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THE ROLE OF EDUCATION AND COGNITIVE SKILLS IN UNDERSTANDING MORTALITY INEQUALITIES

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Stockholm 2011

Published by Karolinska Institutet. Printed by US-AB, Stockholm

ISBN 978-91-7457-532-3

"Outside of a dog, a book is man's best friend.
Inside of a dog, it's too dark to read."

(Groucho Marx)

ABSTRACT

The overall aim of this thesis is to improve our understanding of the association between cognitive skills and mortality by epidemiological analyses of their relationship. Related factors, especially own and father's education, are also analysed. This field or research is approached in three observational studies and one quasi-experimental study.

Previous research suggests that higher cognitive skills as measured by IQ tests in childhood predict lower risk of premature mortality. A related field of research demonstrates how schooling is associated to increases of IQ. Longer schooling in itself is also known to be related to longer life. Still how these associations should be understood is not clear.

In this thesis it is argued that education is indeed casually related to lower mortality - and that this can be partly explained by the effect of schooling on cognitive skills. It is also argued that the association between cognitive skills and health cannot be reduced to people being 'clever because they are healthy' or to the position of one individual relative to other individuals.

Since cognitive skills relate to every individual in a population and since they can be improved, new ways of thinking about promotion of population health are implied. Improvements of both cognitive skills and average life expectancy in a population could in principle be achieved at the same time as differences between individuals in a population are reduced.

LIST OF PUBLICATIONS

I. Lager A, Bremberg S, Vågerö D. The association of early IQ and education with mortality: 65 year longitudinal study in Malmö, Sweden. BMJ. 2009;339(b5282).

II. Lager A, Vågerö D, Bremberg S. The effects of own childhood intelligence, own education and partner's education on mortality between age 54 and 78: A prospective study. Submitted.

III. Lager A, Torssander J. The causal effect of education on mortality: 58-year follow-up of a quasi-experiment on 1.2 million Swedes. Submitted.

IV. Lager A, Modin B, De Stavola B, Vågerö D. Social origin, schooling and individual change in intelligence during childhood influence long-term mortality: a 68-year follow-up study. Int J Epidemiol. 2011:1-7.

CONTENTS

1 INTRODUCTION

An idea underpinning this thesis is that humans can control their environment and their conditions, and, with the help of such things as new technology and research, make the world a better place. This may seem a bit pretentious or somewhat obvious statement to make here. After all, the belief that we can take control over our environment is like a definition of what characterizes our modern societies (1). But not all research aims explicitly at such change: some research may instead consider establishing the 'truth' to be its ultimate target. In public health, however, scholars tend to believe - for better or for worse - that we can make things better, and this is a thesis in public health. Whether or not the specific area of public health research that is dealt with here - education, cognitive skills and mortality - can contribute towards this aim, is of course still an open question.

If the aim of gaining control over our conditions is to make things better, this immediately prompts questions such as "better in what way?" and "better for whom?". Richard Rorty, a philosopher and pragmatist, suggests that we can answer such questions quite directly; by promoting conditions that make our children or grand-children behave a little more decently towards each other than we ourselves have managed to do (2). Even if we cannot agree on what constitutes a good life or well-being, most of us do agree on what a good life is not or what constitutes an obstacle to well-being. For example, most people would probably agree that premature mortality, the outcome studied in this thesis, is something we should try to prevent or that such factors as malnutrition, lack of clean water, diseases and violence are hindering well-being.

Interestingly, Rorty also asserts that the question about which accounts of the world are 'true' may simply be wrongly put. The real question, he suggests, is what descriptions of our world can help us to make the world a better place (2). To confess to such a relativistic view of truth may again seem an unstrategic thing to do in the introduction to a PhD thesis. However, it is not a relativistic view in the sense of 'everything goes' or that all 'truths' are equally good - on the contrary.

In addition, Rorty's view does not seem that far removed from views recently put forward in the scientific discussion about causality. Judea Pearl, a computer scientist by training, has noted that, for engineers, the question about causality has never been problematic (3). Engineers often use diagrams or calculations that show causal associations, e.g. between signals in a circuit or over forces affecting different parts of a building. For engineers, to conclude by saying "we cannot prove causality so please be aware that everything we say might be completely false", as we often do in our areas of research (at least indirectly) would not make sense. The 'proof of causality' is that the circuit works or that the skyscraper remains standing. In other words, what determines the value of the descriptions is that they are useful in changing our conditions for the better.

The scientific discussion on education, cognitive skills and health, is clearly interesting from this perspective – at least for those of us who believe that *new* descriptions of the

world are more likely to bring about change than the descriptions that have been around for a while. Traditional ways of approaching health inequalities by focusing on education, social class or income, may still prove to become important for policy (and indeed one of the studies here (III) focuses entirely on education) in the same way that traditional research on intelligence may. But both fields are also politicised, and combining them could possibly provide policy leverage that they could not provide on their own.

The two differing accounts of the world as being either 'truly causal' or 'useful in changing our conditions' are not contradictory. In fact, incorporating more of the pragmatic approach in population health research, could even put more pressure on us to ensure that our research really holds and describes 'true causal' relationships. After all, failed attempts to construct an electric circuit do not imply the same potentially devastating consequences as failed attempts to improve childhood conditions.

2 BACKGROUND AND THEORETICAL FOCUS

When aiming to understand and ultimately promote population health, it may help to conceptually separate between upstream and downstream health determinants. The first include factors at societal or cultural level and the latter factors that are closer to the individual, or even within them. Although upstream factors can be thought of as including most, or at least many, of the characteristics of cultures, countries and societies, some of the most studied factors in the empirical cross-national literature on mortality are education, gross national product (including its distribution) and healthcare (4). Further, a typical focus of health equity studies and studies in social epidemiology is on upstream factors at the individual level such as education, occupation/social class and income/wealth (5, 6).

Among the downstream factors, those closer to the individual, that today are considered to be most important for global health are childhood underweight (which is estimated to account for around eight percent of the full disease burden), high blood pressure, unsafe sex, smoking (accounting for around five-six percent each), alcohol intake, high cholesterol, contaminated water, overweight and indoor smoke (accounting for around three-four percent each) (7).

The theoretical focus of this thesis is on a recent addition to this broad thinking about population health, namely what has been referred to as ‘abilities’ (8), ‘capabilities’ (9), ‘competencies’ (10) or ‘skills’ (11). The specific focus is on cognitive skills, measured by intelligence tests.

Cognitive skills apply to *all* individuals in a population. Since they are also possible to *improve*, for example by education, they have the two fundamental qualifications for potentially being very important population health determinants. In addition, cognitive skills have the (for quantitative researchers) attractive characteristics of being both measurable and quantifiable.

2.1 UPSTREAM AND DOWNSTREAM FACTORS

Upstream factors have the potential to explain more of the health variation than downstream factors because of the role they play at the beginning of many separate chains of events. From that perspective, they are also the most interesting health determinants. A possible drawback with upstream factor is that it is not always easy to determine exactly what chain of events that follows from them or even to determine if there is a causal relationship between them and health.

In addition, when it comes to interventions or reforms, upstream factors tend to already be in society’s focus for other reasons than health promotion. This could mean that the same changes that would be health promoting may have already been pushed for (and would perhaps already have come about if it was straightforward) and/or argued against for other reasons. There could, for example, be economical or ideological standpoints present where other values are put above health. In short, the discussion about upstream factors often involves political considerations and conflict.

Downstream factors have the strength that we may be surer about causality and what the causal chains look like. However, when it comes to health promotion, downstream factors have the innate weakness that each one in itself explains far less variation in health than upstream factors do. In addition, several downstream factors which are known to be important, such as health behaviours, have been shown to be notoriously difficult to influence. In fact, some of the most important downstream factors we know of (e.g. alcohol intake or smoking) are perhaps most successfully influenced by policies that change upstream factors (e.g. increasing taxes on alcohol and tobacco). Thus, we are back at the possible complications that go together with changing policies at that level.

Thinking in terms of upstream and downstream factors may be helpful in itself because it focuses attention on the complex chains of events that precede every health outcome and indicates different ways of explaining as well as intervening on the same phenomenon. But perhaps it works best when a single determinant plays a fairly stable role in a number of different chains of events, e.g. in the way smoking can be thought of as a determinant between for example the upstream factor tobacco tax and the more downstream outcomes of bronchitis or lung cancer. Skills are not easily identified in this way, but this is also what makes them interesting.

2.2 COGNITIVE SKILLS

Cognitive skills can play a number of different roles in a downstream-upstream model. It has been suggested that cognitive skills are; important downstream factors in themselves (12), determinants of other downstream factors such as smoking (13), possible mediators of upstream factors such as education (13, 14) and, at the same time, possible determinants of upstream factors such as education or income (15). In this way they may be involved in pretty much every level of an upstream-downstream model. Furthermore, cognitive skills may potentially also be factors which interact with and modify the effect of upstream factors on health, for example social background (16). Last, but not least, cognitive skills may possibly act, at least in the long term, as factors which generate important characteristics at the *aggregate* upstream level, such as economic growth (17).

The present thesis focuses on cognitive skills where they are most directly linked to health: as important downstream factors in themselves and as possible mediators of education. The reason for this specific focus is that establishing whether cognitive skills and education are at all important determinants of health or, here, survival is a sufficiently complicated task.

3 AIM AND GENERAL APPROACHES

The overall aim of this thesis is to improve our understanding of the association between cognitive skills and mortality by epidemiological analyses of their relationship and of related factors, especially own and father's education. This field is approached in three observational studies and one quasi-experimental study.

The observational studies are conducted on a database in which we have linked a longitudinal study that started in 1937 to the Swedish Cause of Death Register with present day information. Cognitive skills were assessed by means of IQ (intelligence quotient) tests in 1938 at age 10 for both boys and girls, and for the boys also in 1948, at age 20. In addition, we use information on the subjects' conditions during childhood, their way through the education system and their situation in adulthood, including information on their partners.

The quasi-experimental study uses register data on 13 birth cohorts, from 900 Swedish municipalities. These children were affected by a reform in which the old system with elementary and junior secondary school was replaced by the new comprehensive school. This reform was initiated in 1949 and its consequences for mortality until the present day are again identified by help of the Cause of Death Register.

The objective of the first section of this thesis is not to provide a more detailed account of these four studies - which can hopefully stand on their own legs – but rather to describe one way of thinking about a more general context.

4 PREVIOUS RESEARCH

Research on cognitive skills is often met with some suspicion due to the murky history associated with the tests in this area. Intelligence tests have for example been used to decide whether or not to let immigrants enter the USA (18), to decide which military conscripts can become officers and which can become 'cannon fodder' (19), to describe differences between races or nationalities (20) and - with milder consequences but wider use - to decide who to choose for educations and jobs (21, 22).

From these examples it is clear that the very purpose of the tests, when used in this way, has been to differentiate between individuals *within* a population, or to 'separate the wheat from the chaff'. In line with this, many researchers have focused other things than the possibilities to improve the *average* intelligence level of a whole population. Further, much research in this area have, either explicitly or implicitly, been based on the ideas that IQ is stable and "substantially" (23) or "highly heritable" (24) and therefore difficult to influence. The example of Cyril Burt, who was later shown to either have fabricated or, at the very least, used data that were "woefully inadequate" in combination with "grossly inadequate methods" (25) to support his claims about the high heritability of IQ, could perhaps be understood in the light of this attitude. However, it also seems clear that for a long time his 'findings' also asserted important influence on the negative views of the possibilities to improve intelligence by means of changing the environment.

There is however a parallel and completely different strand in the history of intelligence testing – a strand that may have started with Alfred Binet, often considered the father of group intelligence testing (26). *His* aim was to produce a rational and less prejudiced foundation for the allocation of children to different educational tracks. A similar aim guided Siver Hallgren, the founder of the Malmö Longitudinal Study (27) used in three of the studies in this thesis (I, II & IV). Hallgren initiated his study because he suspected that the reasons for more often placing children from working class families *in* support-classes - and for keeping them *out* of the more academic junior secondary school - were based on subjective judgements from individual teachers rather than an objective assessment of the child's actual cognitive skills.

Binet stated that *"Some recent philosophers seem to have given their moral approval to these deplorable verdicts that affirm that the intelligence of an individual is a fixed quantity, a quantity that cannot be augmented. We must protest and react against this brutal pessimism; we will try to demonstrate that it is founded on nothing."* (28). Later colleagues working in the field of education, including those behind the Malmö-study (29), have indeed demonstrated that IQ can be improved, among other things by education itself (30). Numerous other field of research have also provided direct or indirect evidence of this, with one general support for the malleability of cognitive skills coming from what has been termed the 'Flynn-effect' (31).

4.1 THE FLYNN EFFECT

Flynn noted in 1987 that historical data from 14 different countries all demonstrated the same increase in average test-scores over time, with each new generation on average

out-performing previous ones (32). Since then, a number of other studies have demonstrated the same phenomenon, recently for example in Austria, Germany, Switzerland (33), Estonia (34), France (35) and Kenya (36). The Flynn-effect has also been detected in results from some other neuropsychological tests, for example of memory (37, 38). It has even been suggested that the Flynn-effect lies behind a major part of what has previously been interpreted as cognitive decline with age (39). Also the more recent suggestions of a flattening out of, or even decreasing, average test-results, notably demonstrated in for example Sweden (40), Norway (41), Denmark (42) and Britain (43) over the last couple of decades, provide a kind of support for the idea that intelligence is malleable.

4.2 COGNITIVE STUNTING

Less encouraging, but in a way more convincing, empirical support for the idea that cognitive skills can be modified comes from work on factors that have a hampering impact on cognitive development. Firstly, there is an extensive literature on the devastating effects on cognitive skills of toxic substances such as lead (44-46), fluoride (47), arsenic (48) and mercury (49).

In addition, more and more studies are identifying links between cognitive development and more general conditions in childhood. Of around 560 million children under the age of five growing up in developing countries, more than 30 percent, or around 180 million children, live under such circumstances that their height-for-age is below two standard deviations under the normal (50). Low birth weight and undernutrition seem to have direct consequences for both cognitive skills (51, 52) and later educational attainment (53). Other studies show that lack of iodine is related to poor cognitive development (54, 55).

Breastfeeding is known to be positively related to later cognitive ability, but causal interpretations have earlier been questioned since breastfeeding is also related to possible confounders such as the mother's cognitive skills. However, recent studies explicitly targeting this problem with better designs seem to corroborate a causal association (56, 57). Infections during childhood seem to have a negative impact on cognitive ability (51, 58, 59). Finally, there are associations between sleep and cognitive performance, although we may have to wait for research that can disentangle the complex associations here (60-63).

4.3 TWIN- AND OTHER FAMILY-BASED STUDIES

Further support for the idea that environmental factors generally influence intelligence comes from adoption studies, with French researchers as far back as the 1970s showing how adopted children's intelligence correlated more closely to the levels in their new families than in their birth families (64).

The best-known design in the field of family-based studies, however, is the twin-design in which mono- and di-zygotic twins are compared in order to isolate genetic, shared environmental and non-shared environmental influences on IQ. Since the view that IQ is "substantially" (23) or "highly heritable" (24) - and implicitly then difficult to improve - relies on this research, it deserves some extra attention here. More

specifically, the statements about heritability stem from the fact that genetic component seem to explain up to 80 percent of the variation in such studies (65).

An initial reaction from a health promoting perspective could then be to target the 20 percent of the variation that is left for environmental influences. *Childhood obesity* has a genetic component that is *at least as high* according to the same kind of twin-studies (66). The prevalence of childhood obesity has tripled or so during the last three decades (67) - and no-one claims that this is due to a deterioration of the gene pool.

However, there are also more fundamental problems with the gene-environment distinction. In fact, if one wants to think in terms of percentages of variation due to environment and genes, it may make sense to think of most phenomena as *both* 100 % genetic and 100 % environmental. In a scenario in which everyone in a population smokes a lot of cigarettes, the environmental contribution to lung-cancer *in twin-studies* would be very low, and the genetic very high. In other words; if everyone smokes, what determines whether you get lung-cancer is primarily other risk factors, including your genetic make-up. In this scenario, it seems likely that we would draw the devastating conclusion that lung-cancer cannot be prevented by environmental measures.

A second example that could be useful when trying to avoid the risk of drawing unfortunate conclusions from twin-studies comes from paediatrics. The serious condition of phenylketonuria (a disorder in which a problem with a liver enzyme makes you unable to digest an essential amino acid) is purely genetic (it is in fact thought to be caused by a *gene mutation*). However, a combination of purely environmental modifications (the most important aspects of which are screening and change of diet) can prevent most negative consequences of the disorder, of which the most serious is mental retardation (68).

A similar but more specific possible concern with twin-studies of intelligence worth mentioning, because it relates directly to one of the studies of this thesis (IV), is the fact that 'gene-environment interactions' are being increasingly acknowledged as important, while typically being very difficult to detect statistically (69, 70). Our study suggests that school, an environmental measure that everyone is exposed to, may make relatively highly performing children perform even better on IQ tests with age, while it will make children who are already performing poorly perform even worse relative others (14).

This consequence of schooling could mean that monozygotic twins, who tend to have a quite similar IQ already at low ages (whether performing relatively well or relatively poorly), will be even more similar at higher ages, due to the environment, i.e. the school. This will, however, have the counterintuitive effect of the *genetic* component in twin-studies becoming *more* important in explaining the variation with increasing age. This is indeed also what has been detected in twin-studies (71, 72). But this has then been interpreted as some kind of heritage which increases with age (73) and/or even that *also schooling* can be explained by genetic influences (74). However, an interaction can also be interpreted in the opposite way, e.g. that the *environment* has influence also over what has previously been considered genetic. That this problem is inherent in heritability analyses of IQ has been suspected for a long time (75).

A clear gene-environment interaction has also been documented in family-based studies where biological bonds of other kinds than twin-ship are used. It has for example been shown that the amount of the variation in intelligence that can be accounted for by the genetic component *varies* with the social background of the person, so that it is high among children from affluent families (who might enjoy a more optimal environment) but low in more socially deprived groups (76, 77). That the home environment is linked to cognitive development is also demonstrated in an increasing number of more regular observational cohort studies (78-81) including ones where maternal IQ can be controlled for (82, 83) and Norwegian register studies drawing on birth order and age differences between biological brothers (84, 85).

Finally, it could be pointed out that, in line with the reasoning in the introduction, the different possible ways of interpreting the results of twin-studies may seem quite contradictory. It is not, however, necessarily so that they are more or less 'true'. But, different descriptions may nevertheless be more or less useful. This is true also of other areas: *unsafe sex* is arguably both causally related to HIV-infections and an explanation for much of the incidence, and, as already mentioned, it is considered one of the most important health determinants at the global level. Maybe today 'unsafe sex' is a useful description when trying to prevent further spread. However, *'lack of complete vaccination with an efficient vaccine'* is *also* causally related to the HIV-infection, and a factor that explains *all* incidence. Both descriptions are true - but in twenty years time the latter may prove to be more useful.

4.4 EDUCATION AND COGNITIVE SKILLS

Another body of work on the malleability of cognitive skills comes from the educational field. In 1951, Torsten Husén presented international empirical support for the contention that education affects intelligence and discussed the new results from the Malmö-study (29). Forty years later, another reviewer of the literature demonstrated that education has consistent effects on IQ in studies with eight different kinds of design, including natural experiments (86). Recent studies suggest that much of the differences in average IQ between countries can be explained by differences in education and literacy (87, 88).

A Swedish study shows how being exposed to different educational tracks and different numbers of months in senior secondary school influenced intelligence at conscription (89). Further, the very purpose of using cognitive tests in Swedish military conscription is to judge the potential for *training* the conscript, i.e. to develop some kind of skills (90). That higher initial skills seem to make it easier to also *further develop* skills has been suggested by the economist James Heckman and colleagues under the theme 'skills formation' (11, 91). The interesting conclusion they draw from this is that optimal investments for children in socially deprived backgrounds should be made *early* and *followed up* by further investments, in for example education, at later ages.

Furthermore, we may believe that school should equip students to, for example, "use mathematical thinking in ordinary life", "solve problems and implement ideas in a creative way", "learn, explore and work both independently and together with others

and feel confidence in their own ability" or "use critical thinking and in an independent way formulate stand-points based on knowledge and ethical considerations". These very aims are presented under the heading 'knowledge' in the new Swedish national curriculum for compulsory schooling (92) but it can be noted that they, in fact, very much resemble the *definition of intelligence* proposed by 52 prominent scholars in the mid 1990s:

"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." (93)

From this perspective, it makes sense that education could improve intelligence. According to the curriculum just mentioned, Swedish schools shall also "deliberately influence and stimulate the students to embrace our society's common values" (92). The aims in *this* section of the curriculum, however, do not include *any* reference to critical thinking, problem solving, analysis or suchlike. When it comes to ethics, our future citizens are apparently not expected to use their cognitive skills. That could of course be challenged (94-96).

The Swedish curriculum also aims to foster the ability to "enter into and understand other peoples situation…" (92). At best, this could be interpreted as a support for efforts to promote *social and emotional skills* - an area where the scientific evidence for positive effects of educational efforts (97-100) may very well outweigh the evidence in any other school subject. Other skills that we tend to hope that education will promote are the abilities to take responsibility, be independent, keep deadlines and suchlike. Such characteristics are also related to health (101, 102).

4.5 COGNITIVE SKILLS AND HEALTH

Strictly speaking, we do *not* need to know exactly what *mediates* the effect of cognitive skills-on health in order to promote health by improving skills. What is needed is rather convincing support for the relationship being causal. This is not as strange as it might sound. Consider for example the Swedish official description of how the painkiller paracetamol works: "the analgesic effect is *probably correlated* to the paracetamol molecule's ability to… …it is however *possible* that the analgesic effect partly can be explained by *other influences* on…" (my italics) (103). Clearly, exact knowledge of the mechanisms is not always needed for something to be useful. Another example is the fact that we now put our babies to sleep on their back to prevent cot deaths. It is not clear exactly *why* this works; only that it does. In fact, when it comes to sleeping position, the opposite idea – that children should sleep on their stomachs – was based on quite specific ideas about mediators, or mechanisms, on the part of clinicians. What were missing were studies of good design demonstrating a causal association.

At the same time, we also, for good reason, want to identify some plausible mediators of an association. In addition, there is at least one hypothetical scenario in which

cognitive skills might be causally related to health but where improving them on average would *not* lead to population health gains – namely if what one person *gains* from higher skills is another person's *loss*. Such effects could in addition *increase* health inequalities.

A recent systematic review showed that IQ measured in childhood or early adulthood was related to all-cause mortality later in life (12). In another study early IQ predicted cardiovascular disease better than most other established risk factors (104). How should relationships such as these be understood? Two possible explanations are *confounding* and *reverse causality* and these will be discussed further on. But if the association is causal, intelligence must be a (more or less valid) measure of a characteristic that helps us (in one way or another) in different aspects of life.

Karl Popper suggested that we could view 'all life as problem-solving'; from the actions of the smallest amoeba to the scientific inquiry of humans (105). In a broad sense, life and organisms are typically defined as objects capable of reacting on the environment in a way that make them sustain (106, 107). If intelligence is something like - or at least partly tapping – an ability to react to the complex environment of human life (93) it makes sense that intelligence could help us become healthier, at least if we think of health as something quite close to what it means for an organism to sustain.

4.5.1 Social adjustment as a possible mediator

More and more research seems to suggest that much of the differences in health *over cognitive skills* might be mediated by 'social adjustment', here understood in a wide sense, such as the way it is defined by the U.S. National Library of Medicine; *"Adaptation of the person to the social environment. Adjustment may take place by adapting the self to the environment or by changing the environment"* (108).

The first kind of empirical support for this interpretation comes from studies where the association between early intelligence and health, or health-relevant outcomes, is accounted for by adult socioeconomic position (13, 16, 109-113). As already mentioned IQ-tests are sometimes used in order to give some, but not all, people access to military training or jobs. When tests are used in this way it makes sense that the tests could play a part in social stratification. This would in fact hold even if the tests *did not* reflect true cognitive skills. The results of IQ tests are also correlated with other mechanisms that could act in the same way, especially school grades.

But in addition, most intelligence researchers would probably suggest that there are also 'real' links to social outcomes that are not dependent on the actual use of IQ-tests (or, for that matter, the use of school grades). For example, intelligence might be linked to getting a better job and social mobility (114) at least partly because the tests are actually tapping into something that is indeed related to your capability at performing the job. To most, it would also make sense to expect exactly such consequences of the abilities that our schools aim to provide.

The beneficial outcomes related to higher intelligence discussed so far could possibly be gained at the expense of others. In the first examples, where IQ-tests are used in order to differentiate between people, it even seems likely; if you get the attractive job, someone else will not. Also in the example where IQ is related to social outcomes in more 'real' ways, e.g. because it is related to your ability to do the job well, it could be at the expense of others. Yet, here it is also possible to imagine situations where this would not be the only consequence - for example if cognitive skills help the performance of the economic system as a whole and hereby make the situation a little better for everyone. Something similar could also be hypothesised to hold in other situations, for example in the every-day life of a family where improving one person's cognitive skills by for example education could have *positive* effects also for other members of the family, such as the child (115), the partner (116) or even the parents (117).

The suggestions of links between cognitive skills and health via *other* forms of social adjustment, again understood in the wide sense above, make it even clearer that what would be better for one individual does not have to involve a worsening of the situation for someone else. Cognitive ability has for example been linked to social adjustment in the form of behaviour problems (118, 119), drug intake (120), and the risk of being assaulted (121) or even murdered (122). There is also a growing literature that links cognitive ability to mental health in general (123-127). These diagnoses tend to demand "impairment in social, occupational or other important areas of functioning" (128) or, in other words, clearly include an aspect of social adjustment. In all these examples, improved cognitive skills of one person seem likely to be related to a *better* situation also for people in his or her immediate surroundings.

4.5.2 Other potential mechanisms and concluding remarks

There might also be mechanisms linking cognitive skills and health that would imply *direct* population health improvements of improved general skills. For example, studies linking cognitive ability to obesity and the metabolic syndrome (129-131) may suggest that some traditional health behaviour such as eating habits or physical activity act as mediators. Unintentional injuries, which might in turn be related to such things as ability to calculate risks, are also related to cognitive skills (132-134). Finally, if attempted suicide is linked to the ability to generate and choose between different possible solutions to a situation, this could also be a more direct mediator (135).

To sum up, even if the IQ tests themselves are both designed to differentiate *between* individuals, it is entirely possible that the *health effects* of higher skills are not mainly due to the relative position between individuals. Thus, average improvements of cognitive skills could mean average improvements of health. What would happen to differences *between* individuals, if average skills were improved is partly a different question. However, there is no innate contradiction between high averages and low differences. One illuminating example is provided by the so called PISA studies (the OECD Programme for International Student Assessment) where school performance in reading, mathematics and science among 15-year-olds is assessed, in the last round, 2009, in 67 countries (www.oecd.org) . Of the 17 countries that scored the *highest* on average in reading (reading scale result>=500) *two*, Korea and Shanghai-China, were

also among those that had the absolutely *lowest differences* between individuals (standard deviation<=80, in total seven countries). Another *six* of the high-performing countries, Estonia, Hong Kong-China, Finland, the Netherlands, Poland and Canada, are on the lower half on this scale, with another high-performer, Norway, being the median country. Thus, for these countries, performing well on average went hand in hand with low inequalities.

5 METHODOLOGICAL CONSIDERATIONS

Helping to change our conditions may be one ultimate goal of scientific descriptions of the world, i.e. by trying to predict what *will* happen to an outcome (such as mortality) if we manipulate one of its determinants (such as education and/or cognitive skills). However, since our complete knowledge-base is entirely made up of events that have already happened, a more basic question needs to be dealt with first, namely what *would have* happened under other conditions.

In research, we are typically not satisfied with concluding what happened in an exposed group, but also that these events did happen *because of* the exposure. What we are saying then, is that under other conditions, the change would not have come about. Here we are typically referring to a more stable situation in some kind of a control or reference group, i.e. those who were not exposed but were similar in other ways.

However, even in the best-designed kind of study, the randomized controlled trial, the control group is *not* the experimental group - and the situation in which the experimental group did not become exposed did simply *not* occur. In other words, that situation is 'counter-factual'. What we do have, if we are lucky, is a substitute for the counter-factual situation that is similar enough in all other aspects than the actual exposure.

5.1 REVERSE CAUSALITY AND CONFOUNDING

There are two common potential pitfalls in trying to determine causality. The first is reverse causality, e.g. that what looked like a causal link from an environmental exposure to health, as described, for example, by a statistical correlation, was in fact due to an influence *in the other* direction; from health to the exposure. One field of research where this problem may very well have caused spurious relationships is observational research into the outcomes of drinking small quantities of alcohol. Where consuming at least small amounts of the alcohol is the norm, the comparison group – the total abstainers - is likely to include some people who are different in other ways as well, and some of these characteristics may be health-related.

Some of the total abstainers may, for example, take medication or may even be reformed alcoholics, who, even if they have stopped drinking, might be expected to experience poorer than average health in the future. Such circumstance could give the false impression that drinking very small amounts of alcohol is better than drinking nothing at all, while it might actually be the case that not drinking alcohol at all is a marker of some health problems. Again, the problem is that we cannot have information about the counter-factual situation, in which those who drank very little instead abstained from all alcohol; we can only have better or worse substitutes for this situation.

The example of the potential problem of determining the effect of low doses of alcohol from observational studies is used here purely as a thought experiment. However, the findings on low doses of alcohol are not yet convincingly corroborated in randomized controlled trials. It should also be mentioned, that although this example concerns

longitudinal studies, the potential problem of reverse causality is greatest in cross-sectional studies, in which both the exposure and the outcome are measured at the same time and where one often cannot determine which phenomenon, the exposure or the outcome, that manifested itself first.

The second over-arching pitfall in determining causality is often referred to as confounding, or the third variable problem. Here, a third variable is associated with both the exposure and the outcome. In *nutritional epidemiology* it has for example been shown how associations that have been repeatedly replicated in *observational* studies of the effect of vitamin supplements could *not* be replicated in randomized controlled trials, possibly due to unmeasured confounding of socioeconomic or other variables in the observational studies (136).

An example of confounding from the literature on cognitive skills comes from a study that demonstrated how an earlier observed association between mothers' smoking during pregnancy and their children's IQ, an association that could have been causal, was *completely* confounded by the mothers' own IQ (137). Thus, the mothers' IQ was statistically associated with both their smoking and their children's IQ (through other ways than smoking), and the association between smoking and their children's IQ was spurious.

For us to believe something to be causal, we tend to require ideas about mechnisms that are biologically plausible (138). However, this is not a quick solution for any of the problems that we come across in determining causality. The problem with this requirement is that it is not difficult to construct "a posteriori" explanations for unexpected findings. In our two examples above, one could quite easily think of mechanisms which might mediate associations between small amounts of alcohol and health, or between smoking and IQ, irrespective of whether these findings are causal or not. Also, what we are sometimes mistaken about what *is* biologically plausible.

Finally, there is an increasing awareness of the potential gravity of even weak tendencies to make biased decisions (conscious or unconscious), on behalf of the researcher (139, 140). The problem is greatest in areas of research where there are many testable associations and where the correlations are weak. That this risk is real has become obvious, for example, in failed efforts to replicate findings in the new field of genetic epidemiology (141).

5.2 STUDYING SKILLS, EDUCATION AND HEALTH

The best substitute for the counter-factual situation is given by randomized controlled trials. This is reflected in the grading of evidence, applied for example to interventions in public health (142). Thus, the optimal design for studying the causal effect of cognitive skills on mortality would be to conduct mortality follow-up of randomized controlled trials that have been successful in improving cognitive skills. Such analyses will perhaps come in the future, for example by following up mortality in eight existent American studies in which children have been randomly exposed to pre-schools (143).

Another possibility is to take advantage of quasi-experiments, in which the exposure has been manipulated (the very definition of an experiment) and there are control groups (in contrast to pre-post experiments), but where the allocation to the experiment and control groups is not random. If studies in cognitive epidemiology continue to deliver interesting results, it is probably also only a matter of time before such studies, will begin to show up.

In the meantime, however, we are referred to purely observational studies. The basis for conducting such studies could, on the other hand, hardly be better. Thanks to longitudinal studies which began just after the Second World War or even earlier, the field of cognitive epidemiology has recently been able to expand rapidly. Of the studies briefly reviewed above, the vast majority have been published during the last five years.

In longitudinal observational studies there are two main strategies for dealing with the potential problems of reverse causality and confounding. The first is to ensure that the potentially problematic phenomena are measured so they can be taken into account in the statistical analysis. The second is to take advantage of time by measuring the exposure first and the outcome later, hereby in principle eliminating the risk that the outcome preceded the exposure in time.

5.3 THE MALMÖ LONGITUDINAL STUDY

The Malmö Longitunal Study, MLS, used in three studies of this thesis (I, II & IV), includes all children that attended third grade in any of the schools in the Swedish city of Malmö in the spring term of 1938. Most of these 1530 children were born in 1928, and thus around ten years old in 1938, when they took an intelligence test. The study has since then been supplemented with rich information about the subjects' educational attainment.

A possible *confounder* in the association between cognitive skills, education and mortality, is childhood social conditions. Because of its original purpose, the MLS contains good information about the children's parents, collected before the IQ testing began. The possible *reverse-causality* explanation in cognitive epidemiology has been termed the 'system integrity hypothesis'. The idea here is that intelligence may, right from the start, be one marker of a "general latent trait of a well-functioning body" (144). There are also studies linking cognitive test results of older people with disease, where the results of cognitive tests before the actual diagnosis is sometimes referred to as 'pre-morbid intelligence' (145). Here, the risk of reverse causality is arguably high; someone with undiagnosed dementia for example, will on average perform less well on cognitive tests *and* have an increased risk of developing the clinical manifestations.

The health information in the original Malmö-study is limited, which is a problem in relation to the potential risk of reverse causality. It does, however, have an early measurement (age ten) of the cognitive skills. The risk of disease having influenced cognitive ability is arguably therefore lower than when results from tests in older ages are used. It can also be mentioned that no period in life is as free from disease as the pre-school and early school-years. When the subjects in the Malmö-study where asked

at age 36 if they had been seriously ill for any long period before or during their school years, only four percent gave a positive response (146).

5.4 A NATIONWIDE QUASI-EXPERIMENT

In one of the studies of this thesis (III), the effect of education on mortality is studied by following up of a Swedish reform of the educational system that started in 1949, and was implemented as a quasi-experiment. When determining the causal effects of education on health, the two potential problems are again reverse causality (e.g. that disease will make you less able to complete an education) and confounding; where cognitive skills may actually be one of the best candidates of potential confounders.

In a quasi-experiment, these problems are approached by assigning the experimental and control conditions to groups in ways that are thought not to be different. Since this allocation is not random, however, one can never be completely confident that the groups really are similar on all other aspects. The remedy for this problem is to take measurements of both groups both before and after the experiment. By assessing mortality in all municipality both before and after the reform, our study mimics a controlled pre-post experiment, and provide a unique possibility to study education in a setting were there is a far better substitute for the counter-factual situation than purely observational studies can provide.

5.5 INTELLIGENCE TESTS

Intelligence tests are both designed and shown to be quite successful in measuring *one* underlying intelligence. Despite this, there has been an intense discussion about exactly how - and over *how many* abilities - the scores should be structured (90, 147). This goes back to the fact that there is a tension built into the very concept of intelligence in that it is both viewed, by researchers and laymen alike, as 1) a single general ability, e.g. in the consensus definition listed above (93) or in the Swedish version of Wikipedia (which defines it simply as “mental ability”) and 2) as an ability that can be expressed in many ways, and therefore needs to be measured by a set of different tests, as argued for example by Charles Spearman (148).

One thing that has followed from this tradition of measurement is that the complexity of human abilities has become visible in the empirical results. Besides variation in general ability, there is a tendency for test-takers to be more or less capable with regard to a number of other, nowadays considered subordinate but nevertheless definable, abilities. For example one could, at the same level of general intelligence, be more or less good on spatial or at verbal tasks. One of the first and most influential theorists to expand the general model in this way was Louis Leon Thurstone (90, 147, 149). The Enlistment Battery of 1948, used in one of the studies in this thesis (IV), was based on his work while at the same time aiming to measure one general factor (90).

Over time, many different models have been presented. An interesting systematic approach to the whole question was taken by John Carroll, who collected more than 460 international datasets, and conducted exploratory factor analyses on them. He suggested that the results could best be understood as describing one general intelligence, eight broad abilities (fluid intelligence, crystallized intelligence, memory

and learning, visual perception, auditory perception, retrieval ability, cognitive speediness, and processing speed) and 69 more narrow abilities (8).

However, exactly how the results are structured in a factor analysis depends on the technique used; exploratory (that Carroll used) or confirmatory factor analysis. In 1984, Jan-Eric Gustafsson, demonstrated empirical support for a more parsimonious model with one general, two abilities at the second level (verbal and spatial), and a third (or first) level with more primary abilities, by using the latter method (150).

Much work is being done to further develop such models and maybe this can, as one researcher puts it, provide "an unprecedented opportunity to extend and refine our understanding of human intelligence" (151). Others remain more sceptical, and claim that we need experimental and theoretical analysis in order to decide between alternative interpretations (147).

In any case, the aim of the present thesis is not to provide new insights in the structure of IQ-test results. Both tests used in this thesis are designed to measure one intelligence. Many of today's theorists and psychometricians also seem to agree that IQ-tests at least do *not* measure several different parallel abilities *at the highest level*. Therefore, we have settled for two simple ways of providing variables to use in the analyses. The original IQ-score in 1938, constructed by Hallgren by standardising the score of each subtest relative to its mean and standard deviation and then summing up the scores (152), was used in Study I and II (116, 153). For the children born in 1928, the correlation between this original IQ-score and a predicted score from a principal factor analysis was r=0.97, and r=0.98 for a principal component analysis (analyses conducted for this summary). In Study IV, latent intelligence was identified by means of measurement models, or factor analyses, for both this test and the conscript test, identified by the scores on their respective subtests (14).

5.6 EDUCATION

The Swedish school system has gone through some dramatic changes during the lives of the subjects followed in the studies of this thesis: from a system with early tracking (typically starting at age ten) and many different school-forms, to one inspired by the American educational system with one comprehensive and compulsory school for nine years. This is nowadays followed by a senior secondary school that means an additional three years of schooling that are nearly as comprehensive, and in practice nearly as compulsory, as the preceding nine-year school.

Both the complexity and the subsequent reform of the old system provided the very foundations for work presented in this thesis. The initial complexity meant that the length of education varied substantially between age 10 and 20 in the Malmö-cohort - that is between the IQ measurements - and variation is a requirement for analysis. At the same time, complexity also means that measurement becomes difficult. Here, we are deeply indebted to the great efforts of Torsten Husén and his colleagues who in detail have mapped the Malmö-children's paths through the educational system.

The reform, thanks to being implemented as a huge quasi-experiment, provided a unique foundation for studies of the causal effect of education on different outcomes including, as we have shown, mortality.

5.7 MORTALITY

It is sometimes argued that mortality is a problematic outcome because it confuses incidence and prognosis (154). *All-cause* mortality, the primary outcome in all studies in this thesis, is from this perspective even more inappropriate, since it also seems to confuse incidence and prognosis from different causes. However, such assessments are based on the idea that the best way forward is to look more closely at the details. This is not obvious, and others have approached the issue in quite another way. Leonid and Natalia Gavrilova, for example, have argued that the aging of a human population, dying from different causes, might be understood as the result of *one* underlying process and that the aging of populations corresponds to the predicted failure of systems (such as the human body) that consists of elements that are on one hand irreplaceable (if they have failed, they have failed) but redundant (which here does not mean 'superfluous', but rather 'have backup' in the sense that another element can compensate) (155).

Furthermore, as mentioned above, skills could perhaps be understood as measures of an ability to react to the complex environment, that together with the ability to sustain (i.e. *survive*) make up the very definition of life. In any case, cognitive skills apply to all individuals in a population and are potentially being involved at several places in different causal chains. From these perspectives, all-cause mortality makes sense. A strength with all-cause vis-à-vis cause-specific mortality, is also that there are less problems with potential miss-classification; you are (typically) either dead or not.

The relative risk of dying – at any given point in time - can be estimated with the help of Cox proportional hazards regressions. This statistical approach does not assume any specific form of the underlying hazard. Further, it helps us to use much of the available information, especially the knowledge that censored individuals (internal drop-outs) were alive at the time of censoring – even though we do not know what happened to them next. Another strength of the method is that it makes maximum use of the information about *when* someone dies, so that deaths occurring during a time when there are few deaths occurring over-all (in population studies typically early in the follow-up) influence the estimate more than deaths during periods where the overall risk of dying is higher (typically at the end of the follow-up period). This is probably in line with how most people judge the gravity of early vis-à-vis late deaths. The method assumes that the risk of dying in one group relative to another stays proportional, i.e. consistent through-out the time studied, an assumption that can be tested.

Translating the hazard ratio to other interpretable estimates is not always straight-forward. However, a hazard ratio of two does, for example, imply that there is a point in time when two-thirds of the exposed groups have died (or experienced the event studied) but only one third of the control group. Further, when the groups are of equal size, a hazard ratio of two implies that whenever one person dies in the control group, two die in the exposed group.

6 OVERVIEW OF THE STUDIES

The most important theoretical associations that were analyzed in at least one of the studies in this thesis are described in Figure 1.

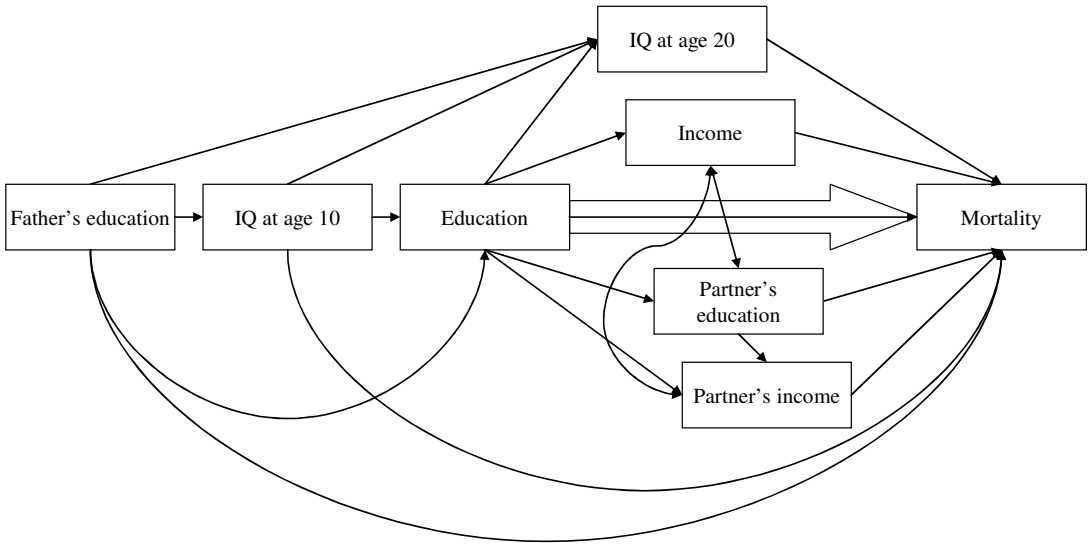

Figure 1. Studied theoretical associations between father's education, IQ at age 10, education, IQ at age 20, income, partner's education, partner's income and mortality: the double arrows between income and partner characteristics illustrate that the direction of these links are not certain and the block arrow illustrates the total effect of education, irrespective of mediators (analyzed in Study III).

In short, at least some support was found for all associations except for the *direct* link between father's education and mortality. Note, however, that clear indirect (i.e. mediated by other factors) effects of this factors existed, see below.

6.1 STUDY I

The specific aims of the first paper was to establish whether differences in early IQ explain why people with longer education live longer, or whether differences in father's or own educational attainment explain why people with higher early IQ live longer. These questions were approached by analysing all-cause mortality among the individuals of the Malmö Longitudinal Study, MLS. We linked the MLS to the Swedish Cause of Death Register including deaths until December 31, 2003, when the subjects were around 75 years old.

The analyses showed that longer education was associated with lower mortality, also after statistical adjustment for intelligence at age ten and their father's education. This finding applied to both sexes. Higher early IQ was linked to a reduced mortality risk among men, even when own educational attainment and father's education were adjusted for, but not among women.

The most general findings from Study I concern the associations between father's education, IQ, own education and all-cause mortality. These are illustrated in Figure 2 and 3, for men and women, respectively.

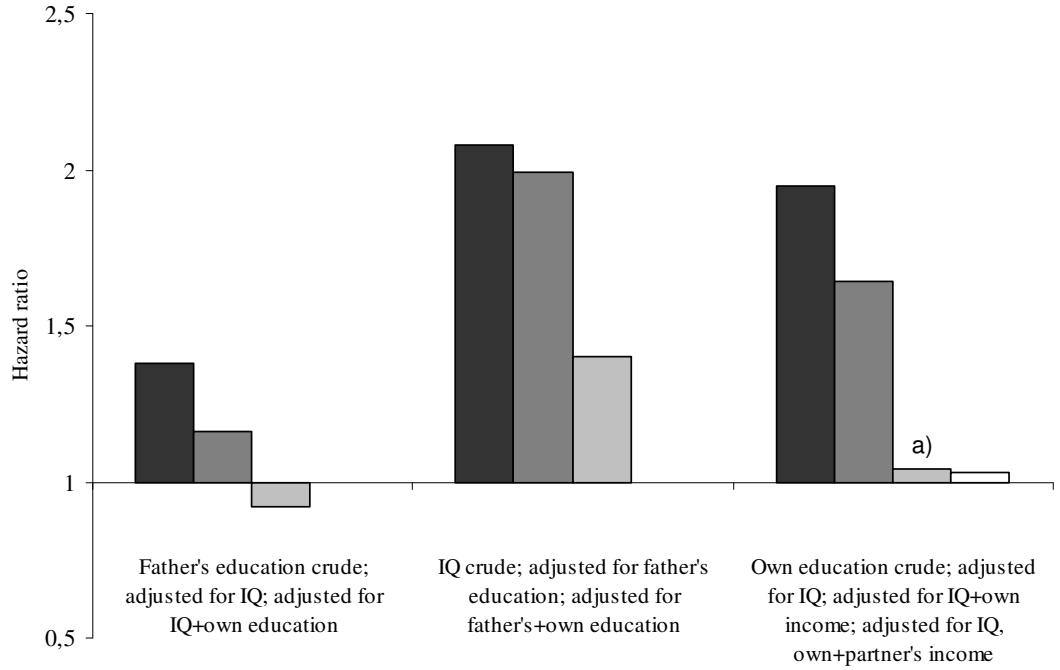

Figure 2. General finding*s from Study I on the a*ssociations between father's education (basic vs theoretical), IQ (lowest vs highest quartile), own education (basic vs theoretical) and all-cause mortality *among men*; crude and adjusted associations. [a)] *Both income-adjusted estimates are from Study II, and the initial 'adjusted for IQ' estimate was here already a bit lower (1.40) than in Study I (1.64).*

6.2 STUDY II

The specific aims of the second paper was to test whether (a) own early cognitive skills have an effect on mortality after adjustment for both partner's education and income, (b) whether own education affects mortality after adjustment for own cognitive skills, partner's education, and partner's income, and (c) whether partner's education influences own mortality after adjustment for own cognitive skills and for both partners' income.

Again, the MLS was used, this time with mortality follow-up until December 31, 2006, i.e. until the subjects were around 78 years old. In addition to the results on the cognitive tests, information about own educational attainment, own income, the partner's education and the partner's income was used in the analyses. Information about the partner was collected in 1964. The mortality follow-up started in 1983, right after the last income data were collected, that is around age 55.

The analyses showed that cognitive skills at age ten were related to own survival even after adjustment for own and partner's, education and income – but only among men. The effect of education seemed to be mediated by own income among men, but not among women. The latter's educational effect on mortality instead seemed to be mediated by their *partner's* income. Furthermore, there were suggestions of a direct effect of women's education on their *husbands'* mortality.

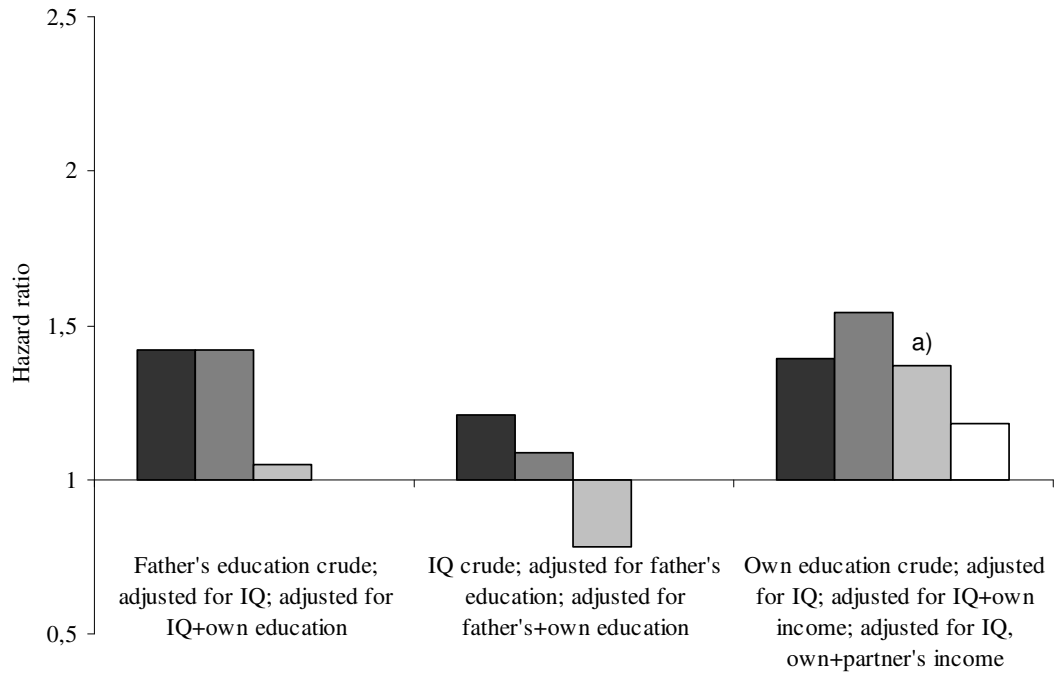

Figure 3. General findings from Study I on the associations between father's education (basic vs theoretical), IQ (lowest vs highest quartile), own education (basic vs theoretical) and all-cause mortality *among men*; crude and adjusted associations. [a)] *Both income-adjusted estimates are from Study II, but the initial 'adjusted for IQ' estimate were comparable.*

6.3 STUDY III

The aim of the third paper was to test the hypothesis that education is causally related to mortality.

Here, Swedish registers of the total population was used. All children born between 1943 and 1955, in 900 municipalities that introduced a new comprehensive nine-year school for one of the cohorts born between 1944 and 1955 were eligible for the study. The reform was carried out as a quasi-experiment, each year exposing children in some new municipalities to the new school form, while keeping whole other municipalities as controls. Information about all deaths occurring until December 31, 2007, was collected from the Cause of Death Register. Thanks to the design, birth-cohort and municipal differences in mortality could be statistically adjusted for.

The analyses showed that the reform was associated with reduced mortality after age 40, but not before or during the whole follow-up. After age 40, the reform was associated with reduced mortality in overall cancer, lung cancer, ischemic heart disease, overall external causes and accidents.

6.4 STUDY IV

The aim of the fourth paper was to assess the hypothesis that individual *change* in the performance on IQ tests between ages 10 and 20 years is associated with mortality later in life.

The study used information about the boys in the MLS, for whom information about the results of a second IQ test, taken in 1948, was available. The mortality follow-up was carried out until December 31, 2006.

The analyses suggested that change in intelligence *was* in itself associated with lower mortality. These changes in intelligence partly mediated the association between own education and mortality. The father's education was related to both initial intelligence, and to own education, also after initial intelligence had been accounted for. Through these links, father's education was also related to greater increases in intelligence, and consequently, lower mortality. There was no evidence of any other link between father's education and mortality, other than those that follows from the results that was just mentioned.

7 DISCUSSION

The results from Study I suggested that early cognitive skills were associated with mortality for boys. This finding is in line with the literature reviewed in the background section. The database that was constructed was based on the Malmö Longitudinal Study, MLS, with children born in 1928, for whom intelligence was assessed at an early age and rich information on childhood social conditions collected. According to a recent systematic review, only three studied cohorts in the world are older, namely individuals in the Scottish Mental Survey of 1932, the Terman life cycle study of children with high ability, and a study of twin World War II veterans (only men) (12). Thus, Study I means an addition to the general literature. Furthermore, of the three mentioned studies, only one study could adjust its models for childhood socio-economic status.

In the background section it was argued that the association between cognitive skills and health could perhaps be understood by social adjustment. All three studies of the MLS in this thesis (I, II & IV) produced results that could be viewed in this light. Firstly, social adjustment is arguably dependent on the prevailing social conditions. If cognitive skills are related to health in ways that at least involve the social environment, this clearly opens up for what is sometimes referred to as 'heterogeneity of effect', i.e. in this case different associations between skills and health in different groups, defined for example by gender or time-period. The studies on the MLS cohort in this thesis found that there was *not* any association between cognitive skills and all-cause mortality for women - while in some other studies such associations have been found (12). One possible interpretation of this is that the effect of cognitive skills on health is dependent on the different social conditions which vary across for example time-periods, countries and genders.

Furthermore, the association between intelligence and all-cause mortality for men seemed to be partly mediated by their later educational attainment. Successful schooling could be viewed as a kind of social adjustment. Interestingly, this social adjustment in turn also seemed to *increase* cognitive skills (Study IV). That IQ can be raised in such a way is completely in line with literature reviewed in the background section. This increase could be *validated* here, in the sense that it was shown to be related to mortality. This is maybe the most important contribution of this study.

In Study II, the question about the possible mediating role of education and income was studied in more detail. Here, information about the partner was also added. These analyses suggested that a substantial part of the association between own education and later all-cause mortality *for men* could be understood by using information on own income. In contrast, cognitive skills remained associated with all-cause mortality throughout all models for men, including those in which information about the partner's education and income was taken into consideration.

Interestingly, for women there is a clear association between *education* and all-cause mortality in the MLS, despite the fact that their cognitive skills were not associated with mortality (Study I & II). It seemed to be possible to explain this association by

their partners' income, *but not* by own income. This finding could reflect the pooling of resources by couples and the relatively greater importance of men's income during the studied period. Furthermore, a finding that is in line with the potential *pooling of skills* by couples was detected: men's mortality was namely associated with *their wives'* educational level. Unfortunately, we could not directly study the association between own mortality and partner's cognitive skills, but this would be a natural next step in this field.

In Study III, the question about possible *causal* effects of education was studied. The study suggested links with mortality from lung cancer, ischemic heart disease, overall external causes and accidents. Thanks to the unique design of this study, these findings provide a clear contribution to the overall literature. In relation to mediation, the findings in Paper III are certainly consistent with skills being possible mediators, but it should be noted that this issue is not at all analysed in this study. Whether these causes of death suggest mediation by 'social adjustment' or not is open to discussion.. If smoking (the major cause of lung cancer) is a result of such processes (poor social adjustment), it is, for example, in line with this idea, but the link to accidents may be more direct, perhaps via risk-taking behaviours.

The final study (IV) attempted to model a more full possible causal chain between social background, initial cognitive skills, subsequent education, *changes* in the cognitive skills and later all-cause mortality. Two measures of skills are needed for this kind of analysis and we were therefore limited to the male half of the MLS, who were conscripted for military service at age 20. Few studies have previously been designed in this way, and some of the findings are intriguing. Not only was longer education related to greater increases in intelligence, but this increase in itself predicted all-cause mortality, and in this way may be thought of as one mediator of the association between education and health. Further, social differences in access to education had effects on social differences in both adult intelligence and adult health.

7.1 CONCLUSION

The field of cognitive epidemiology is new and this summary only provides a very tentative suggestion of one way of thinking about a frame of reference to this research. However, the four papers in this thesis are in line with this general context, in which education is linked to improved cognitive skills and cognitive skills, in turn, are linked to health, whether mediated by 'social adjustment' or more directly. Potentially then, skills in general - and the cognitive ones in particular - could prove to be important for our understanding of and efforts to improve population health.

As mentioned in the background section, generally improved cognitive skills in a population could well be combined with smaller differences between individuals in that population. Then, if the health effects of cognitive skills are not completely due to the position of one individual relative to other individuals, improved skills would mean both better health on average *and* lower health inequalities, if other factors remain the same. If this reasoning holds, it provides clear support for the benefits of the increasingly longer average education adopted over time for example in Sweden. It can of course also be noted here that this historical trend has been parallel both to a trend

towards increasing average IQ scores and increasing average life expectancy. For mortality, it is also clear that higher average life expectancy in general is accompanied by lower overall individual differences in life expectancy, both today and historically, as shown by data from 212 countries during the last 200 years (156).

At the same time, such positive trends are not fixed. It has been reported that the long term trends towards convergence in global mortality may have switched into divergence in the late 1980s (157). As we have shown, a school system with great variation in length of education may produce *increasing* social differences in both cognitive skills and health. It has even been suggested, as mentioned in the background section, that the Flynn-effect towards higher average IQ has levelled off or even changed direction in our part of the world during the last decades. Schooling may have affected this development, not due to a reduction in educational length, but perhaps due to changes in teaching practices. A recent thesis suggests such an explanation for the recent negative trends in Swedish students' mathematics performance (158). A natural policy response to this negative development that is yet to be seen in Sweden would be to implement the results from a vast international quantitative literature on what actually works in increasing school achievement (159).

Some possible ways forward *for research* are directly or indirectly indicated by the results in this thesis. Firstly, even if there is quite consistent evidence that cognitive skills can be improved *and* that skills are related to health, few studies have so far attempted what we did in Study IV, that is try to model a more full potential causal chain. More studies of this kind could be a natural next step. But there is also a need for further studies of the more straight-forward type, i.e. analysing the association between cognitive skills and mortality, in more materials - especially because of the suggestions that this association may function differently in different groups, e.g. between men and women. It has recently also been pointed out that if there is *not* a difference between men and women in the effect of cognitive skills on *all-cause* mortality, this implies that there must be differences in relation to specific causes of death, since what kills men and women differs (160).

One could also speculate about whether improved skills mean positive spill-over effects onto other people. Arguably, positive *effects* of own cognitive skills (e.g. higher income) affect other people. But it may very well be that skills are also 'contagious' in the way we tend to think of health behaviours such as smoking, so that one person improving her skills also improves the skills of those around her. In some materials, it should be possible to study such possible effects.

A final concrete way forward could be to merge the discussion of intelligence and health with the research into other skills. One such field is research into literacy where, interestingly, the idea that skills can be improved is one of the very starting points. The Organisation for Economic Co-operation and Development, OECD, is at the moment conducting a very large study on adult competencies including assessment of literacy, numeracy skills and problem solving, called PIAAC (www.oecd.org). Five thousand individuals aged 16-65 years in each of the 25 participating countries will be visited, interviewed and tested. The possibilities of linking this data to health and health-relevant outcomes are especially great in the Nordic setting and steps in this direction

have already been taken. Although this will not automatically make the world a better place, the descriptions that may result from such a work could very well prove useful in such efforts.

8 ACKNOWLEDGEMENTS

This thesis has come about much thanks to my main supervisor, colleague and friend Sven Bremberg. His unsentimental and clear-sighted views of the way things are and genuine will to improve conditions for children are contagious - and suddenly I had given up my conviction not to set foot in academia. I am equally thankful to my other supervisor Denny Vågerö who is always open to wild brainstorms and tirelessly provides insightful input - despite all my sudden turnarounds of the manuscripts. Thanks to my other co-authors Bitte Modin, Bianca De Stavola, Jenny Torssander and my mentor Bjöörn Fossum. Thanks Marit Gisselman, Kristiina Rajaleid and Jenny again for reviewing and commenting on my summary.

Furthermore, I am grateful to all those whom I have harassed with the daily problems of this thesis, including all the above-mentioned - and then pretty much everyone else who has either been placed at the Centre for Health Equity Studies, CHESS, or have been exposed to me in my spare time during these years.

Thanks to the Swedish National Institute of Public Health for financing me during the major part of this time. A special thanks to the former general director Gunnar Ågren and the present Sarah Wamala. Thanks to CHESS for financing the remaining part. A special thanks to Denny again as well as the present director Olle Lundberg and assistant director Viveca Östberg.

Thanks Felicia Lager and Josefine Lager for putting up with a father that is sometimes so absent-minded in the mornings that you risk being handed over at the wrong pre-school. Thanks to my parents and parents-in-law for turning out and solving everyday practical situations. Thanks to Erika Lager for standing by me in this work - and for making my life meaningful.

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