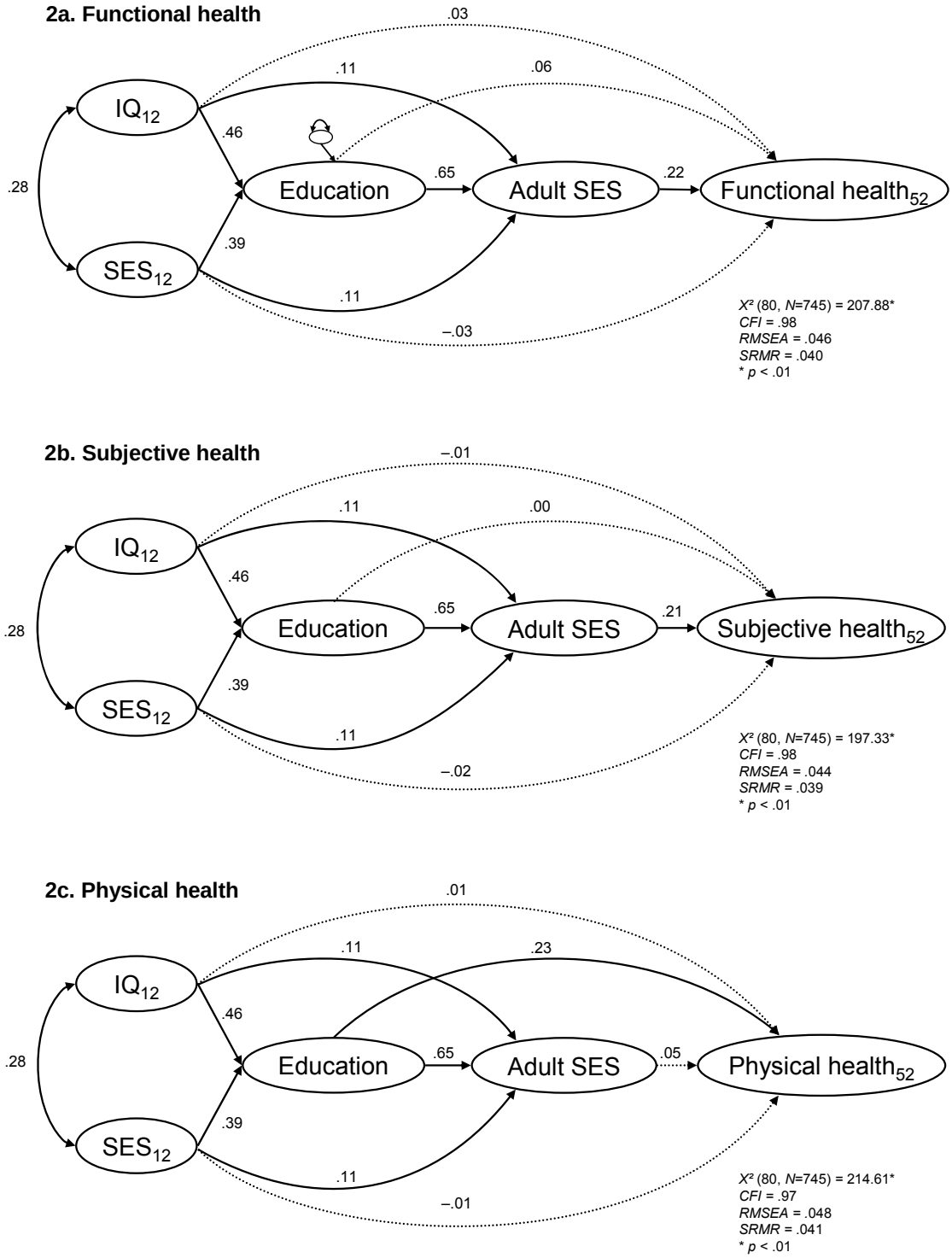
To explore potential gender differences in the direct and indirect effects of childhood intelligence on adult health, we employed multiple-group models. Multiple-group models allow the user to test whether SEM parameters (e.g., estimates of direct effects; Muthén & Muthén, 1998–2010; Vandenberg & Lance, 2000) differ significantly in magnitude across groups (e.g., between genders). See Section S3 in the online supplementary materials for a full description of the methods and results of these multiple-group models.

3. Results

Our analyses showed that the three models provided reliable parameter estimates for analyzing the mediation processes linking childhood intelligence and adult health via educational attainment and adult SES. Specifically, the model fit was good for all three models because, although the χ^2 values were significant, all CFI values were larger than .95, and all RMSEA and SRMR values were smaller than .05 and .08, respectively. Further, all latent variables were well-defined, as indicated by the substantial factor loadings (see Table S3 in the online supplementary materials). The results are shown in Figure 2 and Table 1. Figure 2 contains the standardized coefficients for each direct path in the three models that investigated the direct and indirect effects of intelligence at age 12 on functional health (Figure 2a), subjective health (Figure 2b), and physical health (Figure 2c) at age 52. Table 1 shows the standardized direct, specific indirect, total indirect, and total effects of intelligence at age 12 on the three health dimensions at age 52.

As predicted, we found that higher intelligence at age 12 was significantly associated with (a) higher educational attainment and (b) better SES in adulthood (see Figure 2). Notably, these estimates represent the positive effects of childhood intelligence on socioeconomic outcomes later in life while adjusting for differences in parental SES at age 12.

Figure III-2. *Structural equation models for determining how the effects of intelligence at age 12 on functional health (Figure 2a), subjective health (Figure 2b), and physical health (Figure 2c) at age 52 are mediated via educational attainment and adult SES*



Note. Figure 2 shows standardized coefficients for each direct path. Dashed arrows indicate nonsignificant paths ($p > .05$, two-sided testing). Latent factors are depicted as ellipses. For clarity of presentation, manifest indicators and variances of latent factors have been omitted. Double-headed arrows represent correlations. Goodness-of-fit indices for each model are included. Key: IQ = Intelligence; SES = Socioeconomic Status; Education = Educational Attainment.

Crucially, our results confirmed the prediction that a substantial part of the relations between intelligence at age 12 and health at age 52 would be mediated via educational attainment and SES. Before estimating a full mediation model, we estimated the bivariate regressions of the three health dimensions on childhood intelligence, excluding educational attainment and adult SES from the model (see Figure S1 in the online supplementary materials). Childhood intelligence significantly predicted adult functional health (β = .18), subjective health (β = .11), and physical health (β = .17). These positive effects seemed to be entirely explained by the indirect effects of intelligence on the three health dimensions. Specifically, the direct effects of intelligence on health were close to zero and failed to reach significance in the full mediation model (see Table 1, row "Direct"). By contrast, intelligence showed small yet significant total indirect effects on adult functional health (β = .12), subjective health (β = .09), and physical health (β = .12). Moreover, the total indirect effects of intelligence on health were nearly as large as the total effects of intelligence (see rows "Total indirect" and "Total"). Thus, the indirect effects constituted by far the largest portion of the total effects of intelligence on health. Finally, the percent mediated (i.e., the proportion of the total effect of intelligence on health that was mediated via educational attainment and adult SES) was large for all three health outcomes: 78% for functional health, 91% for subjective health, and 95% for physical health.

Table III-1. *Standardized direct, specific indirect, total indirect, and total effects of intelligence at age 12 on functional, subjective, and physical health at age 52*

	Functional health (Model 2a)			Subjective health (Model 2b)			Physical health (Model 2c)		
Effect of intelligence at age 12 on health at age 52	β	95% CI	% mediated	β	95% CI	% mediated	β	95% CI	% mediated
Direct	0.03	–0.09, 0.16		–0.01	–0.13, 0.11		0.01	–0.13, 0.14	
Specific indirect 1 (via education)	0.03	–0.05, 0.10		0.00	–0.07, 0.15		0.11	0.02, 0.19	
Specific indirect 2 (via adult SES)	0.03	–0.01, 0.06		0.02	–0.01, 0.06		0.01	–0.02, 0.03	
Specific indirect 3 (via education and adult SES)	0.07	0.01, 0.12		0.06	0.01, 0.12		0.01	–0.04, 0.07	
Total indirect (sum of specific indirect effects 1-3)	0.12	0.06, 0.18		0.09	0.03, 0.15		0.12	0.06, 0.18	
Total (sum of direct and total indirect effects)	0.15	0.06, 0.25	77.92	0.08[a]	–0.01, 0.17	90.91	0.13	0.02, 0.24	94.62

Note. Effect estimates are from the completely standardized solution of the corresponding model (i.e., Models 2a, 2b, and 2c). The percent mediated is calculated by dividing the total indirect effect by the total effect (cf. MacKinnon et al., 2001) but may differ slightly from the quotient total indirect effect/total effect in Table 1 due to rounding errors.

[a] Because the direct and total indirect effects were in opposite directions, the mediation of the total effect was based on the absolute value of the sum of the direct and total indirect effects (i.e., 0.10 in this case; cf. Judge, Ilies, & Dimotakis, 2010).

To further investigate the specific mediational pathways by which childhood intelligence influences adult health, we examined the specific indirect effects of childhood intelligence on the three adult health dimensions. The results showed that the mediation did not work in the same way for all three health dimensions. Adult functional and subjective health showed similar mediation patterns. Specifically, the positive effects of childhood intelligence on adult functional and subjective health were mediated via the effect of intelligence on educational attainment and the effect of educational attainment on subsequent SES as indicated by the significant specific indirect effects of childhood intelligence on adult functional health ($\beta = .07$) and subjective health ($\beta = .06$) via both mediators (see Table 1, row "Specific indirect 3"). The physical health dimension showed a different pattern of mediation. Specifically, the positive effect of childhood intelligence on adult physical health was mediated via the effect of intelligence on educational attainment only, not via the effect of educational attainment on subsequent SES. We found a significant specific indirect effect of childhood intelligence on physical health via educational attainment ($\beta = .11$; see Table 1, row "Specific indirect 1"), whereas the specific indirect effect of childhood intelligence on physical health via educational attainment and adult SES was close to zero.

Auxiliary analyses showed that these findings held up when considering adult intelligence and gender differences (see Sections S2 and S3 in the online supplementary materials, respectively). The positive effects of childhood intelligence on adult health were entirely mediated via educational attainment and adult SES in both women and men. The inclusion of adult intelligence as an additional mediator between childhood intelligence and adult health did not alter this pattern of results. Figure 3 in Appendix A shows the results of the analyses including adult intelligence.

4. Discussion

The main goal of this prospective cohort study was to examine whether and the extent to which educational attainment and adult SES mediate the impact of childhood intelligence on adult functional, subjective, and physical health. A major contribution of the present study is the investigation of the longitudinal relations between intelligence and health in a country that offers universal access to quality health care, as quality health care may compensate for the differential effects of intelligence on health. A further contribution is the detailed investigation of potential differences in the specific mediational pathways that link childhood intelligence and different dimensions of adult health.

4.1 Direct and indirect effects of childhood intelligence on adult health

Our main result was that childhood intelligence had significant positive effects on all three dimensions of adult health 40 years later and that these positive effects were entirely mediated via educational attainment and adult SES. Specifically, childhood intelligence had significant positive indirect effects on social-functional health (i.e., fewer limitations in everyday activities due to health problems), subjective health (i.e., a better subjective evaluation of one's own health status), and physical health (i.e., lower numbers of doctor visits, sick-leave days, and nights in the hospital). The direct effects of childhood intelligence on these three health dimensions were reduced to near zero in the full mediation models. In general, these mediational processes operated in similar ways in women and men, and the inclusion of adult intelligence for a subgroup of our study population did not alter these findings. These results support the main cognitive epidemiological findings of positive associations between higher childhood intelligence and lower adult morbidity risk (Deary, 2012; Deary et al., 2010).

Importantly, our results were obtained after adjusting for participants' socioeconomic family backgrounds and in a country with universal access to quality health care. Thus, our

results support the generalizability of a "chain reaction" model (Gottfredson, 2002 p. 369). The protective effects of childhood intelligence on adult health accumulate across the life span. Early advantages in intelligence translate into a more successful educational career and subsequently into higher socioeconomic status in adulthood. These socioeconomic outcomes are in turn related to better adult health (Deary, 2010). Even high-quality public health care cannot fully offset the impact of these socioeconomic life outcomes on adult health (Lleras-Muney, 2005).

Our finding of near zero direct effects of intelligence on health does not seem to be consistent with results from studies that have found direct effects of intelligence even after controlling for subsequent socioeconomic life outcomes (Batty et al., 2007b; Batty, Gale, et al., 2008; Johnson et al., 2011). This may be due to various reasons. First, to alleviate the problem of measurement error (Deary & Johnson, 2010; Deary et al., 2010), we specified all variables as latent factors that were defined by several indicators. This may have contributed to a more precise estimation of the direct and indirect effects. Compared to using manifest variables, the direct effects of intelligence may thus have been attenuated to a greater extent. Second, we exclusively employed self-reported health measures in our study. Thus, it is possible that with different health measures (e.g., objective health indicators such as the metabolic syndrome; Batty, Gale, et al. 2008), we would indeed have found small direct effects of childhood intelligence.

4.2 Potential differences in the indirect effects of childhood intelligence on different adult health dimensions

Next, we examined the specific mediational pathways that link childhood intelligence to the three adult health dimensions. Childhood intelligence influenced adult functional and subjective health mainly through the positive effect of childhood intelligence on educational attainment and the positive effect of educational attainment on subsequent SES. By contrast,

childhood intelligence influenced adult physical health mainly through the positive effect of educational attainment, without additional positive effects of subsequent SES. Below, we interpret these differences for each health outcome and discuss potential microprocesses that may account for them.

4.2.1 Functional health. The finding that the positive effects of higher childhood intelligence on functional health were transmitted via higher adult SES may be due to the higher incomes associated with higher SES occupations. Participants with higher incomes may have more resources at their disposal to prevent or to overcome potential functional limitations due to health problems. For instance, a high income may provide the resources to switch between leisure activities in a flexible manner or to use different means of transportation to maintain social relations and mobility. In addition, higher SES occupations may provide working environments that help preserve good functional health. By contrast, lower SES occupations may be associated with more strenuous and monotonous working environments, which may go along with an increased risk of functional impairment in everyday life. For instance, several studies found that a greater proportion of workers involved in monotonous and strenuous occupations needed to rest after work and before leisure or other activities compared to workers in less strenuous occupations (Gardell, 1982).

4.2.2 Subjective health. The finding that the significant positive effects of higher childhood intelligence on subjective health were transmitted via higher adult SES may in part be due to the same aspects of high adult SES that could account for the positive effects on functional health. Specifically, just as the higher income associated with high SES may help a person to prevent and cope with functional limitations, it may also lead to a more positive subjective evaluation of one's health status. The less strenuous, less monotonous, and less functionally limiting working environments of high SES occupations may also contribute to higher subjective health. Finally, several additional advantages associated with high SES

occupations may positively influence subjective health. For instance, Near, Rice, and Hunt (1978) found that compared to blue collar occupations, white collar occupations were associated with higher job satisfaction and higher occupational prestige. These factors were found to predict a more positive subjective evaluation of a participant's health status.

4.2.3 Physical health. The finding that the significant positive effects of higher childhood intelligence on physical health seemed to be entirely transmitted through educational attainment and not adult SES may point to potential effects of the Luxembourgish health care system. As universal access to quality health care is provided for every citizen, the costs for the preservation and restoration of physical health (doctor visits, treatments, etc.) are borne by society. As a consequence, having a low income poses no hurdle for access to quality health care and hence may not substantially contribute to individual differences in physical health. Thus, individual differences in educational attainment may contribute to a greater extent to individual differences in physical health in different ways. First, education helps to prevent actual physical diseases (e.g., via health-related knowledge and behavior; Cutler & Lleras-Muney, 2006). Second, in the case of a manifested disease, individuals with more education may know when it is necessary to consult a doctor, leading to a more efficient use of the health care system. Further, their education may provide better verbal and communicative skills, enabling them to communicate symptoms more efficiently, understand medical advice, and correctly follow prescriptions (Cutler & Lleras-Muney, 2006; Johnson et al., 2011). Third, education may also improve more general skills, such as critical thinking skills, problem solving skills, and efficient learning. These skills have been shown to positively influence health (Cutler & Lleras-Muney, 2006), and are in turn closely related to early life intelligence (Gottfredson, 2004). Thus, higher childhood intelligence that translates into a higher educational attainment may ultimately help a person to manage the "job of being a patient" (Gottfredson, 2004, p.

175).

4.3 Strengths and limitations

This study has several limitations that can be used to derive recommendations for future studies on the direct and indirect effects of early intelligence on later health. First, the results of our and other studies suggest that educational attainment and adult SES are important mediators of the intelligence-health relation (Deary, 2010; Deary et al., 2010). However, the present study was not designed to address the interplay of education and SES with other microprocess variables, such as health-preventive and health-compromising behaviors (e.g., attending preventive doctor appointments, physical activity, smoking, and alcohol intake), measures of social integration (social support or influences on health behavior), and risk factors (e.g., blood pressure, obesity). An inclusion of these variables could help researchers to gain a better understanding of how intelligence translates into health. Second, participants self-reported their health. Self-report measures may be subject to reporting biases. However, empirical research has confirmed that self-reported health ratings are reliable and valid measures of health (Hultsch et al., 1999; Liang, 1986). Nevertheless, for a more comprehensive assessment of health, additional health indicators should be included, such as diagnosed diseases and measures of mental health. Third, childhood intelligence was assessed at the average age of 12 years. This age guaranteed a comparable educational starting point for all participants. However, at the age of 12, reciprocal effects between intelligence and education are still possible. That is, some variability in the intelligence scores obtained at the age of 12 may be due to the differential effects of education on intellectual development up to this time point. Thus, the effects of educational attainment on health found in our study may be lower bound estimates. Therefore, it would be preferable to measure intelligence early on in order to minimize differential effects of education on intellectual development. Moreover, intelligence should best be measured at several time

points across the (early) life course. Several measurements would allow for a more detailed investigation of the interplay between intelligence and environmental influences such as education and for a detailed investigation of the reciprocal influences between intelligence and health. Altogether, cross-lagged designs seem preferable for investigating the "chain reaction" between intelligence and health. Fourth, even though our sample size was comparatively large and should have been sufficient for detecting even small effects (Cohen, 1988), the confidence intervals for the nonsignificant direct effects of intelligence on health were relatively wide. Thus, the possibility of small direct positive effects of childhood intelligence on adult health in the population cannot be ruled out completely. Therefore, large sample sizes seem advisable for obtaining precise estimates of the direct and indirect effects of intelligence on health.

Despite its limitations, this study features several strengths. First, the present study used a prospective longitudinal cohort design spanning 40 years, thus adding to the small number of studies that have investigated the longitudinal relations between childhood intelligence, subsequent socioeconomic life outcomes, and health. Second, the present study investigated a nationally representative sample and was thus the first to investigate the key assumptions of cognitive epidemiology in a Central European country with universal access to quality health care. Third, the present study simultaneously investigated three dimensions of adult health, which was shown to be important in light of the fact that the specific mediational pathways linking intelligence and adult health were found to differ between the different health dimensions. Fourth, childhood intelligence was assessed several decades before the assessment of health, thus ruling out the possibility of reverse causality (i.e., a detrimental effect of deteriorating health on intelligence test performance; Deary et al., 2010).

4.4 Conclusion

In conclusion, our study showed that educational attainment and subsequent SES are central in explaining the relation between childhood intelligence and adult health. However, it remains a challenging task for future studies to examine the microprocesses involved in how intelligence, education, and SES translate into better adult health and the relative importance and interplay of different mediating processes.

Our results also have potential policy implications. The cumulative influence of intelligence on such important life outcomes as education, socioeconomic success, and adult health suggests that societal investments in maternal nutrition and early child rearing, a reduction in risk factors for childhood intellectual and physical development, and other early human capital investments may provide manifold benefits to their recipients and, ultimately, to society (Heckman, 2006; Judge, Ilies, & Dimotakis, 2010). The benefits of intelligence are not limited to economic factors, and investments in intelligence may produce economic, health, and social benefits.

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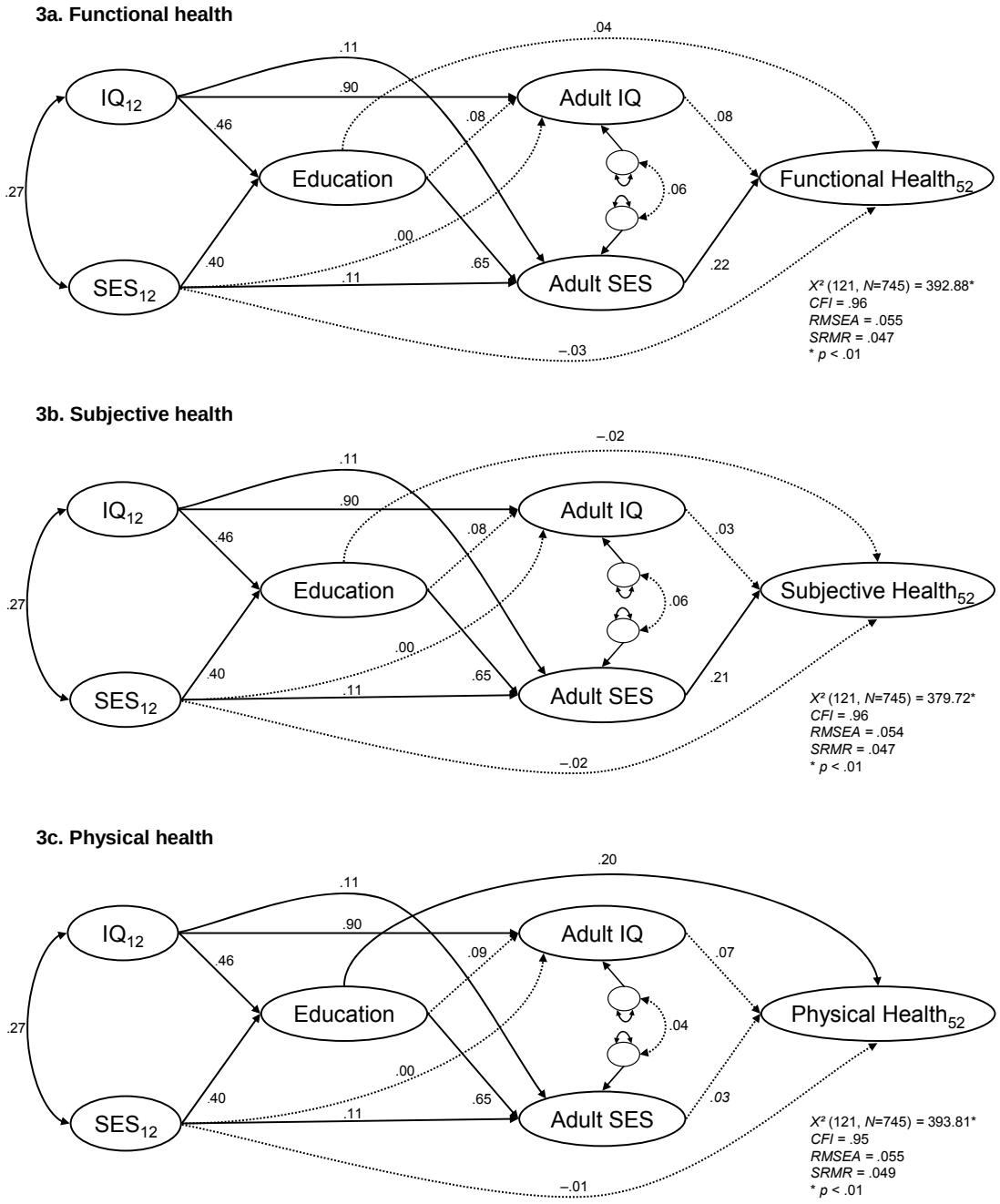
Appendix A: Results of auxiliary analyses including adult intelligence as additional mediator

Figure III-3. *Auxiliary analyses to investigate potential effects of adult intelligence on the direct and indirect effects of intelligence at age 12 on three dimensions of health at age 52*



Note. Figure 3 shows standardized coefficients for each direct path. Dashed arrows indicate nonsignificant paths ($p > .05$, two-sided testing). Latent factors are depicted as ellipses. For clarity of presentation, manifest indicators and irrelevant variances of latent factors have been omitted. Double-headed arrows represent correlations. Goodness-of-fit indices for each model are included. IQ = Intelligence; SES = Socioeconomic Status; Education = Educational Attainment.

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Online Supplementary Materials

1. Section S1: Comprehensive description of the measures of childhood intelligence, childhood SES, and adult health

Childhood Intelligence

Childhood intelligence was assessed by the Leistungsprüfsystem (L-P-S [Performance Test System]; Horn, 1962, 1983). The L-P-S is a standardized, objective, and comprehensive German intelligence test based on the model of primary mental abilities formulated by Thurstone (1938). Its 14 subtests provide measures of general intelligence and more specific intellectual abilities, such as verbal abilities, reasoning abilities, figural-spatial abilities, and perceptual speed. Each subtest contains 40 items that have to be completed within strict time constraints as specified in the test manual. For the purpose of the present study, performance on seven of the L-P-S subtests was summarized in terms of three scale scores: verbal ability, reasoning ability, and visual-spatial ability. The scale score for verbal ability was based on three subtests. Two subtests consist of misspelled six-letter words; participants have to identify the appropriate words as well as the spelling errors. The other subtest consists of anagrams (Borkenau & Liebler, 1993). The scale score for reasoning ability was based on two subtests inspired by Raven's Progressive Matrices (Horn, 1983). For both subtests, participants have to identify the inappropriate element in a series of eight elements, the elements of the first subtest being geometric figures and those of the second subtest being letters and digits (Borkenau & Liebler, 1993). The scale score for visual-spatial ability was based on two subtests. The first subtest is a mental figure folding task. Participants have to fold figures in relation to marker points on the surfaces of both the unfolded and folder figures. The second subtest is a task that taps the ability to grasp spatial relations. Participants have to identify the number of all hidden and unhidden surfaces of an object (Schalke et al.,

2012).

Split-half reliability of the overall test is .99, parallel-forms reliability is .94. Retest reliability across a time span of 32 months is .83 for the overall test score (Horn, 1983; Tent, 1969). There is ample evidence for the construct validity of the L-P-S. Specifically, the correlation of the L-P-S total score with the total score on the German version of the Wechsler Adult Intelligence Scale (WAIS)—the Hamburg Wechsler Intelligenztest für Erwachsene (HAWIE-R; Tewes, 1991)—is .94 (Sturm & Büssing, 1982). Furthermore, the correlation of the standardized L-P-S total score with the standardized total score of the Intelligenz-Struktur-Test (IST; Liepmann, Beauducel, Brocke, & Amthauer, 2001) is .72. The IST is another well-validated and widely used German intelligence test that also correlates substantially with the HAWIE-R (Tewes, 1991). In a recent meta-analysis, Hülsheger, Maier, Stumpp, and Muck (2006) compared the predictive validity of the L-P-S and five other intelligence tests widely used in German-speaking countries, including the IST and Raven's Progressive Matrices (Kratzmeier, 1979), for the outcomes of vocational education. The authors found the L-P-S to be one of the instruments with the highest criterion-related validity. Further, the total and subtest scores of the L-P-S showed high correlations with grades in various school subjects (Horn, 1983). For instance, the total score showed a correlation of .55 with grade point average in Grade 4 of elementary school (Tent, 1965). Given the strong empirical evidence for its reliability and validity, the L-P-S is widely employed in various areas of current research, such as research on gender differences in cognitive functions (Weiss, Kemmler, Deisenhammer, Fleischhacker, & Delazer, 2003) or clinical and neuropsychology (Kuelz, Hohagen, & Voderholzer, 2004).

Childhood Socioeconomic Status (SES)

Participants' childhood SES was measured by three indicators. First, in 1968, participants reported their parents' current occupation. These occupations were later mapped onto the categories of the International Standard Classification of Occupations (ISCO-88; Elias, 1997). Ganzeboom and Treiman (1996) showed that the ISCO-88 scheme also applies to occupational data from 1968. These classifications were then transformed into the widely used International Socio-Economic Index of occupational status (ISEI; Ganzeboom, de Graaf, Treiman, & de Leeuw, 1992; Ganzeboom & Treiman, 1996). The ISEI scale takes into account the income and educational levels of occupations . It has interval scale properties and a theoretical range from 16 (e.g., cleaners, unskilled agricultural laborers) to 90 (e.g., judges). With its grounding in international occupational classification schemes, the ISEI scale is internationally comparable; it has been demonstrated to be a reliable and valid indicator of socioeconomic status in many international large-scale assessments (e.g., PISA; Organisation for Economic Co-operation and Development, 2004). In the present study, we used the highest ISEI value in a family (usually the father's ISEI value) as a first indicator of childhood SES. Interrater reliability of this ISEI coding was tested for two independent groups of raters and was satisfactory (.72).

In 2008, participants provided further information about their childhood SES. Specifically, they reported the occupation for which their father had been trained. These data were again mapped onto the ISEI scale and used as a second indicator of childhood SES. As a third indicator of childhood SES, participants reported their fathers' highest academic qualification, which was mapped onto the International Standard Classification of Education (ISCED) scale (UNESCO, 1997). ISCED scores range from 0 (preprimary education) to 6 (second stage of a tertiary education), with higher values indicating a higher educational level.

Adult Health

The conceptualization and measurement of adult health in the present study was based on the multidimensional model of health developed by Liang and colleagues (1986, 1991). In this model, health has a (social-)functional dimension, a subjective dimension, and a physical dimension. Liang (1986) based his model on the World Health Organization's definition of health as "[...] a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity" (World Health Organization, 1958, p. 469). This multidimensional approach to the measurement of health has been exceedingly influential in the social sciences. Liang's (1986) paper is, for instance, among the 20 most frequently cited social science articles ever published in the *Journal of Gerontology* (Ferraro & Schafer, 2008).

Functional health. The functional health measure was adapted from an influential study by Hultsch, Hertzog, Small, and Dixon (1999). Specifically, participants used a 5-point rating scale (1 = *not at all*, 2 = *hardly*, 3 = *moderately*, 4 = *significantly*, 5 = *strongly, gave up the activity*) to answer the question "Looking back over the past two years, how much did your health status interfere with the following activities?" Nine activities suitable for indicating the functional health of adults aged 52 years were examined: (1) work activities, (2) household tasks (e.g., cleaning, doing laundry, making repairs), (3) mobility (e.g., running errands, visits to the authorities), (4) maintaining relations (e.g., family reunions, meeting friends), (5) travel (e.g., holidays, excursions), (6) intellectual activities (e.g., playing chess, crossword puzzles), (7) sports activities (e.g., riding a bike, hiking), (8) leisure activities (e.g., going to the movies, to the theater), and (9) other hobbies (e.g., do it yourself, painting). The reliability of this nine-item scale was $\alpha = .93$.

Our measure of functional health has certain similarities with other established scales, such as the SF-36 (Ware & Gandek, 1998). Specifically, the SF-36 physical functioning scale

assesses health-related limitations in physical activities such as lifting heavy objects, sports, climbing stairs, kneeling, and walking various distances. Furthermore, it includes two questions assessing health-related limitations in social activities and one question concerning limitations in work activities. These aspects were also covered in the present study. Specifically, we assessed limitations in sports and mobility in a manner similar to the SF-36 physical functioning scale, limitations in the ability to maintain relations in a manner similar to the SF-36 social functioning scale, and limitations in work activities in a manner similar to the SF-36 question on work activities. However, the present study also went beyond the aspects that were similar to the SF-36 scales by assessing further aspects of functional health, such as traveling, intellectual activities, specific leisure activities, and hobbies.

Subjective health. Participants used a 5-point rating scale (1 = *very poor*, 2 = *poor*, 3= *satisfactory*, 4 = *good*, 5 = *very good*) to evaluate (a) their overall health status and (b) their health status relative to peers. These two questions were taken from previous health research (Hultsch et al., 1999; Liang, 1986) and large-scale surveys such as the German ALLBUS survey (Haarmann, Scholz, Wasmer, Blohm, & Harkness, 2006; Terwey, 2000). The ALLBUS survey is similar in theoretical scope and methodology to the American General Social Survey (Davis, Mohler, & Smith, 1994). Further, participants used a 7-point rating scale (1 = *very dissatisfied*, 2 = *dissatisfied*, 3 = *somewhat dissatisfied*, 4 = *neutral*, 5 = *somewhat satisfied*, 6 = *satisfied*, 7 = *very satisfied*) to report (c) their satisfaction with their health status. This question has been widely used in several international representative surveys (e.g., the European Values Study; Halman, 2001). The reliability of this three-item scale was $\alpha = .85$.

Physical health. Reliable and valid indicators of the physical dimension were adapted from previous studies (Hultsch et al., 1999; Liang, 1986) and the large-scale German ALLBUS survey (Haarmann, et al., 2006; Terwey, 2000). Specifically, participants reported:

(a) the number of visits to a doctor in the last 3 months (0 = *none*, 1 = 1, 2 = 2, 3 = 3, 4 = 4, 5 = 5–6, 6 = 7–8, 7 = 9–10, 8 = 11–15, 9 = > 15), (b) the number of sick-leave days in the last 12 months (0 = *none*, 1 = 1–3, 2 = 4–7, 3 = 8–14, 4 = 15–30, 5 = 31–60, 6 = > 60), and (c) the number of nights spent in the hospital in the last 12 months (0 = *none*, 1 = 1–3, 2 = 4–7, 3 = 8–14, 4 = 15–30, 5 = 31–60, 6 = > 60). The response categories for these items were derived from data from the representative ALLBUS survey, on which these questions were presented in an open-answer format. The use of categories facilitated automated processing and scoring of responses in the present study. The reliability of this three-item scale was $\alpha = .68$.

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Table III-S1. *Standard deviations of and intercorrelations between study variables*

Indicator	*SD*	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
1 Gender[a]	0.50	–																				
2 Verbal ability[b]	3.99	**–.11**	–																			
3 Reasoning ability[b]	4.32	.04	**.41**	–																		
4 Visual–spatial ability[b]	7.06	**–.17**	**.45**	**.53**	–																	
5 Father's academic degree	3.22	.03	**.23**	**.13**	**.11**	–																
6 Father's vocational training	1.37	.01	**.20**	**.12**	.07	**.48**	–															
7 Father's occupation	1.29	.01	**.17**	**.14**	**.13**	**.50**	**.65**	–														
8 Years of education	3.59	**–.20**	**.39**	**.32**	**.29**	**.37**	**.32**	**.39**	–													
9 Highest school visited	2.90	**–.10**	**.47**	**.39**	**.32**	**.40**	**.35**	**.39**	**.78**	–												
10 Highest academic degree	3.23	**–.10**	**.37**	**.36**	**.27**	**.36**	**.27**	**.34**	**.81**	**.77**	–											
11 Household income	2.46	**–.12**	**.25**	**.24**	**.22**	**.19**	**.24**	**.25**	**.45**	**.42**	**.43**	–										
12 Current occupation (HS)	1.45	–.00	**.37**	**.28**	**.21**	**.29**	**.32**	**.31**	**.56**	**.57**	**.56**	**.41**	–									
13 Current occupation (CT)	1.45	**–.12**	**.35**	**.29**	**.23**	**.20**	**.27**	**.28**	**.53**	**.54**	**.52**	**.40**	**.70**	–								
14 Functional health 1[b]	0.98	–.08	**.12**	**.15**	**.14**	.06	.06	**.12**	**.21**	**.22**	**.20**	**.17**	**.23**	**.26**	–							
15 Functional health 2[b]	0.82	–.03	.07	**.13**	**.14**	.09	.06	**.11**	**.20**	**.20**	**.17**	**.18**	**.18**	**.21**	**.72**	–						
16 Functional health 3[b]	0.91	.01	.02	**.10**	.08	.07	.01	.07	**.16**	**.16**	**.16**	**.15**	**.15**	**.19**	**.72**	**.78**	–					
17 Judgment of overall health	0.98	.02	.01	.08	**.10**	.09	–.00	.08	**.14**	**.12**	**.13**	**.16**	**.15**	**.15**	**.56**	**.42**	**.46**	–				
18 Health compared to peers	0.98	.04	.01	.06	.07	.07	–.00	.05	**.12**	**.11**	**.11**	**.12**	**.10**	**.09**	**.48**	**.36**	**.41**	**.81**	–			
19 Satisfaction with health	1.61	.05	.01	.08	.08	**.11**	–.01	.09	**.11**	.10	**.11**	**.13**	**.14**	**.14**	**.52**	**.41**	**.44**	**.75**	**.63**	–		
20 No. of doctor visits	2.06	–.08	.06	**.16**	**.12**	**.17**	.04	.09	**.21**	**.16**	**.18**	**.15**	**.13**	**.17**	**.46**	**.35**	**.39**	**.51**	**.47**	**.51**	–	
21 No. of sick leave days	1.70	.03	.06	.09	.03	**.10**	.03	.06	**.15**	**.17**	**.17**	.09	**.13**	**.14**	**.40**	**.29**	**.34**	**.38**	**.34**	**.37**	**.52**	–
22 No. of hospital nights	0.94	.01	.06	.04	.06	.06	.03	.07	**.13**	**.14**	**.12**	.08	.08	.04	**.30**	**.21**	**.27**	**.36**	**.38**	**.31**	**.42**	**.49**

Note. Correlations in boldface are significant at $p < .01$ (two-sided testing). HS = Household Study; CT = Cognitive Testing.
[a] Gender is a dummy-coded variable (1 = male, 2 = female). [b] Parcel score.

Table III-S2. *Frequency distributions of item categories of 15 items measuring functional, subjective, and physical health at age 52*

Item	Category									
	0	1	2	3	4	5	6	7	8	9
Functional health										
1 Work activities	–	59.0	16.4	13.9	6.6	4.2	–	–	–	–
2 Household tasks	–	50.2	20.0	16.7	10.2	2.9	–	–	–	–
3 Mobility	–	65.5	16.8	11.5	4.6	1.6	–	–	–	–
4 Maintain relations	–	63.7	19.3	11.8	4.1	1.1	–	–	–	–
5 Travels	–	66.1	16.1	9.7	6.0	2.2	–	–	–	–
6 Intellectual activities	–	72.8	15.5	6.7	3.9	1.1	–	–	–	–
7 Sports activities	–	48.5	21.2	16.9	9.4	4.0	–	–	–	–
8 Leisure activities	–	64.6	19.6	10.4	3.8	1.6	–	–	–	–
9 Other hobbies	–	63.0	18.8	11.7	5.2	1.2	–	–	–	–
Subjective health										
1 Judgment of overall health	–	3.4	10.9	21.8	48.5	15.4	–	–	–	–
2 Health compared to peers	–	3.4	8.6	19.6	49.2	19.2	–	–	–	–
3 Satisfaction with health	–	3.2	3.9	6.5	12.1	16.8	27.3	30.2	–	–
Physical health										
1 No. of doctor visits	43.3	24.5	9.7	8.2	3.7	4.6	1.4	2.3	1.0	1.5
2 No. of sick-leave days	50.3	19.2	11.4	6.6	5.1	2.7	4.7	–	–	–
3 No. of hospital nights	83.9	7.2	3.8	3.0	.8	.7	.5	–	–	–

Note. Frequencies are reported in percent. Item categories were coded as follows: Functional health items 1–9: 1 = *not at all*, 2 = *hardly*, 3 = *moderately*, 4 = *significantly*, 5 = *strongly, gave up the activity*. Subjective health items 1–2: 1 = *very poor*, 2 = *poor*, 3 = *satisfactory*, 4 = *good*, 5 = *very good*. Subjective health item 3: 1 = *very dissatisfied*, 2 = *dissatisfied*, 3 = *somewhat dissatisfied*, 4 = *neutral*, 5 = *somewhat satisfied*, 6 = *satisfied*, 7 = *very satisfied*. Physical health item 1: 0 = *none*, 1 = 1, 2 = 2, 3 = 3, 4 = 4, 5 = 5–6, 6 = 7–8, 7 = 9–10, 8 = 11–15, 9 = > 15. Physical health items 2–3: 0 = *none*, 1 = 1–3, 2 = 4–7, 3 = 8–14, 4 = 15–30, 5 = 31–60, 6 = > 60.

2. Section S2: Auxiliary analyses to investigate potential effects of adult intelligence on the direct and indirect effects of childhood intelligence on adult health

1. Analyses

In the course of a cognitive testing session during the second wave of the MAGRIP study in 2009, 378 of the 745 study participants completed the same intelligence test as in 1968, the Leistungsprüfsystem (L-P-S, [Performance Test System]; Horn, 1962, 1983). As data on adult intelligence for these 378 participants were thus available, we followed the recommendations of two anonymous reviewers to include adult intelligence in the mediation models that investigated the direct and indirect effects of intelligence at age 12 on health at age 52. Specifically, we investigated whether childhood intelligence indirectly influences adult health via adult intelligence, over and above the indirect effects via educational attainment and adult SES. Further, we investigated whether educational attainment, which was found to be a crucial mediator of the effects of childhood intelligence on adult health, additionally influences adult health via an enhancement of adult intelligence.

To this end, performance on the L-P-S in 2009 was summarized in terms of the same three scale scores that were used to measure intelligence in 1968: verbal ability (3 subtests), reasoning ability (2 subtests), and visual-spatial ability (2 subtests). These three scale scores were applied to measure a latent factor that captured general intelligence in adulthood. We analyzed three mediation models to study the direct and indirect effects of childhood intelligence on adult functional, subjective, and physical health (corresponding to Models 2a–c in the main paper), including adult intelligence as an additional variable. Specifically, adult intelligence was included as an additional mediator in adulthood (next to adult SES) in these models. The residual variances of adult intelligence and adult SES were allowed to correlate freely (see Models 3a – 3c in Figure 3 of the main article). As data on adult intelligence were available for only about half of the study sample, we employed full-information maximum

likelihood (FIML) estimation as implemented in the Mplus software to deal with the missing data (Graham, 2009; Muthén & Muthén, 1998-2010).

When analyzing the models that included both childhood and adulthood intelligence, we encountered a substantial problem, namely, an extremely high collinearity between the two intelligence variables. Our analyses showed that the corresponding latent variables were correlated $r = .95$ (see Table S4, column 1). The high collinearity between the two intelligence variables was due to a very high rank-order stability of the intelligence scores between ages 12 and 52 in the MAGRIP study sample. The development of intelligence across 40 years in Luxembourg has been investigated in great detail in another publication based on the MAGRIP study (Schalke et al., 2012). Stated briefly, the study by Schalke and colleagues showed that substantial gains in individual intelligence scores occurred from ages 12 to 52. However, there was very little change in the rank-order of the study participants with respect to their intelligence scores across this time span.

Crucially, due to the high collinearity of childhood and adult intelligence, we could not include the direct effects of both intelligence variables on adult health in the mediation models. As the two variables were virtually interchangeable on a statistical level, their direct effects would have been uninterpretable when included simultaneously in a model. We therefore omitted the direct effect of childhood intelligence on adult health when investigating adult intelligence as an additional predictor (see Figure 3).

2. Results and Discussion

Importantly, our analyses that included adult intelligence effectively yielded the same results as the analyses without adult intelligence: Even when adult intelligence was included, childhood intelligence had significant indirect effects on all three adult health outcomes (see Table S5, rows “Total indirect” and “Total”). Depending on the health dimension under investigation, these indirect effects of childhood intelligence were mediated via educational

attainment (for physical health, see Table S5, row "Specific indirect 1") or educational attainment and adult SES (for functional and subjective health, see row "Specific indirect 4"). By contrast, adult intelligence did not significantly mediate the effect of childhood intelligence on health (see rows "Specific indirect 3" and "Specific indirect 5"). Further, adult intelligence did not have any significant direct effects on adult health (see Figure 3 in the main article). Moreover, educational attainment did not significantly predict adult intelligence, which indicates that the crucial role of educational attainment in the prediction of adult health from childhood intelligence was not due to an enhancement of adult intelligence through education.

In a nutshell, the core results and the core message of our study remain unaffected by the inclusion of adult intelligence: Childhood intelligence has positive effects on three dimensions of adult health 40 years later. These positive effects seem to be entirely mediated via educational attainment and adult SES. Adult intelligence does not seem to exert any direct or indirect influence on adult health.

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3. Section S3: Multiple-group models for investigating potential gender differences in the direct and indirect effects of childhood intelligence on adult health

1. Analyses

To explore potential gender differences in the direct and indirect effects of childhood intelligence on adult health, we employed multiple-group models. Multiple-group models allow users to test whether SEM parameters (e.g., estimates of direct effects; Muthén & Muthén, 1998–2010; Vandenberg & Lance, 2000) differ significantly in magnitude between genders. To employ multiple-group models, it is necessary to establish *measurement invariance* (i.e., the conceptual equivalence of the underlying theoretical constructs in women and men; Vandenberg & Lance, 2000). In our analyses, it was essential to ensure invariance of the factor structure (i.e., invariant relations between latent factors and their manifest indicators for women and men). Factorial invariance can be assumed when two increasingly restrictive invariance specifications in the measurement part of the SEM, namely, *configural* and *metric* invariance, can be shown to fit the data well. Configural invariance tests the null hypothesis that the pattern of constrained and free factor loadings of each latent factor is the same between groups (e.g., genders). Metric invariance tests the null hypothesis that factor loadings for like indicators are numerically invariant across groups. At least partial metric invariance must be established in order for cross-group comparisons of structural parameters to be meaningful (Vandenberg & Lance, 2000) because this level of invariance ensures that the metric of the latent factors is the same for women and men. As models with metric invariance are nested within models with configural invariance, we used χ^2 difference values ($\Delta\chi^2$) to evaluate the fit of the metrically invariant model specification compared to the configurally invariant model specification. However, as χ^2 difference tests become extremely powerful when sample size is large, differences in model fit should also be evaluated using differences in practical fit indices, such as the CFI and the RMSEA (Widaman, Ferrer, &

Conger, 2010). If sample size is large and a set of invariance constraints leads to a statistically significant worsening of fit in the $\Delta\chi^2$ test but no appreciable change in practical fit indices, it is justifiable to accept the more restrictive model due to its superior interpretive value, despite its significantly poorer statistical fit (Widaman et al., 2010). Following Cheung and Rensvold (2002), we considered a ΔCFI value of ≤ -.01 to be indicative of an equivalent model fit of the metrically invariant model compared to the configurally invariant model. Note that for the RMSEA and the SRMR, interpretative guidelines for ΔRMSEA and ΔSRMR values have not yet been established.

2. Results and Discussion

As was true for the total sample, all latent variables in the multiple-group models were well-defined as indicated by the substantial factor loadings for women and men (see Tables S6 and S7). Before exploring potential gender differences, we first evaluated whether the mediation models for functional, subjective, and physical health (Models S2a–c, see Figure S2) were invariant across genders. Table S8 contains the fit indices for the configural and metric invariance specifications in the three multiple-group models. The descriptive fit indices (i.e., CFI, RMSEA, and SRMR) indicated that configural invariance (i.e., an equal pattern of constrained and free factor loadings for women and men) held for all three models. Metric invariance was obtained by constraining the numerical values of like factor loadings in women and men to be invariant. The descriptive fit indices CFI, RMSEA, and SRMR showed that metrically invariant models provided an acceptable level of approximation to the empirical data. Moreover, although the $\Delta\chi^2$ values were significant, the misfit of the metrically invariant models seemed to be tolerable as ΔCFI values for all three models were comparable to the recommended cut-off value of -.01. Taken together, due to their acceptable level of overall model fit, the tolerable decrease in model fit compared to their configurally invariant counterparts and their superior interpretative value (Widaman et al., 2010), we

applied metrically invariant model specifications to explore potential gender differences in the mediational pathways linking childhood intelligence and adult health.[1]

We assessed the significance of the direct and indirect effects of childhood intelligence on adult health for women and men separately on the basis of bias-corrected bootstrap confidence intervals. In addition, we examined whether any of the direct effects of the predictors in the mediation models differed significantly in magnitude between women and men. To this end, we computed difference parameters using the Mplus software (Muthén & Muthén, 1998–2010). These parameters contained the difference of a certain pair of direct effects between women and men. For instance, we computed a difference parameter that contained the numerical difference between the direct effect of adult SES on functional health in women and the direct effect of adult SES on functional health in men. These difference parameters were again tested for significance using 95% confidence intervals (Muthén & Muthén, 1998–2010). A significant difference parameter indicates that the respective direct effect on health differs significantly between women and men.

The results are shown in Figures S1 and S2 and Table S9. Figure S1 contains the bivariate regressions of the three adult health dimensions on childhood intelligence for women and men. Figure S2 contains the standardized coefficients for each direct path in the three models that compared the direct and indirect effects of intelligence at age 12 on functional health (Figure S2a), subjective health (Figure S2b), and physical health (Figure S2c) at age 52 between women and men. Table S9 shows the standardized direct, specific indirect, total indirect, and total effects of intelligence at age 12 on the three health dimensions at age 52 for women and men.

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[1] Note that we also explored whether models with partial metric invariance specifications, which involve a smaller number of constrained parameters than full metric invariance specifications, showed a better fit in terms of $\Delta\chi^2$. As there were no substantial differences in model fit, and as direct and indirect effect estimates did not differ substantially between mediation models with partial and full metric invariance specifications, we opted for full metric invariance because it represented a more parsimonious explanation of our data.

As observed for the total sample, we found that intelligence at age 12 was significantly associated with higher educational attainment and better adult SES in both women and men. However, an examination of the difference parameters for the direct effects in the mediation models showed that the pattern of results deviated somewhat. Whereas childhood intelligence had a significantly stronger direct effect on educational attainment in women (β = .54; see Figure S2) than in men (β = .37), childhood SES had a significantly stronger direct effect on educational attainment in men (β = .51) than in women (β = .30). Moreover, childhood intelligence had a significantly stronger direct effect on adult SES in women (β = .28) than in men (β = -.01). In men, the effect of childhood intelligence on adult SES was entirely mediated via educational attainment. Furthermore, educational attainment had a significantly stronger direct effect on adult SES in men (β = .74) than in women (β = .51). Altogether, these results suggest that women's educational and socioeconomic success seems to be driven to a larger extent by their own intellectual abilities, whereas men's educational and socioeconomic success is driven to a larger extent by the direct and indirect effects of an advantageous socioeconomic family background.

We now turn to the gender differences in the mediational pathways linking childhood intelligence and adult health. With respect to the bivariate regressions, women's childhood intelligence significantly predicted all three health outcomes (see Figure S1). By contrast, men's childhood intelligence significantly predicted only their functional health, whereas men's childhood intelligence did not significantly predict their subjective and physical health. However, the numerical differences between the bivariate regression coefficients for women and men were not statistically significant.

With respect to the full mediation models, the overall results obtained for women and men mirrored the results obtained for the total sample. Specifically, the positive bivariate effects of intelligence on health seemed to be entirely explained by indirect effects of

intelligence on the three health dimensions. We did not find any significant direct effects of intelligence on health for women or men (see Table S9, row "Direct"). By contrast, the total indirect and total effects of childhood intelligence on the three adult health dimensions were always positive in women and men yet did not always reach statistical significance. Furthermore, the percent mediated (i.e., the proportion of the total effect of intelligence on health that was mediated via educational attainment and adult SES) was always large for women and men for all three health dimensions. Finally, we did not find any significant numerical differences in the direct effects of childhood intelligence, educational attainment, and adult SES on adult health between women and men. Altogether, these results suggest that the "chain reaction" that links childhood intelligence and adult health via educational attainment and adult SES generally seems to operate in a similar way in women and men.

Even though we did not find any statistically significant gender differences in the mediational pathways that link childhood intelligence and adult health, some tendential gender differences were observed. Moreover, it is possible that these gender differences were not significant because splitting the total sample into two separate groups with smaller respective subsample sizes resulted in a loss of power. Thus, it is possible that with a larger sample, these tendencies would have been significant. Below, we therefore outline in more detail the tendential gender differences that were observed in our sample.

For subjective health, the total indirect effect of women's childhood intelligence on their subjective health ($\beta = .18$, see Table S9) was significant and more than three times larger than the nonsignificant total indirect effect of men's childhood intelligence on their subjective health ($\beta = .05$). The total effect of women's childhood intelligence on their subjective health ($\beta = .13$) just barely failed to reach significance, and was also more than three times larger than the nonsignificant total effect of men's childhood intelligence on their subjective health ($\beta = .04$). These results may indicate that childhood intelligence tends to

exert a larger mediated influence on women's subjective health than on men's subjective health. Men's subjective health in general seemed to be hardly influenced by the predictors in the mediation model.

For physical health, the specific indirect effect of men's childhood intelligence on their adult physical health via educational attainment was significant (β = .13), whereas the specific indirect effect of women's intelligence on their physical health via educational attainment was not (β = .07). Moreover, the direct effect of men's educational attainment on physical health was significant (β = .36; see Figure S2), whereas the direct effect of women's educational attainment on their physical health was not (β = .13). These results may indicate that the finding that educational attainment was the crucial mediator in the relation between childhood intelligence and physical health at age 52, as observed for the total sample, tended to be driven by a stronger mediation via education in men.

Altogether, however, further studies with large enough sample sizes are warranted to investigate potential gender differences in the specific mediational pathways that link childhood intelligence and adult health.

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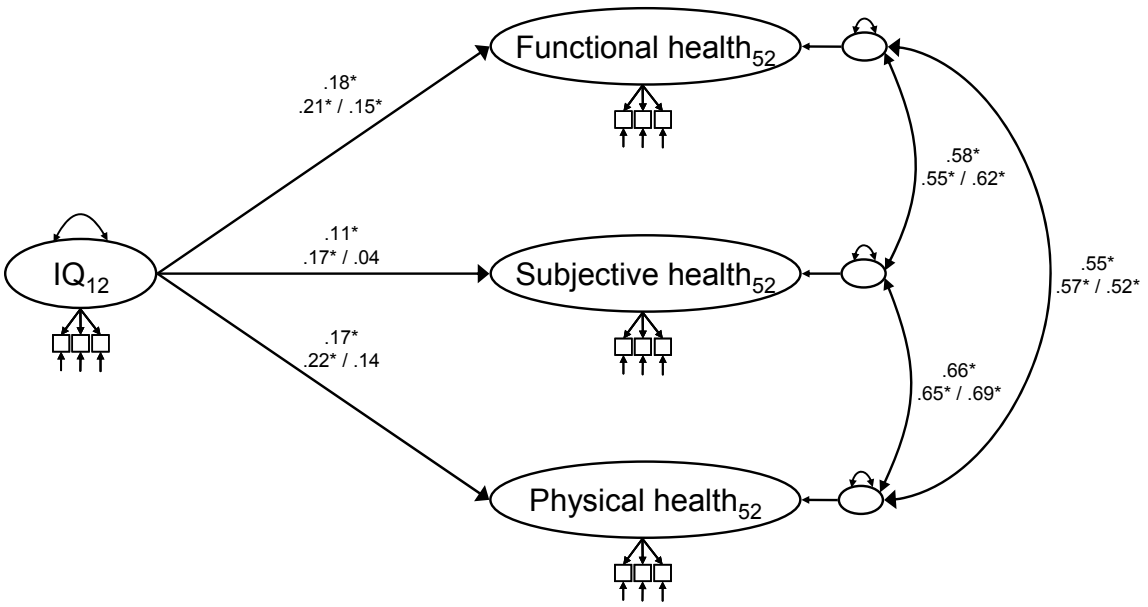
Table III-S3. *Standardized factor loadings of manifest indicators of intelligence and SES at age 12, educational attainment, adult SES, and functional, subjective, and physical health at age 52, as obtained for the three models for the total sample shown in Figure III-2 (Model 2a / Model 2b / Model 2c)*

Indicator	IQ_{12}	SES_{12}	Education	Adult SES	Functional health (Model 2a)	Subjective health (Model 2b)	Physical health (Model 2c)
Verbal ability[a]	.66 / .66 / .66						
Reasoning ability[a]	.70 / .70 / .70						
Visual–spatial ability[a]	.69 / .69 / .69						
Father's academic degree		.63 / .63 / .63					
Father's vocational training		.78 / .78 / .78					
Father's occupation		.81 / .81 / .81					
Years of education			.90 / .90 / .90				
Highest school visited			.88 / .88 / .88				
Highest academic degree			.89 / .89 / .89				
Household income				.54 / .54 / .54			
Current occupation (HS)				.83 / .83 / .83			
Current occupation (CT)				.82 / .82 / .82			
Functional health 1[a]					.82 / -- / --		
Functional health 2[a]					.89 / -- / --		
Functional health 3[a]					.88 / -- / --		
Judgment of overall health						-- / .98 / --	
Health compared to peers						-- / .82 / --	
Satisfaction with health						-- / .78 / --	
No. of doctor visits							-- / -- / .68
No. of sick leave days							-- / -- / .78
No. of hospital nights							-- / -- / .62

Note. HS = Household Study; CT = Cognitive Testing.
[a] Parcel score.

Figure III-S1. *Bivariate regressions of functional, subjective, and physical health at age 52 on intelligence at age 12*



Note. Regression coefficients for each path are first presented for the entire sample, then below for women and men, separated by a slash (coefficient women / coefficient men). Latent factors are depicted as ellipses. Double-headed arrows represent correlations. IQ = Intelligence. * $p < .05$.

Table III-S4. *Intercorrelations between the latent variables intelligence and SES at age 12, educational attainment, adult SES, adult intelligence, and functional, subjective, and physical health at age 52*

Latent variable	1	2	3	4	5	6	7	8
1 IQ_{12}	–							
2 SES_{12}	.27**	–						
3 Education	.57**	.52**	–					
4 Adult SES	.51**	.48**	.77**	–				
5 Adult IQ	.95**	.30**	.60**	.54**	–			
6 Functional $health_{52}$	.18**	.12**	.24**	.28**	.24**	–		
7 Subjective $health_{52}$	.10*	.08	.15**	.20**	.16**	.59**	–	
8 Physical $health_{52}$	.18**	.14**	.27**	.23**	.27**	.57**	.67**	–

Note. IQ = Intelligence; SES = Socioeconomic Status; Education = Educational Attainment.
* $p < .05$. ** $p < .01$.

Table III-S5. *Standardized specific indirect, total indirect, and total effects of intelligence at age 12 on functional, subjective, and physical health at age 52 – including adult intelligence as an additional mediator*

	Functional health (Model 3a)			Subjective health (Model 3b)			Physical health (Model 3c)		
Effect of intelligence at age 12 on health at age 52	β	95% CI	% mediated	β	95% CI	% mediated	β	95% CI	% mediated
Direct	–	–		–	–		–	–	
Specific indirect 1 (via education)	0.02	–0.06, 0.09		–0.01	–0.08, 0.07		0.09	0.00, 0.18	
Specific indirect 2 (via adult SES)	0.02	–0.01, 0.05		0.02	–0.01, 0.05		0.00	–0.02, 0.03	
Specific indirect 3 (via adult IQ)	0.07	–0.04, 0.18		0.03	–0.08, 0.13		0.06	–0.06, 0.19	
Specific indirect 4 (via education and adult SES)	0.07	0.01, 0.12		0.06	0.01, 0.12		0.01	–0.05, 0.07	
Specific indirect 5 (via education and adult IQ)	0.00	–0.00, 0.01		0.00	–0.01, 0.01		0.00	–0.01, 0.01	
Total indirect (sum of specific indirect effects 1-5)	0.18	0.09, 0.26		0.11	0.02, 0.19		0.17	0.07, 0.27	
Total (sum of direct and total indirect effects)	0.18	0.09, 0.26	100	0.11	0.02, 0.19	100	0.17	0.07, 0.27	100

Note. Effect estimates are from the completely standardized solution of the corresponding model (i.e., Models 3a, 3b, and 3c; see Figure 3 in the main article). The percent mediated is 100% because, due to the high collinearity between intelligence at ages 12 and 52, the direct effects of intelligence at age 12 were fixed to zero in the models. As there was no direct effect of intelligence at age 12, the total effect corresponds to the total indirect effect.

Table III-S6. *Standardized factor loadings of manifest indicators of intelligence and SES at age 12, educational attainment, adult SES, and functional, subjective, and physical health at age 52 for women*

Indicator	IQ_{12}	SES_{12}	Education	Adult SES	Functional health (Model S2a)	Subjective health (Model S2b)	Physical health (Model S2c)
Verbal ability[a]	.65 / .65 / .65						
Reasoning ability[a]	.71 / .71 / .71						
Visual–spatial ability[a]	.70 / .70 / .70						
Father's academic degree		.64 / .64 / .64					
Father's vocational training		.79 / .79 / .79					
Father's occupation		.84 / .84 / .84					
Years of education			.92 / .92 / .92				
Highest school visited			.85 / .85 / .85				
Highest academic degree			.87 / .87 / .87				
Household income				.49 / .49 / .49			
Current occupation (HS)				.78 / .78 / .78			
Current occupation (CT)				.77 / .76 / .77			
Functional health 1[a]					.82 / -- / --		
Functional health 2[a]					.92 / -- / --		
Functional health 3[a]					.88 / -- / --		
Judgment of overall health						-- / .97 / --	
Health compared to peers						-- / .83 / --	
Satisfaction with health						-- / .77 / --	
No. of doctor visits							-- / -- / .64
No. of sick leave days							-- / -- / .77
No. of hospital nights							-- / -- / .62

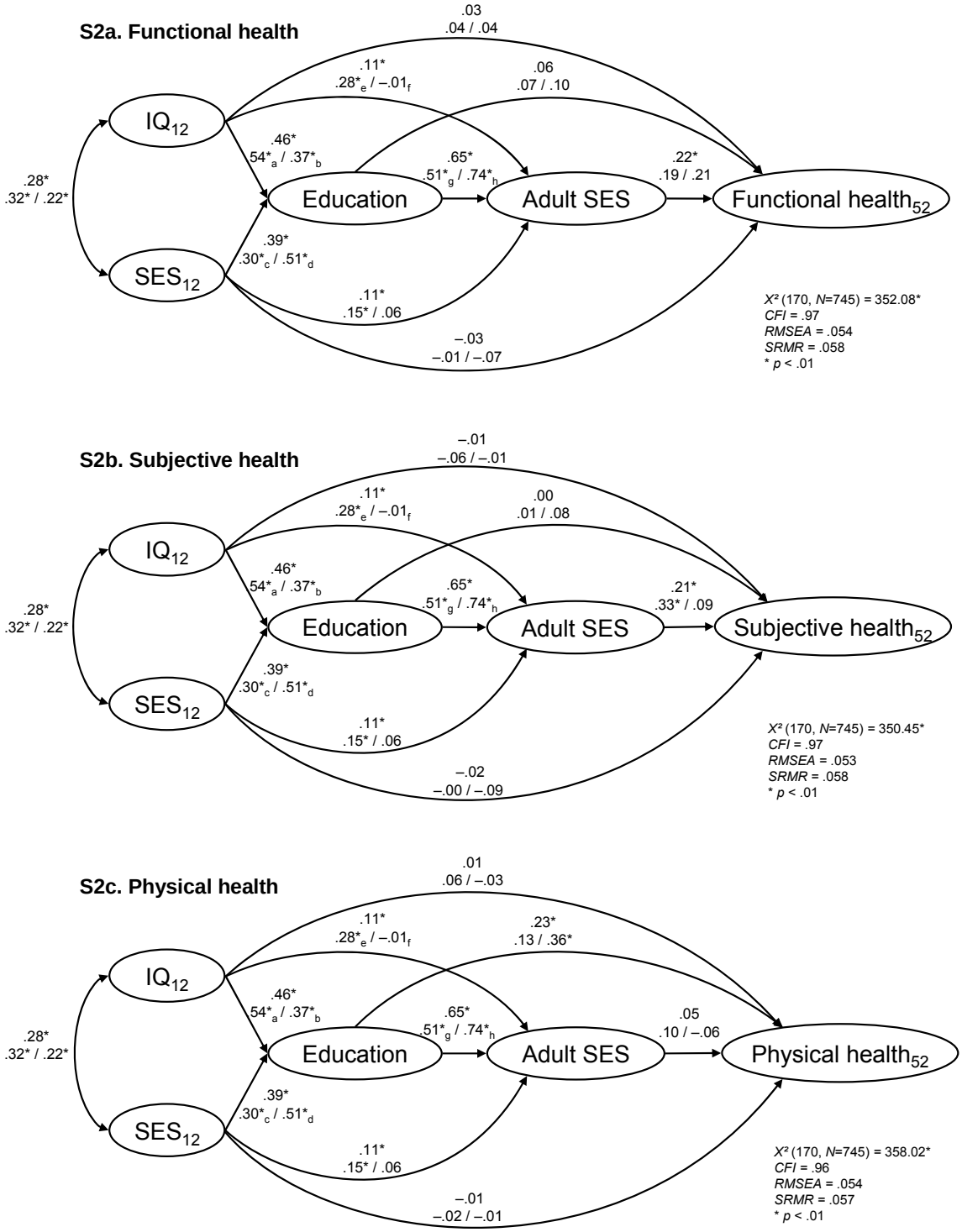
Note. The table shows standardized factor loadings for women as obtained for the three multiple-group models shown in Figure S2 (Model S2a / Model S2b / Model S2c). HS = Household Study; CT = Cognitive Testing.
[a] Parcel score.

Table III-S7. *Standardized factor loadings of manifest indicators of intelligence and SES at age 12, educational attainment, adult SES, and functional, subjective, and physical health at age 52 for men*

Indicator	IQ_{12}	SES_{12}	Education	Adult SES	Functional health (Model S2a)	Subjective health (Model S2b)	Physical health (Model S2c)
Verbal ability[a]	.63 / .63 / .63						
Reasoning ability[a]	.75 / .75 / .75						
Visual–spatial ability[a]	.69 / .69 / .69						
Father's academic degree		.62 / .62 / .62					
Father's vocational training		.74 / .74 / .74					
Father's occupation		.79 / .79 / .79					
Years of education			.88 / .89 / .88				
Highest school visited			.89 / .89 / .89				
Highest academic degree			.90 / .90 / .90				
Household income				.60 / .60 / .60			
Current occupation (HS)				.90 / .90 / .91			
Current occupation (CT)				.83 / .82 / .82			
Functional health 1[a]					.83 / -- / --		
Functional health 2[a]					.86 / -- / --		
Functional health 3[a]					.88 / -- / --		
Judgment of overall health						-- / .99 / --	
Health compared to peers						-- / .81 / --	
Satisfaction with health						-- / .76 / --	
No. of doctor visits							-- / -- / .74
No. of sick leave days							-- / -- / .76
No. of hospital nights							-- / -- / .64

Note. The table shows standardized factor loadings for men as obtained for the three multiple-group models shown in Figure S2 (Model S2a / Model S2b / Model S2c). HS = Household Study; CT = Cognitive Testing.
[a] Parcel score.

Figure III-S2. *Multiple-group models for studying potential gender differences in the direct and indirect effects of intelligence at age 12 on the three dimensions of health at age 52*



Note. Standardized coefficients for each direct path are first presented for the entire sample, then below for women and men, separated by a slash (coefficient women / coefficient men). Latent factors are depicted as ellipses. For clarity of presentation, manifest indicators and variances of latent factors have been omitted. Double-headed arrows represent correlations. Path coefficients for women and men that show different subscripts are statistically different from one another at $p < .05$ (two-tailed). IQ = Intelligence; SES = Socioeconomic Status; Education = Educational Attainment. * $p < .05$.

Table III-S8. *Model fit indices for multiple-group models for studying potential gender differences in the direct and indirect effects of intelligence at age 12 on functional, subjective, and physical health at age 52*

	Fit index						
	χ^2	*df*	$\Delta\chi^2$	CFI	ΔCFI	RMSEA	SRMR
Functional health (Model S2a)							
Configural invariance	308.81*	160		.97		.050	.049
Metric invariance	352.08*	170	43.27*	.97	-.01	.054	.058
Subjective health (Model S2b)							
Configural invariance	310.48*	160		.97		.050	.050
Metric invariance	350.45*	170	39.98*	.97	-.01	.053	.058
Physical health (Model S2c)							
Configural invariance	325.06*	160		.96		.053	.052
Metric invariance	358.02*	170	32.95*	.96	-.01	.054	.057

Note. Models with metric invariance specifications are nested within models with configural invariance specifications. χ^2 difference tests for nested models ($\Delta\chi^2$; Widaman et al., 2010) and CFI difference tests for nested models (ΔCFI; Cheung & Rensvold, 2002) were used to compare the fits of the two invariance specifications. A nonsignificant $\Delta\chi^2$ value is indicative of an equivalent model fit of the metric compared to the configural invariance model. Likewise, a ΔCFI value of ≤ -.01 is indicative of an equivalent model fit of the metric compared to the configural invariance model. * $p < .01$.

Table III-S9. *Standardized direct, specific indirect, total indirect, and total effects of intelligence at age 12 on functional, subjective, and physical health at age 52, obtained for women and men*

	Functional health (Model S2a)			Subjective health (Model S2b)			Physical health (Model S2c)		
Effect of intelligence at age 12 on health at age 52	β	95% CI	% mediated	β	95% CI	% mediated	β	95% CI	% mediated
Women									
Direct	0.04	–0.16, 0.23		–0.06	–0.27, 0.16		0.06	–0.17, 0.29	
Specific indirect 1 (via education)	0.04	–0.08, 0.15		0.00	–0.14, 0.14		0.07	–0.06, 0.19	
Specific indirect 2 (via adult SES)	0.05	–0.04, 0.15		0.09	–0.02, 0.20		0.03	–0.07, 0.13	
Specific indirect 3 (via education and adult SES)	0.05	–0.04, 0.14		0.09	–0.03, 0.21		0.03	–0.07, 0.12	
Total indirect (sum of specific indirect effects 1-3)	0.14	0.02, 0.26		0.18	0.04, 0.33		0.12	–0.02, 0.26	
Total (sum of direct and total indirect effects)	0.18	0.05, 0.31	79.10	0.13 [a]	–0.01, 0.26	76.67	0.19	0.04, 0.33	66.13
Men									
Direct	0.04	–0.11, 0.20		–0.01	–0.15, 0.13		–0.03	–0.21, 0.15	
Specific indirect 1 (via education)	0.04	–0.06, 0.13		0.03	–0.06, 0.12		0.13	0.02, 0.25	
Specific indirect 2 (via adult SES)	0.00	–0.03, 0.03		0.00	–0.02, 0.02		0.00	–0.02, 0.02	
Specific indirect 3 (via education and adult SES)	0.06	–0.01, 0.13		0.02	–0.04, 0.08		–0.02	–0.08, 0.04	
Total indirect (sum of specific indirect effects 1-3)	0.09	0.01, 0.17		0.05	–0.02, 0.12		0.11	0.03, 0.19	
Total (sum of direct and total indirect effects)	0.13	0.00, 0.26	68.42	0.04 [a]	–0.08, 0.16	82.54	0.08 [a]	–0.07, 0.24	78.08

Note. Effect estimates are from the completely standardized solution of the corresponding multiple-group model for women and men (i.e., Models S2a, S2b, and S2c). The percent mediated was calculated by dividing the total indirect effect by the total effect (cf. MacKinnon et al., 2001), but may differ slightly from the quotient total indirect effect/total effect in Table S9 due to rounding errors.

[a] Because the direct and total indirect effects were in opposite directions, the mediation of the total effect was based on the absolute value of the sum of the direct and total indirect effects (cf. Judge, Ilies, & Dimotakis, 2010).

Chapter IV

Study III:

Childhood Intelligence Predicts Premature Mortality: Results from a 40-Year Population-Based Longitudinal Study

Abstract

Objectives: To study whether childhood intelligence predicts the risk for all-cause premature mortality 40 years later in a country with universal access to health care when controlling for childhood socioeconomic status and gender differences. ***Design:*** Nationally representative prospective cohort study. ***Setting:*** Luxembourg. ***Participants:*** 1,408 women and 1,416 men at age 12 in 1968 were followed for 40 years until 2008. ***Main Outcome Measures:*** Logistic regression odds ratios (OR) for all-cause premature mortality. ***Results:*** Higher childhood intelligence predicted a lower risk for premature mortality, even when childhood socioeconomic status and gender were controlled for (OR for a one standard deviation increase in childhood intelligence: 0.82 (95% CI 0.71 to 0.95)). Analyses by intelligence groups identified a high-risk group. Men belonging to the group of the lowest 20% in intelligence were at the highest risk for mortality, compared to men higher in intelligence and women in general (OR 2.37 (95% CI 1.03 to 5.48)). ***Conclusion:*** Childhood intelligence predicts the risk for premature mortality especially in men—even in a country that provides all citizens with access to health care and education. Individuals with low intelligence scores may be exposed to persisting disadvantages in later life. To reduce the risk for premature mortality, interventions that pay special attention to the one in five men with lower cognitive abilities should be considered, including efforts to reach, engage, and retain them in health care.

Keywords: childhood intelligence, adult mortality, childhood socioeconomic status

Childhood Intelligence Predicts Premature Mortality:

Results from a 40-Year Population-Based Longitudinal Study

1. Introduction

Even though the average life expectancy in Western countries is rising, systematic inequalities in mortality continue to exist between citizens with a higher and a lower socioeconomic status. These inequalities amount to a 5- to 10-year difference in average life expectancy (Mackenbach, 2012). Premature mortality is of particular concern as it causes a substantial loss in productive years of life and adds many years fraught with serious difficulties for those left behind. Furthermore, the majority of premature deaths seem preventable and would thus be suitable for public health interventions that target those at risk (Eurostat, 2009). However, universal access to quality health care, generous welfare policies, and other public health interventions have so far been unsuccessful at eliminating socioeconomic inequalities in mortality (Batty, Kivimäki, & Deary, 2010; Mackenbach, 2012).

In recent years, intelligence has been investigated as one factor that could help to explain these inequalities (Deary, 2012; Deary, Weiss, & Batty, 2010; Mackenbach, 2010, 2012). Some have even labelled intelligence the "fundamental cause of social class inequalities in health" (Gottfredson, 2004, p. 174). Although it seems more likely that intelligence is but one factor among many for explaining socioeconomic differences in mortality (Batty et al., 2010), several studies have suggested that lower intelligence in childhood and early adulthood is predictive of increased risk for all-cause and cause-specific mortality across the adult life span (Calvin et al., 2011; Hart et al., 2003, 2005; Kuh, Richards, Hardy, Butterworth, & Wadsworth, 2004; Lager, Bremberg, & Vagerö, 2009; Leon, Lawlor, Clark, Batty, & Macintyre, 2009). These effects of childhood intelligence on later mortality remain even when accounting for the effects of childhood socioeconomic status on mortality

(Calvin et al., 2011).

However, a number of open questions remain regarding the relation between intelligence and mortality risk. First, all previous studies on this relation have been conducted in English-speaking or Scandinavian countries (Calvin et al., 2011; Deary, 2010). It remains to be shown whether these findings can be generalised to countries with different cultural backgrounds, health care systems, and levels of social mobility. Second, the shape of the intelligence-mortality relation is unclear (Batty, Deary, & Gottfredson, 2007). Does this relation exist across the entire spectrum of the intelligence distribution as some studies suggest (Batty et al., 2010; Lager et al., 2009; Martin & Kubzansky, 2005), or is there a high-risk group of individuals at the lower end of the intelligence distribution with elevated mortality, thus pointing to a potential threshold effect (Hart et al., 2003, 2005; Kuh et al., 2004)? Identification of a specific group with an elevated risk for premature mortality would provide important information about who should be targeted in particular by health care interventions and preventive measures. Finally, few studies have systematically investigated potential gender differences in the relation between intelligence and mortality. The results seem inconclusive as some studies have found gender differences and others have not, therefore warranting further investigation (Calvin et al., 2011; Lager et al., 2009; Pearce, Deary, Young, & Parker, 2006; Whalley & Deary, 2001). The examination of potential gender differences is important for formulating explanatory models for the intelligence-mortality relation. Universal effects for women and men may indicate that intelligence has an association with mortality largely because it may be a marker of a healthy body in general (i.e., of "system integrity"; Batty et al., 2007; Lager et al., 2009). Differential relations between intelligence and mortality in women and men may be indicative of environmental and behavioural factors that may be modifiable and thus targeted by interventions.

The present study aimed to address three research questions: (1) Does childhood

intelligence predict risk for all-cause premature mortality in Luxembourg, a country that offers universal access to quality health care (World Health Organization, 2000)? (2) Is there a high-risk group at the lower end of the intelligence distribution? (3) Does the intelligence-mortality relation differ between women and men?

2. Methods

2.1 Participants

Participants were individuals enrolled in a longitudinal prospective cohort study (the MAGRIP study) initiated in 1968 in Luxembourg (Bamberg, Dickes, & Schaber, 1977). The MAGRIP study was a school-based study designed to investigate the determinants of children's school careers in Luxembourg. In 1968, detailed data on intelligence and socioeconomic family background were collected on a randomly selected nationally representative sample comprised of 2,824 children at the end of their primary education (M_{age} = 11.9 years; *SD* = 7.2 months; 50.1% male). In 2008, mortality rates for this cohort were retrieved from the database of the social security agency of Luxembourg.

2.2 Measures

2.2.1 Childhood intelligence. In 1968, trained test personnel administered a standardized, objective, and comprehensive German intelligence test, the Leistungsprüfsystem (L-P-S, [Performance Test System]; Horn, 1962, 1983), to the children in classroom sessions. The L-P-S is comprised of 14 subtests that provide measures of general intelligence and more specific intellectual abilities, such as verbal abilities, reasoning abilities, figural-spatial abilities, and perceptual speed. To obtain a measure of childhood intelligence, we summarized participants' performance on the 14 subtests in terms of a total intelligence score. We standardized this score to generate a mean of 100 and a standard deviation of 15 for the entire 1968 sample. The reliability of the total intelligence score in the

present sample was satisfactory with α = .85. Previous research has shown that this total intelligence score has excellent psychometric properties (split-half reliability = .99, parallel-forms reliability = .94, retest reliability across a time span of 32 months = .83; Horn, 1983). The correlation of the L-P-S intelligence score with the total score on the German version of the Wechsler Adult Intelligence Scale (WAIS)—the Hamburg Wechsler Intelligenztest für Erwachsene (HAWIE-R; Tewes, 1991)—is .94 (Sturm & Büssing, 1982).

2.2.2 Childhood socioeconomic status (SES). In 1968, children reported their parents' current occupation. These occupations were mapped onto the categories of the International Standard Classification of Occupations (ISCO-88; Elias, 1997). Ganzeboom and Treiman (1996) showed that the ISCO-88 scheme can be appropriately applied to occupational data from 1968. These classifications were then transformed into the widely used International Socio-Economic Index of occupational status (ISEI; Ganzeboom, de Graaf, Treiman, & de Leeuw, 1992; Ganzeboom & Treiman, 1996). The ISEI scale takes the income and educational levels of occupations into account. It has interval scale properties and a theoretical range from 16 (e.g., cleaners, unskilled agricultural labourers) to 90 (e.g., judges). With its grounding in international occupational classification schemes, the ISEI scale is internationally comparable; it has been demonstrated to be a reliable and valid indicator of socioeconomic status in many international large-scale assessments (e.g., PISA; Organisation for Economic Co-operation and Development, 2004). In the present study, we used the highest ISEI value in a family (usually the father's ISEI value) as an indicator of childhood socioeconomic status. Interrater reliability of the ISEI coding was tested for two independent groups of raters and was satisfactory at .72.

*2.2.3 Mortality***.** In 2008, a second wave of the MAGRIP study was initiated (Brunner & Martin, 2011). Data on the all-cause mortality rate among the MAGRIP participants in the period between 1968 and 2008 were obtained from the database of the social security agency

of Luxembourg (permission was granted by the Luxembourgish data protection committee "Commission Nationale Pour la Protection des Données"). Of the 2,824 former participants, 2,377 (84%) were alive, and 166 (6%) had died by 2008. The remaining 281 (10%) former participants could not be traced by their social security ID and had most probably left the country. Thus, the analyses for the present study were based on those 2,543 former participants for whom information was available regarding whether they were alive or deceased.

2.3 Statistical Analyses

To quantify how childhood intelligence predicted premature mortality in Luxembourg, we ran two series of logistic regression models. In the first series, we applied logistic regression models using the full range of the continuous intelligence score as a predictor. In Model 1, we used a bivariate logistic regression model to study how this intelligence score would predict mortality. In Model 2, we included gender as an additional predictor and controlled for childhood socioeconomic status. To further investigate potential gender differences in the relations between childhood intelligence or socioeconomic status and mortality, we added the interaction between gender and intelligence and the interaction between gender and socioeconomic status in a third model (Model 3). All models were computed with mean-centered intelligence and socioeconomic status variables.

To explore the shape of the intelligence-mortality relation, we divided all participants into equal-sized groups according to their intelligence scores. This resulted in five groups with increasing mean intelligence scores (i.e., quintiles), with each group comprising 20% of the participants of our total sample[2]. Mortality rates in these five groups were graphed for the general sample (Figure 1a) and for men and women separately (Figure 1b). In the second

[2] Using quintiles is a standard statistical technique applied when a major goal of the grouping process is to retain as many of the properties of the original variable's distribution as possible (Austin, 2011).

series of logistic regression models, we then explored statistically whether individuals with low levels of intelligence would exhibit a particularly increased mortality risk. To this end, we repeated the logistic regression Models 1-3 using an intelligence grouping variable as a predictor (Models 4-6). This dichotomous intelligence grouping variable was based on the five equal-sized intelligence groups and coded whether a participant belonged to the lowest 20% or to the remaining 80% of the intelligence distribution.

We included all 2,543 participants for whom data on mortality were available. To account for missing data in childhood intelligence (3% missing data) and childhood socioeconomic status (1% missing data), we applied multiple imputation (Schafer & Graham, 2002). We conducted 10 cycles of imputations using the Amelia II package for the R software (Honaker, King, & Blackwell, 2011; R Core Team, 2012). In each cycle, the missing values were estimated based on the available data in the predictors. This process resulted in 10 imputed data sets, each one containing slightly different versions of the imputed values. The software Mplus 7 (34 Muthén & Muthén, 1998–2007) was then used to conduct the logistic regression analyses. Mplus allows for the combination of the results from imputed data sets to obtain overall parameter estimates and standard errors that reflect uncertainty in the imputation as well as uncertainty due to random variation (Muthén & Muthén, 1998–2007; Schafer & Graham, 2002).

3. Results

Table IV-1 shows the descriptive statistics for the entire MAGRIP study sample in 1968 (N = 2,824), for all participants included in the present study (n = 2,543), and separately for those participants in the present study who were still alive in (n = 2,377) or who had died (n = 166) by 2008. Mean childhood intelligence, mean childhood socioeconomic status, the ratio of men to women, and the percentage of native Luxembourgers were highly similar across the entire 1968 study population, the sample in the present study, and the survivors in

2008. These results indicate that the sample in the present study was representative of the original sample. However, those 166 participants who had died by 2008 had a lower mean childhood intelligence (Cohen's $d = 0.22$) and childhood socioeconomic status ($d = 0.19$). Further, a substantial majority of the deceased were men ($\varphi = .10$). These results indicate that lower childhood intelligence, lower socioeconomic status, and being a man could be risk factors for premature mortality in adulthood.

Table IV-1. *Descriptive statistics for the original sample in 1968, for participants in the present study, and separately for participants alive or deceased in 2008*

	Original sample in 1968 (N = 2,824)	Study participants		
		Total (n = 2,543)	Alive (n = 2,377)	Deceased (n = 166)
Childhood intelligence				
M	100	99.5	99.7	96.3
SD	15	15.2	15	17
Childhood socioeconomic status				
M	39.2	39.2	39.4	36.6
SD	13.7	13.5	13.5	12.6
Sociodemographic characteristics				
Percentage of men	50.1	50.8	49.4	69.9
Percentage native	84.1	85.7	85.9	82.5

3.1 Childhood intelligence and risk of premature mortality: General and gender-specific relations

Table IV-2 (upper panel) shows the results of the first series of logistic regression models that investigated the impact of the full-range childhood intelligence predictor on mortality risk. The bivariate logistic regression (Model 1) showed that higher childhood intelligence significantly predicted a lower premature mortality risk in adulthood. Specifically, participants with a higher childhood intelligence had a lower risk of having died by 2008 (OR 0.80, 95% CI 0.69 to 0.92). The effect of childhood intelligence on mortality risk remained robust when controlling for childhood socioeconomic status (Model 2). Further, gender had a significant effect on mortality risk in Model 2. Specifically, even when controlling for socioeconomic status and intelligence, men had a higher risk of having died by 2008 than women (OR 2.43, 95% CI 1.72 to 3.42). Model 3 showed a tendency for stronger effects of intelligence on mortality risk in men than in women, as reflected in the odds ratio for the interaction (OR 0.80, 95% CI 0.56 to 1.14). However, this interaction failed to reach significance.

Table IV-2. *Odds ratios (95% confidence intervals) for the relation of a 1 Standard Deviation increase in full-range childhood intelligence or of belonging to the lowest childhood intelligence group versus all higher childhood intelligence groups, a 1 Standard Deviation increase in childhood socioeconomic status, and gender with premature all-cause mortality*

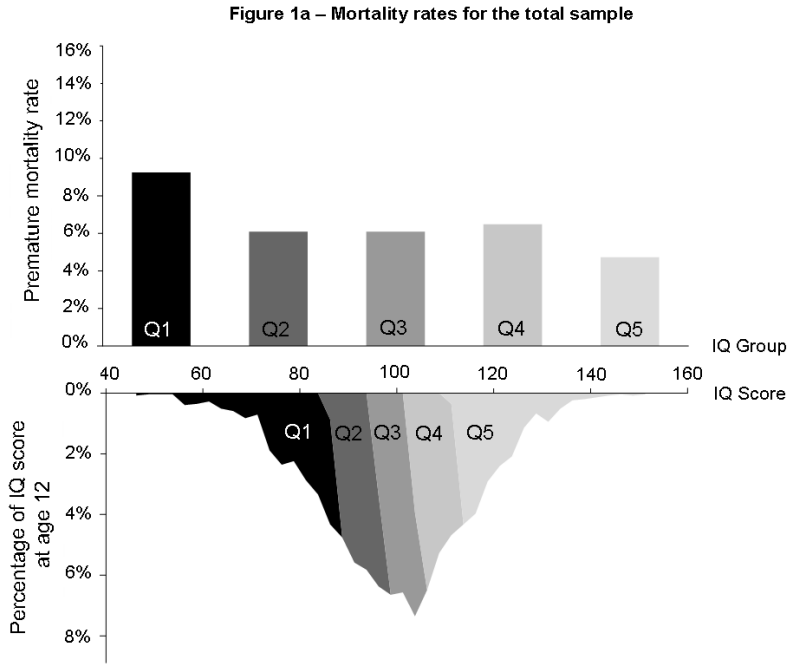
	Predictor of premature all-cause mortality				
	IQ	SES	Gender	IQ*Gender	IQ*SES
Full-range IQ					
Model 1	0.80 (0.69 to 0.92)				
Model 2	0.82 (0.71 to 0.95)	0.84 (0.70 to 1.01)	2.43 (1.72 to 3.42)		
Model 3	0.96 (0.71 to 1.29)	0.70 (0.50 to 0.99)	2.45 (1.71 to 3.51)	0.80 (0.56 to 1.14)	1.29 (0.87 to 1.92)
Lowest vs. higher IQ groups					
Model 4	1.63 (1.14 to 2.32)				
Model 5	1.52 (1.06 to 2.20)	0.83 (0.69 to 1.00)	2.40 (1.70 to 3.38)		
Model 6	0.83 (0.40 to 1.70)	0.69 (0.49 to 0.97)	2.04 (1.37 to 3.04)	2.37 (1.03 to 5.48)	1.31 (0.88 to 1.94)

Note. Gender was coded 0 = women, 1 = men. Lowest IQ group vs. higher IQ groups was coded 0 = higher IQ groups, 1 = lowest IQ group. Models 2-3 and 5-6 adjusted for childhood SES and Gender. Key: IQ = intelligence; SES = socioeconomic status.

3.2 Is the lowest intelligence group at particularly high risk of mortality?

Figure IV-1 shows premature mortality rates in five intelligence groups for the total sample (Figure 1a) and for women and men separately (Figure 1b), as well as the frequency distribution of intelligence scores in the five groups for the total sample. A visual analysis of these plots indicates that participants at the lower end of the intelligence distribution, and particularly men, seemed to constitute a risk group with a particularly increased mortality risk. Specifically, when investigating mortality rates for all participants in the five equal-sized groups with increasing mean intelligence scores, the mortality rate seemed to be particularly high in the lowest intelligence group compared to the remaining four intelligence groups, which in turn showed similar mortality rates (see Figure 1a). Moreover, an investigation of the mortality risk for women and men indicated that men in the lowest intelligence group seemed to exhibit an increased mortality risk compared to all other groups. Specifically, the mortality rate in men belonging to the lowest intelligence group was substantially higher than the mortality rate in women belonging to the lowest intelligence group (see Figure 1b). The mortality rates for men in the remaining four groups were also mostly higher than those for women, yet the differences between men's and women's mortality rates were smaller in these groups. This result pointed to an increased mortality risk for men in the lowest intelligence group.

Figure IV-1. *Premature mortality rates and frequency distribution of intelligence (IQ) scores in five equal-sized intelligence groups for the total sample (Figure 1a) and for women and men (Figure 1b)*



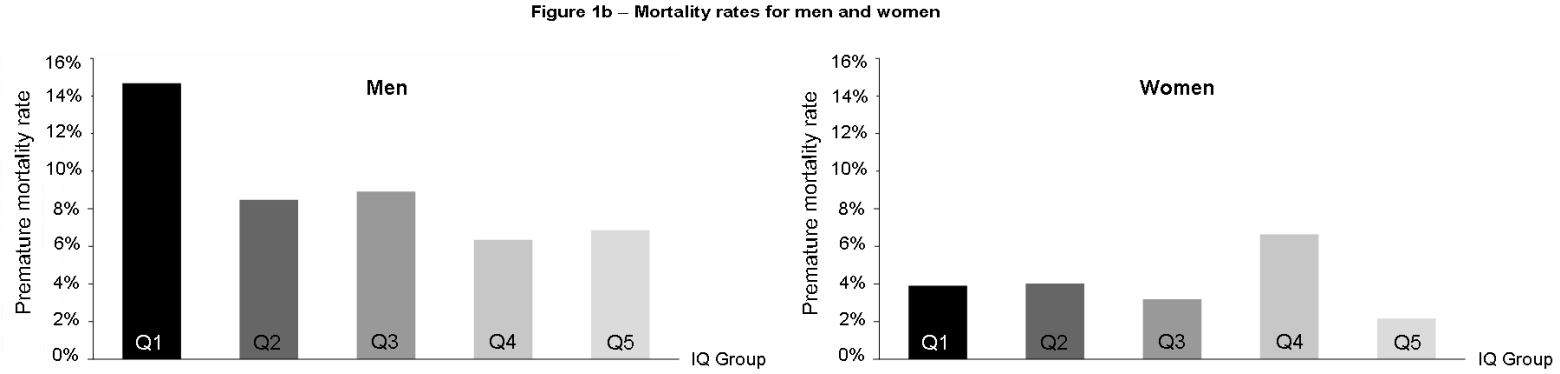


Table IV-2 (lower panel) shows the results of the second series of regression models that back up these conclusions. Our analyses suggested that the intelligence grouping variable significantly predicted mortality risk. Specifically, being in the lowest intelligence group increased the risk of dying by 2008 compared to being in the remaining four intelligence groups (Model 4; OR 1.63, 95% CI 1.14 to 2.32). This relation remained robust when controlling for childhood socioeconomic status and including gender in the model (Model 5). Importantly, there was a significant interaction between the intelligence grouping variable and gender (Model 6). Being a man in the lowest intelligence group increased the risk of dying by 2008 compared to being a man in the remaining intelligence groups or to being a woman in any group (OR 2.37, 95% CI 1.03 to 5.48).

4. Discussion

4.1 Discussion of the main findings

The principal findings of this prospective cohort study were: (1) Childhood intelligence predicted risk for all-cause premature mortality in Luxembourg, a country that offers universal access to quality health care. (2) The results are in line with the assumption that there is a high-risk group at the lower end of the intelligence distribution: Men, but not women, at the lower end of the intelligence distribution are at higher risk for premature mortality.

The first finding substantiates the broad generalizability of one of the core findings of the research on intelligence and mortality: Childhood intelligence does not lose its predictive power for mortality even in a country with universal access to quality health care. The finding of intelligence-mortality effects among comparatively young individuals before the regular onset of chronic diseases highlights the importance of intelligence as a predictor of mortality. Notably, our results were obtained when controlling for another important childhood variable,

namely, childhood socioeconomic status. This finding is important as Luxembourg has a level of social mobility below the OECD average (Organisation for Economic Co-operation and Development, 2010). Luxembourg's low social mobility indicates that—contrary to many modern societies (Mackenbach, 2010)—an individual's social achievement across the life course depends largely on the socioeconomic position of the individual's family of origin. Thus, childhood socioeconomic status could have acted as a partial proxy for adult socioeconomic status in our study and therefore could have been expected to yield the strongest effects on mortality. As a consequence, the impact of intelligence as a personal factor on health could have been smaller or even negligible in Luxembourg, compared to more meritocratic societies in which crucial life outcomes such as socioeconomic achievement depend more on personal factors. Importantly, intelligence had incremental effects on mortality in our study. Therefore, individual differences in childhood intelligence provide information that can be used to predict premature mortality over and above the socioeconomic background of a person's family.

The second finding, which indicated that individuals with low childhood intelligence exhibited an increased mortality risk, is in agreement with the results of other studies that have pointed towards a potential threshold effect (Hart et al., 2003, 2005; Kuh et al., 2004). Interestingly, men but not women in the lowest group of the intelligence distribution showed an increased mortality risk, in line with prior studies that found gender differences in the intelligence-mortality relation (Kuh et al., 2004; Lager et al., 2009; Pearce et al., 2006), and with gender differences in the rates and causes of premature mortality. In Europe, premature mortality rates for men are about twice as high as for women (Eurostat, 2009). The most important causes of premature mortality in Luxembourg are external causes of death (transport accidents, unintentional injuries), intentional self-harm (e.g., suicides), and alcohol-related mortality (Eurostat, 2009). For men between the ages of 25 and 64, factors

related to working environments also play an important role (Statec, 2009*)*. Crucially, the significant gender differences in the intelligence-mortality relation found in our study may be the result of a stronger association between intelligence and the risk factors and causes of premature mortality in men. First, many of the causes of premature mortality are strongly related to behavioral risks (e.g., risky driving), psychological and social risks (e.g., hopelessness and depression), or both (e.g., suicides), and all of them are more pronounced in men than in women (Eurostat, 2009; Statec, 2009). Importantly, intelligence may be directly and indirectly related to these causes. For instance, intelligence is inversely related to psychiatric disorders and suicide (Deary et al., 2010), unintentional injuries (Lawlor, Clark, & Leon, 2007; Osler, Nybo Andersen, Laursen, & Lawlor, 2007), motor vehicle accidents (O'Toole, 1990), and alcohol intake (Batty, Deary & Macintyre, 2006; Kubička, Matějček, Dytrych, & Roth, 2001). Intelligence as the ability to think, reason, deal with novel, complex, and unpredictable situations, identify problems, and react accordingly is one important factor that influences the probability of experiencing several of these events (e.g., unintentional injuries and accidents; Gottfredson, 2004). Therefore, low intelligence among men may have increased their risk for premature mortality.

Another important reason why men but not women with lower intelligence were at increased mortality risk may result from men being the principal earners in our study cohort. Consequently, the detrimental consequences of lower childhood intelligence, such as a lower educational attainment, a lower socioeconomic status in adulthood, and low problem solving, reasoning, and thinking skills (Gottfredson, 2002, 2004), may have been worse for men than for women. For instance, in the 1970s and 1980s, the iron, steel, and chemical industries in Luxembourg were important employers. Thus, men with lower childhood intelligence may have entered manual occupations with unsafe working environments that may have proven hazardous to their health. Crucially, as most industrial accidents happen while workers are

performing tasks that are complex or nonroutine and thus require them to solve new problems and use less-exercised skills (Gottfredson, 2004; Hale & Glendon, 1987; Saari, Tech, & Lahtela, 1981), men with lower intelligence may have had insufficient cognitive resources to deal with hazardous and novel situations, and thus may have been particularly at risk for work accidents. This finding is supported by another study on this cohort that showed that low childhood intelligence was significantly related to an increased number of sick days and doctor visits in adulthood (Wrulich et al., 2012). Moreover, men in the lowest intelligence group may have even been unemployed after primary school. These factors may have increased mortality risk (Kuh et al, 2004; Lundin, Lundberg, Hallsten, Ottosson, & Hemmingsson, 2010; Voss, Nylén, Floderus, Diderichsen, & Terry, 2004).

Taken together, our results point to a "chain reaction" of cumulative risks across the life course: Men with low levels of childhood intelligence may have unfavorable educational careers and enter low socioeconomic status environments later in life. These environments may be associated with a higher frequency of risk factors such as unemployment or unsafe working environments. These factors are also associated with riskier and unhealthier behavior, low motivation for bodily integrity and survival of the self (Eurostat, 2009), less social support, and worse mental health. Moreover, men's lower intelligence decreases their ability to cope with risk factors and hazards, prevent diseases and accidents, and self-manage health. All these factors may contribute to an increased risk for mortality, and as they are particularly relevant in men of working age, they may also explain the finding that childhood intelligence exerts a particularly strong influence on mortality before the age of 65 but not after the age of 65 (10 Hart et al., 2005). In general, our finding of differential relations between intelligence and mortality in women and men highlights the importance of environmental and behavioral factors in explaining the intelligence-mortality relation rather than intelligence being a marker of a healthy body in general (Lager et al., 2009).

4.2 Strengths and limitations

The current study features several notable strengths. First, we used a prospective longitudinal cohort design, thus adding to the small number of studies that have investigated the longitudinal relations between childhood intelligence and later mortality risk. Second, the present study investigated a nationally representative sample and was thus the first to investigate the intelligence-mortality relation in a Central European country with universal access to quality health care. Importantly, the present study controlled for childhood socioeconomic status, given the high impact socioeconomic family background has on an individual's later life achievement in Luxembourg. Third, whereas many previous studies have been based on data from men only, our study included data on men and women, thus enabling the systematic investigation of gender differences in the intelligence-mortality relation.

One important limitation of our study is the low number of deaths in our study sample. In particular, the lack of an effect in women may be the result of lower statistical power due to a smaller number of deaths in women (Calvin et al., 2011; Pearce et al., 2006). This could be due to the comparatively young age of our study sample in combination with women's higher average life expectancy. Investigating late life mortality instead of premature mortality may yield a higher number of deaths in women and may thus indicate no substantial gender differences in the intelligence-mortality relation. However, previous studies have yielded contradictory results regarding this notion (Kuh et al., 2004; Leon et al., 2009). Thus, further studies with large enough samples of women and men are needed to investigate potential gender differences in the intelligence-mortality relation at different ages. Another limitation of the current study is that we focused on the predictive power of childhood intelligence for mortality without including potential mediators of the intelligence-mortality relation, such as educational attainment and socioeconomic status in adulthood, risky behavior, or mental

health. Whereas it has been shown that educational attainment and socioeconomic status mediate this relation to some extent (Calvin et al., 2011), other studies have suggested an influence of intelligence on mortality independent of these mediators (Batty & Deary, 2005; Lager et al., 2009). Thus, future research should examine in greater detail the mediating processes that link childhood intelligence to later mortality.

4.3 Implications and conclusions

Our findings suggest that intelligence may be one important factor that could help to explain socioeconomic differences in mortality. In line with findings from other studies (Kuh et al., 2004; Lager et al., 2009), the gender differences in our study highlight the importance of behavioral, psychological, and social risk factors in the intelligence-mortality relation. These factors are potentially modifiable, which suggests that intelligence should be considered when devising interventions to promote health and longevity (Lager et al., 2009, 2010). Several implications for primary health care may therefore be derived. First, in light of the fact that intelligence early in life was found to be a risk factor for mortality later on, interventions targeting children (and especially boys) seem warranted. For instance, risk factors for children's intellectual and physical development (e.g., malnutrition) should be minimized. Supplementing pregnant women's, breast-feeding women's, and neonates' diets with long-chain polyunsaturated fatty acids has been shown to positively influence children's intelligence (Protzko, Aronson, & Blair, 2013) and could thus help to prevent health risks later on. Beyond that, interventions could target entire families, familiarizing children and parents alike with a healthy lifestyle (e.g., exercise and a healthy diet), for instance in schools or paediatrician's practices. Second, interventions targeting the risk group of men with low intelligence should be implemented across the life course. The aim of such interventions should be to reach individuals with lower intellectual abilities and make health care and preventive treatments accessible to them. Such interventions could include, for instance,

company doctors taking special care of high risk individuals and reducing the cognitive complexity of work situations in order to minimize the risk for work accidents, sick days, and even mortality. Moreover, interactions between health professionals and clients as well as tasks that are critical to health self-care could be adapted (e.g., obtaining preventative care and managing a chronic illness) with the objective of reducing unnecessary complexity when possible (e.g., simpler documents, fewer medications, or simpler dosing schedules). When possible, supplementary cognitive assistance could also be provided (e.g., more detailed feedback) to patients unable to cope with the inherent complexities of treatment and self-care (Batty et al., 2007). A concrete instance would be "targeted surveillance" (Deary et al., 2010, p. 72): A patient with lower intelligence could have his or her cardiovascular health monitored more regularly. This would be helpful for managing costs because regular and costly monitoring would be targeted towards those most at risk, whereas those who are less at risk could undergo less frequent, albeit still regular, monitoring. Increased surveillance of those at risk, although more costly in the short term, could lead to large savings for health-care organizations and societies gained from a reduced likelihood of hospitalizations and costly treatments.

With respect to more general policy implications, our results suggest that societal investments in early child rearing, a reduction of environmental risk factors for childhood intellectual and physical development (e.g., exposure to toxins; Evans, 2004), early interventions to foster intellectual abilities and education (e.g., the American CARE or Abecederian projects; Campbell et al., 2008), and other early human capital investments may provide manifold benefits to their recipients and, ultimately, to society (Heckman, 2006; Judge, Ilies, & Dimotakis, 2010). Investments in intelligence may produce economic, health, and social benefits.

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Chapter V

General Discussion

The major goal of this Ph.D. thesis was to answer five important open research questions regarding the relation between childhood intelligence and adult health outcomes: (1) Can the effects of childhood intelligence on adult health that were found in other countries be generalized to Luxembourg? (2) Does childhood intelligence predict the three subdimensions of adult physical health (physical, subjective, and functional health) equally well? (3) Do different facets of childhood intelligence, namely, general, fluid, and crystallized intelligence, predict adult health equally well? (4) To what extent do educational attainment and adult SES mediate the effects of childhood intelligence on the three subdimensions of adult health? (5) Does childhood intelligence predict adult mortality risk in Luxembourg, and if so, does it predict mortality risk in an incremental or threshold manner? These five questions were addressed in three distinctive studies. Their main findings will be reviewed below.

1. Review of the main findings

1.1 Does childhood intelligence predict adult health in Luxembourg?

This first research question was motivated by the fact that virtually all studies that have investigated intelligence as a predictor of adult health outcomes have been conducted in English-speaking or Scandinavian countries (Calvin et al., 2011; Deary, 2010). To investigate whether these effects can be generalized to Luxembourg is important for several reasons. First, from a general perspective, the universality of psychological processes can never be assumed in advance (Segall, Lonner, & Berry, 1998). This statement highlights the importance of extending findings gathered in one cultural setting to different settings to

establish that they are indeed universal. Second, investigating which factors influence health in Luxembourg is crucial for determining which factors should be targeted by interventions to improve population health. Further, knowledge of these factors may help to solve the fundamental cause controversy over the origins of socioeconomic health inequalities. Specifically, Luxembourg is a country with low levels of social mobility (Brunner & Martin, 2011; Organisation for Economic Co-operation and Development, 2010), which indicates that an individual's social and economic achievement depends largely on the socioeconomic position of the person's family of origin. Therefore, the impact of intelligence as a personal factor on health could be smaller or even negligible in Luxembourg compared to more meritocratic societies. Furthermore, Luxembourg provides universal access to quality health care (Huber, 1999; World Health Organization, 2000). Universal access to quality health care may compensate for some of the effects of individual differences in intelligence on health. Thus, both the low level of meritocracy and the universal access to health care could reduce or even offset the effects of intelligence on health in Luxembourg. Such a finding would indicate that external factors, such as high health care expenditure or socioeconomic family background, are more important in explaining health inequalities and should be the primary targets of interventions. On the other hand, if intelligence really is one of the fundamental causes of socioeconomic health inequalities, its influence over and above childhood SES should be detectable even in Luxembourg.

The answer to the first research question was positive: Study I showed that childhood intelligence predicted health 40 years later in Luxembourg, over and above childhood SES. Importantly, these effects were found in a comparatively young sample of adults. At the age of 52, chronic diseases that pose a threat to health in later life do not tend to occur very frequently. The study's main finding suggests that intelligence may be a potential cause of social class inequalities in health that cannot be fully offset even by quality public health care.

The finding that adjusting for childhood SES did not alter these effects to a large extent highlights the fact that contrary to other life outcomes in Luxembourg (e.g., educational achievement), which are substantially determined by socioeconomic family background, health across the life course is substantially influenced by personal factors such as intelligence.

1.2 Does childhood intelligence predict the three subdimensions of adult health equally well?

This second research question was motivated by the fact that childhood intelligence has mostly been investigated in relation to indicators of the physical subdimension of physical health in adulthood (*Intelligence*, 2009). However, its effects on the functional and subjective subdimensions have not yet been investigated to a large extent. Other researchers have emphasized that childhood intelligence may be differentially related to different aspects of adult health (Johnson, Corley, Starr, & Deary, 2011). Therefore, Study I investigated the effects of childhood intelligence on all three subdimensions of adult physical health.

The answer to the second research question was also positive: Study I showed that childhood intelligence significantly predicted indicators of all three subdimensions of physical health 40 years later. Specifically, higher general intelligence *g* scores in childhood significantly predicted a lower number of doctor visits and sick leave days as well as better functional and subjective health in adulthood. When controlling for gender and childhood SES, childhood general intelligence *g* still predicted a lower number of doctor visits and better functional health in adulthood.

1.3 Do different facets of childhood intelligence predict adult health equally well?

This third research question was motivated by the fact that most previous studies on the relation between childhood intelligence and adult health outcomes employed composite measures of general intelligence *g* as predictors (Calvin et al., 2011). However, intelligence is

a multifaceted hierarchically structured construct (McGrew, 2009): General intelligence *g* is located at the apex of the hierarchy, whereas more specific facets, such as fluid and crystallized intelligence, are located at the next lower level of the hierarchy. Investigating which facets of childhood intelligence (general, fluid, or crystallized intelligence) predict adult health is crucial as such results can be used to identify which aspects of intelligence are important in personal health management and should be targeted by interventions. If crystallized intelligence were the more important predictor, then interventions to foster education and health literacy could remediate deficits. If general or even fluid intelligence were more important, interventions would have to target general reasoning, abstract thinking, and problem solving skills.

The answer to the third research question was negative: Study I showed that the different facets of childhood intelligence were not equally powerful predictors of adult health. Specifically, childhood fluid intelligence was found to be the most important predictor as higher fluid intelligence significantly predicted a lower number of doctor visits and sick leave days as well as better functional and subjective health in adulthood, even when controlling for crystallized intelligence, childhood SES, and gender. Childhood fluid intelligence was an even stronger predictor of health than childhood general intelligence. Childhood crystallized intelligence did not predict any of the adult health outcomes when controlling for fluid intelligence, childhood SES, and gender.

1.4 To what extent do educational attainment and adult SES mediate the effects of childhood intelligence on adult health?

This fourth research question was motivated by the fact that previous research had yielded inconsistent results regarding the extent of mediation in the effects of childhood intelligence on adult health outcomes. Some studies have reported pronounced mediation, yet others have reported little or no mediation via educational attainment and further indicators of

later SES (Batty & Deary, 2005; Batty, Deary, Schoon, & Gale, 2007b; Batty, Gale, et al., 2008; Calvin et al., 2011; Deary, Weiss, & Batty, 2010). However, establishing the amount of mediation via socioeconomic outcomes is crucial for solving the fundamental cause controversy. Such information can help to determine whether intelligence influences health mainly via a "chain reaction" ranging from childhood intelligence to education and socioeconomic success to health, and/or whether intelligence influences health because it encompasses generic thinking skills that are key in personal health management. Moreover, previous results have indicated that the extent of mediation may depend on the time in life when intelligence is measured. If intelligence were measured after participants had already completed their educations, the intelligence-health relation could be confounded by education (Calvin et al., 2011). Study II appropriately addressed these questions by including intelligence measures that were obtained before the completion of primary education in Luxembourg.

The results of Study II confirmed the results of Study I as childhood intelligence had significant positive effects on all three subdimensions of adult physical health 40 years later. Specifically, childhood intelligence had significant positive effects on physical health (i.e., lower numbers of doctor visits, sick-leave days, and nights in the hospital), subjective health (i.e., a better subjective evaluation of one's own health status), and social-functional health (i.e., fewer limitations in everyday activities due to health problems). Importantly, these positive effects were entirely mediated via educational attainment and adult SES. The direct effects of childhood intelligence on the three health subdimensions were reduced to near zero in the full mediation models. The inclusion of adult intelligence in the mediation models did not alter the finding of complete mediation via socioeconomic outcomes. Further, adult intelligence did not significantly predict adult health in its own right. At first glance, these results could be interpreted as indicating that intelligence exerted its positive effects on health

not because of generic thinking skills that are crucial for health management in adulthood, but only because of is influence on socioeconomic outcomes such as educational and occupational success. However, separating the effects of intelligence and potential mediators (particularly education) on health is a complex issue, which will be discussed in greater detail in Section 2.1 of this chapter. What can be concluded from the results of our mediation analyses, however, is the generalizability of a "chain reaction" model (Gottfredson, 2002, p. 369). The protective effects of childhood intelligence on adult health accumulate across the life span. Early advantages in intelligence translate into a more successful educational career and subsequently into higher socioeconomic status in adulthood. These socioeconomic outcomes are in turn related to better adult health (Deary, 2010). Even high-quality public health care cannot fully offset the impact of these socioeconomic life outcomes on adult health (Lleras-Muney, 2005).

Interestingly, the mediation between childhood intelligence and adult health did not seem to operate in the same way for the three subdimensions of health. Childhood intelligence influenced adult functional and subjective health mainly through the positive effect of childhood intelligence on educational attainment and the positive effect of educational attainment on subsequent SES. By contrast, childhood intelligence influenced adult physical health mainly through the positive effect of educational attainment, without additional positive effects of subsequent SES. Altogether, these results suggest that educational attainment played a crucial role in the prediction of adult health. Therefore, its relations to childhood intelligence and adult health will be discussed in greater detail in Section 2.1 of this chapter.

1.5 Does childhood intelligence predict adult mortality risk in Luxembourg in an incremental or threshold manner?

This fifth research question was motivated by the fact that, as is the case for studies on intelligence and other health outcomes, virtually all studies on intelligence and mortality risk have been conducted in English-speaking or Scandinavian countries (Calvin et al., 2011; Deary, 2010). Thus, it remained unclear whether results that have shown that intelligence predicts mortality risk could be generalized to Luxembourg. Furthermore, there is a controversy in the literature over whether there is an incremental or a threshold effect of intelligence on mortality. Some studies have suggested an effect across the entire intelligence distribution, including the especially gifted (Batty, Kivimaki, & Deary, 2010; Lager, Bremberg, & Vagerö, 2009; Martin, & Kubzansky, 2005), whereas others have suggested a threshold effect, as individuals at the lower end of the intelligence distribution may exhibit a particularly increased mortality risk (Hart et al., 2003, 2005; Kuh, Richards, Hardy, Butterworth, & Wadsworth, 2004). Knowing the shape of the intelligence-mortality relation would provide important information on who in particular should be targeted by interventions and preventive measures: If intelligence influences mortality risk across the entire distribution, then interventions could be applied to anyone. By contrast, if there is a specific risk group at the lower end of the distribution, this group of individuals should be the primary target of interventions.

The answer to the fifth research question was positive: Study III showed that childhood intelligence significantly predicted risk for premature all-cause mortality in Luxembourg. Once again, it is important to note that these effects were shown in a comparatively young sample, before the usual onset of chronic diseases that may lead to higher mortality rates later in life. This finding substantiates the broad generalizability of the core findings of the research on intelligence and health outcomes: Next to three

subdimensions of adult physical health, childhood intelligence significantly predicts adult mortality, even in a country with universal access to quality health care. The results of Study III were obtained after adjusting for childhood SES. Thus, intelligence had incremental effects on mortality in Luxembourg, a country in which other important life domains such as social achievement depend largely on the socioeconomic position of the family of origin. Importantly, the effects of childhood intelligence on mortality risk seemed to be substantially driven by an increased mortality risk in the low intelligence group. This finding may indicate that individuals in the low intelligence group are exposed to persisting disadvantage in later life (Kuh et al., 2004). This lends further support to the notion of a chain reaction (Gottfredson, 2002): Low childhood intelligence leads to unfavorable educational careers and consequently to entering low SES environments later in life, and these environments are in turn associated with unemployment or unsafe working environments. These factors are not only associated with occupational health hazards, but also with riskier and unhealthier behavior, low motivation to preserve bodily integrity and one's survival (Eurostat, 2009), less social support, and worse mental health. All these factors may have contributed to an increased risk for premature mortality within the low intelligence group.

The remainder of this conclusive chapter will be structured as follows: First, I will discuss two important interpretational issues. Specifically, I will discuss the roles of intelligence, education, and their reciprocal relations in the prediction of health as well as the contribution of the present Ph.D. thesis to the controversy surrounding the causes of socioeconomic health inequalities. Second, I will address the limitations of the present Ph.D. thesis and potential implications for future research. Finally, I will outline potential policy implications.

2. Interpretational issues – intelligence, education, and the fundamental cause controversy

2.1 Intelligence, education, and the prediction of health outcomes

One major finding of the present Ph.D. thesis was that educational attainment and adult SES entirely mediated the effects of childhood intelligence on adult health. Specifically, childhood intelligence influenced adult functional and subjective health mainly through the positive effect of childhood intelligence on educational attainment and the positive effect of educational attainment on subsequent SES. By contrast, childhood intelligence influenced adult physical health mainly through the positive effect of educational attainment without additional positive effects of subsequent SES. These findings highlight the crucial role of educational attainment in this mediational chain, as it seemed to act as a kind of "gate keeper" that opened the potential for childhood intelligence to lead to higher SES and better health in adulthood. These results raise the question of which driving forces are responsible for the effects of intelligence and education on health in the present Ph.D. thesis. Is intelligence itself the driving force? Or is the driving force educational attainment? Or maybe even both? Do these two predictors of adult health constitute distinct influences, or are they interchangeable? These questions will be discussed below.

The finding that educational attainment was crucial in mediating the effects of childhood intelligence on later health in the present Ph.D. thesis is not surprising. Childhood intelligence significantly predicts educational success (Deary & Johnson, 2010; Deary, Strand, Smith, & Fernandes, 2007; Kuncel, Hezlett, & Ones, 2004), and educational success significantly predicts health (Cutler & Lleras-Muney, 2006; Cutler, Lleras-Muney, & Vogl, 2008). The positive effects of educational attainment on health may in part be due to the positive influence of education on adult SES and the effects of adult SES on health (Cutler & Lleras-Muney, 2006; Cutler et al., 2008). However, the positive effects of education on health

knowledge and behavior and on the readiness to adopt novel treatments and medical innovations seem to be even more important (Cutler & Lleras-Muney, 2006; Cutler et al., 2008). Moreover, education may provide better verbal and communication skills, enabling educated individuals to communicate symptoms more efficiently, understand medical advice, and follow prescriptions correctly (Cutler & Lleras-Muney, 2006; Johnson et al., 2011). Finally, education may have a positive influence on health because it improves critical thinking and problem solving skills, efficient learning, and the ability to self-manage health, all of which in turn positively influence health (Cutler & Lleras-Muney, 2006; Cutler et al., 2008).

Crucially, intelligence and education are highly correlated (Gottfredson, 2004; Deary et al., 2007). Moreover, a substantial part of the variations that have been found in intelligence and educational outcomes between individuals can be traced back to common genetic variations. For instance, about 40%-60% of the total observed (phenotypic) variation in educational attainment (as measured by years of education completed or tests of educational achievement) can be linked to genetic differences in general intelligence *g*. This does not mean that variations in education (e.g., in the number of years of education) are encoded in our genes, but they may in part reflect variations in intelligence, which in turn is partly genetically determined (Bartels, Rietveld, Van Baal, & Boomsma, 2002; Gottfredson, 2004). Finally, with respect to health, problem solving skills, efficient learning, and the ability to self-manage health are closely related to education, and these skills are crucial in predicting at least some health outcomes (e.g., chronic diseases). However, these skills closely correspond to the definition of intelligence as "[…] the ability to reason, plan, solve problems, think abstractly, comprehend complex ideas, learn quickly and learn from experience" (Gottfredson, 1997, p. 13). This finding points to a substantial overlap between intelligence and education in health matters (Cutler & Lleras-Muney, 2006; Cutler et al.,

2008). Altogether, these results have led to the assumption that education may serve as a partial surrogate for intelligence (Batty & Deary, 2005; Gottfredson, 2004) in that effects of education on health may actually reflect effects of intelligence on health. The notion of the identity of intelligence and education has received support from studies that have shown that tests such as the Scholastic Assessment Test (SAT), which are designed to measure educational achievement, actually seem to measure general intelligence *g* (Frey & Detterman, 2004). Further, analyses at the level of national aggregated data have shown that large-scale student assessment studies, such as the PISA study (Organisation for Economic Co-operation and Development, 2004), which are supposed to measure achievement in distinct academic subjects, may actually measure one single cognitive ability that is practically identical to general intelligence *g* (Rindermann, 2006, 2007; cf. Baumert, Lüdtke, Trautwein, & Brunner, 2009).

However, the notion that intelligence and measures of educational achievement are identical has been challenged. Baumert and colleagues demonstrated that educational achievement outcomes (e.g., success in academic subjects such as reading comprehension or mathematical literacy) can be conceptually distinguished from intelligence. Achievement in these outcomes indeed depends on abilities as measured by general, and more so, by fluid intelligence (i.e., reasoning ability) but also on domain-specific processes of knowledge acquisition and information processing, over and above intelligence (Baumert et al., 2009). Further, the authors provided evidence from construct validation studies that demonstrated that general intelligence *g* and educational achievement outcomes showed different relations to other characteristics such as gender or school grades (Baumert et al, 2009; Brunner, 2008). Finally, it has been highlighted that the high intercorrelations between measures of educational achievement and intelligence at an aggregated level cannot be interpreted as evidence that these tests indeed measure one single ability (Baumert et al. 2009).

Intelligence and educational achievement thus seem to be distinguishable constructs. Importantly, they have reciprocal influences. As mentioned previously, educational achievement depends substantially on intelligence. Intelligent students are able to grasp new tasks more quickly, have access to more effective problem-solving strategies, find it easier to identify relevant rules, and have greater processing capacity and more elaborated memory strategies, all of which are skills that are indispensable for success in education (Baumert et al., 2009; Gustafsson, & Undheim, 1996). In this view, (fluid) intelligence is invested into the acquisition of knowledge or crystallized intelligence (Cattell, 1987; McArdle, Ferrer-Caja, Hamagami, & Woodcock, 2002; see also Chapter I, Section 3.2). However, educational achievement also depends on domain-specific processes of knowledge acquisition, over and above intelligence. Moreover, the influence of intelligence decreases across the school career as students' competences and domain-specific prior knowledge increase. However, the predictive value of intelligence for educational achievement remains stronger in situations in which students are less familiar with the learning content and when less prior knowledge is available in the respective domain (Baumert et al., 2009). In addition to the influence of intelligence on educational achievement via an "investment" in processes of knowledge acquisition during a student's school career, these latter processes also influence the development of intelligence. Intelligence is therefore not only a condition, but also an outcome of academic learning, and different learning opportunities (as reflected, for instance, in different secondary education tracks) differentially influence intellectual and educational development (Ceci, 1991; cf. Baumert et al., 2009). Altogether, when learning opportunities are homogeneous in terms of content and structure—as was probably the case in primary school for the study sample of the present Ph.D. thesis—it is primarily differences in general intelligence at school entry or when new subjects are introduced that lead to differences in educational achievement. However, differences in educational achievement also depend on

prior knowledge. Once students are placed into situations that offer different learning opportunities based on their intelligence and domain-specific prior knowledge (e.g., in different secondary education tracks), these different learning environments differentially influence the development of intelligence. All these distinct processes may collectively lead to the high correlations between educational achievement measures and measures of intelligence (Baumert et al., 2009), and it is these high correlations that have been interpreted by other researchers as evidence for the identity of educational achievement and intelligence (Batty et al., 2007; Frey & Detterman, 2004; Gottfredson, 2004; Rindermann, 2006, 2007).

What are the implications of this controversy over an existing or non-existing identity of intelligence and educational attainment for the interpretation of intelligence-health relations? Were the educational measures included in the present Ph.D. thesis merely a surrogate for intelligence such that including them led to an over-adjustment of otherwise stronger direct effects of intelligence on health? Or were the intelligence differences that predicted health differences actually a partial surrogate for differential effects of education up to the time of measurement, as these differential effects are conceivable even in primary school with its relatively homogenous learning environment? In this case, including intelligence may have led to an over-adjustment of otherwise even stronger effects of education on health. And what potential effects could domain-specific aspects of education have on health, over and above the domain-independent intelligence component that is also important for educational achievement? To answer these questions, it may be helpful to consider how exactly intelligence helps a person to be successful both in educational settings and in health matters. As mentioned previously, intelligence is a key determinant of educational success as it is crucial in the acquisition of domain-specific knowledge and skills. Furthermore, intelligence is particularly important in situations in which a student is not familiar with the learning content and has less prior knowledge available (Baumert et al.,

2009). These are precisely the situations that led researchers to argue for the crucial role of intelligence in explaining socioeconomic health inequalities (see Chapter I, Section 2.3). They observed that intelligence best predicts key life outcomes such as educational achievement and job performance in the most cognitively complex situations (Gottfredson, 2004). For instance, intelligence predicts job performance primarily indirectly by promoting faster and more effective learning of essential job knowledge during both training and experience on the job. Yet, higher levels of intelligence also enhance job performance directly, such as when jobs require workers to solve novel problems, plan, make decisions, and the like. Intelligence appears to have increasing direct effects when jobs are less routinized or less closely supervised, more fraught with ambiguity and novelty (and hence are inherently less trainable), or otherwise require more independent judgment and innovative adaptation (Gottfredson, 2004; Schmidt & Hunter, 2004). Thus, the advantages conferred by higher levels of intelligence are successively larger in successively more complex jobs, tasks, and settings. Greater experience can compensate to some extent for lower levels of intelligence, but experience can never negate the disadvantages of information processing that is slow or prone to errors. To the extent that everyday tasks mirror tasks performed at work, such advantages and disadvantages associated with intelligence are felt in many spheres of life (Gottfredson, 2004). Crucially, it has been suggested that being a patient is one of these spheres of life, a sphere of life that, due to advances in medical and health care, is becoming ever more complex. Analyses of the "job of being a patient" (Gottfredson, 2004, p. 175) show that it requires the same cognitive skills that intelligence represents and that most jobs and also educational success require for good performance: efficient learning, reasoning, and problem solving. For instance, managing chronic diseases such as diabetes is somewhat similar to jobs and school subjects that require considerable knowledge for good performance. However, because conditions keep changing, the job of (chronic) disease management cannot

be routinized. Chronic diseases therefore require constant judgment in applying old knowledge and the need to spot and solve new problems. This always requires, to some extent, the exercise of intelligence (Gottfredson, 2004, 2009). In addition, the job of being a patient may require domain-specific abilities over and above general intelligence, such as reading comprehension. For instance, not being able to read medical prescriptions can be hazardous to one's health. Approximately 10% of all hospitalizations and 23% of all nursing home admissions in the U.S. are attributed to patients' inability to correctly understand prescription labels and take drugs accordingly (Berg, Dischler, Wagner, Raia, & Palmer-Shevlin, 1993). Likewise, reading comprehension is required for understanding informed consent forms. Williams and colleagues showed that 60% of the patients in two urban hospitals in the U.S. did not understand a standard informed consent document (Williams et al., 1995; cf. Gottfredson, 2004). Moreover, even domain-specific mathematical abilities may be important. For instance, calculating the dosage of a drug that should be administered to a sick child with a specific sex, age, or weight is an important task for parents (Gottfredson, 2009). Further, calculating how many pills of a certain drug should be taken per day is a standard task for patients and is therefore also included as an item in the Test of Functional Health Literacy of Adults (TOFHLA; Parker, Baker, Williams, & Nurss, 1995). Like low reading comprehension, low mathematical abilities may therefore contribute to incorrectly following medical prescriptions with potentially serious consequences. Finally, knowledge is one predictor of health. This may be health-related knowledge that is acquired during education or the advantages in the ability to acquire knowledge that go along with educational success. Research has shown that individuals who are well informed in one area are well informed in many other areas, too, and that advantages in the amount of information gathered are due to advantages in the acquisition of knowledge from the mass media. Education has been shown to be an important correlate of such an ability to acquire publicly

available knowledge (Gottfredson, 2004; Tichenor, Donohue, & Olien, 1970).

In a nutshell, it seems probable that the indirect effects of childhood intelligence on health via education that were found in the present Ph.D. thesis encompass both general and domain-specific components. The general component will most likely be comprised of the reasoning, problem solving, abstract thinking, and efficient learning abilities that have been labeled fluid intelligence and that are also at the heart of general intelligence *g* (Blair, 2006; Undheim & Gustafsson, 1987; Valentin Kvist & Gustafsson, 2008). Study I of the present Ph.D. thesis showed that fluid intelligence, rather than general or crystallized intelligence, was the strongest predictor of health. Thus, it was not the differences in general knowledge that stem from prior learning and past experience per se as reflected in tests of crystallized intelligence, but the fluid abilities that are invested into crystallized intelligence that were crucial. Other studies have also shown that fluid intelligence is crucial for educational attainment (Baumert et al., 2009). Further, the general abilities that are subsumed under the term fluid intelligence are also crucial for health management (Cutler & Lleras-Muney, 2006; Gottfredson, 2004). Thus, the effects of educational attainment on health may at least in part have acted as surrogates for the effects of intelligence on health. This may also have been the reason why adult intelligence did not significantly predict adult health in its own right in a complete mediation model that included adult intelligence next to childhood intelligence, educational attainment, and adult SES (see Chapter III on Study II). However, it is also likely that educational attainment had significant unique effects on health over and above the effects of general thinking skills. These effects will most likely have been due to beneficial effects of education on health behavior, a higher general and health-related knowledge conferred by education, a greater readiness to adopt novel treatments and medical innovations (Cutler & Lleras-Muney, 2006; Cutler et al., 2008), and domain-specific abilities acquired during education that may also play a role in health management. These specific abilities may

encompass reading comprehension and communication skills, which enable patients to communicate symptoms more efficiently, understand medical advice, and follow prescriptions correctly (Cutler & Lleras-Muney, 2006; Johnson et al., 2011). To a lesser extent, even domain-specific mathematical abilities may be important for the job of being a patient. However, further causally informative studies are required to disentangle such possibilities (Deary et al., 2010).

The next section will discuss the implications of the results obtained in the present Ph.D. thesis, especially regarding the above-mentioned interplay between intelligence and education in health matters, with respect to the controversy over the fundamental causes of socioeconomic health inequalities.

2.2 Contributions of the present Ph.D. thesis to the fundamental cause controversy

As mentioned in Section 4.3 of Chapter I, intelligence has been suggested as the fundamental cause of socioeconomic health inequalities instead of the "flexible resources" that are associated with a higher socioeconomic status and that are held responsible for health inequalities in sociological approaches (Gottfredson, 2004; Gottfredson & Deary, 2004; Link & Phelan, 1995; Link, Phelan, Miech, & Westin, 2008). This hypothesis is based on several observations. First, intelligence is a content- and context-free ability. Thus, it is useful in different circumstances, times, and places. Second, higher childhood intelligence significantly predicts a wide range of adult health outcomes. Third, intelligence is highly stable across the life course. Thus, childhood intelligence not only predicts adult SES and adult health but adult intelligence as well. Therefore, childhood intelligence could influence adult health through its influence on intellectual ability in adulthood. Fourth, intelligence test scores are socially patterned, as children and adults from socially deprived backgrounds typically have worse results. Thus, low SES groups are more homogenously composed of individuals with low intelligence (Batty et al., 2010; Gottfredson, 2004; Mackenbach, 2012;

Neisser et al., 1996). Fifth, health nowadays depends more than ever on private precaution and health lifestyle, which comprises maintaining one's health, protecting oneself against chronic disease and accidents, and adhering to complex treatment regimens. To accomplish these tasks, cognitive competences as measured by psychometric intelligence tests in childhood and adulthood are required (Gottfredson, 2004; Gottfredson & Deary, 2004; see also Section 2.1 of this chapter).

The results of the present Ph.D. thesis partially support this notion. Childhood intelligence, and especially childhood fluid intelligence, which are largely content- and context-free abilities, significantly predicted different adult health outcomes. These effects were shown in Luxembourg, a country with a low level of meritocracy and with universal access to health care. Both factors could have reduced or even offset the effects of intelligence on health. Thus, the reasoning and problem solving skills associated with intelligence in childhood and adulthood indeed may be responsible for differences in adult health. However, other results of the present Ph.D. thesis challenge the notion of intelligence as the fundamental cause. Most importantly, the effects of childhood intelligence were entirely mediated via education and further socioeconomic outcomes. Further, adult intelligence as an indicator of general thinking and problem solving did not mediate any effects of childhood intelligence on health, nor did it predict adult health in its own right. These results highlight the importance of childhood intelligence for adult socioeconomic and health outcomes but could be interpreted as supporting the sociological approach, as the advantages and flexible resources associated with a higher education and a better subsequent SES could be the key factors in that influence health (Link et al., 2008). However, as pointed out in Section 2.1 of this chapter, the complex interplay between intelligence and education prevents the drawing of such definite conclusions. Moreover, the limitations of the present Ph.D. thesis with respect to the timing of the intelligence measurement and the use of health

measures also preclude ruling out intelligence as an ability that directly influences health management (see Section 3 below). It seems more plausible that the general thinking and problem solving skills associated with intelligence are crucial for both educational success and good health. Thus, intelligence is most probably not the only but rather one important cause of socioeconomic health inequalities. This interpretation has been supported by other studies that have shown direct effects of childhood intelligence on health over and above its indirect effects mediated via education and adult SES (Hart et al., 2003; Batty, Gale, et al., 2008). Altogether, whether intelligence influences health only indirectly via a chain reaction and/or directly via generic thinking skills could not be determined definitely in the present Ph.D. thesis. However, "[…] what happens in the mind, whether the influences come from the material world or the social, has to be taken into account if we are to understand how the socio-economic circumstances in which people live influence health and well-being" (Marmot & Kivimäki, 2009, p. 1820).

3. Limitations and directions for future research

The three studies that constitute the present Ph.D. thesis showed that childhood intelligence has substantial effects on different socioeconomic and health outcomes in adulthood. However, these studies have several important limitations. The results and limitations of the three studies can be used to derive several directions for future research.

First, with respect to the multidimensionality and the measurement of health, Studies I and II showed that childhood intelligence predicted all three subdimensions of adult physical health, namely, physical, functional, and subjective health. However, the results of Study II showed that these dimensions cannot be treated interchangeably. When considering the mediational pathways by which intelligence influences the three dimensions, Study II showed that educational attainment and adult SES mediated the effect of childhood intelligence on adult functional and subjective health, whereas only educational attainment mediated the

effect of childhood intelligence on physical health. One important limitation of Studies I and II, however, is that they employed only self-reported health measures. These health measures may be subject to several problems, such as reporting biases (e.g., the tendency to exaggerate or conceal a health problem; McDowell, 2006). These biases may be due to different reasons. For instance, personality traits such as stoicism or the need for attention may influence the willingness to report health problems. Likewise, the drive to portray oneself in a good light by providing socially desirable responses or attitudes toward the topics in question may influence self-reports (Bradburn, Sudman, Blair, & Stocking, 1978; McDowell, 2006). Importantly, it is possible that with different health measures (e.g., objective health indicators such as the metabolic syndrome; Batty, Gale, et al. 2008), Study II would indeed have found small direct effects of childhood intelligence instead of complete mediation via education and subsequent SES. However, self-reported health measures also have important advantages. First, they extend the information obtainable from morbidity and mortality statistics by describing the quality rather than the mere quantity of a certain health outcome (McDowell, 2006). Second, empirical research has confirmed that self-reported health measures are reliable and valid measures of health. Specifically, self-reported health measures such as general subjective health ratings have been shown to be independent predictors of a range of health outcomes, including morbidity, use of health services, and mortality (Benyamini & Idler, 1999; Benyamini, Leventhal, & Leventhal, 1999; Idler & Benyamini, 1997; Idler & Kasl, 1995). For instance, in a comprehensive meta-analysis, DeSalvo, Bloser, Reynolds, He, and Muntner (2006) showed that a single general self-rated health question was highly predictive of later mortality risk, even after adjusting for key covariates such as comorbidity, and even in studies with long time periods before follow-up. Some studies have even shown that self-reports have greater predictive utility than more "objective" measures such as reports by medical personnel (e.g., Ferraro & Su, 2000; Mossey & Shapiro, 1982).

Altogether, these results and limitations of Studies I and II suggest that future studies on the intelligence-health relation should simultaneously investigate different dimensions of adult health, if possible, and not focus merely on indicators of physical health. Further, even though subjective, self-reported health measures have important advantages, a variety of subjective and more objective indicators of each health dimension should be included (McDowell, 2006). Objective indicators could include diagnosed diseases or biomarkers associated with chronic disease and health conditions (e.g., Body Mass Index [BMI] or systolic blood pressure). Subjective indicators could include self-reports on the presence or absence of diseases or symptoms, subjective evaluations of an individual's health status, or "functional disability indicators" that have participants report the impact of health problems on various areas of their everyday functioning (McDowell, 2006). Further, measures of mental health (e.g., the Beck Depression Inventory; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), and social health (e.g., indicators of social and instrumental support or the ability to fulfill social roles; Liang, 1986; McDowell, 2006) should be included. To preserve a feasible and economic study design, health measures that combine the physical, mental, and social dimensions of health in one instrument could be employed (e.g., general health status measures such as the SF-36; Ware & Gandek, 1998; or the SF-20; Stewart, Hays, & Ware, 1988). The inclusion of various indicators of different health dimensions would help to shed further light on the different microprocesses by which childhood intelligence influences later health outcomes.

Second, with respect to the multifaceted nature of intelligence, Study I showed that different intelligence facets do not predict adult health equally well. Childhood general intelligence *g* had significant effects on all three subdimensions of adult physical health when investigated as the sole predictor. However, some of these effects of childhood general intelligence were reduced to non-significance when childhood SES and gender were

controlled for. By contrast, the effects of childhood fluid intelligence on all three adult health dimensions were barely affected when childhood SES, gender, or crystallized intelligence were taken into account. Crystallized intelligence in turn did not significantly predict any health outcome when adjusting for childhood SES, gender, or fluid intelligence. These results suggest that it is the skills measured by fluid intelligence tasks, such as reasoning, abstract thinking, and problem solving, that are crucial in the prediction of later health. The observation that the effects of general and fluid intelligence pointed in the same direction yet were stronger for fluid intelligence is not surprising as these two constructs are closely related. Some studies have suggested that general and fluid intelligence may even be identical (Undheim & Gustafsson, 1987). However, this is not a universal finding. It seems more probable that general and fluid intelligence are closely related yet not identical constructs (Blair, 2006; Valentin et al., 2008). Crucially, most studies that have investigated the effects of early life intelligence on later health to date have used composite measures of early life general intelligence *g* (Calvin et al., 2011). These measures reflect the intelligence component that is to some extent present in basically all intellectual tasks (fluid intelligence, crystallized intelligence, etc.) and thus is responsible for the intercorrelations among these tasks (Carroll, 1993). As our results suggest that fluid intelligence seems to be the most important intelligence measure for predicting later health, future studies should employ intelligence measures that reflect this fluid facet. In fact, it is to be expected that the higher the fluid component in these measures, the stronger the effect on later health will be.

Third, with respect to mediation via socioeconomic outcomes, Study II showed that educational attainment and adult SES are important mediators of the intelligence-health relation. This result is in line with other studies that investigated these mediators (Deary, 2010; Deary et al., 2010). However, one important limitation of this Ph.D. thesis is that the interplay of education and SES with other potential mediators could not be addressed. For

instance, health-preventive and health-compromising behaviors (e.g., attending preventive doctor appointments, physical activity, smoking, and alcohol intake; Batty et al., 2007a, 2007b; Batty, Deary, & Macintyre, 2006; Batty, Deary, et al., 2008), measures of social integration (social support or social influences on health behavior; Berkman, Glass, Brissette, & Seeman, 2000), and risk factors for disease (e.g., blood pressure, obesity; Batty et al., 2007a; Chandola, Deary, Blane, & Batty, 2006) could have been further mediators. Future studies should therefore include these and other potential mediators of the intelligence-health relation as the inclusion of these variables would allow researchers to examine the microprocesses involved in how intelligence, education, and socioeconomic status translate into better adult health and the relative importance and interplay of different mediating processes.

Fourth, the results and limitations of all three studies of the present Ph.D. thesis offer directions for the inclusion of education and intelligence in future studies. Specifically, childhood intelligence was assessed at the average age of 12 years. This age marks the end of primary education in Luxembourg and thus guaranteed a comparable educational starting point for all participants, as the different effects of different secondary education tracks on intellectual development were controlled for. However, as was shown in Section 2.1 of this chapter, intelligence and education have reciprocal effects early in life, possibly even at the age of 12. This fact renders the task of disentangling the unique effects of intelligence and education on health extremely difficult, a hurdle that was particularly relevant for Study II, as this study investigated the effects of both intelligence and education on later health. Specifically, some variability in the intelligence scores obtained at the age of 12 may have been due to the differential effects of education on intellectual development up to that time point. Thus, the effects of education on health found in Study II may have been underestimated. On the other hand, it has been suggested that education may act as a

surrogate for intelligence (see Section 2.1). Thus, some of the differences in education may have actually reflected differences in intelligence. As a consequence, the effects of intelligence on health found in Study II may have been underestimated. In future studies, it would therefore be preferable to measure intelligence early in life in order to disentangle reciprocal effects between intelligence and education. Further, intelligence should best be measured at several time points across the (early) life course. Several measurements would allow for a more detailed investigation of the interplay between intelligence and environmental influences such as education. Moreover, measuring intelligence at several points in time would allow for a detailed investigation of the reciprocal influences between intelligence and health. Intelligence may directly or indirectly affect health, but health may also affect performance on intelligence measures. For instance, ill health in childhood or early adulthood may have detrimental effects on intelligence test performance. This effect has been coined "reverse causality" (Deary et al., 2010, p. 61). Altogether, cross-lagged designs seem preferable for investigating the relations between childhood intelligence, different mediators, and different dimensions of adult health.

Fifth, the sample sizes for our Studies I and II were comparatively large and should have been sufficient for detecting even small effects (Cohen, 1988). However, the results of Study II showed that the confidence intervals for the non-significant direct effects of intelligence on health were relatively wide. Thus, the possibility that childhood intelligence has small direct positive effects on adult health in the population could not be ruled out completely. This is of particular importance as ruling out direct effects of intelligence on health was crucial to show that these effects are indeed completely mediated via education and SES. Further, Study II showed some tendential differences between women and men in the prediction of adult health from childhood intelligence and in the mediational pathways linking the two variables. These gender differences were not significant. However, we

investigated the differences between women and men with multiple-group models. For these models, the total sample was split into two separate groups with smaller respective subsample sizes. Thus, the non-significant gender differences in Study II may have been due to the loss of power that occurred when the multiple-group models were applied. Altogether, these results and limitations suggest that large sample sizes seem advisable for obtaining precise estimates of the direct and indirect effects of intelligence on health outcomes and for investigating gender or other group differences with large enough samples in every subgroup.

More generally speaking, our results, as well as the results obtained in other studies on intelligence and health outcomes, suggest that intelligence needs to be incorporated into health and epidemiological research (e.g., Gottfredson, 2004; Gottfredson & Deary, 2004; Lubinski & Humphreys, 1997). Socioeconomic health inequalities are not solely a function of socioeconomic factors (Mackenbach, 2010, 2012; Reeve & Basalik, 2010), and our results suggest that intelligence may be one important potential cause of these inequalities. Thus, leaving intelligence unaccounted for in health research may lead researchers to search for explanations for health inequalities without ever being able to formulate fully explanatory models. Moreover, ignoring intelligence when devising policies and interventions to reduce these inequalities could have serious consequences. The reduction of socioeconomic health inequalities is an important goal of many policy makers, national, and international organizations (e.g., World Health Organization; Wilkinson & Marmot, 2003). To be maximally effective, these interventions must be based on accurate information regarding all factors that cause and maintain health inequalities. Thus, interventions that ignore intelligence would be, to some extent, "flying blind" (Reeve & Basalik, 2010, p. 288), and focusing solely on economic factors would cause them to fall short of their goals or to target the wrong causes. The next subchapter will therefore make several suggestions for policy makers, health care personnel, and others concerned with public and individual health on how

to incorporate intelligence into interventions to reduce socioeconomic health inequalities and to render health care and preventive measures maximally effective for everyone.

4. Practical and policy implications

The practical and policy implications presented in this subchapter will be divided into two broad domains according to the temporal order of their potential application. First, the present Ph.D. thesis demonstrated that intelligence in early life is an important predictor of health outcomes 40 years later. Thus, factors influencing intellectual and physical development early in life and interventions to increase positive and decrease negative influences on this development will be presented. Second, intelligence in adulthood may be important in managing the job of being a patient (Gottfredson, 2004). Specifically, intellectual skills such as reasoning and problem solving skills may substantially influence who will be reached by public health care and preventive measures, who will benefit from quality health care, and who is capable in managing health and disease. Thus, interventions that consider these factors in adulthood will be presented.

4.1 Practical and policy implications that focus on childhood

The first question that may come to mind when thinking about interventions that target early life intelligence may be: Can intelligence be influenced at all? Is our intelligence not something that has been "given" to us at birth, something that is predetermined by our genes, thus being forever unalterable? As a matter of fact, intelligence runs in families. For instance, the average correlation between intelligence test scores of biological parents and their offspring and for siblings raised together is about $r = .45$. In general, about half of the variation in intelligence scores between individuals in a population is attributable to genetic differences among them (Plomin & Petrill, 1997). Importantly, however, this does not at all imply that a particular individual's intelligence is to 50% caused by his or her genetic

makeup and is therefore nonmalleable. Estimates of the heritability of intelligence provide just an average impression of how much of the differences in intelligence scores between individuals in a population are attributable to genetic differences between them. This has two important implications for interventions that target early life intelligence: First, this means that the other half of the variance in intelligence scores between individuals is not genetic. Second, as 50% is an average population estimate, this means that the importance of environmental factors may be even greater than 50% for a particular individual's intelligence. Moreover, the heritability estimates of differences in intelligence between individuals increase with age, suggesting that environmental factors play a larger role in early childhood (Plomin & Spinath, 2004). This finding has a third important implication for interventions that target early life intelligence: Intelligence early in life is particularly malleable and can be influenced by a variety of environmental factors. Given that environments are modifiable, these results suggest a variety of promising starting points for interventions to increase positive influences and decrease negative influences on early life intelligence.

A first starting point for early interventions is the socioeconomic environment a child is raised in. Even though our studies have shown that childhood intelligence influences adult health and mortality over and above the effects of childhood SES, childhood intelligence and SES are interrelated (Bradley & Corwyn, 2002; Johnson et al., 2011). Specifically, the socioeconomic environment a child is raised in has a substantial impact on both the intellectual and physical development of the child. This is particularly important for children from low-SES families. For instance, children from low-SES families experience substantially less cognitive stimulation and enrichment in comparison to children from high-SES families (Coley, 2002; Larson & Verma, 1999). Family turmoil and discord as well as nonresponsive and harsh parenting are more frequent in low-SES families and affect socio-emotional as well as cognitive and physical development (Evans, 2004; Repetti, Taylor, &

Seeman, 2002; Taylor, Repetti, & Seeman, 1997). Further, cognitive enrichment activities such as quantity and quality of parent-to-child speech and exposure to print media are less frequent in low-SES families (Hart & Risley, 1995; Kagan, Tulkin, 1971; Neumann & Roskos, 1993). Moreover, being raised in low-SES families is often associated with polluted, unhealthy environments. For instance, exposure to toxins such as lead and pesticides along with living in areas with poorer air and water quality can cause physical health problems and cognitive deficits in children (Evans, 2004). Adverse physical and cognitive developmental outcomes are accelerated by exposure to multiple risks relative to singular risk exposure (Evans, 2004). Given that the unequal distribution of material resources such as income and occupation are an ecological reality, interventions aimed at reducing the risk factors that have particularly potent risks for both childhood physical and intellectual development seem particularly crucial given their immediate and long-term detrimental effects on health. Those interventions could include nutrition, of both pregnant mothers and the infants themselves. For instance, supplementing pregnant women's, breast-feeding women's, and neonates' diets with long-chain polyunsaturated fatty acids has been demonstrated to raise intelligence (Protzko, Aronson, & Blair, 2013). Moreover, interventions could encourage women to breastfeed as this simple action has been shown to have a positive impact on childrens' intellectual level (Anderson, Johnstone, & Remley, 1999; Lucas, Morley, Cole, Lister, & Leeson-Payne, 1992; Mortensen, Michaelsen, Sanders, & Reinisch, 2002). Other interventions could attempt to reduce environmental toxins and to increase air and water quality. Further interventions could target early child-rearing practices. For instance, parents should be advised to talk more to children, encourage their curiosity, and provide them access to learning materials and outside learning experiences (Hart & Risley, 1995). Moreover, reading to children in an interactive manner has been shown to positively influence children's intelligence (Protzko et al., 2013).

A further potential starting point for early interventions is childhood intelligence itself. Childhood intelligence is fundamentally influenced by experience, schooling, and abstract reasoning. As just one instance, sending a child to preschool significantly raises the child's intelligence (Protzko et al., 2013). This finding is in line with modern ideas about the plasticity of the brain (Vagerö, 2011). Study I showed that fluid intelligence is a particularly powerful predictor of adult health and would therefore be a potential target for interventions. This notion is supported by results suggesting that the utilization and repeated practice of pre-frontally based fluid cognitive skills that begin relatively early in life are likely to lead to relatively enduring changes in performance of measures of fluid intelligence (Blair, 2006: Blair, Gamson, Thorne, & Baker, 2005). This "neurodevelopmental-schooling hypothesis" (Blair et al., 2005, p. 93) has also been used to explain the fact that the mean level of intelligence test performance has been rising worldwide over successive age cohorts throughout the 20th century (the so-called "Flynn effect"; Dickens & Flynn, 2001). Incidentally, mortality fell across much of the same period, giving support to the hypothesis that intelligence and health may be causally connected and that they share closely related environmental determinants (Vagerö, 2011). Moreover, schooling has substantial beneficial effects on different cognitive abilities including fluid intelligence, and there is evidence that schooling causes real changes in these abilities rather than merely surface-level effects due, for example, to the acquisition of specific pieces of information (Cliffordson & Gustafsson, 2008)

Thus, it appears that, if potential problems in a child's intellectual development are detected, early life educational programs could be utilized to foster the child's intelligence, and this fostering in turn may have beneficial effects on the child's adult health. This may be especially true for children at the lower end of the intelligence distribution, as Study III of the present Ph.D. thesis showed that low childhood intelligence is a particular risk factor for

premature mortality. Recent reviews of early learning and school readiness interventions found that these programs resulted in marked improvements on tests of reading, arithmetic ability, fluid intelligence, and general intelligence and that these improvements appeared to extend to secondary school ages (Camilli, Vargas, Ryan, & Barnett, 2010; Ramey & Ramey, 2004). However, with a modest duration to follow-up in these interventions, the extent to which these improvements are maintained across the life course is not clear. If the effects of childhood intelligence on later health outcomes reported in the present Ph.D. project are genuinely causal, these early interventions may have an impact on health many decades later. Although problematic given the logistical considerations, the long-term follow-up of participants in such intervention studies would provide valuable insights into whether this could be the case. Next to these educational programs, childhood intelligence could potentially be enhanced via working memory exercises, which have been shown to increase levels of fluid intelligence (Jaeggi, Buschkuehl, Jonides, & Perrig, 2008; Mackey, Hill, Stone, & Bunge, 2011; Sternberg, 2008), cognitive control (Chein & Morrison, 2010; Klingberg, Forssberg, & Westerberg, 2002), and reading comprehension (Chein & Morrison, 2010) in some studies. Such exercises are easy and quite cheap to introduce from a very young age via games like memo both at home and in school environments. However, the beneficial effects of working memory training are subject to considerable debate. For instance, Chooi and Thompson (2012) attempted to replicate the beneficial effects on fluid intelligence found by Jaeggi and colleagues (Jaeggi et al., 2008) and did not succeed. They concluded that increasing one's working memory capacity by training and practice could not transfer to improvements in fluid intelligence. Moreover, Buschkuehl and Jaeggi themselves (2010) asserted several important limitations of the research on working memory training so far. For instance, they criticized the repeated usage of identical test material within a study in order to measure intelligence because practice on the same items considerably lowers the sensitivity

of the test to assess intelligence processes. They further highlighted that only a very restricted range of intelligence tasks has thus far been used in studies on the effects of working memory training and that there is little evidence that the effects go beyond laboratory tasks to standardized measures or even academic achievement or into daily life in general. Finally, they emphasized that few studies have looked into the long-term effects of interventions. They concluded that to date, it is not known how long potential improvements last or whether "booster-sessions" may be beneficial for maintaining performance (Buschkuehl & Jaeggi, 2010, p. 270). Other critics have expressed concerns regarding the inconsistent use of appropriate control groups in working memory training studies (Shipstead, Redick, & Engle, 2012). The authors concluded that the present literature provides insufficient evidence of the efficacy of working memory training. Until further evidence is provided, these trainings do not constitute the most promising interventions to positively influence childhood intelligence and its long-term effects on health.

Altogether, the development of interventions and policies that create equal opportunities for child development is an urgent priority and could help to reduce health inequalities (Mackenbach, 2010). Improvements in early family and social circumstances as well as in schooling represent opportunities for these kinds of public health interventions. One of the ways through which such interventions will work appears to be through their effect on cognitive development and intelligence (Vagerö, 2011). Promoting optimal early development could thus improve both intelligence and later health. Beyond childhood intellectual and physical development, interventions have further potential starting points in adulthood. More specifically, the goal of such interventions is to make all sorts of health care, preventive measures, and treatments accessible to individuals with lower intellectual abilities.

4.2 Practical and policy implications that focus on adulthood

In contrast to childhood interventions, interventions in adulthood do not target environmental factors that influence intellectual and physical development. Neither do they aim to foster intelligence in a general manner. These interventions rather aim to reach individuals with lower intellectual abilities and make public health care and preventive measures or treatments accessible to them. Ultimately, a major goal of these interventions is to prevent these individuals from "being lost" to health care.

The results of the present Ph.D. project showed that it is general reasoning, abstract thinking, and problem solving skills, as reflected in measures of general, but more so in measures of fluid intelligence, that seem to be important driving forces behind the effects of childhood intelligence on adult health. They may thus also be important in the successful management of a person's health and health behaviors in adulthood. Moreover, our studies showed that universal health care cannot offset these effects. Other studies have shown that other public health care measures, such as health promotion campaigns, do not reach those most at risk and that intelligence is considered to be one explanatory factor for this finding. Even though the public may become better informed about a certain health topic due to these campaigns (e.g., about the warning signs of cancer), knowledge is not equalized. The previously informed (mostly high-SES individuals) become better informed, whereas the previously uninformed (mostly low-SES individuals) remain uninformed. Thus, inequalities in knowledge remain or even grow (Feldman, 1966; cf. Gottfredson, 2004; Tichenor, 1970). This could mean that individual intelligence levels should be considered when preparing health-promotion campaigns and in the health professional–client interaction. For instance, tasks that are critical to health self-care could be adapted (e.g., obtaining preventative care and managing a chronic illness) with the objective of reducing unnecessary complexity when possible (e.g., simpler documents, fewer medications, or simpler dosing schedules). When

possible, supplementary cognitive assistance could also be provided (e.g., more detailed feedback) to patients unable to cope with the inherent complexities of treatment and self-care (Batty, Deary, & Gottfredson, 2007). The strategies for rendering health communications more comprehensible to patients with low intellectual abilities could follow the guidelines for simplifying written materials that were developed by Army researchers who sought to enhance the work literacy of low-ability soldiers (Sticht, 1975; cf. Gottfredson, 2004). These guidelines constitute a primer for reducing the complexity and content of information. Among these guidelines are: Omit all nonessential information; describe the specific behavior required of the individual; use simple vocabulary; require reading no higher than a fifth-grade level; use simple line drawings (photographs contain distracting, irrelevant information); use several headings, arrows, or the like to summarize or draw attention to the most important pieces of information; and limit the number of type fonts and colors to minimize distraction. That is, provide no theory, require no inferences, provide only the bare minimum of information that must be understood to produce the desired behavior, and eliminate all else on the page that might distract rather than draw attention to it (Gottfredson, 2004). In general, health education and patient information should be tailored to meet the needs of people with different intellectual abilities (Kajantie et al., 2009).

How exactly could tailoring and developing more effective intervention strategies for particular patients be accomplished? One instance for a concrete intervention could be derived from our finding that childhood intelligence predicts adult physical health and from the findings of other studies that have shown that childhood intelligence influences diagnosed diseases, such as cardiovascular disease (CVD). This intervention would be considered "targeted surveillance" (Deary et al., 2010, p. 72): A patient with lower intelligence could have his or her cardiovascular health monitored more regularly. This would be helpful in managing costs because regular and costly monitoring would be targeted at those most at risk,

whereas those who are less at risk could undergo less frequent, albeit still regular, monitoring. The increased surveillance of those at risk, although costing more in the short term, could lead to large savings for health-care organizations and societies that would be gained from a reduced likelihood of myocardial infarction. Another instance would be to consider that individuals who are high in intelligence will be able to adhere to a complex treatment regimen, such as highly active antiretroviral therapy, whereas individuals who are low in intelligence could have difficulties. The results of Study III and of other studies have shown that the contrasting long-term survival likelihood of those who have a higher childhood intelligence versus those who have a lower childhood intelligence is marked (e.g., Calvin et al., 2011; Deary et al., 2010). A patient in the latter group could be supplied with a mnemonic device that reminds him or her of when to take a particular medication or could be the recipient of a newer, less complex treatment. Again, the additional costs borne by such devices or newer treatments are likely to be outweighed by reductions in serious future complications and the evolution of resistance (Deary et al., 2010). Likewise, behavioral modification advice, intended, for example, to modify levels of smoking, physical activity and diet could be redesigned to make it more suitable for individuals with lower intelligence. In principal, this would be a rapidly implemented, adult-targeted intervention to address variation in these behaviors due to variations in intelligence (Batty et al, 2007a). A final instance involves health care at work. Employees with lower levels of intelligence may be more at risk for sick leave, as Studies I and II showed. Furthermore, these individuals may be more at risk for work accidents, especially when tasks are novel and not routinized (Gottfredson, 2004). Thus, company doctors should take special care of these individuals. More generally speaking, societal support to people with low intellectual abilities to help them take responsibility for their health should be encouraged.

Altogether, the results of this Ph.D. project and other studies highlight the fact that

intelligence is an important factor that influences various health outcomes and even the risk of mortality in adulthood. Intelligence may thus be one important factor for explaining socioeconomic health inequalities. Interventions for reducing the detrimental effects of lower intelligence on various health outcomes have numerous starting points both in childhood and adulthood, and these interventions could ultimately help to reduce health inequalities. Thus, human capital investments should take intelligence into consideration (Heckman, 2006; Judge, Ilies, & Dimotakis, 2010; Reeve & Basalik, 2010). The benefits of intelligence are numerous as intelligence influences life trajectories in crucial domains such as educational attainment, socioeconomic success, and health. Investments in intelligence may therefore produce economic, health, and social benefits.

5. General conclusions

The results of the three studies of the present Ph.D. project permit the following conclusions to be drawn:

1. Childhood intelligence is one important factor in explaining socioeconomic inequalities in health and mortality.
2. Low childhood intelligence is at the origin of a "chain reaction" of unfavorable life outcomes that ultimately result in ill adult health and perhaps even premature mortality.
3. General reasoning, abstract thinking, and problem solving skills, as reflected in fluid intelligence tasks, seem to be important driving forces behind the effects of childhood intelligence on adult health outcomes.
4. Interventions for reducing the detrimental effects of low intelligence on health have numerous starting points in childhood and adulthood, and may thus help in reducing socioeconomic health inequalities.

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