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Empirical Essays on Health and Economic Outcomes

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Abstract

This thesis contains four independent research papers, which investigate the causal relations between several aspects of health and economic outcomes at different stages of the life course. The first paper investigates the causal effects of maternal deprivation and maltreatment during various periods of childhood on adolescent health and human capital. Using hospital data and information on ninth year GPA for the entire Swedish population born in 1978-1995, we exploit between-sibling variation in the age at exposure to maternal psychiatric hospitalization. Our results indicate a greatly elevated risk of hospital admission due to self-harm and substance-related diagnoses during late adolescence among individuals exposed to maternal psychiatric hospitalization in childhood. We also find a relatively small negative impact on girls' ninth year GPA. Taken together, the results suggest substantial adverse effects on psychosocial health for individuals exposed to maternal psychiatric hospitalization during childhood. The detrimental effects on child health are especially pronounced for exposure at very early ages, especially for boys.

The second paper uses draft data covering the entire population of Swedish males born in 1965-1975 to study visually impaired individuals' labor market outcomes. A detailed and objective measure of visual acuity lets me distinguish visually impaired individuals whose impairment comprises a work-limitation from those whose productivity remains unaffected. Together with detailed information on occupational categories, this allows me to separate effects of work limitations and selection into professional categories from consequences of discrimination due to wearing glasses. The data contains objective information on cognitive and non-cognitive ability and general health, allowing me to investigate the role of important mediators. While I do not find any evidence of discrimination against individuals wearing glasses, my results suggest that work-limitations adversely affect visually impaired individuals' employment rates and earnings, already at a low level of reduced vision after optimal correction. I also show the importance of, most notably, non-cognitive ability in explaining part of the labor market disadvantage, suggesting difficulties for visually impaired individuals in acquiring this type of skills.

The third paper uses Danish day care teachers as an ideal case for analyzing whether or not work pressure, measured by the child-to-teacher ratio, that is, the number of children per teacher in a day care institution, affects teacher sickness absenteeism. We control for individual teacher characteristics, workplace characteristics, and family background characteristics of the children in the day care institutions. We perform estimations for two time periods, 2002–2003 and 2005–2006, by using generalized method of moments with lagged levels of the child-to-teacher ratio as instrument. Our estimation results are somewhat mixed. Generally, the results indicate that the child-to-teacher ratio is positively related to short-term sickness absence for teachers working with 1/2–3-year old children, but not for teachers working with 3–6-year olds.

In the fourth paper, we study the short-run effect of salary receipt on mortality among Swedish public sector employees. By exploiting variation in paydays across work-places, we completely control for mortality patterns related to, for example, public holidays and other special days or events coinciding with paydays and for general within-month and within-week mortality patterns. We find a dramatic increase in mortality on the day that salaries arrive. The increase is especially pronounced for younger workers and for deaths due to activity-related causes such as heart conditions and strokes. The effect is entirely driven by an increase in mortality among low-income individuals, who are more likely to experience liquidity constraints. All things considered, our results suggest that an increase in general economic activity on salary receipt is an important cause of the excess mortality.

Key words
health, mental health, human capital, children, visual impairment, earnings, employment, discrimination, cognitive ability, disability, work pressure, sickness absence, day care, mortality, consumption, liquidity constraints, permanent income hypothesis

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To my family

Contents

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In the fourth paper, we study the short-run effect of salary receipt on mortality among Swedish public sector employees. By exploiting variation in paydays across work-places, we completely control for mortality patterns related to, for example, public holidays and other special days or events coinciding with paydays and for general within-month and within-week mortality patterns. We find a dramatic increase in mortality on the day that salaries arrive. The increase is especially pronounced for younger workers and for deaths due to activity-related causes such as heart conditions and strokes. The effect is entirely driven by an increase in mortality among low-income individuals, who are more likely to experience liquidity constraints. All things considered, our results suggest that an increase in general economic activity on salary receipt is an important cause of the excess mortality.

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If anyone would have told me fifteen years ago that I would ever hold a Ph.D. in economics, I would have deemed them insane. As most economists are painfully aware, there is a lot of prejudice about our field, and I was one of those who instinctively disliked everything about it. I would never have set my foot at an Economics department, had it not been for that compulsory elementary-level course that I would have to suffer my way through. However, I eventually realized that economics was a much broader and more open field than I thought, and that economists were in general sensible and sympathetic people. Inga Persson, who supervised my bachelor and master theses and who remained a support and a friendly face throughout my Ph.D. studies, had a great part in this process. Nowadays, I try to pass along this message to my students and family members.

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Malmö, April 2, 2017
Elvira

Introduction

1 Background

Health and economic outcomes are heavily intertwined throughout the life course. A large literature documents strong positive correlations between health and various measures of economic and social success, suggesting that healthier individuals have higher earnings and employment rates. While this type of correlations is frequently observed, the issue of what actually causes them remains debated (see, for example, Currie and Madrian (1999) and Smith (1999)). This thesis aims at disentangling and investigating some of these pathways. In four independent research papers, I investigate the causal relations between several aspects of health and economic outcomes at different stages of the life course.

1.1 Health and human capital

The neoclassical economic framework models economic outcomes, such as earnings and employment, as functions of the individual's human capital stock, which determines his/her labor productivity (Becker, 1957, 1964). Building on the human capital framework, Grossman (1972b,a) develops a similar model, where health is interpreted as a capital good. In both models, each individual is assumed to be born with certain endowments, in terms of inherent ability (Becker, 1957, 1964) and an initial health stock (Grossman, 1972b,a). These initial endowments vary across individuals and depend on genetics and external causes. Individuals invest in education, health and other productivity-enhancing characteristics as to maximize lifetime utility, which is determined by consumption possibilities and time preference. The return to these investments depends, in turn, on the individual's initial endowments and on earlier investments.

The human and health capital models build on the notion of a rational individual investing in his/her own health and productivity. However, during childhood, parental investment is likely to be of great importance to children's health and human capital stocks. The level of these investments are in turn likely to be associated with parental productivity-related characteristics. For example, a growing literature establishes that higher levels of parental ability and engagement produces persistently higher levels of cognitive and noncognitive skill in children (see e.g. Carneiro and Heckman (2003), Heckman and Masterov (2007), Cunha et al. (2006), and Cunha et al. (2010)). Importantly, as an individual's human and health capital stocks are partly determined by earlier investments, parental investments in child health and ability are likely to have long-run consequences. Hence, beside a genetic connection between parents' and children's initial endowments, parental health, human capital, and preferences are likely to comprise additional channels of intergenerational correlations in both health and economic outcomes. Using maternal psychiatric hospitalizations as an indicator of low parental investment ability, due to both mother-child separation and ill mental health, paper 1 investigates this issue.

Ill health may in itself reduce labor productivity, thereby reducing earnings and employment prospects. However, it is also possible that ill health reduces the ability to participate in and benefit from productivity-enhancing activities, such as schooling. If this is the case, a low initial health endowment or an impairment acquired in early life may have adverse secondary effects on economic outcomes, running through a lower human capital stock (Johnson and Lambrinos, 1985; DeLeire, 2001; Hotchkiss, 2004). Further, the connection between health, human capital and economic outcomes may not be purely related to the supple side, but also contain demand-side elements. Several studies find that employers' attitudes are more negative towards the disabled than towards many other marginalized groups, such as the elderly or ethnic minorities, indicating the existence of employer taste discrimination (see e.g. Hahn (1983)and Bowe (1978)). Additionally, statistical discrimination may occur if workers with impairments are (correctly or incorrectly) perceived as being on average less productive or more expensive to hire and train than the able-bodied (Johnson and Lambrinos, 1985; Skogman Thoursie, 1999). Expectations of future discrimination may, in turn, reduce human capital investment among low-health individuals, due to lower expected rates of return (Neal and Johnson, 1996). In paper 2, I attempt to disentangle the effects of these mechanisms, analyzing visually disabled individuals' labor market outcomes.

Naturally, individual investment behavior is not the only thing affecting health. More recent theoretical models extend the health-producing units to include employers (see, for example, Bolin et al. (2002)). Work characteristics, such as stress or heavy physical demands, may affect health, leading to both short sick leave spells and long-term disability. Further, according to both the Becker (1957, 1964) and Grossman (1972b,a) models, human and health capital depreciate over time. While human capital depreciates when not being used, health capital requires continuous investments in order to keep the depreciation rate down until the health capital stock reaches the critical point that implies death. Time at work lost due to illness reflects depreciation of both human and health capital, which may in turn have detrimental consequences for future wellbeing and productivity (Grossman, 1972b,a). Paper 3 looks further into the issue of job characteristics as triggers of worker health by investigating the role of work pressure as a determinant of short- and long-term sick leave spells among Danish day care teachers.

Another aspect to take under consideration when analyzing the association between health and economic outcomes is the time frame studied. While the positive long-run association between income and health is well-documented and undisputed (see, for example, Smith, 1999; Deaton, 2003), several studies using data from developed countries show that mortality rates follow a pro-cyclical pattern, suggesting that the positive association between income and health does not apply to temporary income changes at the aggregate level (Ruhm, 2000; Neumayer, 2004; Tapia Granados, 2005; Gerdtham and Ruhm, 2006). A possible explanation of this discrepancy is that income receipt has adverse short-run health effects that partly offset the positive long-run association between income and health. In paper 4, we consider this possibility by studying the short-run effect of salary payments on mortality among Swedish public sector employees.

2 Summary of the papers

Paper 1

The effect of early life conditions on outcomes later in life is widely recognized. A large literature documents long-lasting negative effects of adverse conditions in utero or during early childhood on a broad range of outcomes, related to both health, behavior and human capital (see e.g. Almond and Currie (2011),Hertzman and Boyce (2010), and Fox et al. (2010)). A large and growing literature establishes that parents play an important role in this process, higher levels of parental ability and engagement producing persistently higher levels of both cognitive and non-cognitive skill in children (see e.g. Carneiro and Heckman (2003), Heckman and Masterov (2007), Cunha et al. (2006), and Cunha et al. (2010)). Also within the psychological literature, the importance of parent-child relations is emphasized, a secure parent-child attachment pointed out as being as an important determinant of many aspects of child development (see e.g. Cummings and Cichetti (1990) and Cummings and Davies (1994)). Hence, both quality and quantity of parental interaction is likely to be vital to children's accumulation of health and human capital.

Maternal psychiatric hospitalization may have detrimental effects on both these aspects of parental investment in children. Hospitalizations due to psychiatric conditions typically last longer than hospitalizations due to somatic or external causes, thus separating the child from his/her mother for a relatively long period of time. Further, as remarked by Cummings and Cichetti (1990) and Cummings and Davies (1994), mothers suffering from psychiatric illness may not be able to provide optimal emotional care and support for their children, and may be more likely to turn to neglect or physical or verbal abuse.

In this paper, we assess the causal effects of maternal deprivation and maltreatment during various periods of childhood on adolescent health and human capital. Using hospital data and information on ninth year GPA for the entire Swedish population born in 1978-1995, we exploit between-sibling variation in the age at exposure to maternal psychiatric hospitalization. Our results indicate a greatly elevated risk of hospital admission due to self-harm and substance-related diagnoses during late adolescence among individuals exposed to maternal psychiatric hospitalization in childhood. We also find a relatively small negative impact on girls' ninth year GPA. Taken together, the results suggest substantial adverse effects on psychosocial health for individuals exposed to maternal psychiatric hospitalization during childhood. The detrimental effects on child health are especially pronounced for exposure at very early ages, especially for boys.

Paper 2

In order to reduce the labor market disadvantages of workers with impairments or disabilities, learning why such disadvantages occur is an important task. A vast body of research has documented a negative association between weak health and labor market outcomes (see Currie and Madrian (1999) for an overview), but much less is known about what causes these differentials. Moreover, whereas much of the literature and policy debate focuses on the effects of severe health issues, less is known about the labor market consequences of relatively minor impairments, which are not acknowledged as disabling, but may still affect labor market possibilities.

Physical impairment may affect labor market outcomes through several channels. First, it may limit productivity. The reduction may occur either directly, or indirectly, for example, by making it difficult to attain and profit from schooling or other activities crucial to human capital accumulation (Johnson and Lambrinos, 1985; DeLeire, 2001; Hotchkiss, 2004). Second, more severe impairments could entail additional costs of entering the labor market, as eligibility for disability insurance may raise reservation wages (see e.g. Hotchkiss (2004), Autor and Duggan (2003), Bound and Waidmann (2002), Kruse and Schur (2003), and Burkhauser and Gumus (2003)). Further, labor market discrimination against people with impairments may be prevalent (Hahn, 1983; Bowe, 1978; Johnson and Lambrinos, 1985; Skogman Thoursie, 1999).

This paper uses draft data covering the entire population of Swedish males born in 1965-1975 to study visually impaired individuals' labor market outcomes. A detailed and objective measure of visual acuity allows me to distinguish visually impaired individuals whose impairment comprises a work-limitation from those whose productivity remains unaffected. Together with detailed information on occupational categories, this allows me to separate the effects of work limitations and selection into professional categories from the consequences of direct discrimination due to wearing glasses. The data contains objective information on cognitive and non-cognitive ability and general health, allowing me to investigate the role of important mediators of the between-group differentials.

While I do not find any evidence of discrimination against individuals wearing glasses, my results suggest that work-limitations comprise significant adverse effects on visually impaired individuals' employment probability and earnings, already at a low level of reduced vision after optimal correction. I also show the importance of, most notably, non-cognitive ability in explaining part of the labor market disadvantage, suggesting difficulties for visually impaired individuals in acquiring this type of skills. Taken together, the results of this study underlines the importance of acknowledging the work-limitations of individuals suffering from visual impairment, even at a relatively minor level, suggesting labor market and school policies targeting such groups.

Paper 3

Sickness absence comprises significant costs for society, implying costs related to health care, sickness benefits, substitute employees, and reduced productivity. Moreover, repeated long-term sickness periods comprise substantial costs also for the affected individual, by depreciating human capital, reducing wages and enhancing the risk of leaving the labour force. Sickness absence also harms the productivity and labour-market outcomes of sick-listed individuals' colleagues and families (Tompa, 2002).

The literature on occupational health points at pressure of work as a trigger of absence due to sickness (Lund et al., 2005). Unfortunately, reliable, objective measures of work pressure are in short supply. This paper uses Danish day care teachers as an ideal case for analyzing whether or not work pressure, measured by the child-to-teacher ratio, that is, the number of children per teacher in an institution, affects teacher sickness absenteeism. Controlling for individual teacher and workplace characteristics, we perform estimations for two time periods, 2002–2003 and 2005–2006. We exploit the panel dimension of the data using both a within estimation and a generalized method of moments approach with lagged levels of the child-to-teacher ratio as instrument, in order to account for possible endogeneity of the child-to-teacher ratio arising from healthy teachers selecting into municipalities with favourable working conditions.

Our estimation results are somewhat mixed. Generally, the results indicate a positive relation between child-to-teacher ratio and short-term sickness absence among teachers working with 1/2-3-year old children, but not among teachers working with 3-6-year olds. This discrepancy may be related to the greater dependency among younger children.

Paper 4

A large and growing literature has established a positive relationship between health and income, showing that mortality and morbidity rates are lower for high-income individuals (see, e.g., Smith, 1999; Deaton, 2003). However, several studies using data from developed countries show that mortality rates follow a procyclical pattern, suggesting that the positive association between income and health does not apply to temporary income changes at the aggregate level (see e.g. Ruhm (2000), Neumayer (2004), Tapia Granados (2005), and Gerdtham and Ruhm (2006)) A possible explanation of this discrepancy is that income receipt has adverse short-run health effects that partly offset the positive long-run association between income and health. In this paper, we consider this possibility by studying the short-run effect of salary payments on mortality among Swedish public sector employees.

We combine register data with survey-based information on exact paydays for the entire population of Swedish public sector employees between 1995 and 2000. Exploiting variation in paydays across public sector units, we employ a date-fixed effects strategy, i.e. we include a separate fixed effect for each day, to identify the mortality effect of salary receipt. This strategy allows us to completely control for mortality patterns related to, for example, public holidays and other special days or events coinciding with paydays and for general within-month and within-week mortality patterns.

Our findings indicate that the mortality consequences of salary receipt are large. We find a 23% increase in total mortality, corresponding to approximately 96 premature deaths per year if extended to include the entire Swedish working-age population, on the day that salaries are paid. Circulatory conditions are the main reason behind the excess mortality, representing an entire 83% of the increase. The effect is driven by a mortality increase among low-income individuals and is especially pronounced for young workers.

We connect the increase in mortality to a rise in consumption following income receipt, which has been documented by a an extensive literature (Shea, 1995; Shapiro and Slemrod, 1995; Parker, 1999; Souleles, 1999; Stephens Jr., 2003; Shapiro, 2005; Johnson et al., 2006; Stephens Jr., 2006; Elger, 2012; Huffman and Barenstein, 2005; Zhang, 2013; Stephens Jr. and Unayama, 2011). The increase in consumption has been shown to be greater for young individuals and for households who are likely to experience liquidity constraints, i.e. who have low incomes or liquid wealth (see, e.g., Stephens Jr., 2006; Johnson et al., 2006; Mastrobuoni and Weinberg, 2009). If consumption increases upon salary receipt, a temporary rise in activity, due to, for example, an increase in travel and the pursuit of leisure activities, is likely to arise. As previously dicussed by Evans and Moore (2011) and Miller et al. (2009), the raised activity level may cause a short-term increase in mortality due to causes that are activity-related and characterized by a short space of time between onset and death. All things considered, our results suggest that an increase in general economic activity on salary receipt is an important cause of the excess mortality.

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Paper I

Consequences of Early Life Adversity - Evidence from Maternal Psychiatric Hospitalizations

with Petter Lundborg

1 Introduction

The long-run effects of early life conditions are widely recognized. A large literature documents long-lasting negative effects of adverse conditions in utero or during early childhood on a broad range of outcomes, related to both health, behavior and human capital (see e.g. Almond and Currie (2011),Hertzman and Boyce (2010), and Fox et al. (2010)). A specific branch of this literature deals with the effects of parental deprivation during childhood. An extreme example of this relates to children raised in impoverished Romanian orphanages, for whom follow-up studies indicate adverse effects on both physical, socioemotional and cognitive development (see e.g. Bakermans-Kranenburg et al. (2008) and Rutter (2010)). In addition, numerous observational studies document adverse developmental and behavioral patterns for children who were abused, neglected or otherwise lacked secure attachment to their primary caregivers during childhood (see Hildyard and

Wolfe (2002) for an overview).

Simple comparisons between exposed and non-exposed individuals may fail to identify a causal effect due to unobserved between-group differences, however. Conti et al. (2012) avoid this selection problem in a study on primates, where rhesus monkeys were randomly allocated across mother rearing, peer rearing, and surrogate peer rearing. Their results show that monkeys who were deprived of their mothers displayed worse health and higher levels of aggression and self-harm behavior.[1] However, although these findings suggest lasting adverse effects of maternal deprivation for rhesus monkeys, to which extent they translate to the human species is yet to be revealed.

In this paper, we use between-sibling variation in exposure to maternal psychiatric hospitalization during childhood to study its effects on later health and human capital. We show that while exposure to maternal psychiatric hospitalizations is far from random between children of different families, exposure within families is much less related to an extensive set of predetermined maternal and child characteristics. We exploit this as-if randomness to identify the causal effect of exposure to maternal psychiatric hospitalization during different phases of childhood.

We find dramatic increases in the risk of hospitalization due to self harm and substance-related conditions at age 15-20 for children who were exposed to maternal psychiatric hospitalization during early life. We also find modest negative effects on girls' ninth year school grades. The detrimental effects on child health are generally greater for exposure at very early ages, especially for boys. However, they do not appear to be driven by exposure during the postnatal year, suggesting a limited role for postpartum depression and psychosis. The effects are not dri-

[1]Mother reared monkeys stayed with their biological mothers from birth and were raised together with other monkeys. Peer-reared monkeys were individually raised in a nursery until 37 days old and were subsequently placed in groups with the three other peer-reared monkeys who were closest in age. Surrogate peer-reared monkeys were placed alone in a cage with a surrogate mother consisting of a cloth-covered hot water bottle for 22 hour a day and spent the remaining two hours a day in a play cage with three other surrogate peer-reared monkeys. Between the age of six months and one year, all monkeys who were born during the same year were put together in a mixed group. All outcomes were measured after the first year, ensuring exposure to a normal environment for all monkeys. The authors find a significant reduction in physical health among surrogate peer-reared male monkeys, and higher levels of aggression and self harm behavior among female monkeys who were deprived of their mothers. Peer- and surrogate peer-reared monkeys of both sexes also displayed a higher probability of developing stereotypies, which are in turn associated with autism and cognitive and language deficits in humans.

ven by a correlation with adverse life events or paternal psychiatric hospitalization, suggesting that mothers' absence and mental illness affect child outcomes in their own right.

The remainder of the paper unfolds as follows. Section 2 describes the theoretical background and summarizes previous research. Section 3 describes our data. Section 4 discusses our empirical strategy. Section 5 reports our main results. In Section 6, we report results from separate analyses of specific sample subgroups and variation in treatment intensity. Section 7 reports the results from various robustness checks. In Section 8, we analyze the mechanisms behind the effects. Section 9 investigates the role of fathers. In Section 10, we discuss our findings.

2 Background

A large and growing literature emphasizes the role that parents play for children's production of human capital (see e.g. Carneiro and Heckman (2003), Heckman and Masterov (2007), Cunha et al. (2006), and Cunha et al. (2010)). The psychological literature also points to the importance of a secure parent-child attachment for many aspects of child development (see e.g. Cummings and Cichetti (1990) and Cummings and Davies (1994)). Hence, both quality and quantity of parental interaction are likely to be vital to children's accumulation of health and human capital.

Maternal psychiatric illness may affect both the quality and quantity of child-parent interactions. Severe psychiatric illness may result in long episodes of hospital care, thus separating mother and child for a relatively long period of time. Mentally ill mothers may also be less able to provide optimal emotional care and support for their children, and may be more likely to turn to neglect or abuse. This may in turn impair mother-child attachment and lead to emotional stress in the child (Cummings and Cichetti, 1990; Cummings and Davies, 1994). Further, maternal psychiatric illness could be related to other adverse conditions, such as substance abuse or family disruption, leading to additional strains on the child's home environment.

Emotional stress during childhood may have long-run effects on the formation of human capital. An extensive literature within neuroscience, shows that early emotional stress permanently alters brain function, resulting in reduced cognitive ability and higher levels of anxiety and aggressiveness. It has also been shown to trigger various psychiatric and somatic conditions, including, for example, depression, cardiovascular disease, asthma, diabetes, and chronic lung disease (see e.g. Hertzman (1999) and Shonkoff et al. (2009) and the references therein).

The sensitivity to emotional stress may depend on the age at exposure. A large and growing literature shows that the plasticity of the human brain varies throughout life, identifying sensitive periods for certain developmental phases during childhood. Knudsen et al. (2006)Cunha and Heckman (2008), Cunha et al. (2010), and Heckman and Mosso (2014) relate these findings to investment models in economics, establishing evidence of sensitive periods for investment in children's ability.

Several empirical studies confirm the importance of sensitive periods in development. Beckett et al. (2006) find much greater gains in cognitive ability for children who were adopted from Romanian orphanages before the age of six months than for those adopted at older ages. Further, Ashman et al. (2002) find that the negative association between exposure to maternal depression and stress reactivity is at its peak between birth and age two. In line with these findings, Kotulak (1996) argues that the period between birth and age three is vital for the production and later retention of synapses, which transmit information inside the brain, suggesting that inadequate mental stimulation during this period adversely impacts future development.[2]

This study contributes to the literature by providing new evidence on the long-lasting effects of parent-child relations during specific periods of childhood. We are the first, to our knowledge, to study the impact of maternal psychiatric hospitalization on child health and human capital outcomes using as-if random variation in exposure between siblings.

[2]Other studies suggest that while cognitive skill rank is relatively stable from an early age, noncognitive skills and traits are more malleable during later periods (Cunha et al., 2010, Cunha and Heckman, 2008). As noncognitive characteristics are related to psychosocial function, these results suggest that inputs in later parts of childhood could affect psychosocial wellbeing. Similarly, sensitive periods for several aspects of physical health and development have been documented past the very first years of life. For example, van den Berg et al. (2014) find a threshold at age 9 in the development of adult height, Sparén et al. (2004) find that a sensitive period for future around age 9, and van den Berg et al. (2009) show that children who reach the age of 3 during an economic downturn have greater mortality rates in later life.

2.1 Psychiatric illness

In order to identify mediators and confounders affecting the relationship between maternal mental illness and child outcomes, understanding the causes of psychiatric disorders is important. In this section, we discuss the diagnosis categories included in our study and previous research on the determinants of mental illness. Knowledge about these determinants is crucial to our study design, which exploits between-sibling variation in exposure to mothers' psychiatric hospitalization under the assumption that no unobserved factors correlate with both maternal psychiatric hospitalization and child outcomes within families.

The conditions included in our study belong to the affective disorder, neurosis, and psychosis categories of the ICD8-ICD10 classifications.[3] In order to ensure between-sibling variation in exposure, we restrict our analysis to diagnoses which do no imply a constant state of illness. For this reason, we exclude schizophrenia, which often leads to severe chronic symptoms. Further, we exclude diagnoses directly induced by substance addiction, injury or somatic disease, and also somatoform conditions, in order to limit the influence of factors other than psychiatric illness.[4] In order to limit the influence of stressful life events that may have a separate influence on child outcomes if experienced at a family level, we also exclude diagnoses directly induced by stress, such as post-traumatic stress syndrome.

It is well known that psychiatric disorders display a great degree of heredity, and the main determinant of all conditions included in our study is thought to be biological/genetic. Their onset may also be triggered by stressful life events, such as the

[3] The data does not allow us to construct subcategories that are consistent across ICD versions. However, hospitalizations classified according to ICD10, which was implemented in 1997, can be divided into three subcategories. Various types of *psychosis* accounts for 14% of the post-1996 psychiatric hospital admissions in our population of mothers. Psychoses include abnormal perceptions and beliefs, and frequently also social withdrawal and cognitive deficits. *Affective disorders* amount to 64% of the psychiatric hospital admissions. This category includes depressive disorder and bipolar disorder, amounting to 43% and 21% of the total number, respectively. While depressive disorder manifests through low mood and energy, bipolar disorder includes manic or hypomanic episodes, that is, periods of elevated mood and energy, in addition to recurrent depressive episodes. *Neurotic disorders* account for 21% of the psychiatric hospitalizations. Unlike psychoses, neurotic disorders do not incorporate disillusions, but the patient apprehends the abnormality of his/her perceptions. Various anxiety syndromes account for 18% of the total hospitalizations. 3% are split among obsessive-compulsive disorders, phobia, dissociative syndrome, and other neuroses.

[4] Somatoform conditions are disorders where the patient worries excessively about symptoms that suggest physical illness, but where no such illness can be found.

death of a family member, divorce, or financial problems, or related to long-lasting social adversity, such as deprivation or isolation, however. The relative importance of these factors vary between diagnoses. Affective disorders and neuroses are believed to display a greater relationship with life events (Marneros and Brieger, 2002; Kendler and Gardner, 2016; Monroe et al., 2007; Beard et al., 2008), while lasting social adversity is of greater importance for the onset of psychosis (Bebbington et al., 1993; Mueser et al., 1998; Bebbington et al., 2004; Spauwen et al., 2006; Garety et al., 2007).

Both heredity and the correlation between mental illness and adverse events and situations may give rise to biased estimates of the effects of maternal psychiatric hospitalization on child outcomes. Parents and children may share both genetic markers and environmental factors related to mental illness. Therefore, simple comparisons across families of children exposed and unexposed to maternal psychiatric hospitalization is especially prone to bias, as the researcher is unable to account for all between-family differences related to both psychiatric conditions and child outcomes.

In order to account for such confounders at the family level, our empirical design employs family-fixed effects, which rely on within-family variation in child exposure to maternal psychiatric hospitalization. This design helps us in several ways. First, if a mother has a genetic predisposition to mental illness, the same predisposition is allocated randomly across the children (due to Mendellian randomization). Second, to the extent that environmental factors, such as low income or social isolation, are shared by all family members, we can rule these out as confounders. We also control for a number of maternal life events prior to each childbirth. The underlying assumption is that child exposure to maternal psychiatric hospitalization is largely random within families, at least conditional on a set of observable and known risk factors. We investigate this assumption in detail in our empirical analysis.

2.2 Maternal psychiatric illness and child outcomes

Studies addressing the causal relationship between maternal mental illness and child outcomes are in short supply. However, numerous observational studies document negative associations between maternal mental illness and a broad range of short- and long-term child outcomes.

Studies on mothers' mental health and child development primarily focus on depression, documenting strong negative associations between self-reported depressive symptoms and numerous aspects of child development and wellbeing. Compared to the offspring of nondepressed mothers, studies document more difficult temperaments and lower scores on mental and motor development tests among infants, delayed development of self-regulation strategies and elevated stress-sensitivity among preschool age children, and increased rates of emotional and behavior problems among school age children and adolescents exposed to maternal depression (see e.g. Cummings and Davies (1994) and Gotlib and Goodman (1999)).

Few studies address the relationship between maternal mental health and school achievement. Also, their results are inconclusive, Claessens et al. (2015) and Dahlen (2016) finding small reductions in math and reading test scores for 6-11 year-old children exposed to maternal depression, whereas Frank and Meara (2009), find no such effects. Similarly, the relationship between mothers' mental health and children's physical health is relatively unexplored. The existing evidence, a study based on British survey data, documents a strong correlation between general health among 7-year-olds and their mothers' mental health status (Propper et al., 2007).

A study by Johnston et al. (2013) also suggests that children exposed to maternal mental illness tend to fare worse in adulthood than their non-exposed peers. The authors find a great intergenerational correlation in mental health. Critically, they also show that maternal mental health predicts a range of adult outcomes, including educational attainment, income, and criminal behaviour, and that this association persists when controlling for mental health. Hence, they argue that the observed relationships do not just reflect heredity in mental health, but that a long-term association between maternal mental health and child behavior exists in its own right.

3 Data

The paper uses data from the Swedish Interdisciplinary Panel (SIP) database, which contains longitudinal data from several nation-wide registers. Our data set covers the entire population born in 1978-1995 and their parents. In order to ensure complete co-verage of outcomes and explanatory factors, we only include individuals who lived in Sweden throughout the period between birth and the age at which the outcome is measured (age 15 in the analysis of school achievement and age 20 in the analysis of health outcomes). These individuals constitute 96% of our population. We also restrict our sample to persons whose mothers lived in Sweden during the five years prior to their birth (95% of the remaining population).

As our identification strategy relies on within-family variation, we exclude individuals whose biological father is unknown and restrict our sample to individuals have at least one full sibling represented in the data (about 67%). This restriction is not straightforward, as mothers who experience psychiatric hospitalization during their child's early years are less likely to have more children. Hence, if the consequences of exposure to mothers' psychiatric hospitalization differ between children with and without and siblings, excluding one-child families could limit external validity. We discuss this issue in section 7.1.

In order to ensure genetic similarity between siblings and treatment exposure at an early age, and to avoid selection issues, we restrict the sample to individuals who were raised by their biological mothers (over 99.9% of the population). In order to avoid reverse causality, we also exclude children who were hospitalized due to perinatal conditions prior to age 15 (approximately 5%), as consistent illness in children may trigger maternal mental illness. Further, we exclude individuals with missing records of any explanatory variable used in our main analysis.[5] This results a sample size of 806,326 observations for our analysis of health outcomes. Also excluding individuals without a ninth grade GPA record (1% of our population) leaves us with 989,546 observations for our analysis of human capital outcomes.

3.1 Health records

From the *Inpatient register*, we have information on the main and up to 21 contributing diagnoses for each hospitalization. Using ICD8, ICD9 and ICD10 codes, we create broad diagnosis categories to be analyzed separately. We base these categories on ICD standard classifications, including psychiatric conditions (excluding diagnoses related to intellectual disability), somatic disease, accidents, self harm, abuse, and substance-related diagnoses. Using this information, we design a set of binary outcome variables, which take on the value one if the individual has any hospital record where at least one contributing cause belongs to the selected

[5] We drop 3% of our population due to missing records of apgar score or birth weight, and 7% due to missing income records.

category at age 15-20.

Our treatment variables are more narrowly defined. In order to capture the effects of psychiatric hospital admission, rather than hospitalization due to related conditions, we construct a set of indicator variables which take on the value one if the mother has any hospital record with a main diagnosis that belongs to the psychosis or neurosis subcategories, excluding schizophrenia, somatoform conditions and conditions directly induced by substance use, somatic disease, stress or trauma, or somatic disease, during different periods of the child's early life. In order to investigate whether the consequences differ by age at exposure, we use three levels of aggregation. Our first outcome variable takes on the value one if the individual's mother was admitted to hospital due to a psychiatric condition during the postnatal year. For our second group of outcome variables, the aggregation levels are birth to age 4, age 5 to 9, and age 10 to 14. Finally, we create a dummy variable indicating exposure at any point between birth and age 14.[6] For a complete list of the diagnoses included in each category, see tables A.1 and A.2 in the Appendix.

3.2 School grades

From the *SIP register*, we have access to information on ninth-year school grades for the years 1989-2011 for the entire population. he ninth school year is the final year of compulsory primary school in Sweden, and is generally completed in June the year the individual turns 16.

In 1998, the Swedish school system underwent an extensive assessment reform, where a grading system based on relative assessment on a 1-5 point scale was replaced by one based on central assessment criteria with the possible grades 0, 10, 15, and 20. As the systems are not entirely comparable, we transform the GPA variable into percentiles and assign each student a rank between 1 and 100, relative to his/her peers the same year, in order to allow comparisons over time.[7]

[6] For both treatment and outcome variables, hospitalizations which overlap a birthday are assigned to the child's age at its beginning.

[7] This method has previously been used by Nordenskjöld et al. (2015).

3.3 Background variables

Combining two nation-wide registers, we have access to a rich set of background variables for our population of mothers. Tax records allows us to control for pre-conception family income.[8,9] Based on census data, we construct a set of indicator variables representing stressful events that may trigger psychiatric illness for the five-year period prior to childbirth. These events include divorce, the death of a close family member (parent, spouse, or child), large income loss, and moving between municipalities.[10,11]

The *Medical Birth register* provides detailed information on health at birth for every child born in Sweden. This allows us to control for characteristics related to future health and cognitive outcomes, such as apgar scores, gestation, and birth weight.

3.4 Descriptive statistics

Table 1 provides a statistical description of our population. 14,498 out of the 989,546 individuals included in our analysis, amounting to approximately 1.5%, were exposed to maternal psychiatric hospitalization at some point prior to their fifteenth birthday. 20% of these individuals experience repeated spells. The between-group differences in socioeconomic and demographic characteristics are relatively small, including slightly lower family incomes, poorer maternal education levels, and an overrepresentation of individuals with at least one foreign-born parent among exposed children. Further, the average family size is slightly larger, and high-parity and youngest children are overrepresented in the exposed group. On average, exposed children are also in slightly poorer health at birth than their non-exposed peers.

[8] More precisely, the income measure included is the percentile of maternal disposable family income the year before conception.

[9] The reason for using family income for the year prior to conception rather than the prenatal year is to rule out effects of sick leave spells due to pregnancy-related health issues on income.

[10] Large income loss equals a reduction of 25% or more in disposable family income compared to the previous year.

[11] Information that is collected on a yearly basis is typically collected at the end of the year. Therefore, we connect this information to the life-year that is the closest to the end of the calendar year in question. Hence, parental background variables connected to the year before childbirth stem from the actual year prior to birth for children born during the second half of each year, and from two years prior to birth for children born during the first half of the year.

Table 1: Sample statistics.

	Full sample	Exposed	Unexposed
Observations	989,546	14,498	975,048
Recurrent maternal hospitalizations	0.0003	0.200	
Income quartile 1	0.290	0.285	0.291
Income quartile 2	0.220	0.224	0.220
Income quartile 3	0.263	0.249	0.263
Income quartile 4	0.227	0.242	0.227
Low maternal education	0.602	0.685	0.600
Immigrant background	0.094	0.133	0.094
Female	0.492	0.495	0.492
# children in family	2.884	3.093	2.881
Parity	2.008	2.224	2.005
Spacing	3.048	3.006	3.049
Maternal age at birth	28.61	28.74	28.61
Youngest sibling	0.402	0.422	0.402
Low birth weight	0.020	0.024	0.020
Apgar at 5 min	9.614	9.597	9.615
Premature birth	0.016	0.022	0.016
Divorce	0.021	0.044	0.020
Inter-municipal move	0.398	0.415	0.359
Family death	0.002	0.003	0.002
Income loss > 25%	0.136	0.173	0.135
Psychiatric hosp prenatal year	0.0005	0.0157	0.0003
Psychiatric	0.0234	0.0525	0.0229
Accidents	0.0453	0.0504	0.0452
Self harm	0.0062	0.0172	0.0061
Abuse	0.0028	0.0052	0.0028
Substance-related	0.0197	0.0418	0.0197
Somatic	0.0846	0.0956	0.0844
Percentile; 9th year GPA	51.006	43.876	51.112

Notes: Exposure relates to the entire prenatal year to age 14 period. Low maternal education corresponds to the mother having post-secondary education or less. Immigrant background corresponds to having at least one foreign-born parent. Spacing is the age difference to the sibling closest in age. Divorce, inter-municipal move, family death, and income loss > 25% are average incidence of the specified event 1-5 years before conception. Psychiatric, accidents, self harm, abuse, substance-related, and somatic are the incidence of hospital admissions to the specified diagnosis category during the age 15-20 period.

The groups differ substantially with respect to maternal exposure to stressful life events. For example, maternal divorce during the five-year period prior to childbirth is more than twice as prevalent in the exposed group than among the non-exposed. Importantly, children who were exposed to maternal psychiatric hospitalization during their first fifteen years are also much more likely to have been so in the womb. This reflects a recurrent pattern in mental illness and potential differences in intrauterine environments between the treatment and control groups.

The between-group differences in outcomes are substantial. Exposed children are more likely than the non-exposed to experience hospitalization at age 15-20 due to all diagnosis categories included in our analysis. The differences are especially pronounced for psychiatric conditions, self-inflicted injuries, and substance-related conditions, amounting to 129%, 182%, and 112%, respectively. Further, exposed children are approximately 7.2 percentage points below their non-exposed peers in the relative GPA distribution during their ninth school year.

4 Empirical strategy

Health and school achievement are known to be associated with both biological and socioeconomic characteristics, which, in turn, predict maternal psychiatric hospitalization. Hence, a simple comparison of exposed and unexposed individuals may fail to identify a causal effect due to endogeneity stemming from heredity and family environment. In order to control for such factors, we exploit variation in exposure between siblings.[12] We estimate the model

$$y_{ij(t+\tau)} = \alpha + \beta PSmother_{ijt} + \delta C_{ij} + \gamma X_{ij} + \theta_j + \varepsilon_{ij(t+\tau)} \tag{1}$$

where ij is an index for individual i belonging to family j. $PSmother_{ij}$ is a dummy variable that takes on the value one if the individual's mother was hospitalized due to a psychiatric condition at any point during time period t. θ is a family-fixed effect that accounts for all genetic and social characteristics shared by siblings.

For the fixed effects specification to provide causal estimates, exposure to mothers' psychiatric hospitalization must be as good as random within families. We address this in section 5, where we find that whereas a number of predetermined characteristics and life events predict exposure between families, exposure within families appears much less related to the same set of control variables.

Although most of our control variables were shown to be unrelated to our treatment variables, we include them all in our regressions in order gain precision. The vector C represents child characteristics, including parity, apgar score five minutes after birth, age difference to the closest sibling, year of birth-fixed effects and a set of binary variables representing sex, premature birth, low birth weight, and being the youngest sibling. X is a vector of maternal characteristics, including age at childbirth, family income percentile the year before conception, and a set of indicator variables representing stressful life events that may trigger maternal psychiatric illness during the five-year period before childbirth. These events include the death of a close family member, divorce, large income loss, and moving

[12] We perform our analysis on full siblings, i.e. siblings who share both biological parents.

between municipalities. Also, psychiatric illness is frequently recurrent and an individual may be more sensitive to relapse shortly after an illness episode. Hence, in order to rule out effects of prenatal exposure to maternal mental illness, we add an indicator variable which takes on the value one for mothers with a psychiatric hospital record during the prenatal year.[13] $\varepsilon_{ij(t+\tau)}$ is an idiosyncratic error term. For completeness, Table A.3 in the Appendix reports the results of similar regressions including only a very limited set of baseline control variables.

The outcome variable y_{ij} differs between the analyses; when assessing health outcomes, y_{ij} is the probability of individual i being hospitalized due to the specified condition at any point during age 15-20, whereas in the analysis of human capital outcomes, y_{ij} is the ninth-year GPA rank for individual i. The coefficient of interest is β, which can be interpreted as the remaining effects on health/human capital conditional on the confounders reported above.

As we rely on within-family variation for identification, the control group for each age period consists of siblings who were unexposed during that specific period. Hence, for all periods except the birth to age 14 period, siblings included in the control group may be exposed during another period. As these siblings may also suffer negative consequences, our estimates can be viewed as lower bounds for the causal effects. For the analysis of the entire birth to age 14 period, between-sibling variation in exposure implies that siblings belonging to the control group are unexposed to maternal psychiatric hospitalization prior to age 15. However, between-sibling variation over this age span occurs only when siblings are very far apart in age, when an older sibling is exposed early, or when a younger sibling is exposed late during the period. Hence, for this age period, the variation originates in a rather specific group of individuals. We further discuss this issue in Section 7.2.

[13]Mental illness during pregnancy has been shown to affect child health and development through several channels. A growing literature establishes a connection between prenatal stress and future psychiatric disease in children, likely due to low serotonin exposure in utero (see St-Pierre et al. (2016) for a recent overview). Mentally ill mothers may also less nutritiously during pregnancy. The resulting antenatal nutritional deficit could lead to poor behavioral and cognitive outcomes (Korenman et al., 1995), and adversely affect neurodevelopment and later life health (Barker, 1998).

5 Results

Before turning to our main results, we investigate the randomness of our treatment variables. Table 2 reports the results of regressing an extensive set of socioeconomic and demographic factors, child characteristics, and stressful pre-childbirth life events on maternal psychiatric hospital admissions during different periods, ranging between childbirth and the child's 15th birthday. The explanatory variables are identical to those included in Model 1, through which we perform our main analysis. Columns 1 to 5 report the results from an OLS model, investigating variation between families, while columns 6 to 10 report the results from a model including family-fixed effects to assess variation within families. In order to capture time trends in, for example, the probability and efficiency of psychiatric hospital treatment, all specifications include fixed effects for year of birth. Further, as relapse of prenatal psychiatric conditions may constitute a disproportionate share of hospitalizations during early periods, we include a dummy variable representing psychiatric hospitalization during the prenatal year in all specifications.

As expected, the OLS results, reported in columns 1 to 5, display significant associations between maternal psychiatric hospitalization and both socioeconomic and demographic factors, life events, and child health. However, when adding family-fixed effects to the specification, as displayed in columns 6 to 10 of Table 2, the point estimates turn substantially smaller and statistically insignificant in nearly all cases.

Two variables are strong predictors of maternal psychiatric hospitalization within families. Parity is positively related to exposure during all periods except age 5 to 9. Further, youngest siblings are at greater risk of exposure prior to age 5, and conversely at lower risk during later periods, probably due to a reduced future fertility among mothers experiencing postpartum mental illness (see Section 3 for a further discussion). Apart from these two factors, none of our explanatory variables predicts maternal psychiatric hospitalization during the postnatal year (see column 6), and only one variable does so, at the 10% significance level, for the birth to age 4 and age 5 to 9 periods (see columns 7 and 8). For the age 10 to 14 and birth to age 14 periods, displayed in columns 9 and 10 of Table 2, low birth weight has some predictive power, suggesting that having a less healthy child triggers future mental illness. Taken together, exposure to maternal psychiatric hospitalization appears much more random within than between families, at least with respect to the control variables reported above. However, as complete randomness is not

Table 2: Estimates of the probability of maternal psychiatric hospitalization during different periods of the child's early life

	(1) Postpartum	(2) Birth to age 4	(3) Age 5 to 9	(4) Age 10 to 14	(5) Birth to age 14	(6) Postpartum	(7) Birth to age 4	(8) Age 5 to 9	(9) Age 10 to 14	(10) Birth to age 14
Female	4.78e-05	-2.98e-05	-8.93e-07	8.09e-05	4.14e-05	-2.99e-05	-0.000132	-9.32e-05	5.10e-05	-3.72e-05
	(7.25e-05)	(0.000137)	(0.000156)	(0.000168)	(0.000239)	(9.22e-05)	(0.000133)	(0.000154)	(0.000165)	(0.000144)
Low birth weight	2.46e-05	-8.40e-05	0.00192***	0.00192***	0.00275***	-8.19e-05	-0.000929	0.00110	0.00181***	0.00196***
	(0.000272)	(0.000501)	(0.000647)	(0.000688)	(0.000945)	(0.000375)	(0.000606)	(0.000674)	(0.000682)	(0.000604)
Apgar at 5 minutes	-7.80e-05	-0.000251**	-0.000227	-0.000352**	-0.000593***	-9.82e-05	-0.000120	3.76e-05	-0.000185	-0.000131
	(6.57e-05)	(0.000121)	(0.000140)	(0.000152)	(0.000215)	(8.37e-05)	(0.000118)	(0.000130)	(0.000150)	(0.000129)
Premature	0.000252	0.00164**	0.000576	0.00145*	0.00359***	-5.63e-05	-7.86e-05	0.000188	0.000597	0.000867
	(0.000327)	(0.000645)	(0.000677)	(0.000757)	(0.00108)	(0.000448)	(0.000642)	(0.000732)	(0.000746)	(0.000692)
Parity	0.000112	0.00128***	0.00123***	0.00138***	0.00291***	0.000435***	0.00107***	8.13e-05	0.000625**	0.00139***
	(8.42e-05)	(0.000161)	(0.000185)	(0.000201)	(0.000284)	(0.000157)	(0.000249)	(0.000287)	(0.000297)	(0.000294)
Youngest sibling	0.000604***	0.000663***	-0.000106	-0.000435	0.000194	0.000683***	0.000535***	-0.000550**	-0.000646***	-0.000326
	(0.000126)	(0.000240)	(0.000275)	(0.000297)	(0.000421)	(0.000135)	(0.000207)	(0.000240)	(0.000249)	(0.000244)
Spacing	0.000125***	-7.36e-05	-2.39e-05	1.40e-05	-9.63e-05	3.09e-05	-4.75e-05	0.000118	-2.29e-05	9.97e-05
	(3.01e-05)	(5.08e-05)	(5.45e-05)	(5.91e-05)	(8.37e-05)	(5.34e-05)	(0.000107)	(0.000114)	(0.000122)	(0.000131)
Maternal age at childbirth	4.19e-05***	7.04e-05***	-1.31e-05	-9.17e-05***	-8.87e-05**	-0.000164	-0.000137	0.000323*	-0.000120	5.52e-05
	(1.18e-05)	(2.21e-05)	(2.44e-05)	(2.63e-05)	(3.73e-05)	(0.000118)	(0.000168)	(0.000195)	(0.000212)	(0.000186)
Income percentile	-8.14e-06***	-2.09e-05***	-1.55e-05***	-1.85e-05***	-4.43e-05***	-3.36e-06	-4.38e-06	1.86e-06	-2.69e-06	-4.57e-06
	(1.88e-06)	(3.49e-06)	(3.91e-06)	(4.24e-06)	(6.01e-06)	(2.80e-06)	(3.99e-06)	(4.60e-06)	(4.81e-06)	(4.26e-06)
Divorce	0.000971***	0.00409***	0.00473***	0.00491***	0.0110***	0.000754	-0.000546	0.000112	-0.00155	-0.00201
	(0.000375)	(0.000736)	(0.000812)	(0.000856)	(0.00123)	(0.000735)	(0.00121)	(0.00129)	(0.00146)	(0.00129)
Inter-municipal move	0.000340***	0.00119***	0.00139***	0.00175***	0.00317***	0.000101	-8.53e-06	4.09e-05	-7.63e-07	1.99e-05
	(8.04e-05)	(0.000153)	(0.000173)	(0.000189)	(0.000266)	(0.000141)	(0.000224)	(0.000259)	(0.000275)	(0.000238)
Family death	-3.58e-05	-0.00131	0.00174	0.000748	0.00239	0.000171	-0.00307	0.000282	0.00561	0.00349
	(0.000804)	(0.00154)	(0.00211)	(0.00210)	(0.00310)	(0.00142)	(0.00228)	(0.00337)	(0.00342)	(0.00332)
Income loss > 25%	0.000204*	0.000397*	0.00130***	0.00181***	0.00276***	3.14e-05	-0.000489*	0.000171	0.000146	-0.000303
	(0.000118)	(0.000221)	(0.000260)	(0.000287)	(0.000398)	(0.000180)	(0.000284)	(0.000336)	(0.000355)	(0.000305)
# children in family	3.12e-05	-6.59e-05	0.000216	0.000213	0.000560***					
	(6.49e-05)	(0.000118)	(0.000139)	(0.000153)	(0.000215)					
Primary schooling	0.000198	0.00214***	0.00292***	0.00292***	0.00630***					
	(0.000150)	(0.000298)	(0.000341)	(0.000360)	(0.000513)					
Postsecondary schooling	-0.000390***	-0.00105***	-0.00163***	-0.00181***	-0.00328***					
	(7.98e-05)	(0.000151)	(0.000171)	(0.000185)	(0.000263)					
Primary schooling father	-4.39e-05	4.67e-05	-0.000341	-0.000272	-0.000212					
	(0.000101)	(0.000195)	(0.000220)	(0.000235)	(0.000337)					
Postsecondary schooling father	3.46e-05	-5.20e-05	-0.000105	0.000192	-5.52e-05					
	(8.69e-05)	(0.000165)	(0.000187)	(0.000202)	(0.000287)					
Foreign-born	2.73e-05	0.00107***	0.00111**	0.00160***	0.00371***					
	(0.000209)	(0.000414)	(0.000457)	(0.000499)	(0.000713)					
Foreign-born father	0.000678***	0.00149***	0.00140***	0.00143***	0.00303***					
	(0.000193)	(0.000351)	(0.000388)	(0.000418)	(0.000593)					
Family-fixed effects						Yes	Yes	Yes	Yes	Yes
Means	0.0013	0.0049	0.0063	0.0071	0.0148	0.0013	0.0049	0.0063	0.0071	0.0148
Observations	980,877	980,877	980,877	980,877	980,877	989,546	989,546	989,546	989,546	989,546

Notes: *, **, and *** denote significance at the 10, 5, and 1 percent levels. All specifications control for maternal psychiatric hospitalization during the prenatal year and year of childbirth fixed effects. Spacing is the age difference to the sibling closest in age. Income percentile is measured the year before conception. Divorce, inter-municipal move, family death, and income loss > 25% are indicator variables representing the specified event 1-5 years before childbirth. Robust standard errors in parentheses.

present, and for efficiency reasons, we include all variables reported above in our main specification.

5.1 Health and human capital

Maternal psychiatric hospitalization may affect several aspects of child health and human capital, and may do so in different ways. Further, the consequences could differ by the timing of exposure, due to sensitive periods for physical, cognitive and socioemotional development. In order to investigate these potential effects, Table 3 displays the results of separate estimations of Model 1 using the probability of hospital admission due to each of our selected diagnosis categories at age 15-20 and cohort-specific ninth grade GPA rank as outcome variables.

In column 1, we investigate the effect of exposure to mothers' psychiatric hospitalization on the probability of hospitalization due to mental conditions. The point estimates are generally large and positive, but statistically insignificant, suggesting that childhood exposure is not the main force behind the strong intergenerational correlation in mental health reported in the psychological literature (see e.g. Johnston et al., 2013). Column 2 reports large and negative, but statistically insignificant, estimates regarding hospital admissions due to accidents.

Column 3 displays a statistically significant 160% increase of the probability of hospitalizations due to self-inflicted injuries during adolescence for children who were exposed to maternal psychiatric hospitalization before their fifth birthday. It is worth noting that, although imprecisely measured, the point estimate for exposure during the postnatal year is nearly as great as for the birth to age 4 period, suggesting that postpartum psychiatric hospitalizations account for part of the effect. A borderline significant 129% increase is also evident for children exposed at any point prior to their fifteenth birthday.

The estimates regarding hospital admissions due to abuse, displayed in column 4 of Table 3, are large and predominantly negative, although imprecisely measured. Column 5 reports a borderline significant 68% increase in substance-related hospitalization rates among individuals who were exposed to maternal psychiatric hospitalization before age 5, and a corresponding 49% increase for individuals exposed at any point during the birth to age 14 period. Conversely, we find no evidence of consequences regarding somatic hospitalization during adolescence (see column 6). However, this does not necessarily contradict the findings reported in

Hertzman (1999) and Shonkoff et al. (2009), which suggest detrimental effects of early-life stress on a number of somatic conditions, as many of them, such as type II diabetes and cardiovascular disease, predominantly manifest later in life.

Column 7 of Table 3 reports the results regarding school achievement. As maternal deprivation at early ages have been shown to hinder social and cognitive development (Bakermans-Kranenburg et al. (2008), Rutter (2010)), early exposure to maternal psychiatric hospitalization may manifest in a lower human capital level later in life. It is also possible that exposure during later childhood immediately affects school work, resulting in a stagnated human capital level. However, although the point estimates are nearly consistently negative, our main results reveal no conclusive evidence of adverse effects on GPA rank.

Taken together, our results are consistent with the literature reporting an increase in psychosocial problems among children raised under adverse conditions. The increase in hospitalizations due to self-inflicted injuries points at an increase in internalizing behaviors among exposed children. Similarly, the increase in substance-related hospitalizations indicates substantial negative effects on psychosocial wellbeing, substance use being associated with both depressive symptoms, anxiety, and aggressive or antisocial behavior (see e.g. Hussong et al., 2011). Also consistent with the existing literature, the effects appear to be greater at early ages, suggesting a critical period for psychosocial development during the first few years of life. However, the postnatal year does not appear to be of special importance. Rather, our results suggest that, if anything, the sensitivity to childhood adversity is lower during the postnatal year than during the following years. Hence, at least with regards to outcomes related to psychosocial health, our results lend support to Kotulak (1996), who emphasizes the importance of the entire first three-year period for the development of the information-processing system within the brain.

Our main results are similar to those of similar regressions excluding all but our baseline control variables, which are reported in Table A.3 in the Appendix. In some cases, however, the results from the reduced model display greater statistical significance than the estimates from our main specification. The higher significance level is likely due to the exclusion of the variables representing birth order and being the youngest sibling, and reflects the lower health and school achievement that has been documented among younger siblings.[14] A model including these

[14]See (Black et al., 2016) for evidence of the consequences of birth order for health and (Black et al., 2005) for evidence on the effects of birth order on school achievement.

Table 3: Estimates of the probability of hospital admission at ages 15-20 and 9th year GPA related to exposure to maternal psychiatric hospitalization at specific ages.

	(1) Psychiatric	(2) Accidents	(3) Self harm	(4) Abuse	(5) Substance-related	(6) Somatic	(7) GPA percentile
Postnatal year	0.00531	-0.0104	0.00922	0.00423	0.00972	0.000639	0.729
	(0.00968)	(0.00878)	(0.00611)	(0.00298)	(0.00846)	(0.0123)	(0.912)
Postnatal year to age 4	0.00812	-0.00472	0.00990**	-0.00279	0.0134*	-0.00411	-0.939
	(0.00746)	(0.00763)	(0.00451)	(0.00241)	(0.00693)	(0.00996)	(0.699)
Age 5 to 9	0.000284	-0.000386	0.00704	-0.000942	0.00633	-0.00332	-0.215
	(0.00699)	(0.00722)	(0.00429)	(0.00230)	(0.00647)	(0.00952)	(0.641)
Age 10 to 14	-0.00472	0.000923	0.00228	-0.00150	0.00755	-0.00559	-0.504
	(0.00612)	(0.00656)	(0.00397)	(0.00172)	(0.00555)	(0.00828)	(0.544)
Postnatal year to age 14	0.00146	-0.00728	0.00797*	-4.02e-05	0.00974*	-0.00394	-0.358
	(0.00637)	(0.00697)	(0.00412)	(0.00177)	(0.00582)	(0.00879)	(0.575)
Maternal and child background	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Family-fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Means	0.0234	0.0453	0.0062	0.0028	0.0197	0.0846	51.006
Observations	806,326	806,326	806,326	806,326	806,326	806,326	989,546

Notes: *, **, and *** denote significance at the 10, 5, and 1 percent levels. All specifications control for maternal psychiatric hospitalization during the prenatal year, maternal age at childbirth and apgar score 5 minutes after birth and include fixed effects for year of birth, sex, low birth-weight, premature birth, parity, age difference to the sibling closest in age, maternal income percentile the year before conception (based on disposable family income), and divorce, death of a close family member, large income loss and change of municipality of residence during the five-year period before childbirth. Robust standard errors in parentheses.

two variables in addition to our baseline control variables confirms this hypothesis (results are available on request).

6 Heterogeneous effects across subgroups and treatment spells

In order to design policies effectively alleviating the adverse consequences of maternal deprivation and maltreatment, a thorough understanding of between-group differences in responsiveness is crucial. In this section, we address potential heterogeneity related to sex and socioeconomic status.

6.1 Heterogeneous effects by sex

The response to mothers' psychiatric hospitalization may differ between the sexes due to both biological and social factors. The existing literature provides somewhat conflicting results regarding sex differences in stress sensitivity among younger children. While some studies find no such differences, others suggest a greater stress sensitivity among boys at very early ages.[15] Conversely, numerous studies find a greater response to acute stressors among girls from adolescence and into adulthood. The literature also suggests that, at least from early adolescence, females tend to be more sensitive to stress related to social interaction, while males are more responsive to stress related to achievement (see Panagiotakopoulos and Neigh (2014), Kudielka and Kirschbaum (2005), and Ordaz and Luna (2012) for overviews). In line with these findings, several studies point at girls showing greater sensitivity to child abuse than do boys (see e.g. Cooke and Weathington, 2014).[16]

In Table 4, we address gender-related heterogeneity in responsiveness to maternal psychiatric hospitalization by adding interaction terms between female sex and each of our explanatory variables. The variable $Female{*}PsMother_{ijt}$ represents the additional effect of exposure to maternal psychiatric hospitalization during the selected period for girls.

While no pattern is evident for hospitalizations related to psychiatric conditions or accidents at age 15-20 (see columns 1 and 2 of Table 4), column 3 reveals a greater sensitivity among girls with regards to self harm-related hospital admissions. Whereas the response to exposure during the birth to age 4 period appears relatively uniform across the sexes, the effects are substantially greater for girls during all other periods. For example, self harm-related hospitalizations are 812% higher among girls exposed during the postnatal year than among their siblings who were not exposed during this period, while the estimates related to exposure during ages 5 to 9 and 10 to 14 are 435% and 596%, respectively. The corresponding estimates for boys are small and statistically insignificant.

Similarly, as shown in column 5, our result document a greater sensitivity to maternal psychiatric hospitalization during mid-childhood for girls' substance-related hospitalizations. Further, column 6 displays a borderline significant 44% increase in the risk of somatic hospitalization at age 15-20 among girls exposed to maternal psychiatric hospitalization at age 10 to 14, while no such effect is evident for boys.

[15] For example, Davis and Emory (1995) document greater cortisol levels among male newborns undergoing a developmental assessment examination.

[16] This association has been documented both for stress hormone production among 7-11-year olds (Doom et al., 2013), and the probability of developing mood disorders or substance abuse in adulthood (McClellan et al., 1997).

Table 4: Estimates of the probability of hospital admission at ages 15-20 and 9th year GPA related to exposure to maternal psychiatric hospitalization at specific ages. Heterogeneous effects by sex.

	(1) Psychiatric	(2) Accidents	(3) Self harm	(4) Abuse	(5) Substance-related	(6) Somatic	(7) GPA percentile
Postnatal year	0.0104	-0.00906	-0.00196	0.00255	0.00387	-0.00764	1.663
	(0.0123)	(0.0121)	(0.00672)	(0.00396)	(0.0108)	(0.0160)	(1.224)
Female*Postnatal year	-0.0102	-0.00266	0.0224*	0.00337	0.0117	0.0165	-1.882
	(0.0193)	(0.0167)	(0.0117)	(0.00601)	(0.0162)	(0.0234)	(1.682)
Postnatal year to age 4	0.00305	-0.00265	0.0110**	-0.00337	0.0114	0.00303	-0.0780
	(0.00846)	(0.00906)	(0.00496)	(0.00311)	(0.00814)	(0.0114)	(0.805)
Female*Postnatal year to age 4	0.0105	-0.00405	-0.00224	0.00126	0.00427	-0.0147	-1.910**
	(0.00994)	(0.00929)	(0.00538)	(0.00323)	(0.00868)	(0.0129)	(0.885)
Age 5 to 9	-0.00160	0.000590	0.000443	-0.000390	-0.00212	-0.00108	0.554
	(0.00807)	(0.00859)	(0.00493)	(0.00283)	(0.00720)	(0.0108)	(0.733)
Female*Age 5 to 9	0.00381	-0.00208	0.0135***	-0.00110	0.0172**	-0.00450	-1.622**
	(0.00890)	(0.00868)	(0.00518)	(0.00298)	(0.00815)	(0.0119)	(0.811)
Age 10 to 14	-0.00922	0.00488	-0.00439	-0.00126	0.00354	-0.0146	0.159
	(0.00716)	(0.00827)	(0.00406)	(0.00233)	(0.00638)	(0.00978)	(0.644)
Female*Age 10 to 14	0.00906	-0.00801	0.0135***	-0.000535	0.00822	0.0181*	-1.356*
	(0.00808)	(0.00844)	(0.00479)	(0.00246)	(0.00765)	(0.0109)	(0.717)
Postnatal year to age 14	8.67e-05	-0.00623	0.00500	0.000338	0.00658	-0.00683	0.175
	(0.00661)	(0.00746)	(0.00401)	(0.00198)	(0.00600)	(0.00919)	(0.613)
Female*Postnatal year to age 14	0.00428	-0.00345	0.00931**	-0.00120	0.00986	0.00962	-1.622***
	(0.00679)	(0.00672)	(0.00413)	(0.00183)	(0.00647)	(0.00906)	(0.613)
Maternal and child background	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Family-fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Means	0.0189	0.0562	0.0030	0.0047	0.0175	0.0737	44.952
Observations	806,326	806,326	806,326	806,326	806,326	806,326	989,546

Notes: *, **, and *** denote significance at the 10, 5, and 1 percent levels. All specifications control for maternal psychiatric hospitalization during the prenatal year, maternal age at childbirth and apgar score 5 minutes after birth and include fixed effects for year of birth, sex, low birth-weight, premature birth, parity, age difference to the sibling closest in age, maternal income percentile the year before conception (based on disposable family income), and divorce, death of a close family member, large income loss and change of municipality of residence during the five-year period before childbirth, and interaction terms between the female sex indicator and all remaining control variables. Robust standard errors in parentheses.

Column 7 of Table 4 displays the results regarding gender differences in human capital responses to mothers' psychiatric hospitalization. The point estimates of the $Female * PsMother_{ijt}$ variables are consistently negative and statistically significant for all periods except the postnatal year, suggesting decreases of between 1.1 and 2.0 percentage points in GPA rank among exposed girls. Conversely, no effects are evident for boys. Hence, consistent with the existing literature, we document greater adverse effects of exposure to maternal psychiatric hospitalization on adolescent health and human capital for girls than for boys, at least beyond the first few years of childhood.

6.2 Responsiveness among disadvantaged populations

Vulnerability to maternal psychiatric hospitalization may vary between children of different socioeconomic backgrounds. A greater likelihood of exposure to, for example, substance abuse and violence may result in a riskier environment for socioeconomically disadvantaged children. On the other hand, an extensive literature documents a greater efficiency of investments in child ability among more resourceful parents (see e.g. Carneiro and Heckman, 2003, Heckman and Masterov, 2007, Cunha et al., 2006, and Cunha et al., 2010). A similar association may exist for health capital, arising both through health-related parental investment and through ability formation. If this is the case, children of privileged backgrounds may be more responsive to maternal psychiatric hospitalization than their disadvantaged peers.

In order to assess these potential differences, we estimate two separate models including interaction terms between all explanatory variables and indicator variables representing children of mothers with no postsecondary schooling and children belonging to the bottom income quartile, respectively. Table 5 reports the results from this exercise. The upper panel addresses heterogeneity related to maternal education, while the lower panel displays the results across income brackets.

Some between-group differences in responsiveness are evident. In comparison to children whose mothers hold a postsecondary degree, children of low-educated mothers display greater resilience to maternal psychiatric hospitalization with regards to hospital admissions due to psychiatric conditions (see column 1), accidents (column 2), self-inflicted injuries (column 3), and substance-related diagnoses (column 5). The only diagnosis category for which children of low-educated mothers appear more responsive than their more privileged peers is abuse, which increases substantially for children of low-educated mothers who were hospitalized during the postnatal year (see column 4). No consistent patterns are evident for hospitalizations due to somatic diagnoses or GPA rank (see columns 6 and 7, respectively).

The difference in responsiveness between children of low-income backgrounds and their higher-income peers, displayed in the bottom panel of Table 5, is less clear. Statistically significant differences occur only in two cases.[17] Column 2 displays

[17] Instead using the median as the cutoff point yields similar results. Hence, the lack of statistically significant between-group differences is unlikely to be due to a small sample size for the low-income group. Results are available on request.

Table 5: Estimates of the probability of hospital admission at ages 15-20 and 9th year GPA related to exposure to maternal psychiatric hospitalization at specific ages. Heterogeneous effects by income and education level.

	(1) Psychiatric	(2) Accidents	(3) Self harm	(4) Abuse	(5) Substance-related	(6) Somatic	(7) GPA percentile
LOW MATERNAL EDUCATION							
Postnatal year	0.00842	-0.00679	0.00607	-0.00312	0.00516	-0.0202	0.419
	(0.0171)	(0.0137)	(0.0101)	(0.00342)	(0.0146)	(0.0231)	(1.597)
Low education*Postnatal year	-0.00444	-0.00517	0.00449	0.0104**	0.00647	0.0295	0.449
	(0.0207)	(0.0176)	(0.0126)	(0.00521)	(0.0179)	(0.0273)	(1.944)
Postnatal year to age 4	-0.00393	0.00775	0.000627	-0.000141	-0.00133	-0.0227	-1.197
	(0.0121)	(0.0143)	(0.00790)	(0.00431)	(0.0114)	(0.0186)	(1.267)
Low education*Postnatal year to age 4	0.0165	-0.0170	0.0129	-0.00372	0.0205	0.0256	0.373
	(0.0152)	(0.0169)	(0.00965)	(0.00520)	(0.0143)	(0.0219)	(1.512)
Age 5 to 9	-0.00155	0.0220*	0.0118	-0.000308	0.0137	-0.0248	0.274
	(0.0114)	(0.0128)	(0.00733)	(0.00364)	(0.0103)	(0.0171)	(1.103)
Low education*Age 5 to 9	0.00196	-0.0317**	-0.00721	-0.000874	-0.0113	0.0301	-0.726
	(0.0143)	(0.0155)	(0.00899)	(0.00462)	(0.0130)	(0.0205)	(1.348)
Age 10 to 14	0.00861	0.00524	0.00704	-0.000329	0.0253***	-0.0115	-0.488
	(0.00949)	(0.0109)	(0.00602)	(0.00194)	(0.00935)	(0.0142)	(0.965)
Low education*Age 10 to 14	-0.0193	-0.00618	-0.00699	-0.00165	-0.0258**	0.00829	-0.0311
	(0.0122)	(0.0136)	(0.00786)	(0.00302)	(0.0116)	(0.0174)	(1.167)
Postnatal year to age 14	0.00835	-0.0107	0.0114***	0.000703	0.0137**	-0.00212	-0.277
	(0.00702)	(0.00770)	(0.00435)	(0.00199)	(0.00661)	(0.00964)	(0.659)
Low education*Postnatal year to age 14	-0.0155**	0.00779	-0.00765*	-0.00166	-0.00889	-0.00417	-0.189
	(0.00775)	(0.00818)	(0.00450)	(0.00246)	(0.00701)	(0.0101)	(0.684)
Means	0.0252	0.0479	0.0069	0.0035	0.0219	0.0883	44.048
BOTTOM INCOME QUARTILE							
Postnatal year	0.00360	-0.0110	0.0113	0.00450	0.0138	0.00302	0.648
	(0.0113)	(0.0108)	(0.00748)	(0.00379)	(0.0104)	(0.0142)	(1.046)
Low income*Postnatal year	0.00613	0.00148	-0.00726	-0.000956	-0.0146	-0.00794	0.356
	(0.0213)	(0.0189)	(0.0109)	(0.00478)	(0.0173)	(0.0259)	(2.021)
Postnatal year to age 4	0.0114	-0.00788	0.0116**	-0.00410	0.0196**	-0.00731	-0.796
	(0.00878)	(0.00854)	(0.00523)	(0.00291)	(0.00819)	(0.0111)	(0.777)
Low income*Postnatal year to age 4	-0.00971	0.0118	-0.00500	0.00400	-0.0195*	0.00879	-0.477
	(0.0122)	(0.0112)	(0.00705)	(0.00366)	(0.0104)	(0.0155)	(1.117)
Age 5 to 9	0.000802	-0.00977	0.00708	-0.000210	0.00660	0.00218	-0.581
	(0.00797)	(0.00782)	(0.00487)	(0.00261)	(0.00745)	(0.0105)	(0.696)
Low income*Age 5 to 9	-0.000446	0.0280***	0.000588	-0.00259	0.00124	-0.0179	1.242
	(0.0101)	(0.0106)	(0.00538)	(0.00354)	(0.00903)	(0.0140)	(0.959)
Age 10 to 14	-0.00486	0.00348	0.00192	-0.00217	0.00950	-0.00745	-0.555
	(0.00690)	(0.00725)	(0.00454)	(0.00209)	(0.00642)	(0.00921)	(0.595)
Low income*Age 10 to 14	0.00112	-0.0112	0.00148	0.00223	-0.00521	0.00725	0.0861
	(0.00968)	(0.0105)	(0.00548)	(0.00258)	(0.00832)	(0.0128)	(0.894)
Postnatal year to age 14	0.00250	-0.00716	0.00856**	-0.000264	0.0111*	-0.00436	-0.378
	(0.00652)	(0.00722)	(0.00422)	(0.00189)	(0.00603)	(0.00902)	(0.596)
Low income*Postnatal year to age 14	-0.00499	-0.000216	-0.00279	0.00125	-0.00649	0.00249	0.0922
	(0.00827)	(0.00950)	(0.00448)	(0.00211)	(0.00700)	(0.0113)	(0.824)
Means	0.0257	0.0465	0.0070	0.0031	0.0233	0.0839	49.516
Maternal and child background	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Family-fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	806,326	806,326	806,326	806,326	806,326	806,326	989,546

Notes: *, **, and *** denote significance at the 10, 5, and 1 percent levels. All specifications control for maternal psychiatric hospitalization during the prenatal year, maternal age at childbirth and apgar score 5 minutes after birth and include fixed effects for year of birth, sex, low birth-weight, premature birth, parity, age difference to the sibling closest in age, maternal income percentile the year before conception (based on disposable family income), and divorce, death of a close family member,large income loss and change of municipality of residence during the five-year period before childbirth and interaction terms between the indicators for low maternal education and low maternal income, respectively, and all remaining control variables. Robust standard errors in parentheses.

an 81% increase in accident-induced hospital admissions for low-income children exposed during the age 5 to 9 period, but none for their higher-income peers. Further, we find an 84% increase in substance-related hospitalizations among higher-income individuals exposed prior age to 5, but none for children belonging to the lowest income quartile (see column 5).

While the between-group differences in effects on substance-related hospital admissions are similar along both dimensions of socioeconomic inequality, the differences regarding accident-induced hospitalizations are somewhat puzzling. However, the expected sign of the effect is not clear. Jokela et al. (2009) show that externalizing behaviors, such as aggression and antisocial behavior, lead to an increased injury risk, while internalizing behaviors, for example, withdrawal, passivity, and anxiety, are instead related to a decreased injury risk. Hence, the between-group differences may reflect differing response patterns to stressful events during mid-childhood, externalizing behaviors dominating among children of low-income backgrounds, while children of low-educated mothers are more prone to internalizing behaviors.

Taken together, our results indicate a greater overall resilience to mothers' psychiatric hospitalization with regards to health outcomes among socioeconomically disadvantaged children. Hence, it appears as though differences in loss of parental investment due to maternal psychiatric hospitalization, which is likely to be greater for more privileged children, outweighs the potential additional risks faced by socioeconomically disadvantaged children. The differences are more apparent across maternal education levels than across income brackets, consistent with a literature documenting stronger effects of education and ability than of income on the efficiency of parental investment in children (see, for example, Heckman and Mosso (2014) and Cunha et al. (2006)).

6.3 Length of hospitalization spells

The consequences of maternal psychiatric hospitalization may be affected by treatment intensity. Longer hospitalization spells imply longer periods of separation. They could also indicate more severe illness episodes, which may in turn be associated with more severe maltreatment. In order to investigate the role of treatment intensity, we reestimate Model 1 using two separate treatment variables representing hospitalization spells shorter and longer than the median. Table 6 reports

Table 6: Estimates of the probability of hospital admission at ages 15-20 and 9th year GPA related to exposure to maternal psychiatric hospital admission at specific ages. Heterogeneous responses related to treatment intensity.

	(1) Psychiatric	(2) Accidents	(3) Self harm	(4) Abuse	(5) Substance-related	(6) Somatic	(7) GPA rank
Postnatal year. Low.	0.0168	0.00464	0.0165	0.00461	0.0167	0.000556	0.663
	(0.0133)	(0.0116)	(0.0103)	(0.00414)	(0.0120)	(0.0168)	(1.257)
Postnatal year. High.	-0.00587	-0.0274**	0.00226	0.00393	0.00314	0.00542	0.355
	(0.0136)	(0.0125)	(0.00616)	(0.00377)	(0.0121)	(0.0167)	(1.290)
Birth to age 4. Low	0.00865	0.00835	0.00949	-0.00195	0.00750	-0.00470	-0.719
	(0.00988)	(0.0101)	(0.00642)	(0.00255)	(0.00912)	(0.0125)	(0.879)
Birth to age 4. High.	0.00614	-0.0233**	0.0107*	-0.00443	0.0210**	-0.00659	-1.355
	(0.0106)	(0.0101)	(0.00582)	(0.00401)	(0.0100)	(0.0141)	(1.087)
Age 5 to 9. Low.	0.00688	0.00568	0.00726	-0.00195	0.00412	0.00812	-0.166
	(0.00872)	(0.00909)	(0.00557)	(0.00291)	(0.00799)	(0.0124)	(0.786)
Age 5 to 9. High.	-0.0106	-0.00959	0.00692	-0.000224	0.00893	-0.0199	-0.213
	(0.0105)	(0.0101)	(0.00624)	(0.00320)	(0.00974)	(0.0133)	(0.946)
Age 10 to 14. Low.	-0.00350	0.0138	0.00408	-0.000356	0.00719	0.00268	-0.606
	(0.00794)	(0.00840)	(0.00507)	(0.00241)	(0.00732)	(0.0108)	(0.669)
Age 10 to 14. High.	-0.00840	-0.0157*	0.000285	-0.00406	0.00838	-0.0120	-0.674
	(0.00852)	(0.00873)	(0.00569)	(0.00250)	(0.00801)	(0.0112)	(0.802)
Birth to age 14. Low.	0.00224	0.00151	0.0111**	-0.000959	0.0123	0.00268	-0.232
	(0.00812)	(0.00843)	(0.00538)	(0.00239)	(0.00770)	(0.0108)	(0.672)
Birth to age 14. High.	-0.00379	-0.0234**	0.00388	0.000112	0.00608	-0.0101	-1.149
	(0.00930)	(0.00989)	(0.00541)	(0.00267)	(0.00841)	(0.0126)	(0.902)
Maternal and child background	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Family-fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Means	0.0234	0.0453	0.0062	0.0028	0.0197	0.0846	51.006
Observations	806,326	806,326	806,326	806,326	806,326	806,326	989,546

Notes: *, **, and *** denote significance at the 10, 5, and 1 percent levels. All specifications control for maternal psychiatric hospitalization during the prenatal year, maternal age at childbirth and apgar score 5 minutes after birth and include fixed effects for year of birth, sex, low birth-weight, premature birth, parity, age difference to the sibling closest in age, maternal income percentile the year before conception (based on disposable family income), and divorce, death of a close family member, large income loss and change of municipality of residence during the five-year period before childbirth. Robust standard errors in parentheses.

the results from this exercise.

Our results suggest that treatment intensity plays a role in determining child outcomes. Column 2 documents reductions in accident-induced hospitalization rates related to high-intensity treatment during all periods but age 5 to 9, possibly reflecting an increase in internalizing behaviors (see Jokela et al. (2009)). The effects are substantial, ranging between 35 and 60%, and greater during early periods.

The pattern regarding hospitalizations due to self-inflicted injuries, displayed in column 3, is less clear. The relative magnitudes of the high- and low treatment estimates differ across periods, displaying no consistent pattern. Hence, we find no evidence of differential responses depending on treatment intensity for this diagnosis category.

Our results regarding substance-related hospitalizations suggest greater consequences related to longer hospitalization spells (see column 4). Individuals exposed to high intensity treatment during the birth to age 4 period are more than twice as likely as their unexposed peers to be hospitalized for substance-related diagnoses at age 15-20, while no such effect of low intensity treatment is evident. Although statistically insignificant, the point estimates are also substantially greater for high intensity treatment during the age 5 to 9 and 10 to 14 periods.

All in all, our results provide evidence of greater consequences for children exposed to longer maternal hospitalization spells with regards to certain diagnosis categories. While accident rates and substance-related hospitalizations appear to be heavily related to treatment intensity, the extensive rather than intensive margin seems to play the major role for self harm behavior.

7 Robustness checks

7.1 External validity

Since our identification strategy relies on within-family variation in exposure to maternal psychiatric hospitalization, our sample only includes individuals with at least one sibling represented in the sample. Hence, external validity will be limited if the consequences of exposure among children who have siblings differ from the effects among those who do not. Such a situation could occur due to, for example, between-group differences in type or severity of maternal mental illness, or if the presence of siblings mitigates the effects of maternal deprivation and maltreatment.

In order to evaluate the external validity of our results, we estimate the associations between exposure to mothers' psychiatric hospitalization and child outcomes separately for children with and without siblings. To this end, we use an OLS model with the same set of control variables used in our main specification and an additional set of socioeconomic and demographic characteristics, including family size, parental education levels, and indicator variables representing foreign-born parents. Table 7 reports the results. The results are similar, apart from a lower statistical significance level for children without siblings, most likely due to a smaller sample size. Hence, our findings indicate a high level of external validity in our main results.

Table 7: Estimates of the probability of hospital admission at ages 15-20 and 9th year GPA related to exposure to maternal psychiatric hospital admission at specific ages. Heterogeneous effects between children with and without siblings.

	(1) Psychiatric	(2) Accidents	(3) Self harm	(4) Abuse	(5) Substance-related	(6) Somatic	(7) GPA rank
SIBLINGS							
Postnatal year	0.0277***	-0.00244	0.0132***	0.00207	0.0145**	0.00226	-3.474***
	(0.00683)	(0.00600)	(0.00417)	(0.00214)	(0.00570)	(0.00832)	(0.758)
Birth to age 4	0.0178***	0.000160	0.00692***	-0.000294	0.0124***	0.00241	-3.486***
	(0.00364)	(0.00347)	(0.00212)	(0.00108)	(0.00326)	(0.00473)	(0.417)
Age 5 to 9	0.0214***	0.00259	0.00769***	0.00350***	0.0141***	0.00895**	-3.840***
	(0.00337)	(0.00321)	(0.00195)	(0.00120)	(0.00298)	(0.00441)	(0.369)
Age 10 to 14	0.0158***	0.00796***	0.00665***	0.000770	0.0156***	0.00631	-4.244***
	(0.00294)	(0.00305)	(0.00175)	(0.000893)	(0.00272)	(0.00397)	(0.338)
Birth to age 14	0.0269***	0.00440**	0.0102***	0.00190***	0.0201***	0.0102***	-5.537***
	(0.00202)	(0.00201)	(0.00118)	(0.000649)	(0.00183)	(0.00269)	(0.229)
Means	0.0234	0.0453	0.0062	0.0028	0.0197	0.0846	51.006
Observations	796,151	796,151	796,151	796,151	796,151	796,151	989,546
NO SIBLINGS							
Postnatal year	0.00505	0.0102	0.00689	-0.00317*	0.00258	-0.00692	-2.485**
	(0.00843)	(0.00991)	(0.00554)	(0.00185)	(0.00728)	(0.0119)	(1.022)
Birth to age 4	0.0103*	0.00694	0.00499	-0.000280	0.00676	0.0112	-2.477***
	(0.00544)	(0.00551)	(0.00307)	(0.00171)	(0.00471)	(0.00727)	(0.561)
Age 5 to 9	0.0219***	0.00321	0.00819***	0.00152	0.00996**	0.00632	-3.494***
	(0.00558)	(0.00510)	(0.00315)	(0.00176)	(0.00470)	(0.00679)	(0.527)
Age 10 to 14	0.0324***	0.0105**	0.00726**	0.00214	0.0218***	0.0175***	-4.575***
	(0.00540)	(0.00498)	(0.00284)	(0.00168)	(0.00468)	(0.00652)	(0.502)
Birth to age 14	0.0348***	0.00842***	0.0100***	0.00173	0.0217***	0.0167***	-5.498***
	(0.00341)	(0.00321)	(0.00187)	(0.00107)	(0.00294)	(0.00424)	(0.328)
	(0.00550)	(0.00662)	(0.00282)	(0.00178)	(0.00494)	(0.00938)	(0.812)
Means	0.0273	0.0471	0.0072	0.0035	0.0201	0.0921	46.713
Observations	250,117	250,117	250,117	250,117	250,117	250,117	295,418
Maternal and child background	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Demographic/socioeconomic background	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Notes: *, **, and *** denote significance at the 10, 5, and 1 percent levels. All specifications control for family size, maternal psychiatric hospitalization during the prenatal year, maternal age at childbirth and apgar score 5 minutes after birth and include fixed effects for year of birth, sex, low birth-weight, premature birth, parity, age difference to the sibling closest in age, maternal income percentile the year before conception (based on disposable family income), maternal and paternal educational level, maternal and paternal immigrant background, and divorce, death of a close family member, large income loss and change of municipality of residence during the five-year period before childbirth. Robust standard errors in parentheses.

7.2 Non-treated siblings as control group

In our main analysis, the control groups for all age periods except the birth to age 14 period include both non-exposed siblings and siblings who were exposed to maternal psychiatric hospitalization during other age periods. In this section,

Table 8: Estimates of the probability of hospital admission at ages 15-20 and 9th year GPA related to exposure to maternal psychiatric hospital admission at specific ages. Including families where only one sibling was exposed before age 15.

	(1) Psychiatric	(2) Accidents	(3) Self harm	(4) Abuse	(5) Substance-related	(6) Somatic	(7) GPA rank
Postnatal year	0.0118	0.0102	0.00495	0.000335	0.0151	0.00553	-0.842
	(0.0217)	(0.0212)	(0.0156)	(0.00693)	(0.0178)	(0.0273)	(1.960)
Birth to age 4	0.00250	0.0118	0.00556	0.00275	0.00832	0.0158	0.367
	(0.0149)	(0.0150)	(0.00935)	(0.00532)	(0.0135)	(0.0188)	(1.352)
Age 5 to 9	-0.0361	-0.0447	-0.0335**	0.00105	-0.00341	-0.0575	-0.284
	(0.0330)	(0.0272)	(0.0160)	(0.0131)	(0.0275)	(0.0405)	(2.198)
Age 10 to 14	0.00127	-0.00455	0.0158***	-0.00311	0.0118	-0.00676	-0.587
	(0.00901)	(0.00987)	(0.00607)	(0.00247)	(0.00809)	(0.0124)	(0.780)
Birth to age 14	0.000419	-0.00281	0.0106**	-0.000797	0.0110	-0.00349	-0.295
	(0.00753)	(0.00799)	(0.00494)	(0.00230)	(0.00676)	(0.0101)	(0.653)
Maternal and child background	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Family-fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Means	0.0230	0.0452	0.0062	0.0028	0.0197	0.0845	51.093
Observations	794,785	794,785	794,785	794,785	794,785	794,785	976,454

Notes: *, **, and *** denote significance at the 10, 5, and 1 percent levels. All specifications control for maternal psychiatric hospitalization during the prenatal year, maternal age at childbirth and apgar score 5 minutes after birth and include fixed effects for year of birth, sex, low birth-weight, premature birth, parity, age difference to the sibling closest in age, maternal income percentile the year before conception (based on disposable family income), and divorce, death of a close family member,large income loss and change of municipality of residence during the five-year period before childbirth. Robust standard errors in parentheses.

we estimate Model 1 on families where at most one sibling experienced maternal psychiatric hospitalization before age fifteen, in order to ensure a control group consisting solely of non-exposed individuals. This implies that only siblings very far apart in age, oldest siblings exposed early, or youngest sibling exposed late during the birth to age 14 period are included in the treatment group.[18]

Table 8 reports the results from the restricted sample. Similar to our main results, self harm is the most heavily affected diagnosis category (see column 3). The estimates for the age 10 to 14 and birth to age 14 periods suggest great increases in self harm-related hospitalizations related to exposure. Surprisingly, the results from the restricted sample display reduced rates among individuals exposed in mid-childhood. It is worth noting, however, that the sample is especially small and selective for this period, as only siblings who are very far apart in age are included.

While our main estimates generally indicate greater effects of exposure at younger ages, the results from the restricted sample indicate the opposite. The results of a model investigating potential heterogeneity in responsiveness for youngest si-

[18] The restricted sample consists of 976,454 observations. 2941 individuals, corresponding to 20% of the treated individuals in the full sample, belong to the treatment group.

blings, displayed in Table A.5 in the Appendix, indicate a greater sensitivity for this group to early-childhood exposure. As youngest siblings exposed during early periods are (in general) excluded from the restricted sample, the differences in results are likely due to sampling differences.

8 Mechanisms

8.1 Channels

Mothers' psychiatric hospitalization may be associated with stressful life events, such as marital discord, financial problems, somatic disease, or moving to a new town and having to build a new life. Further, mothers suffering from mental illness may be more likely than others to turn to substance use or child abuse. These events may in turn directly affect child wellbeing and thus act as channels of the effect of maternal psychiatric hospitalization.

In order to investigate which channels may be at play, we estimate the associations between maternal psychiatric hospitalization and indicator variables representing maternal substance-related hospitalization, divorce, large income loss, death of a close family member, inter-municipal move, child hospitalization due to abuse, and maternal non-psychiatric hospitalization, between the beginning of the time period of exposure and the child's fifteenth birthday. We find strong correlations between maternal psychiatric hospitalization and all events apart from child hospitalization due to abuse. Table A.4 in the Appendix displays the results.

We then reestimate Model 1 including indicator variables representing the occurrence of these events between birth and age 14. The results, reported in Table 9, are very similar to our main results. This suggests that the effects of maternal psychiatric hospitalization are not mainly due to its association with stressful life events, but that it predominantly affects child outcomes in its own right.

8.2 Separation, shock or maltreatment?

Direct effects of maternal psychiatric hospitalization on child outcomes could arise through three mechanisms. First, separation may decrease parental investment in

Table 9: Estimates of the probability of hospital admission at ages 15-20 and 9th year GPA related to exposure to maternal psychiatric hospital admission at specific ages. Controlling for adverse life events.

	(1) Psychiatric	(2) Accidents	(3) Self harm	(4) Abuse	(5) Substance-related	(6) Somatic	(7) GPA rank
Postnatal year	0.00876	-0.0142	0.00754	0.00331	0.00588	0.00198	0.770
	(0.00982)	(0.00905)	(0.00621)	(0.00292)	(0.00844)	(0.0126)	(0.931)
Postnatal year to age 4	0.00745	-0.00637	0.00951**	-0.00340	0.0127*	-0.00211	-0.829
	(0.00762)	(0.00775)	(0.00461)	(0.00248)	(0.00707)	(0.0101)	(0.707)
Age 5 to 9	0.00130	-0.00146	0.00710	-0.00163	0.00557	0.000300	-0.107
	(0.00718)	(0.00736)	(0.00438)	(0.00236)	(0.00662)	(0.00969)	(0.652)
Age 10 to 14	-0.00554	0.00216	0.00187	-0.00256	0.00661	-0.00481	-0.284
	(0.00622)	(0.00667)	(0.00402)	(0.00171)	(0.00562)	(0.00837)	(0.551)
Birth to age 14	0.000503	-0.00699	0.00762*	-0.00114	0.00825	-0.00406	-0.189
	(0.00647)	(0.00707)	(0.00412)	(0.00176)	(0.00593)	(0.00895)	(0.585)
Maternal and child background	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Family-fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Means	0.0233	0.0454	0.0063	0.0029	0.0200	0.0847	51.036
Observations	799,348	799,348	799,348	799,348	799,348	799,348	983,796

Notes: *, **, and *** denote significance at the 10, 5, and 1 percent levels. All specifications control for maternal psychiatric hospitalization during the prenatal year, maternal age at childbirth and apgar score 5 minutes after birth and include fixed effects for year of birth, sex, low birth-weight, premature birth, parity, age difference to the sibling closest in age, maternal income percentile the year before conception (based on disposable family income), divorce, death of a close family member, large income loss and change of municipality of residence during the five-year period before childbirth, and substance-related maternal hospitalization, non-psychiatric maternal hospitalization, divorce, death of a close family member, large income loss and change of municipality of residence between childbirth and the child's 15th birthday. Robust standard errors in parentheses.

children.[19] Second, maternal mental disorders may adversely affect parenting behavior. For example, maternal depression has been linked to an increased likelihood of hostility, insensitivity and emotional unavailability towards the child, maladaptive parenting methods, with both non-responsive, submissive and coercive elements, and child abuse. These types of maltreatment could, in turn, impair mother-child attachment (see, for example, Cummings and Davies (1994) and the references therein). Finally, ill maternal health could induce shock and worries, creating emotional stress in children.

In order to disentangle the effects of these mechanisms, we compare the effects of maternal psychiatric hospitalization to the consequences of maternal hospitalization due to accidents.[20] To avoid bias due to comorbidity, the sample used in the analysis of accidents excludes children whose mother was hospitalized due to psychiatric conditions prior to their fifteenth birthday. For similar reasons, the

[19] The positive correlation between psychiatric and non-psychiatric hospitalizations, which is documented in Table A.4 in the Appendix, enhances this risk.

[20] Similar to maternal psychiatric hospitalization, exposure to mothers' accident-induced hospitalization is relatively randomly distributed between siblings (estimates are available on request).

accident category excludes diagnoses related to substance use.

As psychiatric hospital spells typically last longer than hospitalizations due to accidents, separation spells may differ between categories. To account for this difference, we control for the total number of days spent at a hospital during each age period. Hence, our estimates can be interpreted as measures of exposure at the purely extensive margin. These estimates contain effects of to all aspects of exposure, such as shock, worries, and maltreatment. While other aspects are unlikely to be completely uniform across diagnosis types, it seems reasonable to assume that the greatest difference relates to maltreatment. If this is the case, the differences between the estimates yielded by the two models represent lower bounds of the effects of maltreatment.

Table 10 displays the results. The upper panel displays the results regarding psychiatric hospitalizations, while the lower panel reports the estimates for hospital admissions due to accidents. The results suggest adverse effects of both types of maternal hospitalizations on adolescent health. For hospitalizations due to self harm and substance-related diagnoses, most estimates regarding maternal psychiatric hospitalization are quantitatively greater than the corresponding estimates for mothers' accident-induced hospitalization (see columns 3 and 5, respectively). However, in some cases, the statistical significance level is higher in the analysis of maternal accident-induced hospitalizations, probably due to its greater incidence. Further, we find borderline significant increases in hospital admissions due to mental disorders for individuals exposed to maternal accident-induced hospitalization at age 10 to 14 and due to somatic disease for individuals exposed at any point prior to age 15.

Not all between-category differences are statistically significant, but for some outcomes and age periods, they are striking. For example, the great increase in hospital admissions due to self-inflicted injuries related to exposure to maternal psychiatric hospitalization during early periods can be put in contrast to the corresponding, small and statistically insignificant, estimates for children exposed to maternal accident-induced hospitalization (see column 3). These differences suggest that maltreatment accounts for over 90% of the effect of exposure to maternal psychiatric hospitalization in early childhood on self harm behavior during late adolescence.

The pattern of associations differ between treatment categories. While the effects of maternal psychiatric hospitalization is generally greater at younger ages, the

Table 10: Estimates of the probability of hospital admission at ages 15-20 and 9th year GPA related to exposure to maternal hospital admission due to psychiatric conditions and accidents at specific ages.

	(1) Psychiatric	(2) Accidents	(3) Self harm	(4) Abuse	(5) Substance-related	(6) Somatic	(7) GPA rank
PSYCHIATRIC							
Postnatal year	0.00866	-0.00887	0.0119*	0.00321	0.0120	-0.00342	0.737
	(0.00987)	(0.00900)	(0.00633)	(0.00303)	(0.00863)	(0.0127)	(0.926)
Postnatal year to age 4	0.00872	-0.00464	0.00960**	-0.00313	0.0134*	-0.00702	-0.769
	(0.00759)	(0.00774)	(0.00449)	(0.00241)	(0.00693)	(0.0102)	(0.715)
Age 5 to 9	7.11e-05	-0.000208	0.00678	-0.000681	0.00395	-0.00296	0.136
	(0.00715)	(0.00743)	(0.00425)	(0.00224)	(0.00649)	(0.00980)	(0.657)
Age 10-14	-0.00404	0.00421	0.00323	-0.00101	0.00928	9.56e-05	-0.238
	(0.00623)	(0.00678)	(0.00401)	(0.00173)	(0.00565)	(0.00854)	(0.558)
Postnatal year to age 14	0.00197	-0.00614	0.00811*	0.000151	0.00979*	-0.00276	-0.106
	(0.00643)	(0.00708)	(0.00416)	(0.00176)	(0.00584)	(0.00890)	(0.582)
Maternal and child background	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Family-fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Means	0.0234	0.0453	0.0062	0.0028	0.0197	0.0846	51.006
Observations	806,326	806,326	806,326	806,326	806,326	806,326	989,546
ACCIDENTS							
Postnatal year	0.00243	-0.000864	0.000451	-0.000451	-0.000952	0.00582	0.566
	(0.00481)	(0.00648)	(0.00259)	(0.00165)	(0.00469)	(0.00819)	(0.508)
Postnatal year to age 4	0.000667	-0.00153	0.000679	-0.000580	-0.00108	0.00506	-0.210
	(0.00320)	(0.00420)	(0.00176)	(0.00104)	(0.00294)	(0.00540)	(0.329)
Age 5 to 9	0.00132	0.00299	0.00155	0.000682	0.00579*	0.00465	0.136
	(0.00318)	(0.00421)	(0.00176)	(0.00115)	(0.00300)	(0.00535)	(0.333)
Age 10-14	0.00478*	-0.00136	0.00349**	-0.000117	0.00497**	0.00432	-0.0884
	(0.00271)	(0.00368)	(0.00153)	(0.000921)	(0.00253)	(0.00462)	(0.288)
Postnatal year to age 14	0.00294	0.000866	0.00224*	-0.000118	0.00363	0.00778*	-0.202
	(0.00244)	(0.00334)	(0.00133)	(0.000833)	(0.00227)	(0.00421)	(0.262)
Maternal and child background	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Family-fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Means	0.0228	0.0453	0.0061	0.0028	0.0196	0.0844	51.155
Observations	780,138	780,138	780,138	780,138	780,138	780,138	958,131

Notes: *, **, and *** denote significance at the 10, 5, and 1 percent levels. All specifications control for total days of hospitalization during the treatment period, maternal psychiatric hospitalization during the prenatal year, maternal age at childbirth and apgar score 5 minutes after birth and include fixed effects for year of birth, sex, low birth-weight, premature birth, parity, age difference to the sibling closest in age, maternal income percentile the year before conception (based on disposable family income), and divorce, death of a close family member, large income loss and change of municipality of residence during the five-year period before childbirth. Robust standard errors in parentheses.

opposite appears to be true for maternal accident-induced hospital admissions. For later age periods, the effects are also similar in magnitude across treatment categories. As separation is unlikely to be of greater importance at older ages, a possible interpretation of this finding is that maltreatment is the main mechanism behind the effects of exposure to maternal psychiatric hospitalization in early childhood.

Conversely, the adverse effects of shock and worries, which are likely to be more uniform across hospitalization types, may be of greater importance at older ages, when children have a greater ability to grasp the situation.

9 What about the fathers?

For the majority of children, not only mothers, but also fathers or other caregivers provide physical and emotional care. This may be especially true in the Swedish setting, where men and women traditionally share care-giving duties relatively equally. It is possible that paternal care mitigates the effects of maternal psychiatric hospitalization. Conversely, in case of assortative mating or spillover effects on spouses' mental health, our main results may overestimate the effects of maternal psychiatric hospitalization. Further, paternal psychiatric hospitalization may have independent effects on child outcomes. If mothers' and fathers' respective roles differ, these effects may differ from the consequences of exposure to maternal psychiatric hospitalization.

In order to investigate the role of fathers, we estimate Model 1 including both maternal and paternal psychiatric hospitalization during various age periods as our treatment variables.[21] Table 11 reports the results. Column 1 displays the results regarding hospital admissions due to mental conditions at age 15-20, reporting no statistically significant effects of exposure to psychiatric hospitalization of either parent. The same appears to be true for accident-induced hospitalizations (see column 2).

Column 3 displays dramatic increases in hospital admissions due to self-inflicted injuries related to maternal psychiatric hospitalization during several periods. The estimates are slightly greater in terms of both magnitude and statistical significance than the corresponding estimates from our main specification (see column 3 of Table 1). This suggests that paternal care partly compensates for maternal absence and mental illness, although to a minor degree. The estimates regarding exposure to paternal psychiatric hospitalization are much smaller and statistically insignificant.

As displayed in column 4 of Table 11, we find no evidence of childhood exposure

[21] Exposure to paternal psychiatric hospitalization displays a degree of randomness similar to that of maternal psychiatric hospitalization between siblings (estimates are available on request).

to either mothers' or fathers' psychiatric hospitalization affecting hospitalization rates due to abuse in late adolescence. Conversely, exposure to both maternal and paternal psychiatric hospitalization appears to increase the risk of substance-related hospital admissions, albeit during different parts of childhood (see column 5). Also in this case, the estimated effects of exposure to maternal psychiatric hospitalization gain slightly in both magnitude and precision when controls for paternal psychiatric hospitalization are added to the model. Hence, also in this case, fathers appear to compensate slightly for maternal psychiatric hospitalization. We also find an entire 164% increase for children exposed to paternal psychiatric hospitalization during the postnatal year, indicating adverse effects of very early exposure to paternal deprivation and mental illness on psychosocial health. Further, surprisingly, column 6 reveals a 14% reduction in somatic hospitalization rates for individuals exposed to fathers' psychiatric hospitalization prior to age 15.

Interestingly, while we find no evidence of maternal psychiatric hospitalization affecting school achievement for our full population, column 7 reports negative effects of exposure to paternal psychiatric hospitalization during several age periods. Individuals exposed during the postnatal year are 2.7 percentage points below their siblings in GPA rank, suggesting substantial effects on human capital accumulation related to early exposure. We also find modest reductions in ninth grade GPA rank among individuals exposed at age 10 to 14 and during the birth to age 14 period.

In sum, we find that both maternal and paternal deprivation and maltreatment adversely affect children's psychosocial wellbeing. Mothers' psychiatric hospitalization appears to have a greater overall impact on psychosocial health and exposure over a greater age span seems to affect outcomes, consistent with a role as the primary caregiver. On the other hand, the effects of fathers' psychiatric hospitalization seem to be broader in scope, also affecting human capital accumulation. This result is consistent with a number of studies using twin or adoption designs, which find that fathers provide a stronger influence on children's educational attainment than do mothers (see Holmlund et al. (2011) for an overview).[22] Further, the effects of exposure to paternal psychiatric hospitalization seem more confined to the postnatal year and, at least in the case of somatic hospitalizations, late child-

[22]However, Amin et al. (2015) find a greater maternal influence using Swedish twin data. Further, most studies relying on instrumental variable designs find that mothers influence children's educational attainment more than fathers. Holmlund et al. (2011) find that the discrepancy between different-design studies is due to bias inherent in their respective estimation methods.

Table 11: Estimates of the probability of hospital admission at ages 15-20 and 9th year GPA related to exposure to maternal and paternal psychiatric hospitalization at specific ages.

	(1) Psychiatric	(2) Accidents	(3) Self harm	(4) Abuse	(5) Substance-related	(6) Somatic	(7) GPA rank
Mother postnatal year	0.00358	-0.00741	0.00810	0.00344	0.00871	0.00209	0.540
	(0.00983)	(0.00888)	(0.00590)	(0.00298)	(0.00843)	(0.0124)	(0.930)
Father postnatal year	0.0226	-0.00100	0.0117	-0.00226	0.0327**	-0.00682	-2.690**
	(0.0145)	(0.0143)	(0.00982)	(0.00490)	(0.0134)	(0.0179)	(1.343)
Mother birth to age 4	0.00877	-0.00444	0.0107**	-0.00312	0.0154**	-0.000161	-0.926
	(0.00775)	(0.00784)	(0.00462)	(0.00246)	(0.00713)	(0.0103)	(0.711)
Father birth to age 5	-0.00423	0.00367	0.000382	0.00320	-0.00782	-0.0117	-0.423
	(0.00897)	(0.00918)	(0.00518)	(0.00308)	(0.00783)	(0.0124)	(0.829)
Mother age 5 to 9	0.000847	0.000970	0.00754*	-0.000375	0.00690	0.000224	-0.242
	(0.00731)	(0.00742)	(0.00440)	(0.00235)	(0.00665)	(0.00987)	(0.656)
Father age 5 to 9	0.000154	0.00104	0.00318	0.000381	0.00123	-0.00841	-0.284
	(0.00866)	(0.00813)	(0.00452)	(0.00289)	(0.00776)	(0.0112)	(0.729)
Mother age 10 to 14	-0.00287	-0.000134	0.00383	-0.000925	0.0112**	-0.00550	-0.449
	(0.00629)	(0.00677)	(0.00404)	(0.00173)	(0.00565)	(0.00839)	(0.555)
Father age 10 to 14	-0.00159	-0.00246	0.00126	0.00201	-0.00574	-0.0142	-1.028*
	(0.00712)	(0.00678)	(0.00381)	(0.00271)	(0.00627)	(0.00911)	(0.597)
Mother birth to age 14	0.00263	-0.00580	0.00929**	0.000172	0.0123**	-0.00159	-0.389
	(0.00661)	(0.00715)	(0.00425)	(0.00182)	(0.00598)	(0.00898)	(0.588)
Father birth to age 14	-0.000620	-0.00220	0.000817	-0.00229	-0.00376	-0.0221**	-1.340**
	(0.00734)	(0.00752)	(0.00395)	(0.00239)	(0.00636)	(0.00994)	(0.638)
Means	0.0232	0.0453	0.0062	0.0028	0.0199	0.0845	51.099
Observations	780,423	780,423	780,423	780,423	780,423	780,423	960,180

Notes: *, **, and *** denote significance at the 10, 5, and 1 percent levels. All specifications control for maternal and paternal psychiatric hospitalization during the prenatal year, maternal and paternal age at childbirth and apgar score 5 minutes after birth and include fixed effects for year of birth, sex, low birth-weight, premature birth, parity, age difference to the sibling closest in age, maternal and paternal income percentile the year before conception (based on disposable family income), and divorce, death of a close family member, large income loss and change of municipality of residence during the five-year period before childbirth for both parents. Robust standard errors in parentheses.

hood. Fathers' greater impact during the postnatal year is surprising, as children typically spend more time with their mothers during this period.[23] Hence, it appears as though mentally ill fathers tend to exhibit behavior that is has especially detrimental effects during very early life. Further, it is possible that part of the effect runs through mothers' reactions to fathers' absence and mental illness.

[23]In 1974, a gender neutral parental insurance scheme, with six months of paid parental leave, was implemented in Sweden. This period was stepwise extended, consisted of 12 months during 1980 to 1989, and was extended to 15 months thereafter. Throughout the study period, a majority of mothers spent a long period at home with their infants before returning to the labor market. The takeup rate for parental benefits was very low among fathers, amounting to 0.5% of the total amount in 1974, and reached 10% in 1998 (Försäkringskassan, 2014).

10 Concluding remarks

This study investigates the causal effects of maternal deprivation and maltreatment during various periods of childhood on adolescent health and human capital, exploiting variation in exposure between siblings. We provide evidence of highly increased hospitalization rates due to self harm and substance-related diagnoses at age 15-20 related to early-life exposure to maternal psychiatric hospitalization. We also find a small negative impact on girls' school achievement at age 15–16. Conversely, while paternal psychiatric hospitalization appears to play a less important role for child psychosocial health, it seems to have greater importance for human capital formation, pointing at gender differences in parenting roles. Hence, our results reveal a potential need for differential policies targeting affected families depending on which parent suffers from mental disorder.

Exposure to maternal psychiatric hospitalization appears to generate more severe effects at very early ages. However, the effects do not seem to be driven by exposure during the postnatal year, but spread out during the first few years of life, consistent with the literature suggesting a sensitive period for psychosocial development between birth and age 3 (see, for example Kotulak (1996)). Whereas mothers' psychiatric hospitalization is strongly related to adverse life events, it appears as though it is the illness and separation in itself, rather than the associated events, that mainly affect child outcomes. While we cannot rule out a negative impact of separation reducing parental investment in children, our results point at emotional stress being the key mechanism. In line with psychological theories emphasizing the importance of a secure mother-child attachment in early childhood, maltreatment related to mental disorder during the sensitive years of early childhood appears to be especially detrimental to future outcomes. During later childhood, more general stress, such as shock and worries, may be of greater importance.

Consistent with the literature reporting greater sensitivity to interpersonal stress factors during later childhood and early adolescence for females, we find that the treatment effects are generally stronger for girls than for boys, especially during mid- and late childhood. This suggests potential between-sex differences in causal mechanisms and also points to differences in the optimal timing of interventions.

Apart from the well-established link between school achievement and economic outcomes, several studies document strong associations between psychosocial health during adolescence and future income and employment. For example, Goodman et al. (2011) find that psychological problems experienced by age 16 are associated with a 28 percent lower household income by age 50. Similarly, Smith and Smith (2010) find that psychological problems, including substance addiction, before age 16 are associated with a 35 percent reduction in adult family income. Hence, the adverse effects of maternal psychiatric hospitalization on psychosocial health indicate a link between maternal deprivation and maltreatment during childhood and adult economic outcomes. Although our results do not indicate a greater sensitivity to maternal psychiatric hospitalization among socioeconomically disadvantaged children, they are at a greater risk of exposure than their more privileged peers. Hence, if adverse environmental factors alter genome expression, which transmits to future generations, this type of experiences may preserve and enhance economic inequality over time. Hence, providing adequate support and care for children affected by maternal deprivation and maltreatment may contribute to reducing economic inequality.

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Appendix

Table A.1: Diagnoses included in health outcome variables.

	ICD9	ICD10
Psychiatric	290-313	F0-F6 F9 R40-R46
Accidents	E80-E86 E88-E928 E930-E94	V W X1-X5 Y6-Y7 Y80-Y86 Y88
Self harm	E95	X60-X84
Abuse	E96	X85-X89
Substance-related	291 292 303-305 571 962-980 982 E850 E851 E8520-E8525 E8528-E8532 E8538-E8543 E8550-E8556 E8558 E8559 E856-E859 E8600-E8604 E8608-E8616 E8619-E8624 E8629-E8644 E8650-E8655 E8658-E8669 E867 E8680-E8683 E8688-E8693 E8698-E8699 E8780-E8786 E8788-E8799 E9301 E9313 E9319-E9320 E9330 E9332-E9334 E9350-E9352 E9354 E9355 E9358 E9364 E9371 E9378 E9380 E9382 E9390-E9393 E9395-E9401 E9408 E9409 E9411 E9412 E9425 E9426 E9430-E9436 E9438 E9439 E9441 E9444 E9450-E9458 E9461 E9463-E9466 E9470 E9471 E9474 E980	F1 F55 K70 K73 K74 R78 T38-T44 T48 T50 T51 X4 Y1 Y430 Y450 Y451 Y490-Y502 Y508-Y510 Y513 Y525 Y53 Y55 Y560 Y68 Y78-Y84 Y90 Y91
Somatic	000-289 320-629 680-738	A00-E90 G00-N99

Table A.2: Diagnoses included in maternal psychiatric hospitalizations.

ICD8	ICD9	ICD10
296-300 790	296-298 300 311	F22-F24 F28-F33 F40-F42 F44 F48 F53

Table A.3: Estimates of the probability of hospital admission at ages 15-20 and 9th year GPA related to exposure to maternal psychiatric hospitalization at specific ages. Controlling only for maternal psychiatric hospitalization during the prenatal year and year of birth-fixed effects.

	(1) Psychiatric	(2) Accidents	(3) Self harm	(4) Abuse	(5) Substance-related	(6) Somatic	(7) GPA percentile
Postnatal year	0.00534	-0.00987	0.00915	0.00431	0.00993	0.000793	0.185
	(0.00968)	(0.00880)	(0.00611)	(0.00298)	(0.00845)	(0.0123)	(0.957)
Postnatal year to age 4	0.00825	-0.00431	0.00983**	-0.00272	0.0137**	-0.00424	-1.689**
	(0.00747)	(0.00765)	(0.00452)	(0.00241)	(0.00693)	(0.00998)	(0.722)
Age 5 to 9	0.000193	-5.31e-05	0.00691	-0.000887	0.00626	-0.00380	-0.583
	(0.00700)	(0.00722)	(0.00430)	(0.00230)	(0.00647)	(0.00953)	(0.660)
Age 10 to 14	-0.00457	0.000952	0.00230	-0.00151	0.00764	-0.00556	-0.663
	(0.00613)	(0.00657)	(0.00397)	(0.00172)	(0.00555)	(0.00829)	(0.560)
Postnatal year to age 14	0.00167	-0.00707	0.00799*	-2.75e-05	0.00994*	-0.00387	-0.703
	(0.00638)	(0.00698)	(0.00413)	(0.00177)	(0.00582)	(0.00881)	(0.597)
Means	0.0234	0.0453	0.0062	0.0028	0.0197	0.0846	51.006
Observations	929,623	929,623	929,623	929,623	929,623	929,623	1,112,126

Notes: *, **, and *** denote significance at the 10, 5, and 1 percent levels. All specifications control for maternal psychiatric hospitalization during the prenatal year, and include fixed effects for year of birth. Robust standard errors in parentheses.

Table A.4: Estimates of the probability of adverse life events between birth and age 14 related to maternal psychiatric hospitalization during different periods

	(1) Substance-related hosp (mother)	(2) Divorce	(3) Income loss > 25%	(4) Family death	(5) Inter-municipal move	(6) Hosp abuse (child)	(7) Non-psychiatric hosp (mother)
Postnatal year	0.0134**	0.0119	0.0139	0.00207	0.00131	-0.000932	0.0259**
	(0.00606)	(0.00825)	(0.0116)	(0.00254)	(0.0125)	(0.000925)	(0.0110)
Means (birth to age 14)	0.0116	0.1494	0.3900	0.0036	0.3425	0.0005	0.8457
Birth to age 4	0.0288***	0.00814	0.0207**	-0.000684	-0.00477	-1.06e-06	0.0354***
	(0.00518)	(0.00617)	(0.00862)	(0.00182)	(0.00885)	(0.000617)	(0.00734)
Means (birth to age 14)	0.0116	0.1494	0.3900	0.0036	0.3425	0.0005	0.8457
Age 5 to 9	0.0500***	0.0455***	0.0379***	0.00167	0.00984	-0.000159	0.0466***
	(0.00538)	(0.00753)	(0.00869)	(0.00153)	(0.00746)	(0.000161)	(0.00857)
Means (age 5 to 14)	0.0109	0.1289	0.3035	0.0025	0.2079	0.0004	0.4785
Age 10 to 14	0.152***	0.0586***	0.0720***	0.00238**	0.0192***	0.000109	0.0644***
	(0.00714)	(0.00759)	(0.00941)	(0.00112)	(0.00740)	(0.000520)	(0.00957)
Means (age 10 to 14)	0.0080	0.0693	0.1860	0.0014	0.1000	0.0003	0.2287
Birth to age 14	0.163***	0.0671***	0.0704***	0.00311	0.0299***	0.000535	0.0425***
	(0.00821)	(0.00734)	(0.00924)	(0.00204)	(0.00873)	(0.000806)	(0.00695)
Means (birth to age 14)	0.0116	0.1494	0.3900	0.0036	0.3425	0.0005	0.8457
Maternal and child background	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Family-fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	989,546	983,839	984,966	989,546	983,839	989,546	989,546

Notes: *, **, and *** denote significance at the 10, 5, and 1 percent levels. All specifications control for maternal psychiatric hospitalization during the prenatal year, maternal age at childbirth, and apgar score 5 minutes after birth and include fixed effects for year of birth, sex, low birth-weight, premature birth, parity, age difference to the sibling closest in age, maternal income percentile the year before conception (based on disposable family income), and divorce, death of a close family member, large income loss and change of municipality of residence during the five-year period before childbirth. Robust standard errors in parentheses.

Table A.5: Estimates of the probability of hospital admission at ages 15-20 and 9th year GPA related to exposure to maternal psychiatric hospitalization at specific ages. Heterogenous effects for youngest siblings.

	(1) Psychiatric	(2) Accidents	(3) Self harm	(4) Abuse	(5) Substance-related	(6) Somatic	(7) GPA percentile
Postnatal year	0.00742	-0.00853	-0.00220	-0.000388	0.00197	-0.00191	0.229
	(0.0132)	(0.0123)	(0.00892)	(0.00301)	(0.0112)	(0.0165)	(1.244)
Postnatal year	-0.00522	-0.00140	0.0208*	0.00981*	0.0153	0.0140	0.421
	(0.0187)	(0.0159)	(0.0111)	(0.00579)	(0.0159)	(0.0223)	(1.648)
Birth to age 4	0.00235	0.00145	0.00614	-0.00457*	0.00800	0.000234	-1.130
	(0.00859)	(0.00869)	(0.00490)	(0.00265)	(0.00777)	(0.0112)	(0.794)
Youngest*Birth to age 4	0.0174*	-0.00905	0.0174***	0.00754**	0.0208**	-0.00678	1.587*
	(0.0104)	(0.00933)	(0.00567)	(0.00333)	(0.00891)	(0.0133)	(0.891)
Age 5 to 9	0.00431	0.00126	0.0145***	0.00265	0.0132	-0.00227	0.790
	(0.00905)	(0.00900)	(0.00535)	(0.00280)	(0.00838)	(0.0123)	(0.828)
Youngest*Age 5 to 9	-0.00359	0.00213	-0.00551	-0.00685**	-4.47e-05	-0.0130	-1.301
	(0.0100)	(0.00941)	(0.00593)	(0.00329)	(0.00902)	(0.0129)	(0.869)
Age 10 to 14	-0.00417	0.00495	0.00670	-0.00326	0.0142*	-0.0159	-0.270
	(0.00819)	(0.00835)	(0.00539)	(0.00251)	(0.00743)	(0.0105)	(0.713)
Youngest*Age 10 to 14	0.00251	-0.0105	-0.00431	0.00388	-0.00356	0.0173	-0.432
	(0.00894)	(0.00870)	(0.00514)	(0.00249)	(0.00785)	(0.0120)	(0.765)
Birth to age 14	0.000903	-0.00252	0.0100**	-0.000894	0.0107	-0.00540	-0.181
	(0.00747)	(0.00773)	(0.00479)	(0.00228)	(0.00685)	(0.00977)	(0.658)
Youngest*Birth to age 14	0.00241	-0.00765	-0.00236	0.00200	0.00396	0.00684	-0.576
	(0.00749)	(0.00729)	(0.00419)	(0.00223)	(0.00670)	(0.0102)	(0.645)
Observations	792,607	792,607	792,607	792,607	792,607	792,607	972,303

Notes: *, **, and *** denote significance at the 10, 5, and 1 percent levels. All specifications control for maternal psychiatric hospitalization during the prenatal year, maternal age at childbirth and apgar score 5 minutes after birth and include fixed effects for year of birth, sex, low birth-weight, premature birth, parity, age difference to the sibling closest in age, maternal income percentile the year before conception (based on disposable family income), and divorce, death of a close family member, large income loss, and change of municipality of residence during the five-year period before childbirth, and interaction terms between the youngest sibling indicator and all remaining control variables. Robust standard errors in parentheses.

Paper II

A Vision of Success - Visual Impairment and Labor Market Outcomes

1 Introduction

In order to reduce the labor market disadvantages of workers with impairments or disabilities, learning why such disadvantages occur is an important task. A vast body of research has documented a negative association between weak health and labor market outcomes (see Currie and Madrian (1999) for an overview), but much less is known about what causes these differentials. Moreover, whereas much of the literature and policy debate focuses on the effects of severe health issues, less is known about the labor market consequences of relatively minor impairments, which are not acknowledged as disabling, but may still affect labor market possibilities.

Physical impairment may affect labor market outcomes through several channels. First, it may limit productivity. The reduction may occur either as a direct effect of the impairment, or indirectly, for example, by making it difficult to attain and profit from schooling or other activities crucial to human capital accumulation (Johnson and Lambrinos, 1985; DeLeire, 2001; Hotchkiss, 2004). Second, more severe impairments could entail additional costs of entering the labor market, as eligibility for disability insurance may raise reservation wages (see e.g. Hotchkiss (2004), Autor and Duggan (2003), Bound and Waidmann (2002), Kruse and Schur (2003), and Burkhauser and Gumus (2003)). Hiring and training workers with impairments could also add employer expenses, for example, for workplace modification. This may in turn make employers reluctant to hiring these individuals, or to lower their wages to make up for the additional costs (Hotchkiss, 2004). Finally, labor market discrimination against people with impairments may be prevalent. Several studies find that employers' attitudes are more negative towards the disabled than towards many other marginalized groups, such as the elderly or ethnic minorities, indicating the existence of employer taste discrimination (see e.g. Hahn (1983)and Bowe (1978)). Additionally, statistical discrimination may occur if workers with impairments are (correctly or incorrectly) perceived as being on average less productive or more expensive to hire and train than the able-bodied (Johnson and Lambrinos, 1985; Skogman Thoursie, 1999).[a]

Researchers attempting at disentangling these sources of differential labor market success have taken one of two routes. The first method, used by Johnson and Lambrinos (1985), Baldwin and Johnson (1994), and Kidd et al. (2000), identifies two groups of disabled workers; both groups are likely to experience reduced productivity, but only one of them is likely to face discrimination. If these groups are correctly defined, any unexplained differentials in wages or employment between the able-bodied and the group that experiences productivity limitations but no discrimination is entirely due to health effects, while similar unexplained differentials between the able-bodied and the group that is likely to also face discrimination is due to health and discrimination combined. Hence, identification hinges on the health impact on productivity being similar for the two groups.

However, as noted by DeLeire (2001) and Jones et al. (2006), this assumption is most likely violated since impairments associated with more negative attitudes also tend to be more severe. Instead, the authors suggest categorizing individuals with impairments according to whether or not their impairment limits productivity. Under the assumption that these groups face a similar degree of discrimination, any unexplained wage or employment differentials between the able-bodied and non-productivity limited groups are due to discrimination, while any corresponding differentials between the able-bodied and productivity limited groups are due to work-limitations and discrimination combined. Identification in this strategy hinges on truly finding impairments that do not reduce productivity. While it is

[a]Distinguishing between different types of labor market discrimination is beyond the scope of this study. Hence, I define discrimination as negative treatment widely arising either from a preference against persons with disabilities and/or from informational bias about their productivity, see Becker (1957) and Aigner and Cain (1977). Although different types of discrimination comprise distinctly different concepts, they have the same consequences, e.g., permanently lower earnings and fewer job opportunities for disabled workers compared to their able-bodied counterparts.

easy to come up with examples, such as an economist in a wheelchair, aggregating such examples to fit a statistical analysis is more difficult. Some researchers have therefore advocated analyzing each type of impairment separately, (see Currie and Madrian (1999)).

Following the strategy used by DeLeire (2001) and Jones et al. (2006), this study is the first to explore the employment probability and earnings of individuals with various degrees of work-limitations due to visual impairment. For this purpose, I use draft data covering the entire population of Swedish males born in 1965-1975. A unique feature of this data is its detailed and objective measure of visual acuity (VA), which contains information on VA both without any correction, and with the best possible correction. This allows me to distinguish between four different groups; individuals with normal vision, those who can achieve normal vision using glasses or contact lenses, suggesting non-affected work productivity, those whose VA is slightly reduced even with the best possible correction, who may experience slight work limitations, and visually disabled persons, who are likely to face considerable work limitations. While any unexplained differentials in outcomes between the non-work limited group and the normal-sighted is likely to be a result of discrimination due to wearing glasses, the corresponding differentials between the normal-sighted and the two latter groups is likely to be due to discrimination and work-limitations combined. However, both VA and labor market outcomes may be affected by unobserved genetic and social factors, and between-group differences in unobserved characteristics could bias the estimates unless all relevant variables are included in the regression model. In order to further limit the potential effect of between-group differences in, for example, activities, values and norms, I use a sibling-fixed effects approach to control for all genetic and social characteristics shared by siblings.

While my results do not indicate any discrimination against individuals wearing glasses, they suggest the existence of negative direct consequences of visual impairment already at a low level of reduced VA after optimal correction. I find substantial reductions in both employment probability and earnings not only for the visually disabled, but also for individuals who suffer from a slight non-correctable visual impairment, but whose vision is good enough to eligible for driving. The between-group differences in earnings appear to be driven by individuals with very low incomes, suggesting that the results are due to a significantly greater share of short employment spells and/or working hours among individuals with work-limiting visual impairment. Between-group differentials in skills and health, especially non-cognitive ability, can account for part of the raw earnings and employment gaps, suggesting that part of the differentials arise due to difficulties acquiring these traits.

2 Mechanisms

The ophthalmologic literature suggest that vision loss is caused by a mix of genetic and environmental factors (see Wojciechowski (2011) for an overview). Heredity is thought to account for a large share of the variation in refractive error, such as myopia and hyperopia, which is, by far, the most common type of visual impairment, and studies report estimates of heritability ranging from 50 to over 90%.[b] Environmental factors may also impact the development of visual impairment. Several studies find strong positive correlations between refractive errors and near work, such as reading and screen work, while outdoor and sports activities are associated with lower rates of refractive errors. However, this relationship is likely to reflect causality running in both directions, incorporating both effects of activities on vision and also differences in activities chosen on the basis of visual impairment.

Studies attempting to isolate the causal effect of differences in activities on vision typically find that they account for a very small portion of the variation in VA. For example, Guggenheim et al. (2015) find that less than 1% of the cross-population variation in myopia at age 15 is attributable to differences in activities. Jones-Jordan et al. (2014) find that only about 0.5% of the variation in refractive error between siblings can be attributed to differences in activities, suggesting that this share is even smaller, and genetic variation even more important as a determinant of refractive error, within families. This result is not surprising, as siblings typically share their home environment, and hence are more likely to participate in similar activities and share values and norms surrounding, for example, studying and work ethics, which may in turn be associated with VA.

It is possible that some of the genes that affect VA also affect labor market success through separate channels. Also in this case, variation is likely to be substantially smaller between siblings than across the population, since biological siblings

[b] Myopia, or nearsightedness, is a vision condition which causes individuals to see close objects clearly, while objects farther away appear blurred. Conversely, individuals suffering from hyperopia, or farsightedness, see distant objects clearly, while closer objects appear blurry.

share approximately 50% of their genes. Importantly, the remaining genetic variation, which accounts for the vast majority of the variation in VA, is likely to be randomly distributed between siblings.[c] Hence, using within-family variation in VA for identification ensures a more randomly distributed treatment variable than would be the case in a population-based cross-sectional analysis and consequently reduces potential bias in the employment or earnings estimates stemming from both genetics and social factors.

Due to its correlation with differences in activities among children and adolescents, VA may be associated with productivity-related characteristics, such as general health and ability. As individuals with normal vision are more likely to participate in sports and outdoor activities, they are also likely to be more physically fit than their visually impaired peers, at least at a young age. Also, as suggested by Persico et al. (2004), systematic differences in participation in social activities, such as sports or clubs, at a young age may create between-group differences in human capital formation, mostly in the form of non-cognitive skills, such as motivation and sociability. Conversely, it is possible that activities associated with higher prevalence of refractive error, such as reading, foster intellectual development, enhancing cognitive ability and academic achievement. The availability of high-quality measures of health and cognitive and non-cognitive ability in the draft data allows me to control for an extensive set of such productivity-related mediators.

While discrimination is generally thought of as non-preferential treatment against specific groups, the direction of discriminatory behavior against individuals wearing glasses is less clear. It is possible that negative perceptions about the physical attractiveness, productivity, or social skills of glasses-wearers gives rise to non-preferential treatment.[d] However, it may also be the case that positive stereotypes about, for example, glasses-wearers' intelligence and work/study motivation leads to positive discrimination. The direction and mechanisms behind discrimination may also vary across occupational categories. If this is the case, both expectations about future discrimination and actual physical restrictions may cause individuals

[c] In short, a gene consists of two alleles, one of which is randomly inherited from each parent at conception. This phenomenon is known as Mendellian randomization (see e.g. Fletcher and Lehrer (2011)).

[d] In a correspondence study based on fictitious job applications with manipulated photographies, Rooth (2009) finds significant differences in callback rates for obese males. The results suggest that the difference in callback rates is driven by differences in perceived attractiveness. However, to my knowledge, no similar studies using individuals with and without glasses have been conducted.

to self-select into educational and occupational categories based on their impairment. This may in turn affect earnings and employment rates. In order to investigate the role of these potential channels, I use detailed measures of educational attainment and occupational categories based on Swedish register data to control for educational and occupational selection.

3 Data

3.1 The draft data

The empirical analysis is based on a data set constructed by integrating draft data from the Swedish National Service Administration and registers from Statistics Sweden from 2003. The draft data contains records of various tests performed during the enlistment procedure for each person who enlisted during 1984-1997, and who was a Swedish resident in 1999.[e] During this time, enlistment was compulsory for male Swedish citizens, and the only ones granted exemption were those who were living abroad, institutionalized, in prison, or previously convicted of serious crimes.[f] Failure to comply with enlistment led to fines and ultimately to imprisonment (Värnpliktslag, 1967). This implies that the attrition is very low in the data; only about 3% of the males in each cohort did not enlist.

The sample is restricted to males born in Sweden of Swedish-born parents, in order to exclude potential effects of ethnic dicrimination and to avoid the selection that is likely to be present among women and immigrants enlisting for the military. Since carrying out parts of the cognitive ability test requires some vision, this test score is likely to be a biased measure of cognitive ability for blind individuals. For this reason, I also exclude this group. Further, I exclude individuals who have missing records of any of the vision, cognitive or non-cognitive test scores. This procedure results in a total sample size of 443,015 individuals.

The enlistment procedure spans two days with tests of health, physical fitness,

[e] The reason why the study only includes individuals who were Swedish residents in 1999 is that many variables, e.g. the information on enlistment and family background, are collected from the 1999 census.

[f] Institutionalized individuals include, e.g., persons with intellectual disabilities correlated with visual impairment, such as Down's syndrome. The crimes that lead to exemption were mostly violent or drug-related.

and cognitive ability. A certified psychologist also interviews each conscript in order to assess his correspondence to the psychological requirements of serving in the Swedish defence. During the time period considered by this study, nearly everyone who enlisted also went through military service. Therefore, in general, the results on the enlistment tests did not influence the possibility of being selected for military service, but merely the specific placement, worse results leading to a less qualified and meriting type of service. Thus, the incentives to deliberately under-perform on these tests were limited. However, Section 5.3 still addresses this issue by conducting a sensitivity analysis aimed at limiting the influence of potential fakers.

The visual acuity measure

The vision test result combines refraction corrected and non-corrected visual acuity (VA), providing a detailed measure of visual function.[g] VA is in most cases, regardless of the degree of impairment, relatively stable from age 18-20, which is when enlistment takes place, and into adulthood. Hence, the test result is likely to not only capture VA during enlistment, but also at the time that outcomes are measured. The test is performed by having the conscript read different size letters from a VA chart, for each eye independently. Conscripts with reduced vision perform the test both without refractive lenses and after refraction with the optimal correction, using a vision screener.[h] If the conscript cannot read the top line of the test board, a supplementary test is performed, where the test leader asks the conscript to count how many fingers (s)he is holding up from a distance of approximately 2 meters. Combining the results from the tests performed with and without refraction, and the supplementary test, I construct a measure of functional vision, which approximates performance in vision-requiring activities. This measure is

[g] VA is the most common clinical measurement of visual function. It is a measure of clearness of form vision, which is dependent on the sharpness of the retinal focus within the eye, the sensitivity of the nervous elements, and the interpretative faculty of the brain. VA is a quantitative measure of the ability to identify black symbols on a white background at a standardized distance as the size of the symbols is varied. The VA represents the smallest size that can be reliably identified. A VA of 1 (20/20) is defined as normal vision. A person with a VA of 0.5 (20/40) can see detail from a 20 feet distance with the same clarity as a person with normal eyesight would see it from 40 feet away etc.

[h] A vision screener is the standard instrument for vision tests performed by optometrists. Conscripts with reduced VA read the letters from a VA chart looking through the vision screener, like a set of binoculars, while the test leader varies the refraction strength by switching between different-strength glass lenses.

based on a scale that relates VA to functionality, and which is constructed by The International Council of Ophthalmology (ICO).[i] As remarked by Colenbrander (2003, 2005), the limits of this scale do not represent absolute cutoff points for functional vision, but values on a continuous scale (for example, a VA of 0.5 is not an absolute threshold value beyond which one can be judged as a safe driver).[j] This view is supported by West et al. (2002), who find that the association between VA and performance on various mobility, daily living and visually intensive tasks is relatively linear, suggesting that there is no clear cutoff point where a visual impairment becomes disabling.

Table 1: The vision test score groups.

Group	Functional vision	Corrected VA. Max	Corrected VA. Min	Non-corrected VA. Max	Non-corrected VA. Min
1. Normal-sighted	Normal without refraction	1.0	0.8	1.0	0.8
2. Non-work limited	Normal with refraction	1.0	0.8	0.7	0.0
3. Work-limited	Driving vision with refraction	0.7	0.3	0.7	0.0
4. Disabled	Visual disability	0.2	F2	0.2	0.0

Notes: A value of 1.0 corresponds to being able to read the bottom line on a standard vision test board. A value of 0.1 corresponds to being able to read the top line on the vision test board. 0.0 represents absolute blindness. F2 represents a capability to determine how many fingers the test leader holds up from a 2-meter distance.

The ICO defines a corrected VA of 0.8 or above on the best eye as normal vision. A VA within this range generally allows the individual to read regular newsprint at a 80-160 cm distance, thus providing a comfortable reserve given the normal reading distance of approximately 40 cm. Following these standards, I categorize individuals with a VA of 0.8 or above without correction as non-impaired (*Group 1*), while persons who can achieve a VA within this rage using refraction as impaired but not work-limited (*Group 2*).

According to the same standards, individuals with a corrected VA between 0.79

[i]Although VA stabilizes around age 18-20 in most cases, some individuals may experience reductions in VA after this age. In these cases, the VA measure overstates the VA in 2003, which leads to a bias towards zero in the results.

[j]Colenbrander (2005) also remarks that the correspondence between VA and functional vision is not perfect, i.e. despite a strong correlation, functionality varies between individuals with similar levels of VA.

and 0.3 on the best eye suffer from mild vision loss. Most of these individuals are able to read regular newsprint at a 32-63 cm distance at a normal or near-normal speed with refraction. Compared to individuals with normal vision, the comfortable reserve is reduced or gone, but the average reading performance is not seriously compromised and, in general, individuals belonging to this category can perform most working tasks with refraction (Colenbrander, 2003).[k] However, West et al. (2002) find that over 50% of a sample of elderly individuals perform more than one standard deviation below average in a test of reading speed already at a VA of 20/30 (approximately 0.67), suggesting some reduction in reading performance already at low levels of visual impairment.

Individuals at the lower end of the 'mild vision loss' range are likely to experience significant restrictions in everyday life. One such restriction is the eligibility of getting a Swedish driver's license, for which a VA of 0.5 or more is required. This limit suggests that a VA of less than 0.5 comprises a vision loss severe enough to restrict functionality and hence to reduce productivity in certain situations. Additionally, the ineligibility of getting a driver's license may in itself be limiting, as it restricts occupational choice, limits the possibility of working far away from home, etc. For this reason, I categorize individuals with a corrected VA between 0.7 and 0.5 as visually impaired and work-limited (*Group 3*), and persons with a corrected VA below 0.5 as disabled (*Group 4*). This is a somewhat more generous definition of visual disability than the one adopted by the ICO, which defines a corrected VA between 0.3 and 0.02, implying a need for magnifiers while reading and visual aids in order to maintain many types of jobs, as disabling (Colenbrander, 2003).

Individuals with a VA below 0.02 are categorized as (nearly) blind and are excluded from the study.

The ability measures

During the enlistment procedure, the conscripts take the The Swedish Enlistment Battery Test, which is similar to the American Armed Forces Qualification Test (AFQT), in order to evaluate their cognitive skills.[l] The test result, known as the

[k] Colenbrander (2005) compares the degrees of functionality visual functionality for normal-sighted individuals and persons suffering from mild vision loss in terms of physical mobility as 'can walk and run' and 'can walk, but not run', respectively.

[l] The individuals in the sample have taken the Enlistment Battery 80 test, which consists of four parts; instructions, synonyms, metal folding and technical comprehension. The instructions

G factor, is a composite measure derived from the scores of four separate tests and is intended as a measure of general intelligence.[m,n] It is measured on a scale ranging from 1 to 9.

Whereas cognitive skills comprise a clear concept, related to the individual's problem-solving ability, non-cognitive skills are more loosely defined. Generally, the labor-economic literature defines non-cognitive skills as personality traits that are not related to direct problem-solving, but that may still affect productivity, for example, sociability, motivation and persistence. During enlistment, this type of characteristics are assessed through a test aimed at evaluating the conscript's ability to cope with the psychological requirements of military service and potential war. The test consists of a semi-structured interview performed by a certified psychologist and rewards abilities such as social skills, willingness to assume responsibility, independence, emotional stability, persistence, and power of initiative, which are likely to be rewarded not only in the military, but also on the general labor market. Similar to the cognitive ability test, the non-cognitive skill test is graded along a scale ranging from 1 to 9.[o]

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test measures the person's ability to make inductions and, together with the synonyms test, verbal skills. The metal folding test is a spatial test, which measures mathematical/logical ability. The technical comprehension test is related to general knowledge. Each part contains 40 questions and is graded on a 1-9 point scale. The scores of the different parts of the test are normalized into a nine-point scale and, in accordance with the factor analysis method, summed up and transformed into a new nine-point scale. The separate tests and the composite measure have a correlation above 0.9. Visually impaired conscripts perform the test using their regular corrective lenses. According to Björn Bäckstrand, head doctor at the Swedish National Service Administration, the time limit for the test is very generous, so that reading difficulties should not put a restriction on test performance (personal communication April 13th 2016).

[m] For more information on the G factor, see Carroll (1993).

[n] Several studies, opposing the results of Herrnstein and Murray (1994), who suggest that the AFQT mostly measures inherent ability, have shown that achievement test scores such as the AFQT increase with age and schooling (see e.g. Hansen et al. (2004) and Neal and Johnson (1996)). Following this reasoning, concern has been raised about scores from cognitive ability tests possibly being affected by previous schooling and labor market experience. Nordin (2007) remarks, however, that since the enlistment test is generally taken during upper-secondary school, which is attended by the vast majority of Swedish citizens, the test results should not be affected by post-secondary education and labor market experience. The estimate may still be affected by differences in human capital formation before the time of the test, however. Systematic differences in e.g. school quality or parental investment in the human capital of their children may thus affect the test score.

[o] All individuals in the data went through a procedure that was adopted in 1969 and kept unchanged until 1995. A certified psychologist conducted a semi-structured interview with each conscript for approximately 25 minutes. At the time of the interview, the psychologist had access to information on the conscript's results on the test of cognitive ability, physical endurance, muscular

Both cognitive and non-cognitive ability have been shown to be positively related to labor market outcomes (see e.g. Heckman and Rubinstein (2001), Heckman et al. (2006), Borghans et al. (2008)). In a study based on Swedish data, Lindqvist and Vestman (2011) find that a one standard deviation increase in cognitive ability is associated with 8.9 percent higher earnings. For non-cognitive ability, an increase of the same magnitude is associated with 6.9 percent higher earnings. The authors also show that the measures affect labor earnings through (partly) different channels; individuals with high non-cognitive skills are less likely to be unemployed and also have shorter unemployment spells than those with lower non-cognitive skills, while wages are more heavily affected by cognitive than non-cognitive skills.

The health measures

The study uses height and body mass index (BMI) measured during the enlistment procedure and enlistment test scores considering muscular strength and endurance as measures of physical capacity. These variables can be viewed as proxies for general health, and also as indicators of productivity in manual labor.

Adult height has been shown to be related to several aspects of childhood health (see e.g. Bozzoli et al. (2009) and Elo and Preston (1992)). Moreover, a positive association between height and earnings and other measures of social status is one of the most consistent findings within the social sciences (see e.g. Komlos (1990) Persico et al. (2004), Case and Paxson (2008), Steckel (2009), and Lundborg et al. (2014)). Conversely, there is evidence that a high BMI is negatively related to general health (Burton et al., 1998, Pronk et al., 1999), while the evidence on its effect on labor market outcomes is mixed for male workers.[p]

Whereas height and BMI proxy more general aspects of health, physical strength and endurance are more closely related to productivity in manual labor. Despite

strength, school grades, and the answers to 70–80 questions about e.g. friends, family, and hobbies. The details of the non-cognitive enlistment test procedure are confidential and we are referred to a rudimentary description of the procedure and its intentions. For a thorough discussion of the non-cognitive test score, see Lindqvist and Vestman (2011).

[p]Averett and Korenman (1996), Sarlio-Lahteenkorva and Lahelma (1999), and Cawley (2004) find no effect of obesity on earnings for male workers, while D'Hombres and Brunello (2007), Rooth (2009), and Kropfhäuser and Sunder (2015) find some evidence of excess weight leading to lower earnings for both sexes.

the fact that relatively few jobs are physically demanding in the contemporary western world, physical strength has been shown to be positively correlated with earnings for Swedish males (Lundborg et al., 2014)). I use two separate test results as proxies for muscular strength; hand grip strength, measured as the maximum pressure exerted by one hand squeezing a dynamometer, and leg strength, measured as the maximum resistance achieved using a leg extension machine. The endurance variable corresponds to the maximum resistance attained in watts when riding a stationary bike for approximately 5 minutes.

All observations have records of the cognitive and non-cognitive test scores by data design. However, some observations lack information on the health-related variables. This is most common for the hand grip strength measure, for which information is missing for 0.4% of the observations.[q] For the missing records, I impute the variable mean and create an additional binary indicator taking on the value one when information is missing and zero otherwise.

3.2 The Statistics Sweden data

As outcome variables, this study uses annual earnings and employment status in 2003, which is the only year available in the data. These variables are derived from tax records. The earnings variable measures annual labor income including income from self-employment and short-term sick-leave benefits. The employment variable indicates whether or not the individual had a positive labor income during 2003.

The data also allows me to connect family members. This provides an opportunity to control for family background using information on parental income and education levels. Additionally, it is possible to identify 144,788 brothers in the data, which allows me to use a sibling-fixed effects approach to effectively control for a broad range of genetic and family-related confounders.

Some individuals have missing records of certain explanatory variables. This is most common for parental education and income, for which information is missing for at most 11% percent of the observations. Similar to my treatment of missing health variables, I impute the variable mean for the missing records and create

[q]For the remaining physical capacity test scores and BMI, information is missing for 0.1-0.24% of the observations.

an additional binary indicator taking on the value one when information is missing and zero otherwise.

4 Descriptive statistics

A statistical description of the population reveals some between-group differences with respect to characteristics and labor market outcomes. The employment rates are 4.1 percentage points lower for work-limited people and 6.9 percentage points lower among disabled individuals than among those with normal vision. For those who are employed, the average earnings are 11.3% lower for the group that suffers from work-limiting impairment and 12% lower for the disabled. However, workers with a non-work-limiting visual impairment have 2.6% higher average earnings than workers with normal vision. This is also the group with the highest average education and cognitive test score in the sample, possibly reflecting differences in human capital accumulation during childhood due to between-group differences in the choice of activities. The non-work-limited group have a 15 % higher average cognitive test score, while work-limited and disabled individuals tend to have lower scores than individuals with normal vision. A qualitatively similar pattern appears when describing the schooling distribution, which indicates a connection between cognitive ability at the time of enlistment and later investments in schooling. For the non-cognitive test score, the difference between the results of non-work-limited and non-impaired groups' scores is negligible, whereas work-limited and disabled individuals score 21% and 26% lower than the non-impaired, respectively. Additionally, physical capacity appears to be positively related to visual acuity. In general, individuals with better vision are slightly taller and score better on the physical enlistment tests than persons with a lower visual acuity. However, there are no statistically significant differences in the average BMI of the different groups. The siblings sample resembles the full population with respect to between-group differences in characteristics and outcomes.

5 Empirical strategy

Explaining between-group differences in labor market success involves credibly distinguishing the effects of discrimination from those of reduced vision and unobserved characteristics. In order to do this, Neal and Johnson (1996) suggest altering

Table 2: Sample statistics

	(1) Normal vision	(2) Non-work limited	(3) Work limited	(4) Disabled
FULL POPULATION				
Employed 2003	0.959	0.956	0.918	0.890
	(0.000)	(0.000)	(0.008)	(0.023)
Earnings 2003	271,231.8	278,275.9	240,569.6	238,476.6
	(248.070)	(516.417)	(4338.308)	(8827.264)
Years of schooling 2003	12.126	12.972	11.708	11.907
	(0.003)	(0.007)	(0.058)	(0.154)
Cognitive ability	5.031	5.782	4.233	4.158
	(0.003)	(0.006)	(0.063)	(0.178)
Non-cognitive ability	5.175	5.139	4.063	3.814
	(0.003)	(0.005)	(0.053)	(0.141)
Height	179.423	179.868	177.999	177.983
	(0.011)	(0.022)	(0.208)	(0.505)
BMI	21.915	21.705	21.800	21.783
	(0.005)	(0.010)	(0.097)	(0.227)
Muscular endurance	4.284	4.292	4.018	3.957
	(0.001)	(0.002)	(0.023)	(0.056)
Leg strength	597.123	589.784	553.144	536.271
	(0.197)	(0.383)	(3.744)	(10.234)
Hand grip strength	619.283	600.788	576.896	556.593
	(0.161)	(0.311)	(3.164)	(8.007)
Age 2003	33.222	32.491	33.483	33.874
	(0.005)	(0.010)	(0.101)	(0.219)
N	348,701	93,004	1,128	182
SIBLINGS				
Employed 2003	0.963	0.960	0.924	0.909
	(0.001)	(0.001)	(0.013)	(0.039)
Earnings 2003	270,851.4	279,554.7	243,183.4	223,073.3
	(428.811)	(892.377)	(6657.744)	(17,819.230)
Years of schooling 2003	12.091	12.948	11.553	12.110
	(0.006)	(0.013)	(0.109)	(0.306)
Cognitive ability	4.982	5.740	3.995	4.278
	(0.006)	(0.011)	(0.113)	(0.339)
Non-cognitive ability	5.165	5.146	3.921	3.685
	(0.005)	(0.009)	(0.095)	(0.261)
Height	179.351	179.823	177.714	176.944
	(0.019)	(0.038)	(0.377)	(0.966)
BMI	21.865	21.640	21.852	21.702
	(0.008)	(0.016)	(0.167)	(0.348)
Muscular endurance	4.311	4.330	4.016	4.090
	(0.002)	(0.004)	(0.040)	(0.108)
Leg strength	596.489	590.655	540.076	536.204
	(0.339)	(0.668)	(6.652)	(18.966)
Hand grip strength	619.638	602.401	563.507	541.685
	(0.279)	(0.549)	(5.350)	(14.072)
Age 2003	33.213	32.540	33.356	33.836
	(0.009)	(0.017)	(0.171)	(0.394)
N	116,946	30,228	369	55

Notes: Employed 2003 equals having a positive labor income during 2003. Labor income is in Swedish crowns. All ability and health measures stem from enlistment tests performed at age 18 or 19. Hand grip strength is the maximum pressure exerted by one hand squeezing a bar. Leg strength is the maximum resistance achieved using a leg extension machine. Muscular endurance is the maximum resistance attained in watts when riding a stationary bike for approximately 5 minutes. Standard deviations in parentheses.

a traditional Mincer-type equation to include only variables determined before labor market entry, in order to avoid endogeneity in the explanatory variables. The reason for doing so is that many variables commonly used to control for worker productivity, such as occupation, labor market experience and post-secondary education, may be affected by (expected or actual) labor market discrimination. Following this strategy, this paper uses only explanatory variables determined at or before enlistment at age 18 or 19 in its main analysis of the between-group employment and earnings differentials. The baseline model can be written

$$y_i = \alpha + \beta v_i + \delta yob_i + \varepsilon_i \tag{2}$$

where i is an index for the individual, v is an indicator variable representing his degree of visual impairment, and yob represents a year of birth-fixed effect, accounting for age and cohort-specific features. The outcome variable y differs between the analyses; in the analysis of employment, y_i can be interpreted as the probability of individual i having positive earnings during 2003, whereas in the earnings analysis, y_i is the logarithm of annual earnings for individual i. In this model, β picks up the effects of both discrimination, mediating factors, and omitted variables that are associated with both earnings/employment and VA. I then stepwise add variables, such as cognitive and non-cognitive skills and the physical ability measures reported above, which could serve as mediating factors in the relationship between VA and labor market outcomes. Subsequently introducing variables related to parental background, in this case parental schooling and earnings, allows me to control for factors that are likely to be related to both VA, the treatment of visual impairment (for example, check-up frequency and investments in glasses during childhood), and labor market outcomes. The full model is represented by

$$y_i = \alpha + \beta v_i + \gamma X_i + \varepsilon_i \tag{3}$$

where X is a vector of individual characteristics, including year of birth-fixed effects, cognitive and non-cognitive ability, physical capacity, and parental background. In order to allow for non-linear relationships, I also include the squares of age and the two ability measures. In this model, β can be interpreted as the remaining earnings/employment penalty after accounting for the effect of VA that runs through the mediating variables, and conditional on the confounders reported above. However, unless all relevant variables are included in the model, β does not represent pure discrimination for non-work limited individuals, but also includes the effects of omitted variables. In order to account for further confounding factors at the family level, I exploit the siblings sample, constructing my preferred specification

$$y_{ij} = \alpha + \beta v_{ij} + \gamma X_{ij} + \theta_j + \varepsilon_{ij} \tag{4}$$

where ij is an index for individual i belonging to family j. θ represents a family-fixed effect that accounts for all genetic and social characteristics that are shared by brothers. Hence, β should not be biased due to omitted family-level variables that are associated with both VA and earnings. Since all variables included are determined before labor market entry, and thus likely to be unaffected by labor market discrimination, the estimated differentials for the non-work limited group can be seen as upper bounds for labor market discrimination after the time of enlistment, while the estimates for the work-limited and disabled groups include both discrimination and direct effects of visual impairment on productivity and labor supply. Between-group differences in characteristics that arise at an early age are likely to be captured by the control variables.

All specifications include robust standard errors. Further, when analyzing between-group differences in employment, I exclude all cases with a predicted employment probability below zero or above one, in order to minimize the potential issues concerning the linear probability model.[1]

5.1 Occupational and educational sorting

Including schooling and occupational category in the earnings functions may lead to biased estimates of the visual impairment penalties. Neal and Johnson (1996) argue that schooling is a noisy measure of skills and that it is likely to be endogenous if, for example, anticipation of future discrimination causes visually impaired individuals to invest in less education than the normal-sighted. If this is the case, including schooling in the employment or earnings functions would lead to an understatement of discrimination. Conversely, if years of schooling systematically overstates the relative skill of the visually impaired, for example, if this group have problems profiting from education due to their impairment, discrimination is likely to be overstated if schooling is included in the model. A similar situation occurs when controlling for occupational category, as an individual's choice of occupation may be affected by both (actual or perceived) discrimination and could also, as suggested by Skogman Thoursie (1999), be directly affected by the physical

[1]The observations dropped are all due to a predicted employment probability above one, and belong to the normal-sighted and (in a few cases) the non-work limited groups. In my preferred specification, i.e. Model 4, using sibling fixed effects, this only affects the specification including schooling (see Table 4, column 5), where two observations from the normal-sighted group are excluded.

restrictions due to impairment. It is also possible that wearing glasses per se, rather than reduced VA, poses restrictions on productivity in certain professions, which would lead to an overstatement of discrimination if occupational categories are not controlled for. However, if individuals sort themselves into educational and professional categories based on their VA, these may be important channels of any outcome differentials.

To investigate this possibility, I augment Models 3 and 4 to control for schooling and, in the analysis of the earnings gaps, occupational category. Similar to the outcome variables, these characteristics are measured in 2003. Using the Swedish version of the educational attainment variable ISCED97, I construct a years of schooling variable that ranges between nine and twenty. I control for between-group differences in occupational distributions by adding fixed effects for 115 occupational categories based on SSYK (Standard for Swedish Occupational Classification), which is a three-digit occupational classification code similar to the international classification (ISCO). In the models which include both the cognitive test score and schooling, the schooling variable can serve as a proxy for skills that are not captured by the cognitive test or that are attained after the test date Neal and Johnson (1996).[5]

6 Results

Table 3 reports the results from estimating Model 2 on my full population, stepwise adding control variables to form Model 3 in the final column. As a first step, column 1 of Table 3 reports crude estimates of the between-group employment gaps, with age as the only additional control variable. The rows in Table3 and subsequent tables that are denoted "Δ (%)" contains information on the direction and magnitude of change in this crude estimate imposed by each subsequent model. The results show that having a work-limiting visual impairment is associated

[5]As Nordin (2007) remarks, these two variables are affected by the same underlying ability. The joint causality could cause endogeneity problems if they are included in the same model. This has been illustrated by e.g. Angrist and Krueger (1991), who find that including an endogenous test score causes a negative bias in the returns to schooling estimate. However, it may also be the case that visually impaired over-invest in schooling if, as suggested by Lang and Manove (2006), statistical discrimination leads to education being more important as a signal of productivity for disadvantaged groups than for the majority population. If this is the case, jointly including the cognitive test score and schooling is relevant.

Table 3: The employment and earnings gaps. Full population.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
EMPLOYMENT							
Non-work limited	-0.00253**	-0.00574***	-0.00379***	-0.00346***	-0.00316***	-0.00447***	
	(0.00127)	(0.00129)	(0.00128)	(0.00129)	(0.000763)	(0.000767)	
Work limited	-0.0379***	-0.0296**	-0.0157	-0.0151	-0.0199**	-0.0206***	
	(0.0138)	(0.0137)	(0.0135)	(0.0135)	(0.00802)	(0.00801)	
Disabled	-0.0539	-0.0456	-0.0235	-0.0223	-0.0382*	-0.0401*	
	(0.0388)	(0.0393)	(0.0392)	(0.0391)	(0.0230)	(0.0230)	
Cognitive ability		0.0205***	0.00827***	0.00826***	0.00942***	0.00745***	
		(0.00140)	(0.00138)	(0.00139)	(0.000849)	(0.000860)	
Cognitive ability^2		-0.00152***	-0.000668***	-0.000658***	-0.000678***	-0.000659***	
		(0.000126)	(0.000125)	(0.000125)	(7.62e-05)	(7.76e-05)	
Non-cognitive ability			0.0500***	0.0498***	0.0503***	0.0487***	
			(0.00208)	(0.00209)	(0.00123)	(0.00123)	
Non-cognitive ability^2			-0.00397***	-0.00399***	-0.00396***	-0.00388***	
			(0.000187)	(0.000187)	(0.000110)	(0.000110)	
Baseline mean	0.959	0.959	0.959	0.959	0.959	0.959	
Observations	443,015	443,015	443,015	443,011	442,788	440,034	
Δ (%) Non-work limited		-127	-50	-37	-25	-89	
Δ (%) Work limited		22	59	60	47	46	
Δ (%) Disabled		15	56	59	29	26	
EARNINGS							
Non-work limited	0.0221***	-0.0260***	-0.00746**	-0.00549*	-0.00618**	-0.0136***	-0.00588**
	(0.00308)	(0.00309)	(0.00306)	(0.00306)	(0.00306)	(0.00306)	(0.00258)
Work limited	-0.190***	-0.144***	-0.0835***	-0.0773***	-0.0757***	-0.0784***	-0.0589**
	(0.0290)	(0.0285)	(0.0282)	(0.0282)	(0.0281)	(0.0281)	(0.0245)
Disabled	-0.205***	-0.155**	-0.0680	-0.0581	-0.0588	-0.0627	0.0306
	(0.0770)	(0.0764)	(0.0731)	(0.0727)	(0.0729)	(0.0726)	(0.0449)
Cognitive ability		0.0625***	0.00485	0.00205	0.000826	0.00644**	1.70e-05
		(0.00297)	(0.00302)	(0.00302)	(0.00301)	(0.00306)	(0.00263)
Cognitive ability^2		1.82e-05	0.00324***	0.00338***	0.00338***	0.00187***	0.000337
		(0.000281)	(0.000282)	(0.000282)	(0.000282)	(0.000289)	(0.000247)
Non-cognitive ability			0.132***	0.127***	0.123***	0.124***	0.0692***
			(0.00418)	(0.00418)	(0.00418)	(0.00418)	(0.00348)
Non-cognitive ability^2			-0.00565***	-0.00575***	-0.00566***	-0.00603***	-0.00306***
			(0.000384)	(0.000383)	(0.000383)	(0.000383)	(0.000320)
Observations	424,686	424,686	424,686	424,686	424,686	424,686	398,443
Δ (%) Non-work limited		-218	-134	-125	-128	-162	-127
Δ (%) Work limited		24	56	59	60	59	69
Δ (%) Disabled		24	67	72	71	69	115
Health				Yes	Yes	Yes	Yes
Family					Yes	Yes	Yes
Years of schooling						Yes	Yes
Occupation-FE							Yes

Notes: *, **, and *** denote significance at the 10, 5, and 1 percent levels. All specifications include year of birth-fixed effects. The employment analysis estimates the probability of having a positive labor income during 2003. The outcome variable in the earnings analysis is the logarithm of annual earnings in 2003. The sample used in the earnings analysis includes individuals having a positive labor income during 2003. *Health* includes height, BMI, leg strength, handgrip strength, physical endurance, and indicators of missing records of each of these variables. *Family* includes income and years of schooling for the subject's mother and father separately (all measured in 1980) and indicators of missing records of each of these variables. All ability and health measures stem from enlistment tests performed at age 18 or 19. In the employment analysis, all observations with a predicted employment probability above 1 or below 0 are excluded. Δ (%) is the change in estimates compared to the raw estimate displayed in column 1. Robust standard errors in parentheses.

with an 4.0% lower probability of having a job than an individual of the same age with normal vision. The corresponding estimate for the disabled group is a statistically non-significant 5.6%, while individuals with non-limiting impairments have a very slight (0.3%), but statistically significant, lower probability of being employed than normal-sighted persons.[1]

Second, I add the cognitive test score to the specification. As displayed in column 2, this leads to a 22% reduction of the employment gap for the work-limited. The corresponding reduction for the disabled group is 15%, while the minor gap between those with a non-limiting impairment and the normal-sighted more than doubles. This is not surprising, given the higher cognitive skills of the non-work limited group reported in Table 2. As the test score can be seen as a measure of productivity at age 18 or 19, the estimated differentials include effects of all between-group differences that have arisen after the test was taken, including differences in, for example, schooling and labor market experience.

In the third step, I add the non-cognitive test score to the model, in order to account for productivity-related characteristics such as social skills, persistence, and responsibility. Column 3 reports the results. This exercise further reduces the employment gap for all groups. The reductions are large; 34% for the non-work limited, 47% for the work-limited, and 48% for the disabled, suggesting that non-cognitive ability is an important channel of the employment differentials. In line with the findings in Lindqvist and Vestman (2011), the inclusion of non-cognitive ability decreases the return to cognitive ability dramatically, suggesting that the effect of cognitive skills on the probability of having a job mostly runs through it positive correlation with non-cognitive ability.

Column 4 reports results from a specification adding the physical capacity measures, as a measure of general health and manual productivity, along with the cognitive and non-cognitive test scores. Column 5 also adds controls for parental income and parental education in order to reduce the potential bias stemming from between-group differences in family background. These exercises lead to additional, but smaller, reductions in employment gaps, leaving statistically significant employment disadvantages of 0.3, 2, and 4 percent for the non-work limited, work-limited, and disabled groups, respectively, when exploiting the full set of exogenous explanatory variables (see column 5).

[1]The estimates from the linear probability model are similar to the marginal effects yielded by a probit model using the same set of explanatory variables, see Table A.1 in the Appendix.

In column 6, I explore the possibility of self-selection into educational categories being a reason behind the employment differentials by adding years of schooling to the specification. This exercise leaves statistically significant employment penalties of 0.5% for the non-work limited, 2.1% for the work limited, and 4.2% for the disabled group. Hence, it does not appear as if between-group differences in educational length reinforce the between-group differences in employment. Rather, at least for the non-work limited group, it seems like schooling choices instead counteract these differences, although to a modest degree.

The estimated employment differentials suggest that work-limited and disabled individuals who have a job are positively selected. Turning to the earnings differentials between this group and their normal-sighted peers reveals a story qualitatively similar to the one told by the employment gaps. The crude estimates of the earnings gaps within the full population show that workers with a non-limiting visual impairment have slightly (2%) higher unadjusted earnings than normal-sighted individuals of the same age (see column 1). The corresponding estimates for the work-limited and disabled workers are negative, amounting to 19 and 21%, respectively, to their disfavor.

Controlling for cognitive ability reduces the unexplained shares of the earnings differentials for the work-limited and disabled groups, although negative earnings gaps of 14-16% remain. For the non-work limited group, the estimate switches sign, indicating a 2.6% earnings disadvantage, suggesting that workers belonging to this group do not get fully compensated for their relatively greater cognitive skills.

Also adding non-cognitive ability to the specification reduces the estimated earnings gaps substantially for all groups, leaving an unexplained earnings disadvantage of 8.4% for the work limited. The corresponding estimate for the non-work limited is reduced to a modest 0.7%, while the estimate for the disabled group decreases by more than 50%, leaving a statistically non-significant 6.8% earnings penalty. Similar to the results concerning the employment gap, including the non-cognitive test score in the model dramatically reduces the estimated return to cognitive ability, in this case also rendering it statistically non-significant.

Column 4 displays the results from a model adding controls for physical capacity, while column 5 reports results from a specification also including parental earnings and parental years of schooling. Both these exercises lead to marginal reductions in earnings gaps. When controlling for the full set of exogenous explanatory variables, as shown in column 5, the resulting estimates indicate a statistically significant adjusted earnings penalty of 7.8% for the work-limited group, whereas the gap between the non-work limited and the normal-sighted amounts to 1.4%. Although statistically non-significant, the estimated 6.3% earnings gap between the disabled group and the normal-sighted indicate the existence of an earnings penalty for this group.

In order to investigate the role of educational self-selection for the earnings differentials, column 6 adds years of schooling to the specification. This exercise results in a 1.4% earnings disadvantage for this group. The corresponding results for the work limited is 7.8%, while the estimate for the disabled group remains similar. Similar to the results regarding the employment differentials, the increased estimates suggests that, although to a modest degree, between-group differences in schooling evens out, rather than reinforces, the earnings gaps.

Finally, in column 7, I re-estimate the earnings differentials , also controlling for between-group differences in occupational distributions. For the work-limited group, this exercise reduces the size of the earnings gap to 5.9%, while the corresponding estimate for the non-work limited is reduced to 0.6%. Hence, self-selection into occupational categories seems to contribute some to the earnings gaps. All results are similar to those yielded using an alternative income measure that does not include any social benefits as basis for the outcome variables (results are available on request).

6.1 Making use of the siblings sample

It is possible that unobserved characteristics at the family level, such as genetics, values and norms, affect the relationship between visual acuity and labor market outcomes. For example, if visual impairment is more prevalent in more intellectually orientated or hard working families, or in families prone to activities which may potentially be related to both VA and work productivity, such as reading, this could generate between-group differences in productivity which are not captured by the control variables. If this is the case, the OLS results will be biased. In order to limit the influence of possible mediators and confounders related to family background, I limit the sample to individuals who have at least one brother represented in the sample and re-estimate the employment and earnings gaps using a sibling-fixed effects model. This method exploits within-family variation in VA, which is likely to be more randomly distributed than the corresponding variation on the population level (Jones-Jordan et al., 2014). Given this argument, the sibling-fixed effects model is my preferred specification.

First turning to the representativeness of the siblings sample, TableA.2 in the Appendix display the results of an OLS estimation on the siblings sample. These results are similar to the ones reported in Table 3, but display a slightly lower statistical significance level in some cases, possibly due to a smaller sample size. Hence, the siblings sample appears to resemble the full population, and the external validity of the siblings results is likely to be high.

Table 4 reports the results from the siblings-fixed effects model. Column 1 of the top panel reports the crude estimates of the employment rate differentials, suggesting that unobserved heterogeneity on the family level plays no substantial role for the magnitude of work-limited and disabled individuals' unadjusted employment disadvantages. However, the statistical significance of the estimates is reduced compared to the results regarding the full sample. For non-work limited individuals, the addition of sibling-fixed effects reduces the estimated employment differential substantially and also renders it statistically non-significant.

Adding cognitive ability to the specification (column 2) reduces the employment gap between the disabled and normal-sighted groups by 7% and also renders it statistically non-significant, probably due to a small sample size. For the work-limited group, controlling for cognitive ability reduces the earnings gap by 8%. Also adding the non-cognitive test score to the specification reduces the earnings disadvantage of the work-limited and disabled groups by 17% and 20% respectively, as shown in column 3. Hence, differences in ability between siblings with different levels of VA seem to contribute significantly to their respective probabilities of getting a job. Further, similar to the results for the full sample, the return to cognitive ability decreases dramatically when non-cognitive ability is included.

The results of a specification also including physical capacity, displayed in column 4, leave a statistically significant 3.4% reduction in the employment probability for the work-limited group, hence suggesting a slightly smaller employment penalty than the one estimated for the full sample. Also, although statistically non-significant, the estimated 6.3% employment disadvantage of the disabled group is smaller than the corresponding estimate for the full population.

Table 4: The employment and earnings gaps. Siblings.

	(1)	(2)	(3)	(4)	(5)	(6)
EMPLOYMENT						
Non-work limited	-0.00131	-0.00303	-0.00190	-0.00179	-0.00280	
	(0.00197)	(0.00198)	(0.00198)	(0.00198)	(0.00199)	
Work limited	-0.0442**	-0.0405**	-0.0336*	-0.0331*	-0.0332*	
	(0.0181)	(0.0179)	(0.0178)	(0.0178)	(0.0178)	
Disabled	-0.0813*	-0.0757	-0.0603	-0.0593	-0.0603	
	(0.0467)	(0.0472)	(0.0478)	(0.0478)	(0.0481)	
Cognitive ability		0.0138***	0.00684***	0.00671***	0.00519**	
		(0.00205)	(0.00207)	(0.00207)	(0.00208)	
Cognitive ability^2		-0.000833***	-0.000361*	-0.000354*	-0.000372*	
		(0.000188)	(0.000188)	(0.000188)	(0.000190)	
Non-cognitive ability			0.0335***	0.0332***	0.0322***	
			(0.00282)	(0.00283)	(0.00282)	
Non-cognitive ability^2			-0.00260***	-0.00261***	-0.00256***	
			(0.000256)	(0.000256)	(0.000255)	
Baseline mean	0.963	0.963	0.963	0.963	0.963	
Observations	147,598	147,598	147,598	147,598	147,596	
Δ (%) Non-work limited		-131	-45	-36	-113	
Δ (%) Work limited		8	24	25	25	
Δ (%) Disabled		7	26	27	26	
EARNINGS						
Non-work limited	-0.00540	-0.0262***	-0.0169**	-0.0145*	-0.0175**	-0.0136**
	(0.00777)	(0.00777)	(0.00775)	(0.00776)	(0.00777)	(0.00687)
Work limited	-0.200***	-0.181***	-0.151***	-0.143**	-0.144**	-0.149***
	(0.0572)	(0.0569)	(0.0567)	(0.0567)	(0.0567)	(0.0506)
Disabled	-0.330**	-0.286*	-0.221	-0.200	-0.212	0.0754
	(0.150)	(0.149)	(0.149)	(0.149)	(0.149)	(0.142)
Cognitive ability		0.0253***	-0.00548	-0.00837	-3.11e-05	-0.00397
		(0.00721)	(0.00734)	(0.00735)	(0.00741)	(0.00664)
Cognitive ability^2		0.00284***	0.00456***	0.00473***	0.00336***	0.00163***
		(0.000683)	(0.000688)	(0.000688)	(0.000697)	(0.000624)
Non-cognitive ability			0.0959***	0.0908***	0.0934***	0.0567***
			(0.00891)	(0.00894)	(0.00894)	(0.00806)
Non-cognitive ability^2			-0.00476***	-0.00470***	-0.00508***	-0.00324***
			(0.000850)	(0.000850)	(0.000850)	(0.000763)
Observations	141,967	141,967	141,967	141,967	141,967	133,438
Δ (%) Non-work limited		-385	-213	-167	-224	-152
Δ (%) Work limited		10	25	29	28	26
Δ (%) Disabled		13	33	39	36	77
Health				Yes	Yes	Yes
Years of schooling					Yes	Yes
Occupation-FE						Yes
Sibling-FE	Yes	Yes	Yes	Yes	Yes	Yes

Notes: *, **, and *** denote significance at the 10, 5, and 1 percent levels. All specifications include year of birth-fixed effects. The employment analysis estimates the probability of having a positive labor income during 2003. The outcome variable in the earnings analysis is the logarithm of annual earnings in 2003. The sample used in the earnings analysis includes individuals having a positive labor income during 2003. *Health* includes height, BMI, leg strength, handgrip strength, physical endurance, and indicators of missing records of each of these variables. All ability and health measures stem from enlistment tests performed at age 18 or 19. In the employment analysis, all observations with a predicted employment probability above 1 or below 0 are excluded. Δ (%) is the change in estimates compared to the raw estimate displayed in column 1. Robust standard errors in parentheses.

Similar to the results regarding the full population, including schooling in the specification does not significantly contribute to explaining the employment gaps, suggesting that self-selection into educational categories is not the driving factor behind the employment differentials. The results, displayed in column 5, leaves a statistically significant unexplained employment disadvantage of 3.4% for the work-limited group The corresponding, statistically non-significant, estimates for the non-work limited and disabled groups are 0.3 and 6.3%, respectively.

Turning to the results from the earnings specification, displayed in the bottom panel of Table 4, adding sibling-fixed effects to the model reduces the crude estimates of the earnings gaps slightly for the non-work limited and work limited groups compared to the corresponding estimates for the full population. However, adding controls for cognitive and non-cognitive skills (columns 2 and 3, respectively) reduces the estimates less than the corresponding exercises used on the full population, most likely because of a greater similarity between siblings with regards to these traits. When using the full set of exogenous explanatory variables, as displayed in column 4, the unexplained shares of the earnings gaps are larger than their full population counterparts for all groups, leaving statistically significant earnings penalties of 14% and 1.5% for the work-limited and non-work limited groups, respectively. For disabled workers, the coefficients are significantly greater than their full sample counterparts in all specifications, with a (statistically non-significant) 20% earnings gap conditional on all exogenous explanatory variables (column 4).

Also including schooling in the specification, as shown in column 5, leads to a marginal increase in the unexplained earnings gap for the non-work limited group, suggesting that self-selection into educational categories slightly counteracts the earnings penalties of this group. Column 6 reports the results from a specification also including occupation-fixed effects. For the non-work-limited group, the estimated earnings gap decreases slightly compared to the one reported in column 5. For the work-limited, the estimate instead increases slightly, indicating a 15% earnings disadvantage. Hence, self-selection into occupations does not seem to reinforce the earnings disadvantage of this group. Further, it is worth noting, although the results are statistically non-significant, that the coefficient on the earnings disadvantage of the disabled group is dramatically reduced when controlling for occupation-fixed effects, suggesting that self-selection into occupations could be an important factor behind the earnings disadvantage of the visually disabled. Consistent with our main results, an estimation of Model 4 using six professional categories, based on the type of job tasks performed, as outcomes, suggests relatively small differences in the types of jobs held by non-work limited, work limited, and normal-sighted individuals, respectively. For disabled individuals, the point estimates are large, suggesting a statistically significant 48% underrepresentation of visually disabled workers in jobs focusing on data-intensive or administrative tasks. The remaining point estimates are large but imprecisely measured, indicating a potential underrepresentation of visually disabled individuals in leading positions, sales and personal services, and civil services, and an overrepresentation in the cultural and practical sectors. The estimates are displayed in Table A.3 in the Appendix.

The sibling-fixed effects results point at an important role for unobserved heterogeneity across families. Whereas adding parental income and education to the OLS results seems to explain part of the employment and earnings gaps for the full population, the differences between the OLS and fixed effects results suggest that unobserved variables at the family level operate in the opposite direction. This suggests that individuals with visual impairments are more likely to be raised in family environments creating favorable labor market characteristics in a way not captured by the control variables, resulting in a downward bias in the OLS estimates.[u]

6.2 Robustness checks

So far, my results have suggested that having a work-limiting visual impairment reduces both the probability of finding a job and the earnings of those who do. However, it is possible that the negative association between visual impairment and earnings can be attributed to difficulties finding steady employment rather than to lower wages, for example, if a disproportionally large share of visually impaired individuals are employed in short part time positions or experience short employment spells. To account for this possibility, I re-estimate my preferred earnings specification, including sibling-fixed effects, including only individuals with earnings above SEK 50,000 (approximately 6500 USD) in 2003.[v] Further, a potential source of bias in the results is that some individuals may deliberately attain low test scores in order to avoid military service altogether. It seems reasonable to

[u]Also, if parents reinforce rather than compensate for differences in endowments between visually impaired and normal-sighted children, between-group differences may arise to a greater extent in families where the VA varies between siblings.

[v]This reduces the siblings sample by 6312 individuals, corresponding to 4.4 of the sample.

assume that the test score most affected by this type of bias is the cognitive test score, as this is the test performed with the least amount of direct supervision. To account for this possibility, I re-run the estimations excluding individuals with very low cognitive test scores (those who got 1 or 2 out of 9).[w] Table 5 displays the results from these exercises. The top panel reports the estimates of the earnings gap for individuals with yearly earnings above 50,000, while the bottom two panels report the estimates on the earnings and employment gaps, respectively, for individuals with a cognitive test score of three and above.

As reported in column 1, when using SEK 50,000 as an income restriction, the crude earnings differentials between the vision test score groups decrease dramatically and turn statistically non-significant for the work limited and disabled groups, whereas the estimate for the non-work limited group suggests a small but positive impact. When controlling for cognitive and non-cognitive ability, only very small and statistically non-significant earnings gaps remain for all groups. This result remains stable in all later specifications. An estimation of the amount of employment-based transfers included in the income measure on the VA groups suggests that the inclusion of this type of transfers is not the driving factor behind this finding (see Table A.4 in the Appendix). Rather, it appears as though short part-time positions and short employment spells are indeed important factors behind the earnings differentials.

Restricting the sample to include only individuals with cognitive test scores of three and above does not lead to any major differences in estimates, as suggested by the bottom two panels of Table 5. Hence, an uneven distribution of fakers across the VA distribution does not appear to be a driving factor behind the results.

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[w] This reduces the siblings sample by 14,569 individuals, which corresponds to 9.9% of the sample.

Table 5: Robustness checks.

	(1)	(2)	(3)	(4)	(5)	(6)
THE EARNINGS GAP. EXCLUDING INDIVIDUALS WITH EARNINGS BELOW SEK 50,000.						
Non-work limited	0.0121***	-0.00657	0.000339	0.00146	-0.00357	-0.00210
	(0.00448)	(0.00444)	(0.00442)	(0.00443)	(0.00442)	(0.00407)
Work limited	-0.0209	-0.00978	0.00729	0.0124	0.0123	-0.00374
	(0.0332)	(0.0327)	(0.0325)	(0.0325)	(0.0324)	(0.0300)
Disabled	-0.0399	-0.00175	0.0248	0.0332	0.0207	0.0491
	(0.0921)	(0.0908)	(0.0903)	(0.0903)	(0.0899)	(0.0846)
Observations	135,655	135,655	135,655	135,655	135,655	129,157
Δ (%) Non-work limited		-154	-97	-50	-130	-117
Δ (%) Work limited		53	65	135	159	82
Δ (%) Disabled		96	162	183	152	223
THE EMPLOYMENT GAP. EXCLUDING INDIVIDUALS WITH A COGNITIVE TEST SCORE BELOW 3.						
Non-work limited	-0.000989	-0.00220	-0.00122	-0.00120	-0.00204	
	(0.00200)	(0.00201)	(0.00201)	(0.00202)	(0.00202)	
Work limited	-0.0342*	-0.0337	-0.0294	-0.0294	-0.0289	
	(0.0206)	(0.0206)	(0.0205)	(0.0205)	(0.0204)	
Disabled	-0.125**	-0.123**	-0.115**	-0.115**	-0.116**	
	(0.0579)	(0.0579)	(0.0582)	(0.0582)	(0.0583)	
Baseline means	0.966	0.966	0.966	0.966	0.966	
Observations	133,029	133,029	133,029	133,029	133,029	
Δ (%) Non-work limited		-122	-23	-21	-106	
Δ (%) Work limited		1	14	14	15	
Δ (%) Disabled		2	8	8	7	
THE EARNINGS GAP. EXCLUDING INDIVIDUALS WITH A COGNITIVE TEST SCORE BELOW 3.						
Non-work limited	-0.00341	-0.0233***	-0.0138*	-0.0116	-0.0149*	-0.0124*
	(0.00830)	(0.00831)	(0.00829)	(0.00830)	(0.00830)	(0.00736)
Work limited	-0.218***	-0.214***	-0.192***	-0.184**	-0.188***	-0.216***
	(0.0723)	(0.0720)	(0.0717)	(0.0717)	(0.0716)	(0.0635)
Disabled	-0.361*	-0.329*	-0.275	-0.246	-0.263	0.306*
	(0.189)	(0.188)	(0.187)	(0.187)	(0.187)	(0.178)
Observations	128,430	128,430	128,430	128,430	128,430	120,963
Δ (%) Non-work limited		-583	-304	-240	-337	-264
Δ (%) Work limited		2	12	16	14	1
Δ (%) Disabled		9	24	32	27	185
Cognitive ability		Yes	Yes	Yes	Yes	Yes
Non-cognitive ability			Yes	Yes	Yes	Yes
Health				Yes	Yes	Yes
Years of schooling					Yes	Yes
Occupation-FE						Yes

Notes: *, **, and *** denote significance at the 10, 5, and 1 percent levels. The outcome variable is the logarithm of annual earnings in 2003. All specifications include controls for year of birth-fixed effects and sibling-fixed effects. Specifications including cognitive and non-cognitive ability include both linear and squared measures. The sample includes individuals having a positive labor income during 2003. *Health* includes height, BMI, leg strength, handgrip strength, physical endurance, and indicators of missing records of each of these variables. All ability and health measures stem from enlistment tests performed at age 18 or 19. Δ (%) is the change in estimates compared to the raw estimate displayed in column 1. Robust standard errors in parentheses.

7 Discussion and conclusion

Using unique draft data including the entire population of Swedish males born in 1967-1975, this study is the first one to address the causal impact of various degrees of visual impairment on labor market outcomes. Detailed information on corrected and non-corrected visual acuity (VA) measured during the draft procedure allows me to distinguish individuals whose impairment is unlikely to limit labor productivity from those whose impairment is likely to be work-limiting. Using sibling-fixed effects to control for unobserved heterogeneity at the family level, and exploiting detailed and objective measures of cognitive and non-cognitive ability and physical health allows me to distinguish effects of work limitations from consequences of discrimination against glasses-wearers. Further, I investigate the role of self-selection into educational and occupational categories in forming between-group employment and earnings differentials using Swedish register data.

My findings suggest that the earnings and employment rates of individuals who can achieve normal vision using glasses or contact lenses, and who are thus unlikely to experience any work-limitations due to impairment, are similar to those of the normal-sighted. Hence, I do not find any evidence of (positive or negative) discrimination affecting the labor market outcomes of glasses-wearers. While wearing glasses does not seem to affect labor market outcomes, it appears as though having a minor non-correctable visual impairment does. My results indicate that a VA reduction slight enough to not affect driving eligibility reduces employment probability by 3.4% and lowers average earnings by 14% among the, already positively selected, group that is employed. This is comparable to the ethnic earnings gap of 16% found by Nordin and Rooth (2009), the 16% gender earnings gap (Kumlin, 2007) and the 15% earnings gap between non-European immigrants and natives (Le Grand and Szulkin, 2002) in Sweden. The earnings gap does not appear to be mainly due to lower wages, but rather to be a result of a disproportional amount of part-time positions and short employment spells among individuals suffering from work-limiting visual impairment. The same appears to be true, and the earnings and employment penalties even greater, for individuals whose impairment is severe enough to prevent them from driving, although the estimates are imprecise.[x]

[x]The fact that the disadvantages appear to arise on the employment side also reconciles my findings of non-cognitive skills having a larger impact than cognitive ability for both the employment and earnings gaps with the results found by Lindqvist and Vestman (2011), who show that non-cognitive ability mainly affects labor market outcomes by affecting the possibilities of getting a job,

Part of the differential outcomes can be attributed to between-group differences in observable characteristics, most notably non-cognitive skills, that arise before age 18. These differences may in turn be related to between-group differences in activities at a young age.[y] The ophthalmologic literature reports substantially lower participation rates in sports and outdoor activities among visually impaired children (see Wojciechowski (2011) for an overview). Hence, if non-cognitive ability is built through this kind of activities, children with reduced vision may lag behind their normal-sighted peers with respect to these traits. Conversely, visually impaired children are more likely than others to engage in reading and similar activities (Wojciechowski, 2011). If these activities are especially intellectually developmental, this may enhance cognitive ability and academic achievement among visually impaired individuals relative to their normal-sighted peers.

As the onset of impairment occurs before the late teens for the subjects of this study, they have a chance of taking their impairment into account when making schooling and career decisions. However, controlling for schooling and occupation affects the estimates to a relatively minor extent. Schooling choices seem to have a marginal but equalizing effect on both employment and earnings, suggesting a slight over-investment in schooling among the visually impaired. The effect of occupational selection varies by level of impairment. Whereas selection of visually impaired workers into lower-paying jobs appears to explain part of the earnings gaps for non-work limited and disabled workers, it seems to obscure the earnings disadvantage of work-limited individuals, suggesting that this disadvantage occurs to a greater extent within than between occupational categories for this group.

At least for the work-limited group, the weaker labor market status is not likely to be a consequence of a reduced labor supply due to incentives tied to disability insurance, as individuals belonging to this group are highly unlikely to be eligible for disability benefits due to their visual impairment.[z] Also, the possibility of comorbidity affecting visually impaired individuals' labor supply seems unlikely, as between-group differences in a broad range of indicators of general health and physical fitness appear to play only a minor role in explaining their labor market

whereas wages are more heavily affected by cognitive skills.

[y]This mechanism has previously been put forward by Persico et al. (2004) and Lundborg et al. (2014) as an explanation of the height premium in wages.

[z]Eligibility for Swedish disability insurance is based on individual evaluations of work capacity. The only existing formal guidelines concerning disability benefits for the visually impaired that were valid in 2003 state that only (nearly) blind individuals automatically enjoyed eligibility for benefits due to their impairment (Lag om handikappersättning och vårdbidrag, 8703).

disadvantage. Similarly, although some adaptive measures may be needed for individuals with very low vision to maintain certain jobs, this is unlikely to be the case for the work-limited group, whose VA is only slightly reduced when using refraction. Hence, employers' responses to additional costs of hiring and training visually impaired individuals are unlikely to be a reason for their lower earnings and employment rates. Also, as the need for adaptive measures is likely to be similar regardless of the extent of the work position, employers' costs for workplace modification are unlikely to contribute to the higher occurrence of short employment spells and/or working hours among the visually impaired.

The presence of negative labor market effects that do not depend on ability, health, or family background, that do not work through channels of schooling or occupation, and that arise at low levels of visual impairment, suggests that even a mildly reduced VA directly affects an individual's ability to get and maintain a steady job. The effects are likely to be a result of reduced performance and/or raised reservation wages due to an increasing work effort associated with tasks requiring vision. This finding is consistent with a study by Owsley et al. (2001), who find great differences in the time needed to complete various daily life activities among older adults with various degrees of VA, while few of them lacked the ability to complete these tasks altogether. Further, although individuals belonging to the work-limited group can read regular newsprint at a normal or near-normal speed and perform most working tasks with refraction, this may come at a greater effort, as what Colenbrander (2003) refers to as 'the comfortable reserve', experienced by individuals with normal vision, is reduced or gone. Moreover, and consistent with the findings of an over-investment in schooling among work-limited and disabled individuals, if visual impairment affects the ability to benefit from schooling, differences in productivity due to academic achievement may accumulate from an early age. These differences may not be fully accounted for by the cognitive test score, which focuses more directly on intelligence than on general knowledge.

Taken together, the results of this study underlines the importance of acknowledging the work-limitations of individuals suffering from visual impairment, even at

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Diabetes is known to have side effects that could deteriorate vision if not treated correctly. However, according to Carin Gustavsson, MD, consultant at the eye clinic at Malmö university hospital, the number of diabetics with complications severe enough to affect vision at age 18 or 19 is very small and highly unlikely to affect my results (personal communication May 3rd 2016.)

As Colenbrander (2005) points out, performing at one's threshold level, which would be the case for an individual lacking reserves, can be compared to the type of work performed by a Olympic athlete, maximizing his/her physical capacity.

a relatively minor level, suggesting labor market and school policies targeting such groups. Whether the consequences of other types of impairment follow a similar pattern remains to be investigated.

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Appendix

Table A.1: The employment gap. Probit marginal effects. Full population.

	(1)	(2)	(3)	(4)	(5)	(6)
Non-work limited	-0.0324***	-0.0705***	-0.0459***	-0.0408***	-0.0361***	-0.0497***
	(0.00825)	(0.00845)	(0.00859)	(0.00862)	(0.00865)	(0.00870)
Work limited	-0.352***	-0.278***	-0.152***	-0.143**	-0.138**	-0.147***
	(0.0540)	(0.0544)	(0.0558)	(0.0558)	(0.0559)	(0.0559)
Disabled	-0.527***	-0.434***	-0.264**	-0.244*	-0.230*	-0.252*
	(0.124)	(0.125)	(0.132)	(0.132)	(0.131)	(0.132)
Cognitive ability		0.205***	0.0874***	0.0859***	0.0857***	0.0673***
		(0.00728)	(0.00759)	(0.00761)	(0.00764)	(0.00781)
Cognitive ability^2		-0.0149***	-0.00719***	-0.00699***	-0.00593***	-0.00589***
		(0.000713)	(0.000735)	(0.000737)	(0.000743)	(0.000767)
Non-cognitive ability			0.377***	0.371***	0.358***	0.342***
			(0.00828)	(0.00831)	(0.00837)	(0.00840)
Non-cognitive ability^2			-0.0277***	-0.0281***	-0.0265***	-0.0258***
			(0.000848)	(0.000850)	(0.000858)	(0.000861)
Baseline mean	0.959	0.959	0.959	0.959	0.959	0.959
Observations	443,015	443,015	443,015	443,015	443,015	443,015
Δ (%) Non-work limited		-118	-42	-26	-11	-53
Δ (%) Work limited		21	25	57	61	58
Δ (%) Disabled		18	50	54	56	52
Health				Yes	Yes	Yes
Family					Yes	Yes
Years of schooling						Yes

Notes: *, **, and *** denote significance at the 10, 5, and 1 percent levels. All specifications include year of birth-fixed effects. The outcome variable is the probability of having a positive labor income during 2003. *Health* includes height, BMI, leg strength, handgrip strength, physical endurance, and indicators of missing records of each of these variables. *Family* includes income and years of schooling for the subject's mother and father separately (all measured in 1980) and indicators of missing records of each of these variables. All ability and health measures stem from enlistment tests performed at age 18 or 19. The marginal effects are obtained using the margins command in Stata14. Δ (%) is the change in estimates compared to the raw estimate displayed in column 1. Robust standard errors in parentheses.

Table A.2: The employment and earnings gaps. OLS on siblings sample.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
EMPLOYMENT							
Non-work limited	-0.00253**	-0.00574***	-0.00379***	-0.00346***	-0.00303**	-0.00407***	
	(0.00127)	(0.00129)	(0.00128)	(0.00129)	(0.00129)	(0.00129)	
Work limited	-0.0379***	-0.0296**	-0.0157	-0.0151	-0.0144	-0.0142	
	(0.0138)	(0.0137)	(0.0135)	(0.0135)	(0.0136)	(0.0135)	
Disabled	-0.0539	-0.0456	-0.0235	-0.0223	-0.0229	-0.0247	
	(0.0388)	(0.0393)	(0.0392)	(0.0391)	(0.0390)	(0.0390)	
Cognitive ability		0.0205***	0.00827***	0.00826***	0.00808***	0.00588***	
		(0.00140)	(0.00138)	(0.00139)	(0.00138)	(0.00140)	
Cognitive ability^2		-0.00152***	-0.000668***	-0.000658***	-0.000553***	-0.000482***	
		(0.000126)	(0.000125)	(0.000125)	(0.000125)	(0.000127)	
Non-cognitive ability			0.0500***	0.0498***	0.0484***	0.0469***	
			(0.00208)	(0.00209)	(0.00209)	(0.00208)	
Non-cognitive ability^2			-0.00397***	-0.00399***	-0.00384***	-0.00376***	
			(0.000187)	(0.000187)	(0.000187)	(0.000187)	
Baseline mean	0.963	0.963	0.963	0.963	0.963	0.963	
Observations	147,598	147,598	147,598	147,590	147,554	147,086	
Δ (%) Non-work limited		-127	-50	-37	-20	-61	
Δ (%) Work limited		22	59	60	62	63	
Δ (%) Disabled		15	56	59	58	54	
EARNINGS							
Non-work limited	0.0274***	-0.0209***	-0.00340	-0.00154	-0.00228	-0.00870*	-0.00578
	(0.00527)	(0.00529)	(0.00522)	(0.00522)	(0.00522)	(0.00522)	(0.00445)
Work limited	-0.174***	-0.119**	-0.0523	-0.0439	-0.0414	-0.0437	-0.0509
	(0.0524)	(0.0516)	(0.0517)	(0.0516)	(0.0515)	(0.0515)	(0.0474)
Disabled	-0.418**	-0.375**	-0.275	-0.262	-0.273	-0.279*	0.0196
	(0.181)	(0.181)	(0.171)	(0.169)	(0.169)	(0.168)	(0.0669)
Cognitive ability		0.0565***	-0.00134	-0.00416	-0.00518	0.00229	9.07e-05
		(0.00488)	(0.00495)	(0.00496)	(0.00494)	(0.00501)	(0.00431)
Cognitive ability^2		0.000464	0.00371***	0.00383***	0.00380***	0.00222***	0.000350
		(0.000466)	(0.000467)	(0.000467)	(0.000470)	(0.000481)	(0.000413)
Non-cognitive ability			0.136***	0.131***	0.128***	0.130***	0.0742***
			(0.00711)	(0.00709)	(0.00708)	(0.00708)	(0.00590)
Non-cognitive ability^2			-0.00623***	-0.00628***	-0.00624***	-0.00668***	-0.00362***
			(0.000654)	(0.000652)	(0.000652)	(0.000652)	(0.000547)
Observations	141,967	141,967	141,967	141,967	141,967	141,967	133,438
Δ (%) Non-work limited		-184	-112	-106	-108	-132	-121
Δ (%) Work limited		32	70	75	76	75	71
Δ (%) Disabled		10	34	37	35	33	105
Health				Yes	Yes	Yes	Yes
Family					Yes	Yes	Yes
Years of schooling						Yes	Yes
Occupation-FE							Yes

Notes: *, **, and *** denote significance at the 10, 5, and 1 percent levels. All specifications include year of birth-fixed effects. The employment analysis estimates the probability of having a positive labor income during 2003. The outcome variable in the earnings analysis is the logarithm of annual earnings in 2003. The sample used in the earnings analysis includes individuals having a positive labor income during 2003. *Health* includes height, BMI, leg strength, handgrip strength, physical endurance, and indicators of missing records of each of these variables. *Family* includes income and years of schooling for the subject's mother and father separately (all measured in 1980) and indicators of missing records of each of these variables. All ability and health measures stem from enlistment tests performed at age 18 or 19. In the employment analysis, all observations with a predicted employment probability above 1 or below 0 are excluded. Δ (%) is the change in estimates compared to the raw estimate displayed in column 1. Robust standard errors in parentheses.

Table A.3: Selection into professional categories. Siblings.

	(1) Leaders	(2) Sales, personal services	(3) Civil servants	(4) Culture	(5) Data, administration	(6) Practical, agricultural
Non-work limited	-0.00316	0.00162	-0.00103	0.00292**	0.0162***	-0.0188***
	(0.00251)	(0.00305)	(0.00382)	(0.00144)	(0.00444)	(0.00421)
Work limited	-0.00882	0.00743	0.0156	-0.00564	-0.0379	0.0317
	(0.0145)	(0.0201)	(0.0237)	(0.00743)	(0.0246)	(0.0339)
Disabled	-0.0466	-0.0265	-0.0648	0.0294	-0.118**	0.103
	(0.0442)	(0.0575)	(0.0687)	(0.0296)	(0.0594)	(0.0937)
Cognitive ability	0.00537***	0.0242***	0.0344***	0.00418***	-0.0247***	-0.0348***
	(0.00207)	(0.00266)	(0.00324)	(0.00109)	(0.00371)	(0.00412)
Cognitive ability^2	8.80e-05	-0.00236***	-0.00471***	-0.000184	0.00620***	0.000255
	(0.000215)	(0.000253)	(0.000322)	(0.000116)	(0.000379)	(0.000374)
Non-cognitive ability	-0.0190***	0.000643	-0.0122***	-0.00263*	0.0133***	0.0425***
	(0.00269)	(0.00334)	(0.00416)	(0.00155)	(0.00467)	(0.00505)
Non-cognitive ability^2	0.00288***	0.000761**	0.00183***	0.000186	-0.00149***	-0.00547***
	(0.000282)	(0.000331)	(0.000413)	(0.000150)	(0.000462)	(0.000468)
Means	0.060	0.097	0.154	0.015	0.244	0.443
Observations	141,967	141,967	141,967	141,967	141,967	141,967
Health	Yes	Yes	Yes	Yes	Yes	Yes
Years of schooling	Yes	Yes	Yes	Yes	Yes	Yes
Sibling-FE	Yes	Yes	Yes	Yes	Yes	Yes
SSYK codes included	111-131	341 421-422 511 514 521-522 911	0 222-223 231-235 246 249 322-324 341-342 345 348 513 515	245 347	211-214 241-244 247-248 311-313 343 411-414 419	413 415 512 611-834 912-919 921-933 221 314-315 321

Notes: *, **, and *** denote significance at the 10, 5, and 1 percent levels. All specifications include year of birth-fixed effects. The sample includes individuals having a positive labor income during 2003. *Health* includes height, BMI, leg strength, handgrip strength, physical endurance, and indicators of missing records of each of these variables. All ability and health measures stem from enlistment tests performed at age 18 or 19. Robust standard errors in parentheses.

Table A.4: Transfers. Siblings.

	(1)	(2)	(3)	(4)	(5)	(6)
Non-work limited	-0.426***	-0.412***	-0.392***	-0.365***	-0.330***	-0.317***
	(0.0483)	(0.0486)	(0.0486)	(0.0487)	(0.0487)	(0.0513)
Work limited	-0.758**	-0.761**	-0.682*	-0.592*	-0.585*	-0.936**
	(0.356)	(0.356)	(0.356)	(0.356)	(0.355)	(0.378)
Disabled	-1.241	-1.252	-1.073	-0.949	-0.913	-1.060
	(0.933)	(0.933)	(0.933)	(0.933)	(0.932)	(1.062)
Cognitive ability		0.0672	-0.0133	-0.0361	-0.0106	0.0239
		(0.0451)	(0.0460)	(0.0461)	(0.0465)	(0.0495)
Cognitive ability^2		-0.00970**	-0.00482	-0.00317	-4.47e-05	-0.00135
		(0.00427)	(0.00432)	(0.00432)	(0.00437)	(0.00466)
Non-cognitive ability			0.314***	0.275***	0.294***	0.259***
			(0.0559)	(0.0561)	(0.0561)	(0.0601)
Non-cognitive ability^2			-0.0202***	-0.0207***	-0.0208***	-0.0158***
			(0.00534)	(0.00534)	(0.00533)	(0.00570)
Observations	141,967	141,967	141,967	141,967	141,967	133,438
Δ (%) Non-work limited		3	8	14	23	26
Δ (%) Work limited		0	10	22	23	-23
Δ (%) Disabled		-1	14	24	26	15
Health				Yes	Yes	Yes
Years of schooling					Yes	Yes
Occupation FE						Yes
Sibling FE	Yes	Yes	Yes	Yes	Yes	Yes

Notes: *, **, and *** denote significance at the 10, 5, and 1 percent levels. All specifications include year of birth-fixed effects. The outcome variable is the logarithm of the difference between the earnings measure containing social insurance schemes paid for by the employer and an alternative earnings measure based on salary and income from self-employment only during 2003 (SEK). The sample includes individuals having a positive labor income during 2003. *Health* includes height, BMI, leg strength, handgrip strength, physical endurance, and indicators of missing records of each of these variables. All ability and health measures stem from enlistment tests performed at age 18 or 19. Δ (%) is the change in estimates compared to the raw estimate displayed in column 1. Robust standard errors in parentheses.

Paper III

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CHILD-TO-TEACHER RATIO AND DAY CARE TEACHER SICKNESS ABSENTEEISM†

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ABSTRACT

The literature on occupational health points to work pressure as a trigger of sickness absence. However, reliable, objective measures of work pressure are in short supply. This paper uses Danish day care teachers as an ideal case for analysing whether work pressure measured by the child-to-teacher ratio, that is, the number of children per teacher in an institution, affects teacher sickness absenteeism. We control for individual teacher characteristics, workplace characteristics, and family background characteristics of the children in the day care institutions. We perform estimations for two time periods, 2002–2003 and 2005–2006, by using generalized method of moments with lagged levels of the child-to-teacher ratio as instrument. Our estimation results are somewhat mixed. Generally, the results indicate that the child-to-teacher ratio is positively related to short-term sickness absence for nursery care teachers, but not for preschool teachers. Copyright © 2013 John Wiley & Sons, Ltd.

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KEY WORDS: work pressure; sickness absence; day care

1. INTRODUCTION

Sickness absence is an important cost to society, implying costs related to health care, sickness benefits, substitute employees, and reduced productivity. Moreover, repeated long-term sickness periods depreciate human capital, reduce wages and enhance the risk of leaving the labour force. Sickness absence also harms productivity and labour-market outcomes of colleagues and family of the sick-listed people (Tompa, 2002).

The literature on occupational health points at work pressure as a trigger of sickness absence (Lund *et al.*, 2005). Unfortunately, reliable, objective measures of work pressure are in short supply. This paper uses the day care sector as an ideal case as day care teachers' work pressure can be measured by the child-to-teacher ratio. The child-to-teacher ratio, that is, the number of children per teacher, varies over time, across municipalities and possibly also across institutions within municipalities. The paper investigates the role of the child-to-teacher ratio for the incidence and duration of sickness absence in Danish day care institutions, controlling for background characteristics of the staff, the institution and the municipality. A unique feature of the data is that it links children and day care institutions at the institution level. This allows us to control explicitly for the possible impact of family background of the children in day care on sickness absence of the employees. We exploit the panel dimension of the data both in a within estimation as well as in an instrumental variables approach to account for the possible endogeneity of the child-to-teacher ratio arising from healthy teachers selecting into municipalities with favourable working conditions.

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2. PREVIOUS EVIDENCE

Previous research in the occupational health literature points to work pressure as a trigger of sickness absence. Labriola *et al.* (2006) find that 40% of Danish sickness absence can be attributed to workplace conditions. The work environment may affect sickness absence through a number of different channels (Benavides *et al.*, 2001; Afsa and Givord, 2013; Vahtera and Kivimäki, 2001; Lund *et al.*, 2005). A poor working environment can directly cause illness or stress, enhance employees' liability to catch ordinary diseases such as for instance a cold, or have a demotivating effect.

Lindeboom and Kerkhofs (2000) find strong effects on teacher absence of both observed personal characteristics and school characteristics. Moreover, they confirm the more general finding that peer pressure in teams and 'work culture' affects employee behaviour (Mohnen *et al.*, 2008). Clotfelter *et al.* (2007) find that the incidence of teacher absences in North Carolina is regressive in the sense that schools serving low-income pupils have a higher incidence of sickness absence than schools serving high-income families.

By using Norwegian firm-level data for the 1990s, Ose (2005) finds that physical surroundings in the working area, work strain and 'cultural factors' as for instance cooperation and trust among employees and the closest superior, and the potential for employees to influence their own work situation have a significant effect on firms' average sickness absenteeism. Coles *et al.* (2007) use French employer–employee data to investigate the costs of sickness absence across sectors. They find that production technologies impact the costs of sickness absence, and that the overall costs of absenteeism are low. By using employer–employee data, Barmby *et al.* (1991) conclude that, apart from individual and workplace characteristics, financial aspects – including characteristics of the sick pay scheme – contribute in explaining sickness absenteeism.

3. DATA

The data employed in the empirical analysis is a large micro panel based on administrative registers from Statistics Denmark. Our population consists of day care teachers employed in Danish day care institutions in the period 2002–2006. We focus on this period for two reasons. First, the definition of personnel resources in the official statistics of Statistics Denmark changed around 2001, and this shift has implications for our measure of the child-to-teacher ratio. Second, consistent reporting of long-term sickness absence for public-sector employees is only available from 2000 and onwards. The registry has annual information on around 20 000 day care teachers in nursery and preschool, of which approximately ¼ work in nurseries. These teachers include both assistant teachers and trained day care teachers.[1]

The data contain information on periods of long-term sickness absence. Furthermore, we have access to information on short-term absence for 2005 and 2006 for around 2/3 of the employees. A unique feature of the data is the possibility to identify the children who are enrolled in the specific day care centres. Approximately 53% of the day care teachers can be linked to the institution's child group.[2] On the basis of this subset of the data, we can control also for the family background of the enrolled children as work pressure may be impacted by, for example, social background of the children enrolled in specific day care centres.

4. THE DANISH DAY CARE SECTOR

Danish day care offerings are usually run by the municipalities, and parents pay a user fee per child that accounts for 20–30% of the total cost. Municipalities decide the level of the parents' share, but the maximum

[1]Trained day care teachers have completed a 3½–year pedagogical education. Assistant day care teachers are either unskilled or have completed a 2-year education with a combination of school and practice teaching. On average, trained teachers account for almost 50% of the employees, while assistant teachers amount to almost 40% of all employees in day care institutions. The remaining 10% account for management, kitchen personnel, and so on.

[2]Due to data limitations, it was not possible for Statistics Denmark to link all day care institutions to a workplace.

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parental share is set by law to around 1/3. Day care is directed at preschool children aged ½–6 years. Day care consists of day nursery (childminding), nurseries, preschool and age-integrated institutions (institutions that combine nursery care and preschool in one institution for children aged ½–6 years). We focus on institutions that are either nursery care institutions or preschools.

Municipalities decide the level of the child-to-teacher ratio. Our study calculates the child-to-teacher ratio as number of children per full-time day care employee (teachers and assisting teachers with pedagogical functions). Staff occupied with kitchen duties, cleaning, maintenance and repair are not included in the child-to-teacher ratio. The number of staff is registered by Statistics Denmark in November. Information about the number of children is from Statistics Denmark's survey on day care institutions. Because of a change in reporting of the number of children enrolled in day care institutions, a structural break in the child-to-teacher ratio reporting occurs between 2003 and 2004. Therefore, the analysis is carried out separately for the periods 2002–2003 and 2005–2006.[3] There were no actual changes in, for example, managerial practices. On average, the child-to-teacher ratio in nursery-care institutions was fairly constant at a level of 3–3.5 children per teacher or assistant teacher (Figure 1), and the child-to-teacher ratio in preschool was 6–7 children per teacher over the period 2001–2006 (Figure 2).

5. SICKNESS ABSENCE

The paper uses two measures of sickness absence: long-term absence and short-term absence. As shown in Figure 3, during the period 2001–2006, long-term absence periods became more frequent, thus increasing the share of day care teachers who were sometime during a year affected by long-term sickness (more than 2 weeks). Long-term absence is associated with a higher risk of unemployment and early (disability) retirement.

Most employees are not affected by long-term sickness absence at all in a given year but experience shorter periods of sickness absence, so short-term absence is generally much more widespread than long-term absence. Moreover, short-term absence is costly to firms, reduces productivity and the quality of the service provided in day care institutions. Unfortunately, there is only data for short-term sickness absence for 2005–2006.[4] The average number of short-term sickness absence was around 58 annual hours for nursery teachers and around 49 h per year for preschool teachers.[5]

In Denmark, most employees are by law entitled to a full salary during sickness absence periods. For sickness periods of more than 2 weeks (from 2008 : 3 weeks), the employer is liable to a public (municipality financed) refund of part of the employee's salary corresponding to social sickness benefits (around 2000 Euro per month in 2008). With permission from the employee, the employer can apply for a refund already from the first day and can also request a medical doctor's certificate (sick note) from the employee's GP stating that the employee is indeed ill. Employers have the right to fire workers during sickness absence periods longer than 120 days if this was agreed in the employee's contract. Generally, employers can always lay off an employee if the employer can prove that the employee's sickness absence is of an extent and character that is incomprehensible with fulfilling the employee's work duties. The use of the option of laying off workers with many sick days varies across employers in both the public and the private sector.

[3]In our IV analysis, we use lagged levels of the child-to-teacher ratio as an instrument for the present child-to-teacher ratio. Therefore, the analysis for 2002–2003 uses 2001–2002 child-to-teacher ratio levels as instrument, and for the period 2005–2006, levels of the child-to-teacher ratio for 2004–2005 are used as instruments.

[4]Statistics Denmark kindly made this data available for this study. To our knowledge, this analysis is the first to use individual level information on short-term sickness absence.

[5]These numbers encompass some longer sickness periods.

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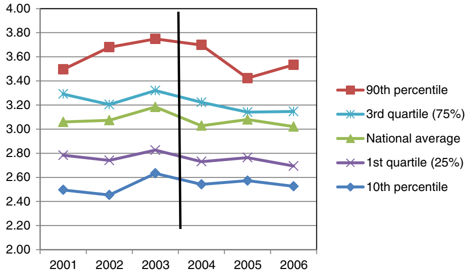


Figure 1. Child-to-teacher ratio in nurseries, 2001–2006

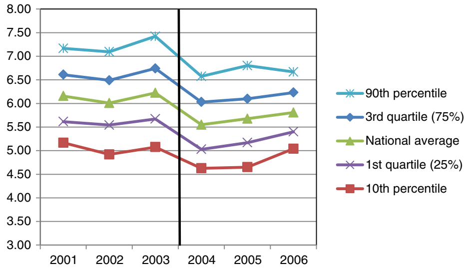
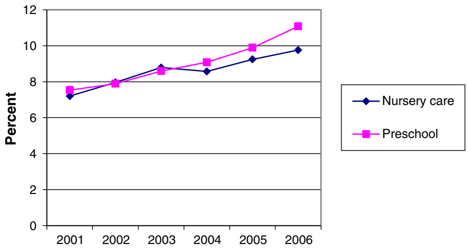


Figure 2. Child-to-teacher ratio in preschool, 2001–2006



Data source: Register data set.

Figure 3. Share of day care teachers affected by long-term sickness

6. EMPIRICAL STRATEGY

6.1. Empirical model

According to Grossman's model of health capital, time lost due to illness reflects a depreciation of health capital that may depend on work characteristics (Grossman, 1972, 2000; Sickles and Taubman, 1986). We formulate

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sickness absence (time lost in the labour market) as a function of municipal level conditions, institution characteristics, individual characteristics, and common national or regional factors. Our dependent variable y_{ijkt} represents teacher sickness absence (measured by number of days or number of hours over a year) for teacher i in workplace j in municipality k, observed in period t:

$$y_{ijkt} = f\left(Z_{it}, S_{jt}, \Pi_{kt}, W_{it}, \eta_i, \phi_t, \varepsilon_{ijkt}\right) \tag{1}$$

Z_{it} represents individual characteristics of teacher i and the local unemployment rate in the municipality of residence. S_{jt} represents institution characteristics (proportion of trained day care teachers in the institution and characteristics of the children) for teacher i in institution j in period t. Π_{kt} is the average child-to-teacher ratio at the municipal level. These explanatory variables are generally state variables observed at the beginning of the year. W_{it} is the individual hourly wage rate. Unobserved heterogeneity at the individual level, η_i, includes, for example, unobserved health characteristics, social competencies, dedication, ability and the individual inclination to report absences when feeling ill. This inclination varies across individuals, across firms and across sectors, depending on, for example, the regional or sectoral unemployment, social norms and attitudes towards absenteeism in the workplace (peer effects), heterogeneity in outdoor facilities, number of square metres, quality of management, unobserved characteristics of children's family background in the institution, proximity to transport opportunities and unobserved variation in employee policies across municipalities. We include municipality dummies to control for municipality fixed effects.

Work pressure may not only depend on the number of children per teacher, but also on the pedagogical challenges in the child group. On the one hand, a high proportion of disadvantaged children may enhance work pressure and therefore induce more sickness absence among staff (directly or through selection). On the other hand, a high proportion of disadvantaged children may promote dedication to work and attract teachers with a high level of work devotion.

Absenteeism varies over the business cycle, as workers may be less worried about losing their job if it is relatively easy to find a job (Shapiro and Stiglitz, 1984). Moreover, absenteeism may rise during an economic boom due to a change in the composition of the work force as the working population will to a larger extent consist of individuals with worse health than in a bust. We control for aggregate time-variant shocks and time trends by using time dummies, ϕ_t. ε_{ijkt} is white noise.

The unobserved individual specific effect in sickness absence may be correlated with some of the explanatory variables. In particular, we speculate that municipalities with favourable working conditions are relatively more successful in attracting teachers with a low propensity to report sick. If this is indeed the case, healthy employees select into municipalities with a low child-to-teacher ratio. Another possible type of endogeneity occurs if municipalities respond to employee sickness absence by reducing the child-to-teacher ratio.

6.2. Estimation techniques

We use a linear specification of the model to ease comparability across different models and to control for municipality fixed effects by including municipality dummies (268 municipalities). These municipality dummies capture variation in working conditions other than the child-to-teacher ratio, for example, senior policies, lay-off policies, sickness policies, and so on across municipalities.

To address selectivity, we consider several alternative estimation strategies, all within a linear setting. One option that we consider and test is to take unobserved heterogeneity at the individual levels into account by exploiting the longitudinal dimension of the data through panel data techniques, allowing for correlation between the unobserved fixed individual heterogeneity and the explanatory variables (including the child-to-teacher ratio). Unfortunately, not only is our panel short (2002–2006), we also have to subdivide our period of analysis into two subperiods, 2002–2003 and 2005–2006, due to the data break mentioned in Section 4. Another drawback when using the within estimator is that it requires that our explanatory variables are strictly exogenous, and this is not necessarily the case for our primary variable of interest, the child-

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to-teacher ratio. We therefore test for strict exogeneity, which is rejected in a number of cases as discussed in the results section (Section 7).

As suggested by, for example, Wooldridge (2003), one may address the problem of having not strictly exogenous (key) regressors by estimating the model by generalized method of moments (GMM), instrumenting for the (endogenous) child-to-teacher ratio by using 1-year lagged levels of the child-to-teacher ratio as an instrument. The argument for using this procedure is that lagged levels of the child-to-teacher ratio are highly correlated with present levels but will not be impacted by potential feedback effects (reverse causality) from higher levels of sickness absence to a lower child-to-teacher ratio. Due to the problem of rejecting strict exogeneity and to the short panel, GMM is our preferred estimation technique.

In the results section, we present ordinary least squares (OLS) and within estimates as a reference, but we stress that these estimates are not consistent in cases where the child-to-teacher ratio is not strictly exogenous.

6.3. Probability of long-term absence

Initially, we analyse the probability of becoming long-term ill. The model for the probability of long-term sickness absence follows a standard linear probability model. In the model, $y = 1$ if an employee has been long-term ill in year t and $y = 0$ if he/she has not been long-term ill. The conditional probability of long-term illness is modelled by:

$$y_{ijkt} = \beta_0 + \beta_1 Z_{it} + \beta_2 S_{jt} + \beta_3 \Pi_{kt} + \beta_4 W_{it} + \eta_i + \phi_t + \varepsilon_{ijkt} \tag{2}$$

6.4. Duration of short and long-term absence

The duration of sickness absence is modelled as in (2). In this case, y signifies duration of sickness absence (measured in days for long-term absence and in hours for short-term absence). As previously mentioned, periods of short-term absence are much more prevalent, and most employees have a positive number of sickness hours in a year.

7. EMPIRICAL RESULTS

We estimate different versions of model (2) discussed in Sections 6.3 and 6.4 separately for nurseries and preschool. Our primary variable of interest is the child-to-teacher ratio, and we control for individual characteristics (age, gender and other personal characteristics), the local unemployment rate, day care institution characteristics, social background of the parents of children in the institutions, year and municipality dummies.[6] Estimations are performed separately for 2002–2003 and 2005–2006 because of the change in the definition of child-to-teacher ratio around 2003–2004 (see Sections 4 and 6.2). Data on short-term sickness absence is only available for the later period (2005–2006). Table I shows summary statistics of the sample. The child-to-teacher ratio is measured by the average number of children per full-time teacher at the municipal level (see Section 4).

There is considerable variation in sickness absence, both across time and within periods across the sample, as shown in Table II.

Estimations are carried out both at the individual (teacher) level and for municipality averages.[7] For the estimation of probabilities, we only use individual data, for the estimation of duration, we use both municipality

[6]Most municipal dummies are not 'effective' in the within-estimation due to (limited) moving across municipalities.

[7]Using aggregated (municipality) data has both pros and cons. On the one hand, aggregating data to the municipality level potentially reduces bias arising from correlation between individual and institution level covariates and variables observed at the municipality level as, for example, the child-to-teacher ratio. Thus, regressions at the municipality level will ideally produce more consistent estimates. On the other hand, exploiting individual variation in the data may produce more efficient estimates. We present both municipality and individual level estimations in the results tables in the paper.

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Table I. Summary statistics

	Nursery care				Preschool			
	Mean	*SD*	Min	Max	Mean	*SD*	Min	Max
Long-term sick days	4.786	19.789	0	180	5.042	20.294	0	180
Short-term sickness hours*	58.730	47.005	1	437.5	50.129	41.696	0	467.5
Child-to-teacher ratio	2.823	0.253	1.61	4.83	5.694	0.807	2.14	10.39
Age	38.385	11.973	18	65	40.206	11.212	18	65
Number of children	0.685	0.942	0	6	0.849	1.021	0	7
Experience	13.316	9.910	0	43	14.233	9.150	0	42.779
Woman	0.941	0.236	0	1	0.897	0.304	0	1
Teacher	0.427	0.495	0	1	0.500	0.500	0	1
Single	0.308	0.462	0	1	0.220	0.414	0	1
Ln net wage	4.078	0.352	−0.806	5.278	4.105	0.346	−0.788	5.291
Unemployment rate	5.720	1.386	2.200	11.500	5.789	1.624	2.200	13.500
Share of trained teachers	0.530	0.142	0	1	0.583	0.164	0	1
Share of male teachers	0.107	0.085	0	1	0.127	0.091	0	1
Share of ethnic minority parents	0.127	0.126	0	0.944	0.101	0.114	0	0.950
Share of parents with short education	0.187	0.044	0.071	0.428	0.195	0.048	0.053	0.456
N	33 398				92 532			

*Short-term sickness absence is only observed in 2005–2006.

Table II. Summary statistics – between and within variation in dependent variables

	Short-term sickness				Long-term sickness			
	Nursery care		Preschool		Nursery care		Preschool	
Overall	58.7	47.0	50.1	41.7	4.8	19.8	5.0	20.3
Between		44.0		39.9		16.6		17.7
Within		19.0		14.6		13.9		14.1

level data and individual level data. Municipality averages correspond to average duration of sickness absence in the municipality as well as municipality averages over the explanatory variables included.

7.1. Probability and duration of long-term sickness absence

We initially analyse the determinants of the probability and duration of long-term sickness periods (Table III for nursery teachers and Table IV for preschool teachers). Parameter estimates of control variables generally show the expected signs. Fully detailed estimation results are available upon request.

In the following, we focus our discussion on the estimation results by using GMM, as GMM is our preferred method of estimation. As discussed in Section 6.2, we also perform estimations by using OLS and the within estimator. The OLS estimates are primarily included to show some simple correlations. The within estimator assumes that explanatory variables are strictly exogenous. Testing for strict exogeneity of the child-to-teacher ratio using the test presented in Wooldridge (2003, Chapter 10), we reject strict exogeneity for nursery care in the early period and for preschool for the late period. This partial rejection of strict exogeneity supports our preference for GMM.

F-values from first-stage statistics (below the parameter estimates for each regression) show that the lagged child-to-teacher ratio is a rather strong instrument in most specifications, except for the municipality level estimation for nursery care for 2005–2006. A rule of thumb has it that the *F*-statistics for the test of the joint significance of the instruments in the first-stage regression should be above 10; this criterion is fulfilled for the majority of the estimations. Moreover, the within estimation suffers from the drawback that strict

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Table III. Long-term sickness absence for nursery teachers

	2002–2003			2005–2006		
	OLS	Within estimator	GMM	OLS	Within estimator	GMM
Individual, probability						
Child-to-teacher ratio	**0.024***	0.031	0.004	**0.029***	**0.045***	−0.006
	(2.41)	(1.03)	(0.18)	**(2.25)**	**(2.50)**	(0.11)
First stage *F*-value			17.2			10.1
R2	0.027	0.023	0.015	0.028	0.019	0.019
N	14 435	14 435	14 435	11 569	11 569	11 569
Individual, number of days						
Child-to-teacher ratio	1.166	2.312	0.965	**2.774***	**4.434*****	1.753
	(1.18)	(1.47)	(0.54)	**(2.59)**	**(3.45)**	(0.70)
First stage *F*-value			17.2			10.1
R2	0.020	0.017	0.013	0.024	0.017	0.017
N	14 435	14 435	14 435	11 569	11 569	11 569
Municipal, number of days						
Child-to-teacher ratio	0.457	2.718	4.683	**2.993****	**6.161**(*)	**6.630****
	(0.43)	(1.16)	(1.09)	**(3.39)**	**(1.87)**	**(2.59)**
First stage *F*-value			4.9			0.9
R2	0.346	0.665	0.271	0.645	0.749	0.577
N	14 435	14 240	14 435	11 569	13 250	11 569

GMM, generalized method of moments.
Estimation controls for teacher gender, ln age, dummy for single, experience, ln net wage, local unemployment rate, share of trained teachers in institution, share of male teachers in institution, share of immigrant parents, share of parents with short education, municipality and year dummies.
Long-term absence is more than 2 weeks in a year.
Standard errors are clustered at the municipality level. *t*-values in parentheses.
Municipality averages are weighted with number of employees in sector in municipality. For the within estimator, weights are calculated in the first year of observation.
(*)$p = 0.10$; *$p = 0.05$; **$p = 0.01$; ***$p = 0.001$.

exogeneity is generally not fulfilled. Consequently, although GMM generally produces insignificant results, GMM is our preferred estimation method.

For nursery care (Table III), we find that there is a positive and significant association between the child-to-teacher ratio and sickness absence when using OLS, but the effects are not significant when accounting for selectivity in the GMM estimation. The only exception is for municipality level averages for the latter period, but given the very low level of the F-statistic in the first stage regression, we do not trust this result.

Turning to the GMM estimates for preschool teachers, we generally find no significant relationship between the child-to-teacher ratio and long-term sickness absence; see Table IV.

We note that estimations of long-term sickness absence measured in days are somewhat different when applying individual and municipality level data, respectively. The difference occurs when individual and institution-level controls are included in the estimation, while a simple estimation including only the municipality level controls gave exactly the same estimates when estimating at the individual and the municipality level, using cluster-robust standard errors and weighting observations in the municipality level estimation with their population weights in municipalities. We suspect that the difference between estimations performed on individual data and municipal data may be attributed to the fact that control variables at the individual or institution level may be endogenous or prone to selection bias. For those estimates that are significant, estimates produced from individual and municipality aggregated data are not that far away from each other. Therefore, we tend to prefer the estimates on the basis of individual data as these are generally more efficient.

We now turn to the results by using within estimation where we find, somewhat surprisingly, a negative association for the periods 2005–2006 between the child-to-teacher ratio. However, as we, as noted, reject strict exogeneity of the child-to-teacher ratio for preschool teachers in the latter period, these estimates are not

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Table IV. Long-term sickness absence for preschool teachers

	2002–2003			2005–2006		
	OLS	Within estimator	GMM	OLS	Within estimator	GMM
Individual, probability						
Child-to-teacher ratio	−0.001	0.003	−0.004	−0.000	**-0.021****	−0.003
	(0.38)	(0.79)	(1.51)	(0.02)	**(3.04)**	(0.68)
First stage *F*-value			166.7			223.7
R2	0.016	0.021	0.008	0.022	0.025	0.013
N	41 119	41 119	41 119	29 367	29 367	29 367
Individual, number of days						
Child-to-teacher ratio	−0.054	0.097	−0.209	−0.149	−0.854	−0.545
	(0.28)	(0.35)	(1.01)	(0.44)	(1.41)	(1.52)
First stage *F*-value			166.7			223.7
R2	0.014	0.021	0.006	0.018	0.021	0.010
N	41119	41119	41119	29367	29367	29367
Municipal, number of days						
Child-to-teacher ratio	−0.197	−0.243	−0.167	**−0.514***	−0.754	−0.670
	(1.37)	(0.72)	(0.71)	**(2.03)**	(1.31)	(1.60)
First stage *F*-value			119.9			182.1
R2	0.134	0.187	0.127	0.176	0.372	0.175
N	41 119	40 976	41 119	29 367	37 462	29 367

GMM, generalized method of moments.
Estimation controls for teacher gender, ln age, dummy for single, experience, ln net wage, local unemployment rate, share of trained teachers in institution, share of male teachers in institution, share of immigrant parents, share of parents with short education, municipality and year dummies.
Long-term absence is more than 2 weeks in a year.
Standard errors are clustered at the municipality level. *t*-values in parentheses.
Municipality averages are weighted with the number of employees in sector in municipality. For the within estimator, weights are calculated in the first year of observation.
(*)$p = 0.10$; *$p = 0.05$; **$p = 0.01$; ***$p = 0.001$.

consistent. The negative association may indeed reflect feedback effects from sickness absence to the child-to-teacher ratio as municipalities may choose to reduce the child-to-teacher ratio to respond to an increase in sickness absence. The same explanation may apply for the negative relationship found when estimating by OLS by using municipality level data. Hence, although these negative estimates are not consistent with our initial hypotheses, they may in fact be attributed to feedback effects from a high level of sickness absence to more personnel resources in terms of replacement teachers that are often hired in a situation with long-term sickness terms. Moreover, many of the individual, institutional and municipality level characteristics that we control for are correlated. We find, for example, that the child-to-teacher ratio is positively correlated with the share of female teachers and negatively correlated with the share of trained teachers, the ethnic background of children in institutions, and the educational background of the parents in the institutions. This suggests that also some of the characteristics that we control for are endogenous because of children's (parents') sorting into institutions with certain qualities.

As we accept strict exogeneity for nursery care in the late period (2005–2006), the result by using the within estimator, which is positive and significant, is in fact consistent for that particular estimation.

As pointed out previously, the child-to-teacher ratio is measured at the municipal level. However, we expect that there is indeed some local variation in the child-to-teacher ratio across institutions in the same municipality. First, the child-to-teacher may be higher than the municipality average in certain institutions to accommodate children with specific needs (diagnoses of autism, ADHD or minor handicaps for which we have no information in the data). We deliberately use the municipality average for the child-to-teacher ratio (which is closer to the municipality 'rule') to avoid having our measure of the child-to-teacher ratio affected by certain institutions serving more children with specific needs. Second, municipalities may try to compensate for institution-based variation in the

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long-term sickness absence among the personnel by hiring replacement teachers (reverse causality) thereby raising the observed child-to-teacher ratio (but not necessarily the effective – or true – child-to-teacher ratio). We have no reason to suspect any systematic correlation between the measurement error and the true child-to-teacher ratio. Thus the estimation is likely to be subject to classical errors-in-variables, creating a downward bias in the OLS estimate of the relationship between the child-to-teacher ratio and sickness absence. Attenuation bias may thus partly explain small and insignificant parameter estimates in the OLS estimation. Using IV can mitigate classical measurement error (Wooldridge, 2002, Chapter 15).

7.2. Short-term sickness absence

We now turn to analysing the relationship between the child-to-teacher ratio and short-term sickness absence (measured in hours per year). While around 10% of the sample experience a long-term absence period sometime during a year, the incidence of short-term periods of absence is much more widespread. On average, teachers in our sample experience 50–60 h per year of short-term sickness absence.

Estimation results in Table V suggest a positive association between the child-to-teacher ratio and short-term sickness absence for nursery care. These results are significant at the 5% level for OLS and significant at the 10% level for GMM when using individual level data. With an F-value of 13.3, the instrument passes the criterion for being a suitably strong instrument. Hence, GMM results for short-term sickness absence suggest a substantial (albeit only marginally significant) increase of 33 h per year (corresponding to a more than 50% increase) associated with a (substantial) increase in the child-to-teacher ratio of 1.

When using municipality aggregate data for nursery care, however, the F-statistic for the first stage regression of the GMM estimation is very low, and GMM produces insignificant estimates. Moreover, there are no significant effects for preschool short-term sickness absence; see Table VI. The results using individual level and municipal level data are rather close in the majority of these estimations.

The fact that we find significant effects for nursery care, but not preschool, is perhaps not that surprising. Intuitively, we would expect the child-to-teacher ratio to be more important for work pressure in nursery care where teachers care for infants and toddlers, while preschool teachers taking care of children aged 3–6 may not to the same extent feel pressed by the number of children per teacher.

Table V. Short-term sickness absence for nursery care teachers

	OLS	Within estimator	GMM
Individual data			
Child-to-teacher ratio	**11.530****	3.339	**33.284**$^{(*)}$
	(2.26)	(0.74)	**(1.76)**
First stage F-value			13.3
R2	0.064	0.065	0.038
N	7335	7335	7335
Municipal averages			
Child-to-teacher ratio	3.661	−10.905	35.297
	(0.97)	(0.60)	(1.58)
First stage F-value			0.1
R2	0.824	0.810	0.67
N	7335	8156	7335

GMM, generalized method of moments.
Estimation controls for teacher gender, ln age, dummy for single, experience, ln net wage, local unemployment rate, share of trained teachers in institution, share of male teachers in institution, share of immigrant parents, share of parents with short education, municipality and year dummies.
Long-term absence is more than 2 weeks in a year.
Standard errors are clustered at the municipality level. t-values in parentheses.
Municipality averages are weighted with number of employees in sector in municipality. For the within estimator, weights are calculated in the first year of observation.
*p = 0.10; *p = 0.05; **p = 0.01; ***p = 0.001.

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Table VI. Short-term sickness absence for preschool teachers

	OLS	Within estimator	GMM
Individual data			
Child-to-teacher ratio	−0.800	0.738	−2.339
	(0.85)	(0.53)	(1.59)
First stage *F*-value			238.4
R2	0.060	0.051	0.023
N	18541	18541	18541
Municipal averages			
Child-to-teacher ratio	−0.759	0.724	−1.77
	(0.62)	(0.47)	(1.25)
First stage F-value			201.0
R2	0.895	0.314	0.393
N	18 541	23 401	18 541

GMM, generalized method of moments.
Estimation controls for teacher gender, ln age, dummy for single, experience, ln net wage, local unemployment rate, share of trained teachers in institution, share of male teachers in institution, share of immigrant parents, share of parents with short education, municipality and year dummies.
Long-term absence is more than 2 weeks in a year.
Standard errors are clustered at the municipality level. *t*-values in parentheses.
Municipality averages are weighted with number of employees in sector in municipality. For the within estimator, weights are calculated in the first year of observation.
(*)$p=0.10$; *$p=0.05$; **$p=0.01$; *** $p=0.001$.

Strict exogeneity of the child-to-teacher ratio is in fact accepted in the short-term regressions, but the results when using the within estimator are not significant.

8. DISCUSSION AND CONCLUSION

This paper analyses the role of work pressure as measured by the child-to-teacher ratio on sickness absence among Danish day care teachers. Teachers may select into institutions/municipalities with favourable work conditions, including a generous child-to-teacher ratio. Thus analysing the relationship between the child-to-teacher ratio and sickness absence by using OLS does not necessarily allow us to make causal interpretations. We consider two modes of dealing with endogeneity: first, we exploit the panel dimension of the data in a within estimation, and second, we use instrumental variable methods with lagged levels of the child-to-teacher ratio as an instrument in a GMM estimation. We reject that the child-to-teacher ratio is strictly exogenous in the estimation of long-term sickness absence for nursery care in the early period (2002–2003) and for preschool in the late period (2005–2006). Thus for these periods, results by using OLS and the within estimator are not consistent.

Estimation results by using our preferred estimation method, GMM, generally indicate that there is no significant relationship between the child-to-teacher ratio and long-term sickness absence. For long-term absence for nursery care teachers in the late period (2005–2006), where we do accept strict exogeneity, the positive and significant result by using the within estimator is in fact consistent.

For nursery teachers' short-term sickness absence, we find a substantial positive albeit only marginally (at the 10% level) significant effect of the child-to-teacher ratio. For preschool teachers, there is no significant association between the child-to-teacher ratio and short-term sickness absence.

In one case, the within estimation for preschool teachers long-term absence in the latter period, we find a negative relationship between the child-to-teacher ratio and long-term sickness absence. However, we speculate that this finding is a result of the child-to-teacher ratio in preschools being not strictly exogenous in the late period (2005–2006), rendering the within estimates inconsistent. Thus the negative association may in fact reflect feedback effects stemming from municipalities or institutions reacting increasing the resources and hence reducing the child-to-teacher ratio as a reaction to an increase in teacher (long term) sickness absence.

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All in all, although most parameter estimates are insignificant, there is some weak indication of a positive and significant relationship between the child-to-teacher ratio and short-term sickness absence for nursery teachers in the late period (2005–2006) when using GMM. Moreover, for nursery teachers' long-term sickness absence, we find for 2005–2006 (where strict exogeneity is accepted) a positive and significant estimate by using the within estimator, but as strict exogeneity is rejected in several cases, we tend to put most emphasis on the GMM estimation results.

In general, our estimation results are thus somewhat mixed. The fact that we find some (weak) indication of a positive and significant relationship between the child-to-teacher ratio and short-term sickness absence for nursery teachers only but not for preschool teachers is perhaps not that surprising. Working conditions are likely to be more severely influenced by the number of children per teacher in nursery care as compared with preschools. Nursery care children, who are usually aged ½–3 years, are much more dependent on constant care and attention by the teachers than the more mature preschool children aged 3–6. Consequently, teachers in nursery care may be more seriously affected by variations in the child-to-teacher ratio than teachers in preschools.

CONFLICT OF INTEREST

There are no conflicts of interest related to any personal or financial relationships affecting the research in this article.

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ETHICS

This research is in accordance with ethical guidelines for research in social sciences. The data used is based on Danish register data, and all guidelines connected to the use of these data have been followed.

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Paper IV

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Income receipt and mortality — Evidence from Swedish public sector employees

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ABSTRACT

In this paper, we study the short-run effect of salary receipt on mortality among Swedish public sector employees. By exploiting variation in paydays across work-places, we completely control for mortality patterns related to, for example, public holidays and other special days or events coinciding with paydays and for general within-month and within-week mortality patterns. We find a dramatic increase in mortality on the day that salaries arrive. The increase is especially pronounced for younger workers and for deaths due to activity-related causes such as heart conditions and strokes. The effect is entirely driven by an increase in mortality among low income individuals, who are more likely to experience liquidity constraints. All things considered, our results suggest that an increase in general economic activity on salary receipt is an important cause of the excess mortality.

1. Introduction

A large and growing literature has established a positive relationship between health and income, showing that mortality and morbidity rates are lower for high-income individuals (see, e.g., Smith, 1999; Deaton, 2003). However, several studies using data from developed countries show that mortality rates follow a pro-cyclical pattern, suggesting that the positive association between income and health does not apply to temporary income changes at the aggregate level.[1]

A possible explanation for this discrepancy is that income receipt has adverse short-run health effects that partly offset the positive long-run association between income and health. In this paper, we consider this possibility by studying the short-run effect of salary payments on mortality among Swedish public sector employees.

Our paper relates to a literature that documents a rise in mortality upon periodic and expected income receipt for specific demographic and socioeconomic groups. A link between income receipt, substance consumption and death is documented by several studies, which find an increase in substance-related mortality and morbidity following income receipt, for example, welfare payments (Verhuel et al., 1997; Riddell and Riddell, 2006; Dobkin and Puller, 2007). In a paper closely related to our study, Evans and Moore (2011) broaden the evidence on the mortality effects of income receipt by showing that the excess mortality is not restricted to welfare clients and drug users, but is also evident in other groups and for various causes of death. Using US data, the authors find a substantial increase in mortality following income receipt (i.e. pay checks and social security checks) among military personnel and social security recipients. Also, investigating the health effects of a one-time income receipt (the 2008 Economic Stimulus Payments), Gross and Tobacman (2014) find a substantial increase in emergency department visits among US residents at all income levels and regardless of health insurance status. The increase is driven by substance-related visits.

Since paydays are not randomly assigned, a simple comparison of mortality rates around salary payments may fail to identify a causal effect for two reasons. First, within-month and within-week mortality patterns due to, for example, habits and health care access may obscure the effect of salary receipt.[2] Second, salary payments may covary with holidays or other days where mortality is exceptionally high or low.

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[1] For example, Ruhm (2000), Neumayer (2004), Tapia Granados (2005), Gerdtham and Ruhm (2006) find evidence of a pro-cyclical mortality pattern. Conversely, Gerdtham and Johannesson (2005) find a counter-cyclical mortality pattern at the individual level among Swedish males.

[2] Such cycles have previously been documented by Evans and Moore (2012) and Phillips et al. (1999).

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0047-2727/

To overcome these difficulties, we combine register data with survey-based information on exact paydays for the entire population of Swedish public sector employees between 1995 and 2000. Exploiting variation in paydays across public sector units, we employ a date-fixed effects strategy, i.e. we include a separate fixed effect for each day, to identify the mortality effect of salary receipt.

Our sample covers approximately 22% of the Swedish work force. This gives us the possibility to both study the mortality effect for a large and heterogeneous population and look into the underlying mechanisms by studying differences in responsiveness between subgroups. We further assess the mechanisms behind the excess mortality by linking information on the causes of death to each deceased individual.

Our findings indicate that the mortality consequences of salary receipt are large. We find a 23% increase in total mortality, corresponding to approximately 96 premature deaths per year if extended to include the entire Swedish working-age population, on the day that salaries are paid. Circulatory conditions are the main reason behind the excess mortality, representing an entire 83% of the increase. The effect is driven by a mortality increase among low-income individuals and is especially pronounced for young workers.

To interpret our findings, we connect our results to several strands of literature. A number of studies provide empirical evidence against the life cycle/permanent income hypothesis (LC/PIH) by showing that households do not smooth consumption, but increase their time-sensitive consumption, such as purchases of perishable goods (e.g., fresh food) and instant consumption items (e.g., restaurant meals and admissions for entertainment events), upon an anticipated income receipt.[3, 4] The increase in consumption has been shown to be greater for young individuals and for households who are likely to experience liquidity constraints, i.e. those who have low incomes or liquid wealth (see, e.g., Stephens, 2006; Johnson et al., 2006; Mastrobuoni and Weinberg, 2009).

If consumption increases upon salary receipt, a temporary rise in activity, due to, for example, an increase in travel and the pursuit of leisure activities, is likely to arise.[5] As previously dicussed by Evans and Moore (2011) and Miller et al. (2009), the raised activity level may cause a short-term increase in mortality due to causes that are activity-related and characterized by a short space of time between onset and death.[6] Consistent with our results, several studies find that deaths due to circulatory conditions exhibit these traits.[7] Additionally, and consistent with our findings, mortality within groups with a higher share of deaths due to acute causes, such as young individuals, should be affected to a relatively greater extent by salary receipt. Conversely, income receipt may relieve economic stress, which could, in turn, reduce mortality. This is supported by a large literature, which documents a strong connection between emotional and financial stress and circulatory conditions (see Steptoe and Kivimäki, 2013 for an overview).

Our results provide new evidence on the mortality effect of periodic and expected income receipt by showing that a large mortality effect is evident for a broad working-age population, and not confined to specific demographic and socioeconomic groups. We also show that a within-month mortality cycle obscures this effect, indicating that the causal effect is greater than previously suggested. Additionally, we provide further evidence against the LC/PIH, suggesting that an increase in economic activity upon income receipt is the main mechanism behind the excess mortality. For our population, a rise in general activity, rather than an increase in health-impeding consumption of specific goods, such as alcohol and illegal substances, appears to be the main cause of the excess mortality.

The remainder of the paper unfolds as follows. Section 2 describes the data. Section 3 discusses our empirical strategy. Section 4 reports our results, including both our main findings on total mortality and results from separate analyses of specific sample subgroups and causes of death. Section 5 compares our findings to estimates of long-run income-related mortality. In Section 6, we discuss our findings.

2. Data

2.1. Payday data

In Sweden, employers have the right to decide which day of the month to pay their employees' salaries. However, in the public sector, paydays are determined at an aggregate level, which means that they vary between (specific) local authorities, counties and the central government (henceforth public sector units), but are shared by all workers within a unit. Using a survey, we collected information on the exact paydays for each unit during 1995 to 2000.[8] We obtained responses from 280 out of 290 local authorities, all 21 counties and the central government.[9]

Paydays vary across units due to differences in regular paydays, rules that apply when this day occurs on a weekend or public holiday, and special rules regarding the December payment. The regular payday occurs on the 25th of each month for central government workers, and varies between the 25th and the 28th for workers employed by local authorities and counties. If the regular payday occurs on a weekend, then some units make salary payments on the preceding Friday. Other units, including the central government, make salary payments on the preceding Friday if the regular payday occurs on a Saturday and on the following Monday if it occurs on a Sunday. Most units pay December salaries before the Christmas holidays, while some occasionally make salary payments after the holidays. Additionally, some units apply specific rules, for example, making salary payments on the second or third last working day of each month.[10]

The differential payment rules generate a great variation in paydays. Table A.1 in the Appendix exemplifies this by reporting the number of workers in our sample who got paid each day in 1995. In April 1995, where the variation was at its lowest, 63.8% were paid on the 27th and 18.4% on 25th, reflecting the most common paydays for local authority and central government employees, respectively. Large shares of workers were also paid on the 24th (10.0%) and 26th (7.8%). The variation was even greater during those months when some of the most

[3] The LC/PIH states that individuals maximize utility by smoothing consumption over time. Thus, an anticipated income receipt should not affect consumption.

[4] See, e.g., Shea (1995), Shapiro and Slemrod (1995), Parker (1999), Souleles (1999), Stephens (2003, 2006), Shapiro (2005), Johnson et al. (2006), Elger (2012), Huffman and Barenstein (2005), Zhang, 2013) and (Stephens and Unayama, 2011) for empirical evidence against the LC/PIH.

[5] An increase in travel could, e.g., be due to shopping. Stephens (2003) also documents an increase in non-shopping activities upon income receipt, finding that the consumption of food away from home and instant consumption items enjoy a greater relative increase than the consumption of, e.g., fresh food to be consumed at home.

[6] Miller et al. (2009) find that the pro-cyclical mortality pattern is stronger among non-working age individuals and that the category of deaths that exhibits the strongest pro-cyclicality among working age individuals is motor vehicle accidents. This suggests that the key mechanisms behind this pattern are not work stress or health behaviors, but mechanisms associated with increased economic activity during economic upturns. Similarly, Evans and Moore (2011) find that the rise in mortality following income receipt is especially pronounced for deaths due to heart attacks and external causes, such as accidents, homicides and suicides, suggesting that the effect is (at least partly) driven by an increase in activity.

[7] An increased risk of onset of circulatory conditions have been connected to various types of activity, e.g., sleep deprivation (Janszky and Ljung, 2008) emotional excitement (Wilbert-Lampen et al., 2008; Carroll et al., 2002; Piira et al., 2012) heavy physical exertion (Albert et al., 2000; Mittleman et al., 1993), eating a heavy meal (Lipovetsky et al., 2004), having sex (Moller et al., 2001), and returning to work after the weekend (Witte et al., 2005; Willich et al., 1994).

[8] Private sector employees are not included in our sample. The reason for this is that information on individual paydays for the entire private sector population (or a representative sample thereof) is extremely difficult to obtain, as paydays are determined at the workplace level.

[9] The local authorities are the main providers of childcare, care for the elderly, and primary and secondary education. The counties primarily provide health care services and public transportation. The central government is responsible for, e.g., the police force and the military and runs the vast majority of universities.

[10] The exact rules for each public sector unit are available from the authors upon request.

common regular paydays occurred on a weekend or holiday, for example December 1995, where the paydays varied between the 21th and 28th.

2.2. Employment records and outcomes

We link the information on unit-specific paydays to individual-level data from three registers. We identify public sector workers through employment records, which link employers and employees on a monthly basis. The register is based on tax records and links each worker to all employers from which (s)he receives any cash transfer. The cash transfers mostly consist of labor income, but also include social insurance schemes paid for by the employer, such as short-term sick leave insurance. Using sector codes and geographical information, we link the employment records to each public sector unit and to our information on paydays. We include workers in our data starting from their second month with the same employer, thus ruling out one-month work-spells. In order to connect each individual to the salary payment that affects him or her the most, we connect workers to employers from the 10th of the month where they receive their salary to the 9th of the following month, i.e. a person employed in unit *x* between January 1st and April 30th is linked to unit *x* between January 10th and May 9th. We further exclude workers with multiple employers, as they may receive salary payments on several occasions each month.

The resulting sample, described in Table 1, consists of 846,916 individuals employed in 298 of the 312 existing public sector units. 55% of the workers are employed by a local authority, while 28% and 16% are employed by a county and the central government respectively. The size of the units varies according to the government level and between individual counties and local authorities. The central government is the largest individual employer, while counties are on average larger than local authorities. The individuals in our sample are of working age (18–66) and heterogeneous.

Our sample resembles the Swedish work force with the exception of male workers being under-represented, amounting to 24% of our sample and 52% of the work force. The share of males differs between the levels; 57% of central government employees, 17% of county employees and 18% of local authority employees are male. The workers are similar in terms of age and family situation across the unit types, while the average income is higher among central government employees than among county and local authority workers.

We further add death records from the *National Causes of Death register* to our data, obtaining a panel with daily information on employment, salary receipt, and mortality for each individual. The death records contain information on date, place, main cause and up to 11 additional contributing causes for each deceased permanent Swedish resident. Using ICD9 and ICD10 codes, we create broad cause of death categories to be analyzed separately. We base these categories on ICD standard classifications, including circulatory conditions, substance-related deaths, external causes, and cancer. We also carry out separate analyses of specific circulatory conditions, i.e. heart conditions and strokes, and specific external causes, including traffic fatalities and suicides. In each category, we include all deaths where at least one of the contributing causes belongs to this category. Table A.2 in the Appendix displays descriptive statistics for each category.[11] A complete list of the ICD codes used for categorization can be found in Table A.3 in the Appendix.

Finally, we add information from the *Population register* (LOUISE), which contains yearly records of a rich set of background variables for the entire Swedish working-age (16–64) population. We use this information to divide our sample into sub-groups based on income, age and sex, to be analyzed separately.

Table 1
Sample statistics.

	All units	Central government	Counties	Local authorities
	(1)	(2)	(3)	(4)
# Units	298	1	24	273
Total # employees	846,916	137,720	241,128	467,922
Average unit size	2842	137,720	10,047	1714
Max unit size	137,720	137,720	32,564	16,945
Min unit size	187	137,720	961	187
With children 0–15	0.35	0.29	0.36	0.36
Male	0.24	0.57	0.17	0.18
Married	0.57	0.54	0.58	0.57
Age <35	0.21	0.22	0.18	0.23
Age 35–50	0.41	0.38	0.45	0.39
Age >50	0.38	0.4	0.36	0.38
Income quartile 1	0.15	0.09	0.13	0.17
Income quartile 2	0.35	0.15	0.34	0.41
Income quartile 3	0.3	.32	0.33	0.28
Income quartile 4	0.21	0.45	0.19	0.15
Daily deaths	0.378	0.448	0.354	0.366

Notes: Daily deaths are daily mortality per 100,000 employees.

3. Empirical strategy

Since all the workers in a public sector unit share the same payday, we aggregate the data. The reason for doing so is purely practical; a data set with one observation per individual-day would be too large to handle. As our dependent variable y_{jt}, we use the daily unit-level mortality per 100,000 employees. Our baseline model for the mortality rate for unit *j* on day *d* in month *s* and year *y* is:

$$y_{jmdy} = \nu_y + \delta_m + \mu_d + \gamma Payday_{jmdy} + \sum_{k=1}^{6} \alpha_k Weekday_{mdy}(k) + \sum_{k=1}^{S} \beta_k Special_{mdy}(k) + \varepsilon_{jmdy} \quad (1)$$

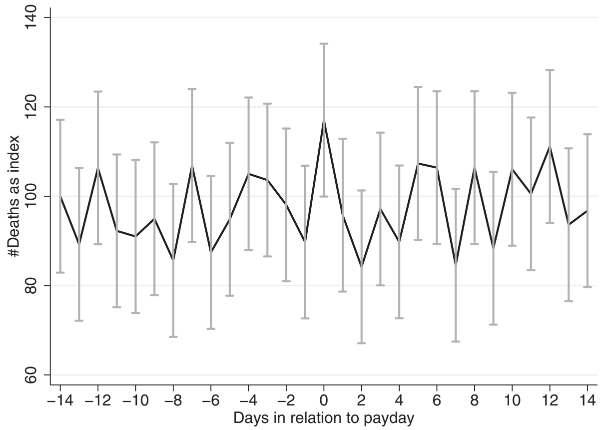
The indicator variable $Payday_{jmdy}$ identifies the day that salaries are paid. Thus, γ captures the mortality effect on this day. However, it is possible that the effect spreads out over the period surrounding a payday. To identify such a case, we include indicator variables capturing the effects on the seven days before and after each payday.

Model (1) includes year- and month dummies (ν_y and δ_m) to control for seasonal variations in mortality. There may also exist, as suggested by Evans and Moore (2011), an independent within-month mortality cycle, which obscures the relation between mortality and salary payments. We control for this possibility using day-of-month fixed effects (μ_d). Further, salary payments are made on weekdays, and occur on a Friday or a Monday when the regular payday is on a weekend. For that reason we use fixed effects for each day of the week ($Weekday(k)$). We also add fixed effects for all public holidays and the two days preceding and two days following each holiday ($Special(k)$).[12] In order to capture regional mortality trends and cycles, we add interactions between the year and month dummies and public sector unit.

Model (1) captures most aggregate changes in mortality correlated with salary payments. However, the between-unit variation in paydays

[11] The annual mortality rate for our sample is approximately 0.1%. We have compared the sample mortality rates within each of our age groups to the mortality rates published by Statistics Sweden for the corresponding age group within the entire Swedish population. For individuals aged 20–35, the annual population mortality rate is 0.075% for males and 0.026% for females, while in our sample the annual mortality rate for this age group is 0.022%. Considering that females constitute 76% of our sample and that this sample only includes employed workers, the differences in mortality rates seem reasonable. We find similar differences for individuals aged 36–50 and 51–66.

[12] The special days include Jan 1st (New Year's Day), Easter (Friday through Monday. Easter Sunday occurs between March 22nd and April 25th), May 1st (Labor day), Ascension Day (a Thursday between April 30th and June 3rd), Whitsuntide (ten days after Ascension Day, Sunday through Monday), Midsummer (a Friday and Saturday between June 20th and 26th), Halloween (in Sweden a Saturday between Oct 21st and Nov 6th), Dec 24th through 26th (Christmas), and Dec 31st (New Year's Eve).



Notes: Mortality rate for Swedish public sector employees, 1995-2000. The mortality on day 14 before payday is standardized to 100. The vertical lined represent 95% confidence bounds for the mortality rate.

Fig. 1. Index for the daily number of deaths by days in relation to payday.

allows us to use a more flexible model, including date-fixed effects, i.e. a separate fixed effect for each day ($Date_{mdy}$). The model

$$y_{jmdy} = Date_{mdy} + \gamma Payday_{jmdy} + \varepsilon_{jmdy} \tag{2}$$

controls not only for reoccurring mortality patterns, but also for additional events that correlate with both mortality and salary payments.

Each observation is weighted by the number of employees in the specific unit. The standard errors are clustered at the public sector unit level, allowing for autocorrelation within the unit mortality rates.

4. Results

A simple plot of mortality rates around payday reveals a sharp, non-lasting peak on the day that salaries arrive (see Fig. 1). This raw increase correspond to a 21% rise in mortality on the day salaries arrive, as shown in Table 2, column 1. In column 2, we add fixed effects for year, month, day of the week and special days. The estimates remain relatively unaffected, suggesting that neither weekday patterns nor annual or monthly variations are a driving force behind the mortality pattern around salary payments.[13]

When adding day-of-month fixed effects to the regression, as shown in column 3 of Table 2, the payday coefficient increases. This suggests that a general decrease in mortality around the time salaries are paid, i.e. at the end of the month, partly offsets the payday effect.[14] Such a mortality cycle may be related to a within-month behavioral pattern. A potential explanation is that most reoccurring monthly payments, such as mortgages and bills, are concentrated at the end of the month. This could mitigate the mortality effects of salary receipt as less money remains for discretionary purposes. This reasoning is in line with the findings of Evans and Moore (2011), who show that mortality in counties with a high proportion of military personnel is more sensitive to a mid-month than an end-of-month military pay check. As displayed in column 4, the results remain unaffected when adding interactions between the year and month dummies and public sector unit.

Our preferred specification, displayed in column 5, adds date-fixed effects to the model. The results reveal a statistically significant 23% increase in mortality on payday, corresponding to 0.66 deaths per payday or 7.92 deaths per year in our sample.[15] Extended to the entire Swedish working-age (18–64) population, a 23% increase in mortality corresponds to 7.98 premature deaths per payday, i.e. nearly 96 deaths per year.[16,17]

In column 6 of Table 2, we use a poisson regression model to estimate the mortality response to salary receipt. For this strategy, we use the total number of deaths in a specific unit on a specific day as our outcome variable and explicitly control for the fact that the number of workers differs across units.[18] This model does not include date-fixed

[13] Fig. A.1 in the Appendix displays the residual mortality controlling for year, month, day of the week and special days.

[14] Fig. A.2 in the Appendix displays this within-month mortality cycle, controlling for payday, various seasonal fixed effects and holiday/special day fixed effects.

[15] Fig. A.3 in the Appendix displays the residual mortality controlling for date fixed effects.

[16] The implications of the results reported above depend partly on whether the excess mortality is mainly due to premature deaths that would not otherwise have taken place, or whether it is a result of harvesting, i.e. a displacement of the deaths of frail individuals by a few days or weeks. If harvesting is the main force behind the excess mortality, the increase in mortality shortly after the income receipt would be offset by a subsequent decline. Since the counterfactual timing of death is unclear, that is, the individual could have died, e.g., two days or two weeks after payday, empirically determining the extent of harvesting is extremely difficult, however. To give some suggestive evidence we have tested for harvesting by estimating the cumulated effects over payday and the six following days. Column 2 of Table A.4 in the Appendix reports the average effect for payday and the following day, column 3 reports the average effect for payday and the two following days, etc. The results show that starting at two days after payday, the cumulated effect is statistically insignificant. This is consistent with harvesting constituting a large share of the excess mortality. However, it is also possible that a zero effect on the days following payday simply blurs out the contribution of the mortality increase on the actual payday to the cumulative estimate even if harvesting is not present. Hence, the extent of harvesting remains inconclusive.

[17] The results are quantitatively similar for units smaller and larger than the median, although a larger variance in daily mortality for smaller units renders the estimates statistically insignificant. This suggests that the mortality pattern is not driven by influential observations such as a small unit with a large number of deaths on a particular day, and thus a very large relative mortality ratio. Table A.5 in the Appendix displays the results from our preferred specification (model (2)) run separately for units smaller and larger than the median.

[18] Specifically, we include log population size with the coefficient restricted to one in our regressions.

Table 2
Estimates of daily mortality per 100,000 employees in relation to payday.

	WLS					Poisson
	(1)	(2)	(3)	(4)	(5)	(6)
Payday − 3 to 7 days	0.00989	0.00412	0.0520**	0.0525**	0.0343	0.0139**
	(0.0140)	(0.0144)	(0.0216)	(0.0217)	(0.0240)	(0.0574)
Payday − 1 to 2 days	−0.0116	−0.0169	0.0403	0.0411	0.00861	0.107
	(0.0173)	(0.0171)	(0.0292)	(0.0296)	(0.0336)	(0.0784)
Payday	0.0778***	0.0744***	0.128***	0.129***	0.0884**	0.323***
	(0.0272)	(0.0285)	(0.0363)	(0.0368)	(0.0399)	(0.0907)
Payday + 1 to 2 days	−0.0267	−0.0306	0.0274	0.0289	−0.0473	0.0694
	(0.0264)	(0.0277)	(0.0352)	(0.0363)	(0.0395)	(0.0983)
Payday + 3 to 6 days	0.0126	0.0159	0.0551**	0.0563**	0.0304	0.145**
	(0.0156)	(0.0160)	(0.0266)	(0.0271)	(0.0361)	(0.0720)
Seasonal FE		Yes	Yes	Yes		Yes
Day of month FE			Yes	Yes		Yes
Year × Unit				Yes		
Month × Unit				Yes		
Date FE					Yes	
Mean	0.378	0.378	0.378	0.378	0.378	0.378
Observations	620,921	620,921	620,921	620,921	620,921	620,921
Public sector units	298	298	298	298	298	298

Notes: Standard errors clustered at the public sector unit level are in parentheses. Columns 1–5 report WLS estimates where each observation is weighted by the number of employees in the specific unit. Column 6 reports Poission regression estimates controlling for the log population rate. Seasonal FE includes dummies identifying year, month, weekday and holidays/other special days. Date FE represents a separate fixed effect for each specific day. The mean is the average daily number of deaths per 100,000 employees. The number of observations is the number of unit-days.
* Significance at the 10% level.
** Significance at the 5% level.
*** Significance at the 1% level.

effects, as a non-linear model with a very large number of fixed effects is too large to handle. The resulting estimates suggest a statistically significant 38% increase in mortality on payday.[19] The estimated effect from the corresponding WLS model (model 3) is 33%.

As an additional robustness check, we drop the days of the week one by one from the model. The results, displayed in Table A.6 in the Appendix, show that a statistically significant payday effect remains when excluding any day of the week except Tuesday.

The inclusion of date-fixed effects yields statistically insignificant coefficients on all days surrounding payday, indicating that the mortality response to salary receipt is immediate.[20] Thus, if the excess mortality is the result of changes in consumption behavior, these changes seem to occur only in the very short run. This finding runs in sharp contrast to Evans and Moore (2011), who find that the mortality level remains elevated for several days after income receipt. The discrepancy could be due to differences in payment methods; during the time period considered, Swedish public sector employers used direct deposit for salary payments, whereas the payments studied by Evans and Moore (2011), i.e. US social security payments and military salaries, were generally made by physical checks. This payment method could potentially delay the behavioral response to income receipt, as individuals have to cash pay checks in order to access their incomes.

Next, we examine the immediate mortality effect displayed in Table 2 in more detail. First, we test for mortality effects during the weekend after salary receipt, by including additional treatment indicators into model ((2)). The results are displayed in Table A.8 in the Appendix. In column 2, we allow for a general effect during the weekend after salary receipt and in column 3 we include an additional interaction effect for weekends when the payday occurred on the preceding Friday. The results from this exercise show no evidence of an increase in mortality during the weekend after salary payments.

Second, we examine whether the mortality effect depends on which day of the week salaries are received. Using Monday as our reference category, we add interactions between the payday indicator and dummies representing the other days of the week (i.e. Tuesday to Friday) to model ((2)). Our results, displayed in Table A.9 in the Appendix, reveal no significant differences in the effect depending on which day of the week salaries are received. This suggests that the mortality response does not vary due to differences in for example habits and access to health care over the course of the week.

These results are somewhat surprising, as workers generally have less opportunities of changing their consumption behavior during weekdays than during weekends. Also, drinking and partying is more likely to take place on Fridays and Saturdays than on any other day of the week. However, earlier studies document an increase in consumption upon income receipt for a broad range of consumption items. Investigating the consumption response to periodic and expected income receipt, Stephens (2003, 2006) find substantial increases in the consumption of instant consumption items, such as admissions for entertainment events, fees for participant sports, etc., food and alcoholic beverages for consumption both at and away from home, and both durable and non-durable goods among UK workers and US social security recipients. Similarly, using Swedish data, Elger (2012) finds an 8%–13% increase in grocery expenditures during the week after salaries are received. These results suggest that the consumption response to income receipt occurs along a broad range of activities that are not necessarily concentrated on weekends.

4.1. Heterogeneous effects by income, age and sex

By studying between-group differences in mortality responsiveness to salary receipt, we assess the mechanisms behind the mortality effect. To this end we use our preferred model including date-fixed effects (i.e. model (2)), using the daily unit-level mortality for each subgroup as our outcome variable.

First, liquidity constraints may pose restrictions on consumption smoothing, which may lead to an increase in activity and a subsequent increase in mortality when salaries are paid. If this is the case, the excess mortality around salary payments is likely to differ across income brackets, with lower-income individuals displaying greater mortality effects. The results presented in Table 3, columns 1 and 2, show that this is the case in our population. We find a statistically significant 35% increase in mortality on the day salaries arrive for workers with below median incomes. Conversely, we find no effect for workers with above median incomes. This is consistent with liquidity constraints contributing to the excess mortality, but may also reflect between-group differences in time preferences.

Second, different-age individuals may respond differently to income receipt due to differences in, for example, current health stocks or the volatility of consumption behavior. The results displayed in Table 3, columns 3–5, show that the excess mortality on payday is substantially higher for younger than for older workers. For the 16–35 age group, mortality increases by a statistically significant 125% on the day salaries arrive, compared to a 29% increase for workers over the age of 50. Conversely, the mortality rate for workers aged 36–50 declines during the days after salaries are received. Our results are in line with the findings in Evans and Moore (2011). Comparing social security recipients to working-age individuals, and comparing different-age social security recipients, the authors find that the association between mortality and income receipt is stronger for younger adults. Evans and Moore (2011) suggest that the differences between age groups reflect more variable activity levels and a larger fraction of deaths due to acute causes for younger individuals. A possible explanation for the decline in mortality on the days following income receipt for 36 to 50-year-old workers is that the relief of economic stress and/or the consumption of health-promoting goods dominate the

[19] The effect in percent is exp(0.323) - 1.
[20] When estimating models (1) and (2) allowing for a separate effect on each of the seven days preceding and following payday, we obtained similar results, with the exception of positive and statistically significant effect three days before payday, see Table A.7 in the Appendix.

Table 3
WLS estimates of daily mortality per 100,000 employees in relation to payday. Total and by subgroup.

	Below median income	Above median income	Female workers	Male workers	Aged 16–35	Aged 36–50	Aged 51–66
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Payday -3 to 7 days	0.0706	0.0152	0.0289	0.0867	−0.0155	0.0462	0.0494
	(0.0659)	(0.0197)	(0.0512)	(0.0764)	(0.0295)	(0.0284)	(0.0569)
Payday -1 to 2 days	0.0266	−0.00111	−0.0296	0.164**	−0.0161	−0.0540	0.0867
	(0.0900)	(0.0239)	(0.0478)	(0.0810)	(0.0329)	(0.0445)	(0.0747)
Payday	0.207**	−0.0146	0.0967*	0.182*	0.0785**	−0.0209	0.215**
	(0.0970)	(0.0328)	(0.0565)	(0.0942)	(0.0398)	(0.0437)	(0.0945)
Payday +1 to 2 days	−0.113	−0.0108	−0.0213	−0.0540	0.0155	−0.0855**	−0.0410
	(0.0975)	(0.0243)	(0.0430)	(0.100)	(0.0429)	(0.0355)	(0.0860)
Payday +3 to 6 days	0.0260	0.0272	−0.00513	0.122	0.0218	−0.0223	0.0933
	(0.0877)	(0.0201)	(0.0346)	(0.0849)	(0.0317)	(0.0263)	(0.0933)
Means	0.587	0.069	0.325	0.537	0.0628	0.194	0.749
Observations	620,890	620,921	620,921	620,890	620,921	620,921	620,921
Public sector units	298	298	298	298	298	298	298

Notes: Standard errors clustered at the public sector unit level are in parentheses. Each observation is weighted by the number of employees in the specific unit. All models include a separate fixed effect for each day. The number of observations is the number of unit-days.
* Significance at the 10% level.
** Significance at the 5% level.
*** Significance at the 1% level.

positive mortality effects of increased activity and unhealthy consumption for this group.[21]

Third, it is possible that mortality responses to salary receipt differ between the sexes, due to differences in, for example, consumption smoothing and risk-taking behavior. However, despite large differences in baseline mortality, we find that the mortality responsiveness to salary receipt is relatively similar for female and male workers, whose mortality rates increase by 30% and 34% on payday respectively (see columns 3 and 4 of Table 3).

4.2. Mortality channels — analysis by cause of death

An additional indication of the mechanisms linking salary receipt to mortality could be obtained by investigating the channels of the mortality effect. If an increase in activity is the underlying reason, the excess mortality is likely to consist of deaths due to activity-related causes characterized by a short space of time from onset to death, such as heart attacks, strokes and external causes, rather than long-term, slowly progressing illnesses.

Table 4 reports the results from model (2), which includes date-fixed effects, run separately for certain cause of death categories. Column 1 is identical to column 4 in Table 2, representing all causes of death. Column 2 presents the results for mortality due to circulatory conditions, such as strokes and heart attacks. We find that mortality due to these conditions increases by 66% on the day salaries are received. The increase corresponds to approximately 83% of the entire mortality response and applies to both heart conditions and strokes, which increase by 66.7% and 118.8% respectively (see columns 3 and 4).[22]

Following previous studies, we also investigate the responsiveness of substance-related mortality to salary receipt. Our results, displayed in column 5 of Table 4, show no evidence of an increase in substance-related mortality upon income receipt. This contradicts the results found by Evans and Moore (2011) for social security recipients and by, for example, Verhuel et al. (1997), Riddell and Riddell (2006) and Dobkin and Puller (2007) for welfare clients and drug users. The discrepancy between the studies could be due to sample differences. As all individuals in our sample are employed, they may be less likely to suffer from grave addiction than the non-employed individuals in earlier studies. Additionally, due to their relatively higher incomes, the individuals in our sample may be less likely than for example welfare clients and retirees to exhibit a substance consumption behavior that is very sensitive to income receipt.

Column 6 shows the estimates for external causes of death, such as accidents, homicides and suicides. These estimates are negative and statistically insignificant, indicating that external causes is not the driving force behind the excess mortality in our sample.

In two cases, we identify significant effects on the days surrounding payday. We find a large and statistically significant increase of traffic fatalities during the week leading up to payday, while no statistically significant effect is evident on payday and during the following week.[23] A possible reason for this is that a reduced amount of traveling before salary receipt reduces congestion and hence increases speed in urban areas, which may lead to an increase in (serious) traffic accidents. However, due to data limitations, we have not been able to look further into this hypothesis. For suicides (column 8), we find a decrease of about 50% during the entire period surrounding payday. A possible explanation is that anticipation effects play a greater role for suicides than for deaths due to somatic conditions. If economic stress contributes to suicide, the suicide rate is likely to decrease during the days leading up to payday, as relief is soon to come.

Finally, we use cancer deaths as a placebo test, as deaths belonging to this category are unlikely to be affected by activity. As expected, we do not find any patterns related to the timing of salary receipt, as shown in Table 4, column 9.

5. Comparison between short- and long-run effects

Our main finding of a 23% increase in mortality following income receipt runs in stark contrast to the well-documented negative long-run association between income and mortality. In this section, we perform a "back-of-the-envelope" calculation, which compares our findings on the

21 Many individuals within the 36–50 age group have young children living at home, causing a strain on the household economy and reducing the possibility of unhealthy consumption patterns. This appears as a credible explanation for the reduction in mortality following income receipt for this group. However, separate estimations of models (1) and (2) on workers with and without 0–15-year-old children do not reveal any significant differences in mortality patterns around income receipt between parents and non-parents.

22 The excess mortality due to circulatory conditions is driven by large increases among workers with below median incomes (63%) and workers above 50 years of age (83%). We do not find any statistically significant effect for high-income workers or for the two younger age groups. These results, which are displayed in Table A.10 in the Appendix, are in line with existing studies, which find a greater incidence of circulatory diseases among lower-income and older individuals (see, e.g., Dawber and Kannel (1958) and Department of Health and Social Security (1980)). Circulatory conditions come through as the main channel of the excess mortality despite its confinement to these groups since they are the groups driving the total effect (mortality among older workers dominate the effect in absolute terms due to its higher mean, although the effect is relatively larger among young workers).

23 A contributing factor to the statistically insignificant result on payday and during the following week could be Sweden's relatively low traffic-related mortality. According to public-use files from the World Health Organization, 6.3 per 100,000 inhabitants died due to road traffic accidents in Sweden during 2002. This can be compared to 15.5 traffic fatalities per 100,000 inhabitants in the USA during the same year (World Health Organization, 2004).

Table 4
WLS estimates of daily mortality per 100,000 employees in relation to payday. Total mortality and by cause of death.

	All deaths	Circulatory conditions	Heart conditions	Strokes	Substance-related	External	Traffic	Suicide	Cancer
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Payday -3 to 7 days	0.0343	0.00711	0.0117	0.00673	0.00231	−0.000529	0.00759**	−0.0161**	0.0346
	(0.0240)	(0.0137)	(0.0120)	(0.00645)	(0.00746)	(0.00976)	(0.00307)	(0.00731)	(0.0219)
Payday -1 to 2 days	0.00861	0.0110	0.0300*	0.00605	−0.00131	−0.00451	0.00887**	−0.0102	0.00320
	(0.0336)	(0.0206)	(0.0172)	(0.00835)	(0.00863)	(0.0112)	(0.00390)	(0.0109)	(0.0263)
Payday	0.0884**	0.0715***	0.0527***	0.0297**	−0.000818	−0.00976	0.00635	−0.0120*	0.0275
	(0.0399)	(0.0261)	(0.0194)	(0.0124)	(0.0107)	(0.0142)	(0.00510)	(0.00729)	(0.0321)
Payday +1 to 2 days	−0.0473	−0.0184	−0.0102	0.0146	−0.00767	−0.0142	0.00492	−0.0146	−0.0329
	(0.0395)	(0.0218)	(0.0182)	(0.00960)	(0.00862)	(0.0114)	(0.00625)	(0.00930)	(0.0306)
Payday +3 to 6 days	0.0304	0.0278	0.0252	0.0117*	−0.000379	−0.00536	0.00512	−0.0172***	0.0124
	(0.0361)	(0.0216)	(0.0188)	(0.00604)	(0.00604)	(0.00813)	(0.00468)	(0.00550)	(0.0264)
Means	0.378	0.109	0.079	0.025	0.025	0.051	0.011	0.024	0.222
Observations	620,921	620,921	620,921	620,921	620,921	620,921	620,921	620,921	620,921
Public sector units	298	298	298	298	298	298	298	298	298

Notes: Standard errors clustered at the public sector unit level are in parentheses. In each specification the number of observations is 620,921. Each observation is weighted by the number of employees in the specific unit. All models include a separate fixed effect for each day. The number of observations is the number of unit-days. In each cause of death category, we include all deaths where at least one of the contributing causes registered belongs to this category. A complete list of the ICD codes used for categorization can be found in Table A.3 in the Appendix.
* Significance at the 10% level.
** Significance at the 5% level.
*** Significance at the 1% level.

short-run effect with estimates of the long-run effect. We construct these estimates by combining results from several studies. The purpose of this exercise is to calculate an approximate total effect of income on health for our population.

Using Swedish data, Eliason and Storrie (2006) exploit plant closures to study the long-run effects of job loss on annual income. The authors find long lasting negative effects; three years after displacement, the total earnings loss for the displaced workers was approximately 9%.[24] In a later study, Eliason and Storrie (2009) investigate the effects of plant closures on long-run mortality rates, using a sample rather similar to that of their 2006 paper.[25] They find that mortality increases by a statistically significant 44% over the first four years after the plant closure for displaced male workers, while for their female counterparts, the effect is nearly zero and statistically insignificant. Combining the estimates from the studies by Eliason and Storrie (2006, 2009), we predict the four-year mortality effect for our sample, which consists of 76% females and 24% males, to 11%.

However, displacement may not only affect mortality through its effect on mean earnings, but also by affecting unemployment and mobility between jobs, occupations, and industries. Displacement may also affect marital stability, fertility, health and social networks, which may in turn affect mortality. Sullivan and von Wachter (2009) examine the relative importance of these channels for US workers, finding that the drop in mean earnings explains 50%–75% of the long-run effect of displacement on mortality.

Assuming that the mean earnings channel is of the same relative importance for Swedish workers, we estimate the long-run effect of income on mortality. From Eliason and Storrie (2006, 2009), we have that displacement leads to an annual income loss of 9% and increases the four-year mortality rate by 11%. If income loss accounts for 50%–75% of this effect, it implies that an income raise equal to a monthly salary (1/12 of annual income) decreases long-run mortality by 5.1%–7.6%. Hence, even though income receipt leads to a sizeable short-run increase in mortality, higher income is probably lifesaving in the long run.

6. Concluding remarks

In this paper, we show that the mortality effects of income receipt are not restricted to certain socioeconomic or demographic groups, but can also be found in a heterogeneous and representative working-age population.

Our results provide further evidence against the LC/PIH for a broad group of individuals, and suggest that the lack of income smoothing has significant adverse health consequences. However, as our entire population gets paid on a monthly basis and we do not have access to data on external income shocks, our data does not allow us to investigate how the size or frequency of income receipt affects the mortality effects. Hence, our analysis does not give any advice on the optimal pay frequency for minimizing these effects. For example, smaller weekly or bi-weekly salary payments may not primarily mitigate the mortality consequences by leading to increased income smoothing, but rather increase mortality by creating further occasions where mortality is elevated.

Rather, the policy recommendations that can be drawn from our findings relate to the staffing of, for example, health care facilities during periods of raised economic activity. Specifically, our findings suggest that an intensified service provision during the end of the month within somatic emergency care and health care facilities focusing on circulatory conditions may decrease the number of premature deaths. Conversely, for psychiatric care facilities, shifting resources away from the period surrounding income receipt and toward mid-month may have a beneficial effect on the suicide rate.

This study also contributes to the reconciliation of the positive (individual and aggregate) long-term relationship between income and health and the pro-cyclical mortality patterns documented in recent studies (Ruhm, 2000, 2003, 2005), Gerdtham and Ruhm (2006), Neumayer (2004), Tapia Granados (2005) and Dehejia and Lleras-Muney (2004)). If an increase in consumption has a negative short-term impact on health for a large share of the population, the increase in economic activity that occurs during an upturn is likely to cause a rise in aggregate mortality. Hence, our findings are consistent with opposite short- and long-term effects of income on health being the explanation of this discrepancy in the health-economic literature.

Our findings also relate to the literature on the gradient between longevity and economic status. For Swedish workers, the probability of survival until age 65 (given survival until age 25) is approximately 5.4 percentage points lower for persons with below average income than for individuals with above average income.[26] An approximate calculation

[24] Eliason and Storrie (2006) do not report separate estimates for men and women, but other studies indicate that the income effects are rather similar for males and females (see, e.g., Jacobson et al. (1993)).

[25] Eliason and Storrie (2009) study workers aged 25–64 who were displaced in 1987 or 1988, while Eliason and Storrie (2006) study 21–50-year-old workers who were displaced during 1987. Both papers use propensity score matching to adjust for differences between workers at closing and non-closing firms.

[26] We divided the Swedish working population into two income groups using employment records from 1998. Using death records from 1999, we predicted the probability of survival until age 65, which is the standard retirement age in Sweden, given survival until age 25 for each income group. The survival rate for individuals with above average income (below average income) is 94.7% (89.3%).

shows that if, as suggested by our results, mortality increases by 35% for individuals with lower than average incomes on each payday, i.e. 12 days each year, while mortality rates remain unaffected by salary receipt for higher income individuals (see Table 3), the excess mortality due to income receipt alone explains approximately 0.09 percentage points or 1.6% of this difference in survival rates. Although only accounting for a modest share of the total income-longevity gradient, this finding points toward a relatively unexplored connection between consumption smoothing behavior and income-health inequalities.

Additionally, the analysis underlines the importance of the time horizon of individual-level studies on the relationship between income and health. As Evans and Moore (2011) point out, the conflicting short and long-term effects require researchers to start measuring the total impact of income on health at the time of receipt, and also make it more difficult to identify a causal link between income and health using exogenous variation in income. Consequently, the distinction between short and long-term effects is likely to be crucial in studies on the income/health relationship at both the aggregate and individual levels.

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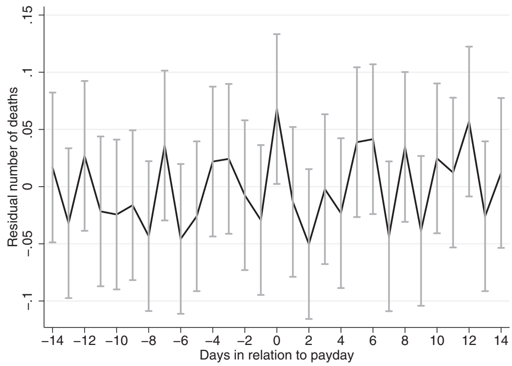
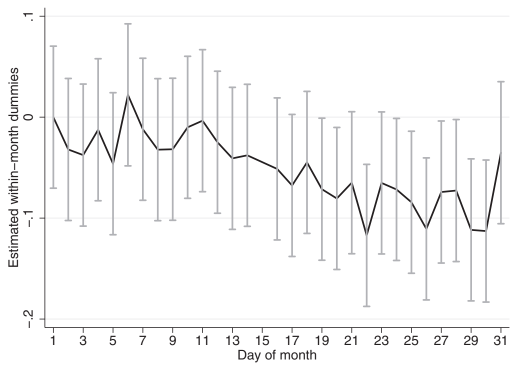
Appendix A



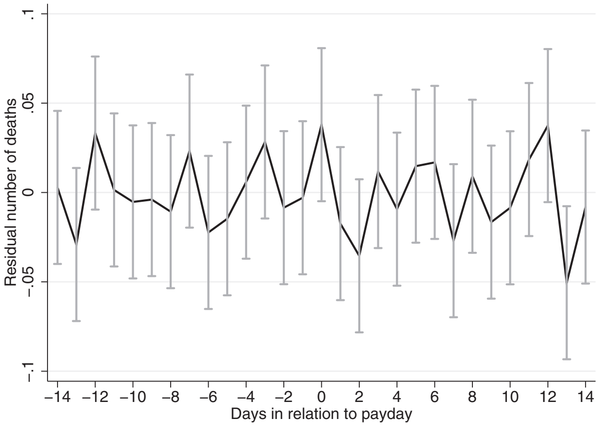
Notes: Residual mortality rate for Swedish public sector employees, 1995-2000, controlling for year, month, weekday and special day fixed effects. The vertical lines represent 95% confidence bounds for the mortality rate.

Fig. A.1. Residual number of deaths in relation to payday. Controlling for year, month, weekday and special days fixed effects.



Notes: Estimated within-month dummies for Swedish public sector employees (1995-2000), controlling for payday effects and year, month, weekday and holiday fixed effects.

Fig. A.2. Estimated within-month dummies controlling for payday effects.



Notes: Residual mortality rate for Swedish public sector employees, 1995-2000, controlling for date fixed effects. The vertical lines represent 95% confidence bounds for the mortality rate.

Fig. A.3. Residual number of deaths in relation to payday. Controlling for date fixed effects.

Table A.1
Number of Swedish public sector employees receiving salary payments on each day of 1995.

Month	Day of month								
	21	22	23	24	25	26	27	28	29
1					128,261	54,344	518,587		
2			527	239,525			432,507	24,810	
3				203,472			501,642	62,052	22,123
4					146,728	80,409	509,689	62,470	
5				145,945		608,737			43,109
6		18,480				202,787	500,579	74,304	
7					144,652	56,663	529,931	61,459	
8					684,073			72,261	38,928
9					146,661	57,359	538,147	62,376	
10					145,790	56,691	595,217		
11				197,924			503,986	81,458	
12	8819	706,972					52,648	6746	

Notes: Own calculations using employment records and the payday survey. No salaries were paid during the 1st through 20th or the 30th through the 31st of the month during this year.

Table A.2
Daily number of deaths. Total and by cause of death. Swedish public sector employees 1995–2000.

	Daily number of deaths	Daily deaths per 100,000 employees
Total deaths	2.94 (1.722)	0.378 (0.222)
Circulatory diseases	0.85 (0.924)	0.109 (0.119)
Heart conditions	0.61 (0.770)	0.079 (0.099)
Stroke	0.20 (0.449)	0.025 (0.058)
Substance-related	0.19 (0.456)	0.025 (0.059)
External causes	0.40 (0.657)	0.051 (0.084)
Traffic fatalities	0.08 (0.296)	0.011 (0.038)
Suicide	0.19 (0.440)	0.024 (0.057)
Cancer	1.73 (1.337)	0.222 (0.172)

Notes: In each cause of death category, we include all deaths where at least one of the contributing causes registered belongs to this category. A complete list of the ICD codes used for categorization can be found in Table A.3 in the Appendix. Standard deviations are in parentheses.

Table A.3
Coding of the cause of death categories.

	ICD9	ICD10
Circulatory	390–459	I00–I99
Heart conditions	390–398, 402, 404, 410–429	I00–I11, I13, I20, I22–I51
Stroke	430–439	I60–I69
Substance related[a]	291–292, 303–304, 305.2–305.9, 357.5–357.6, 425.5, 535.3, 571.0–571.3, 760.7, 779.5, 790.3, 947.3, 962.1, 965, 967–970, 977.3, 980, E850–E858, E860, E863, E935.0–E935.2, E937–E940, E950.0–E950.5, E962.0, E980	F10–F16, F170, F173–F175, F177–F179, F18–F19, F55, G312, G611, G620, G6212, I426, K70, K73–K74, R780, T282, T287, T385, T387, T390–T394, T398–T414, T423–T424, T426–T428, T430–436, T438–T440, T443, T450–T451, T465, T478, T483, T487, T490, T506–T507, T509–T513, T518–T519, X4, Y1, Y430, Y450–Y451, Y453, Y455, Y468, Y478–Y484, Y490–Y502, Y508–Y510, Y513, Y525, Y553, Y557, Y560
External	E870–E869, E880–E929, E950–E999	V01–Y39, Y85–Y98
Traffic	E800–E849	V01–V99
Suicide	E950–E959	X60–X84
Cancer	140–239	C00–C97

[a] The category identifying substance-related deaths is identical to the category used in Evans and Moore (2011, 2012), apart from the exclusion of diagnoses associated with potentially substance-related pregnancy complications and substance-related diagnoses in newborn children. The following ICD codes are excluded in our study, but included in Evans and Moore (2011, 2012); ICD9: 640–641, 6483, 6565; ICD10: O200, O208–O209, O365, O438, O440–O441, O450, O458–O460, O468–O469, O670, O678–O679, O993.

Table A.4
WLS estimates of cumulated mortality effect following payday. Daily mortality per 100,000 employees in relation to payday.

	Payday	Payday and day after	Payday and 2 days after	Payday and 3 days after	Payday and 4 days after	Payday and 5 days after	Payday and 6 days after
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Average effect	0.0884**	0.0639**	−0.00221	0.00602	−0.0289	−0.0142	0.0299
	(0.0399)	(0.0323)	(0.0352)	(0.0380)	(0.0237)	(0.0340)	(0.0316)
Date FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Mean	0.378	0.378	0.378	0.378	0.378	0.378	0.378
Observations	620,921	620,921	620,921	620,921	620,921	620,921	620,921
Public sector units	298	298	298	298	298	298	298

Notes: Standard errors clustered at the public sector unit level are in parentheses. Each observation is weighted by the number of employees in the specific unit. Date FE represents a separate fixed effect for each specific day. The number of observations is the number of unit-days.
* Significance at the 10% level.
** Significance at the 5% level.
*** Significance at the 1% level.

Table A.5
WLS estimates of daily mortality per 100,000 employees in relation to payday. For large and small public sector units.

	All	Small units	Large units
	(1)	(2)	(3)
Payday -3 to 7 days	0.0343	−0.286	0.0410
	(0.0240)	(0.303)	(0.0257)
Payday -1 to 2 days	0.00861	0.239	0.00734
	(0.0336)	(0.404)	(0.0347)
Payday	0.0884**	0.116	0.101**
	(0.0399)	(0.430)	(0.0388)
Payday +1 to 2 days	−0.0473	−1.022*	−0.0388
	(0.0395)	(0.607)	(0.0435)
Payday +3 to 6 days	0.0304	−0.00807	0.0378
	(0.0361)	(0.496)	(0.0383)
Observations	620,921	310,377	310,544
Public sector units	298	149	149

Notes: Small (large) units are all units smaller (larger) than the medium unit size. Standard errors clustered at the public sector unit level are in parentheses. Each observation is weighted by the number of employees in the specific unit. All models include a separate fixed effect for each day. The number of observations is the number of unit-days.
* Significance at the 10% level.
** Significance at the 5% level.
*** Significance at the 1% level.

Table A.6
WLS estimates of daily mortality per 100,000 employees in relation to payday. Robustness analysis dropping the weekdays one-by-one.

	Main	Exclude Mondays	Exclude Tuesdays	Exclude Wednesdays	Exclude Thursdays	Exclude Fridays	Exclude Saturdays	Exclude Sundays
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Payday -3 to 7 days	0.0343	0.0472*	−0.00419	0.0466*	0.0645**	0.0294	0.0307	0.0260
	(0.0240)	(0.0267)	(0.0259)	(0.0254)	(0.0272)	(0.0281)	(0.0276)	(0.0259)
Payday -3 to 7 days	0.00861	0.00243	−0.0350	0.0280	0.0305	0.0134	0.0201	0.00190
	(0.0336)	(0.0381)	(0.0375)	(0.0400)	(0.0368)	(0.0339)	(0.0380)	(0.0352)
Payday	0.0884**	0.0945**	0.0306	0.0776*	0.126***	0.105**	0.0994**	0.0909**
	(0.0399)	(0.0437)	(0.0579)	(0.0419)	(0.0449)	(0.0420)	(0.0421)	(0.0414)
Payday -3 to 7 days	−0.0473	−0.0582	−0.0809*	−0.0633	−0.0293	−0.0350	−0.0277	−0.0350
	(0.0395)	(0.0458)	(0.0441)	(0.0394)	(0.0430)	(0.0439)	(0.0446)	(0.0418)
Payday -3 to 7 days	0.0304	0.00737	0.0208	0.0203	0.0501	0.0428	0.0295	0.0416
	(0.0361)	(0.0475)	(0.0335)	(0.0316)	(0.0307)	(0.0473)	(0.0418)	(0.0398)
Date FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	620,921	532,342	532,050	532,057	532,343	532,340	532,343	532,051
Public sector units	298	298	298	298	298	298	298	

Notes: Column 1 reports our baseline estimate from Table 4. Columns 2–8 each exclude one day of the week. Standard errors clustered at the public sector unit level are in parentheses. Each observation is weighted by the number of employees in the specific unit. Date FE represents a separate fixed effect for each specific day. The number of observations is the number of unit-days.
* Significance at the 10% level.
** Significance at the 5% level.
*** Significance at the 1% level.

Table A.7
WLS estimates of daily mortality per 100,000 employees in relation to payday.

	(1)	(2)	(3)	(4)	(5)
Payday	0.0778***	0.0753***	0.145***	0.146***	0.106**
	(0.0272)	(0.0284)	(0.0410)	(0.0412)	(0.0448)
Payday +1	−0.00436	−0.0129	0.0520	0.0537	−0.0190
	(0.0336)	(0.0338)	(0.0386)	(0.0391)	(0.0499)
Payday +2	−0.0490*	−0.0525*	0.0122	0.0143	−0.0609
	(0.0268)	(0.0288)	(0.0360)	(0.0374)	(0.0454)
Payday +3	0.000107	−0.00176	0.0654	0.0673*	0.0369
	(0.0264)	(0.0273)	(0.0397)	(0.0392)	(0.0523)
Payday +4	−0.0284	−0.0243	0.0140	0.0152	−0.00928
	(0.0235)	(0.0243)	(0.0346)	(0.0352)	(0.0400)
Payday +5	0.0413	0.0434	0.0767*	0.0783*	0.0478
	(0.0310)	(0.0334)	(0.0399)	(0.0410)	(0.0361)
Payday +6	0.0377	0.0472*	0.0636**	0.0649**	0.0499
	(0.0247)	(0.0250)	(0.0294)	(0.0296)	(0.0532)
Payday -1	−0.0276	−0.0274	0.0564	0.0577	0.0313
	(0.0227)	(0.0228)	(0.0434)	(0.0433)	(0.0556)
Payday -2	0.00431	−0.00637	0.0690*	0.0701*	0.0247
	(0.0255)	(0.0251)	(0.0359)	(0.0364)	(0.0391)
Payday -3	0.0256	0.0260	0.0998**	0.101***	0.0967***
	(0.0248)	(0.0254)	(0.0388)	(0.0385)	(0.0330)
Payday -4	0.0309	0.0244	0.0851**	0.0858**	0.0441
	(0.0258)	(0.0264)	(0.0337)	(0.0338)	(0.0362)
Payday -5	−0.00830	−0.0271	0.0380	0.0386	−0.00600
	(0.0238)	(0.0236)	(0.0321)	(0.0321)	(0.0328)
Payday -6	−0.0368	−0.0463*	−0.0137	−0.0132	−0.0261
	(0.0253)	(0.0257)	(0.0309)	(0.0310)	(0.0357)
Payday -7	0.0381	0.0409	0.0805***	0.0808***	0.0575
	(0.0270)	(0.0279)	(0.0308)	(0.0309)	(0.0363)
Seasonal FE		Yes	Yes	Yes	
Day of month FE			Yes	Yes	
Year × Unit				Yes	
Month × Unit				Yes	
Date FE					Yes
Mean	0.378	0.378	0.378	0.378	0.378
Observations	620,921	620,921	620,921	620,921	620,921
Public sector units	298	298	298	298	298

Notes: Standard errors clustered at the public sector unit level are in parentheses. Each observation is weighted by the number of employees in the specific unit. Seasonal FE includes dummies identifying year, month, weekday and holidays/other special days. Date FE represents a separate fixed effect for each specific day. The number of observations is the number of unit-days.
* Significance at the 10% level.
** Significance at the 5% level.
*** Significance at the 1% level.

Table A.8
WLS estimates of daily mortality per 100,000 employees during the weekend after salary receipt.

	(1)	(2)	(3)
Payday	0.0884**	0.0979**	0.0976**
	(0.0399)	(0.0383)	(0.0387)
Weekend after payday			
Payday on Friday same week			0.0101
			(0.0638)
Weekend after payday			
Payday during Mon–Fri same week		−0.0701	−0.0743
		(0.0800)	(0.0941)
Date FE	Yes	Yes	Yes
Mean	0.378	0.378	0.378
Observations	620,921	620,921	620,921
Public sector units	298	298	298

Notes: Standard errors clustered at the public sector unit level are in parentheses. Each observation is weighted by the number of employees in the specific unit. Date FE represents a separate fixed effect for each specific day. "Paid on Friday same week" is an indicator for the weekend after salary receipt given that payday occurred on a Friday. "Paid during Mon–Fri same week" is an indicator for the weekend after salary receipt regardless of which day of the week salary was received. The number of observations is the number of unit-days.
* Significance at the 10% level.
** Significance at the 5% level.
*** Significance at the 1% level.

Table A.9
WLS estimates of daily mortality per 100,000 employees in relation to payday. Heterogeneous effects of a payday occurring on different days of the week.

	(1)	(2)
Payday	0.0884**	0.0469
	(0.0399)	(0.0585)
Payday on Tuesday		0.151
		(0.150)
Payday on Wednesday		0.102
		(0.0765)
Payday on Thursday		−0.0307
		(0.0811)
Payday on Friday		0.0166
		(0.0790)
Date FE	Yes	Yes
Mean	0.378	0.378
Observations	620,921	620,921
Public sector units	298	298

Notes: Standard errors clustered at the public sector unit level are in parentheses. Each observation is weighted by the number of employees in the specific unit. Date FE represents a separate fixed effect for each specific day. The reference day is Monday. The number of observations is the number of unit-days.
* Significance at the 10% level.
** Significance at the 5% level.
*** Significance at the 1% level.

Table A.10
WLS estimates of daily mortality due to circulatory conditions per 100,000 employees in relation to payday. By subgroup.

	Below median income	Above median income	Female workers	Male workers	Aged 16 − 35	Aged 36 − 50	Aged 51 − 66
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Payday -3 to 7 days	−0.00358	0.00695	0.00438	0.0313	0.0174	0.00169	0.00733
	(0.0329)	(0.0123)	(0.0196)	(0.0435)	(0.0111)	(0.0170)	(0.0331)
Payday -1 to 2 days	0.00457	0.00315	0.0186	0.0400	0.0257	−0.0251	0.0417
	(0.0501)	(0.0178)	(0.0196)	(0.0518)	(0.0240)	(0.0197)	(0.0435)
Payday	0.112**	0.0238	0.0749**	0.123*	0.0422	−0.0219	0.189***
	(0.0545)	(0.0246)	(0.0369)	(0.0642)	(0.0290)	(0.0248)	(0.0643)
Payday +1 to 2 days	−0.0818	0.00956	0.00495	−0.0517	0.0144	−0.0159	−0.0388
	(0.0822)	(0.0193)	(0.0212)	(0.0595)	(0.0302)	(0.0171)	(0.0530)
Payday +3 to 6 days	0.0215	0.0200	0.0231	0.0660	0.0152	0.0208	0.0431
	(0.0718)	(0.0143)	(0.0155)	(0.0555)	(0.0160)	(0.0140)	(0.0555)
Date FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Means	0.1792204	0.0392173	0.0128111	0.0475582	0.2268946	0.07510503	0.20993119
Observations	620,921	620,890	620,921	620,890	620,890	620,921	620,921
Public sector units	298	298	298	298	298	298	298

Notes: Standard errors clustered at the public sector unit level are in parentheses. Each observation is weighted by the number of employees in the specific unit. All models include a separate fixed effect for each day. The number of observations is the number of unit-days.
* Significance at the 10% level.
** Significance at the 5% level.
*** Significance at the 1% level.

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