Education, Health, and Earnings – Type 1 Diabetes in Children and Young Adults

Lovén, Ida

2015

Education, Health, and Earnings

Type 1 Diabetes in Children and Young Adults

Ida Lovén

LUND
UNIVERSITY

DOCTORAL DISSERTATION
by due permission of the School of Economics and Management,
Lund University, Sweden.
To be defended at Holger Crafoord EC3:211, Lund
on October 9, 2015 at 10:15.

Faculty opponent
Unto Häkkinen, National Institute of Health and Welfare

Organization Lund University Department of Economics P.O. Box 7082 S-220 07 Lund, Sweden	**Document name** Doctoral dissertation **Date of issue** October 2015
Author Ida Lovén	**Sponsoring organization**

Title and subtitle

Education, Health, and Earnings: Type 1 Diabetes in Children and Young Adults

Abstract

This thesis consists of four independent research papers contributing to the economic literature on child and adolescent health, and adult educational and labor market outcomes. Each paper considers type 1 diabetes as a measure of health and focuses on onset over a specific age span. An overall conclusion of this thesis is that individuals with type 1 diabetes deviate from peers without diabetes, irrespective of which outcome or the timing of onset studied. Though the magnitude of the diabetes related consequences depends on timing of onset and individual characteristics.

The first paper, *Onset of type 1 diabetes in young adults and long-term consequences for annual earnings*, investigates how onset of type 1 diabetes in the age group 28 to 34 relates to adult annual labor earnings over 22 years. First, by focusing on health in young adults (who are generally in the early stages of their careers, but have already made educational choices), I minimize any influence on earnings that otherwise may come from health interacting with education and skill formation during upbringing. Second, by controlling for individual fixed effects, I minimize any influence from time-invariant unobservable factors, such as cognitive and non-cognitive abilities, which have been found to be rather constant throughout adulthood.

The second paper, *Onset of type 1 diabetes in young adults and university education*, assesses how onset of type 1 diabetes at age 17 to 20 relates to subsequent university education and family formation. Unlike children, young adults are themselves responsible for their health behavior and their academic aspirations, while parents' roles are more advisory. Accordingly, the link reflects how type 1 diabetes influences university education when ruling out the influence of earlier academic achievements and minimizing parental involvement.

The third paper, *Labor market consequences of growing up with type 1 diabetes*, explores the long-term labor market consequences of growing up with type 1 diabetes, both for children (6 to 15 years old) developing the disease and their siblings. For siblings of individuals with type 1 diabetes, sisters' outcomes appear unaffected, while brothers' outcomes show, on the one hand, a higher likelihood of being employed, but, on the other hand, lower earnings reminiscent of the earnings decrease for individuals with type 1 diabetes themselves.

The fourth paper, *Early onset of type 1 diabetes and educational field at upper secondary and university level: is own experience an asset for a health care career?*, analyzes the relationship between onset of type 1 diabetes (up to age 15) and the probability of choosing a health-oriented path at upper secondary and university level. By modeling the educational decisions as an unsorted series of binary choices, we shed light on the more qualitative aspects of schooling and assess a potential mechanism linking early life health to adult outcomes.

Keywords

Health, education, earnings, type 1 diabetes, siblings, family formation

Classification system and/or index terms (if any)

JEL Classification: D13, I10, I12, I21, J13, J24, J31

Supplementary bibliographical information	**Language** English
ISSN and key title 0460-0029 Lund Economic Studies no. 189	**ISBN** 978-91-7623-476-1 (print) 978-91-7623-477-8 (pdf)

Recipient's notes	**Number of pages**	**Price**
	Security classification	

Distributed by Department of Economics, P.O. Box 7082, S-220 07 Lund, Sweden

Signature _____ Date _____

Education, Health, and Earnings

Type 1 Diabetes in Children and Young Adults

Ida Lovén

LUND
UNIVERSITY

LUND ECONOMIC STUDIES NUMBER 189

Distributed by the Department of Economics
Lund University
P.O. Box 7082
S-220 07 Lund
SWEDEN

ISBN 978-91-7623-476-1 (print)
ISBN 978-91-7623-477-8 (pdf)
ISSN 0460-0029

Printed in Sweden by Media-Tryck, Lund University
Lund 2015

Contents

Acknowledgment

To write about children and young adults who develop Type 1 diabetes and the changed life circumstances that such a life-long chronic disease brings about has enriched my interest in Economics. Especially when trying to understand the far-reaching impact of health on educational and labor market outcomes. Health affects both existing and future possibilities and balancing different goals in life is hard enough even without the complexities and uncertainties of a chronic disease. Such hardship is affecting not only the children and young adults with the disease but also their friends and family. My goal, to write this thesis, has been influenced by the people around me. I am very much grateful to all of you who have helped, encouraged, and inspired me throughout my work.

My supervisors have always been very helpful and their expertise, comments, and suggestions have surely improved this thesis. I am most grateful to you all. To Carl Hampus Lyttkens for encouraging me to apply to the PhD program. I really appreciate your positive attitude and solution-oriented way of thinking. To Katarina Steen Carlsson for repeatedly placing your trust in me by co-authoring and inviting me to join the Swedish Childhood Diabetes Register Study group and the expert group for the National Guidelines for Diabetes Care at the Swedish National Board of Health and Welfare. Most of all, I appreciate your excellent mix of rationality and empathy and will never forget the heart-warming words you sent me when my first child was born. To Petter Lundborg, I truly appreciate your advice and strategic insights about how to frame a paper.

I am very grateful to Gisela Dahlqusit at the Department of Clinical Sciences in Umeå and all members of the Swedish Childhood Diabetes Register Study group for giving me such a warm welcome to your group, for sharing your knowledge of diabetes, and giving me access to data from SCDR and Statistics Sweden. I am also grateful to Lennarth Nyström at the Department of Public Health and Clinical Medicine, and the Division of Epidemiology and Global Health in Umeå and Mona Landin-Olsson at the Department of Medicine and Clinical Science in Lund for a constructive collaboration and for providing me with the Econ-DISS data. I am much indebted to Terkel Christiansen at Department of Business and Economics at the University of Southern Denmark for valuable comments and suggestions on a thesis draft at my final seminar.

To collaborate with Sofie Gustafsson, co-author of one of the thesis chapters has been a most rewarding experience. Thank you Sofie—you are a true strategist and your excellent cost-benefit analysis of every imaginable choice in life still guides me, both in my professional and personal life. Besides Sofie, I would also like to give special thanks to some other highly altruistic economists and cherished friends: Elvira Andersson, Margareta Dackehag, Lina Maria Ellegård, and Susan Niknami. You always make the benefits outweigh any costs. Many other colleagues (at the department and elsewhere) have contributed to my work and my personal well-being: Tommy Andersson, Karin Bergman, Johan Blomquist,

Jens Dietrichson, Valeriia Dzhamalova, Thomas Eriksson Fast, Ulf Gerdtham, Wolfgang Hess, Ai Jun Hou, Gustav Kjellsson, Lu Liu, Åsa Ljungvall, Kaveh Majlesi, Karl McShane, Caren Yinxia Nielsen, Therese Nilsson, Inga Persson, Sofie Persson, Hilda Ralsmark, Nathalie Stenbeck, Mårten Wallette, Jenny Wate, Anna Welander, and many more. Thank you all for making this an enjoyable time.

I would also like say a few words to all my wonderful friends and family outside academia: I am so fortunate to have one and all of you in my life. My deepest gratitude to my parents Anethe and Stig for always believing in me, to my dear husband Magnus for all your unconditional love and support, and to my darling children Stina and Axel, I love you to the moon and back!

Lund, August 2015
Ida Lovén

Introduction

Introduction

1 Background

This thesis consists of four independent exploratory research papers contributing to the economic literature on child and adolescent health, and adult educational and labor market outcomes. Child and adolescent health is complex and can take many forms. Theoretically, good health is preferable to poor health, because health has a value on its own and facilitates productive use of our time, allowing us to reach other goals in life. Although the papers are individual contributions to the literature, they all consider type 1 diabetes as a measure of health. Each paper focuses on onset over a specific age span and investigates the consequences for either educational or labor market outcomes. Type 1 diabetes is a well-defined disease, with well-documented impacts on everyday life and future health (Sparud-Lundin et al., 2010; Wennick et al., 2009; Wennick & Hallström, 2006). Also, the disease is physician-assessed, and the data on education and labor market outcomes comes from Swedish national population registers, leaving little room for measurement errors and selection bias. Figure 1, an overview

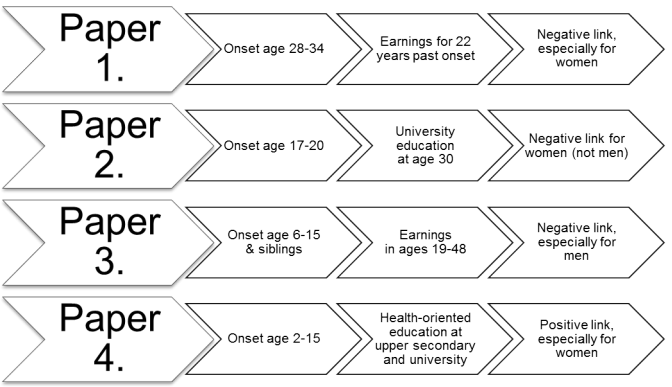

Figure 1: Overview of the four papers included in the thesis.

of the different papers, shows the different onset ages, the outcomes studied, and the main results of each paper. An overall conclusion of this thesis is that individuals with type 1 diabetes deviate from peers without diabetes, irrespective of which outcome or the timing of onset studied. Type 1 diabetes has statistically and quantitatively significant consequences for both education and labor market outcomes, though the magnitude of the impact depends on timing of onset and

individual characteristics. The earnings penalty appears more profound for men than women among those with onset aged six to 15 (Paper 3), whereas women appear to be particularly vulnerable between the ages of 28 and 34 (Paper 1). In terms of education, women appear especially sensitive to onset, both with respect to the likelihood of having a university education (Paper 2) and to the field of education chosen (Paper 4).

1.1 Child and adolescent health and adult outcomes

Childhood and adolescence are formative periods and choices made then may have life-long consequences (Cunha & Heckman, 2008). The choice of educational path is, for example, often decisive for choice of profession and future labor market earnings. To be struck by a health shock early in life may therefore affect adult outcomes. More specifically, first, poor child health may affect future health (see, for example, the fetal origins literature described in Almond & Currie (2011)), which in turn can affect labor supply and productivity (Lundborg, Nilsson, & Rooth, 2014; Johnson & Schoeni, 2011; Smith, 2009; Lindeboom et al., 2006; Case et al., 2005; Currie & Madrian, 1999). Second, poor child health may affect cognition and impair children's ability to learn new skills (Johnson & Schoeni, 2011; Maluccio et al., 2009; Cunha & Heckman, 2008). Third, poor child health may operate via absenteeism in school, affecting educational outcomes (Oreopoulos et al., 2008; Case et al., 2005; Grossman & Kaestner, 1997).

Extensive research confirms the link between child health and adult outcomes for many aspects of health (for reviews, see e.g., Currie & Almond (2011), and Currie (2009)). However, most evidence is based on very early measures of health (e.g., birthweight[1]) or broad measures (e.g., overall health[2] or height[3]) rather than specific diagnoses. At birth, many childhood conditions have not yet emerged, and will not be reflected by, for example, low birthweight. The drawback of broader health measures is the difficulty in knowing what is behind the measure and which mechanisms are in play.

Type 1 diabetes is a well-documented disease with both acute and long-term health complications (Sparud-Lundin et al., 2010; Wennick et al., 2009). High sickness absence, premature retirement, impaired productivity (in school, at work, and at home), and time consuming treatments may cause type 1 diabetes to interfere with individuals' performance and possibilities both in school and on the labor market. Also, childhood health and the circumstances of the entire family are important for children's educational and labor market outcomes (Currie & Almond, 2011; Heckman, 2007). Parents' involvement is crucial for children's

[1] See, e.g., Figlio et al. (2013), Almond & Currie (2011), Royer (2009), Black et al. (2007), Behrman & Rosenzweig (2004).

[2] See e.g., Haas et al. (2011), Smith (2009), and Smith (2007).

[3] See, e.g., Lundborg, Nystedt, & Rooth (2014), and Case & Paxson (2008).

diabetes management, as the burden of managing the disease optimally and maintaining the regularity of routines is a great challenge for the whole family (Wennick & Huus, 2012; Wennick et al., 2009; Wennick & Hallström, 2006). Still, the responsibility of managing diabetes and the focus on a healthy lifestyle are likely to give individuals with diabetes (and their families) useful experience and skills, favoring both educational and labor market outcomes. Thus, consequent educational and labor market impacts of type 1 diabetes might be both positive and negative. This thesis aims to capture the overall impact of type 1 diabetes and encourages future research to assess the different mechanisms at play.

1.2 Type 1 diabetes

Second to Finland, Sweden has the highest annual incidence of type 1 diabetes in the world (Dahlquist et al., 2011). Type 1 diabetes usually develops in childhood or adolescence and is more common in boys. The mean yearly incidence in Sweden between 1983 and 1998 was 28.4/100,000 boys and 27.6/100,000 girls aged zero to 14. The corresponding incidence among those aged 15 to 34 was 16.1/100,000 men and 9.1/100.000 women (Pundziute-Lyckå et al., 2002). Onset of type 1 diabetes can be very dramatic and the symptoms can develop rapidly (over a few days or weeks), as the body stops producing the insulin needed to maintain normal blood glucose levels (Daneman, 2006). Type 1 diabetes requires lifelong disease management with several daily insulin injections and the monitoring of meals, exercise, and blood glucose levels (Wennick et al., 2009). Insulin does not cure the disease but prevents it from causing ongoing damage to the body. Untreated type 1 diabetes can lead to death, while difficulties in achieving glucose control can lead to blindness, heart problems, strokes, nerve damage, amputation, and kidney failure. The choice of insulin regimen is complex and depends on many factors. Technological enhancements (the introduction of take-home blood glucose measures, long-acting insulin, and insulin pumps) have enabled diabetes patients to integrate their diabetes management even further into everyday life. Thereby, children, together with their families (rather than the medical profession), are responsible for daily diabetes management and future health (Wennick et al., 2009).

The heredity of type 1 diabetes is low and more than 90% of newly diagnosed children in Sweden have no close relative with the disease (Dahlquist & Mustonen, 2000). Despite extensive research, the exact combination of environmental and genetic factors, together with the chain of events triggering type 1 diabetes onset, remains unclear. Lifestyle factors (i.e., obesity and physical inactivity) associated with low education (Devaux & Sassi, 2013; Cutler et al., 2003; Molarius, 2003; Molarius et al., 2000; Lissner et al., 2000; Lahmann et al., 2000) do not appear to affect lifetime onset risk (American Diabetes Association, 2008). Factors outside individuals' control, such as genetics, cold climate, and virus infection in

early life, are more likely to be at play (Dahlquist et al., 2011, 2005; Soltesz et al., 2007; The TEDDY Study Group, 2007; Atkinson & Eisenbarth, 2001).

Due to its complexity and sudden onset, type 1 diabetes is generally seen as a health shock that the individual is unable to influence or anticipate (Persson et al., 2013; Minor, 2011; Steen Carlsson et al., 2010). This characteristic is favorable from a methodological perspective because it reduces the need to control for confounding. That is, to control for observable and unobservable factors related to individuals' behaviors and lifestyle choices, which might contribute to disease onset or make individuals respond to a perceived risk of developing type 1 diabetes before onset.

1.3 Previous research on type 1 diabetes and adult outcomes

This thesis confirms previous economic research indicating that individuals with type 1 diabetes have adverse educational and labor market outcomes. Based on the same Swedish register data I use in this thesis, children with onset of type 1 diabetes between the ages of zero and 14 have lower grades from compulsory education (Persson et al., 2013; Dahlquist et al., 2007), and theoretical upper secondary programs preparing for university (Persson et al., 2013), and have a higher risk of unemployment later in life (Persson et al., 2013). Also, adults with onset of type 1 diabetes aged 15 to 34 have a higher risk of unemployment and lower annual labor earnings (Steen Carlsson et al., 2010). Using Swedish enlistment data at age 18 but without stating the type of diabetes studied, Lundborg, Nilsson, & Rooth (2014) reported negative earnings penalties later in life due to diabetes. Minor (2013, 2011) studied both type 1 and type 2 diabetes and concluded that the association between diabetes and labor market outcomes is driven by type 2 diabetes, as no significant association for type 1 diabetes was found. Using US survey data, Minor (2013, 2011) assumes that individuals have type 1 diabetes if they report having diabetes before the age of 20. Though both reasonable and well justified, this assumption implies a high risk of measurement errors due to the fact that incidences of type 1 diabetes also occur in older ages (Dahlquist et al., 2011; Daneman, 2006).

Most other previous studies on diabetes and economic outcomes depend on small-sample surveys (Milton et al., 2006) or cannot discriminate between type 1 and type 2 diabetes[4], likely due to data limitations (Ploug, 2013; Fletcher & Richards, 2012; Maslow et al., 2011; Brown et al., 2010; Latif, 2009; Zhang et al., 2009; Harris, 2008; Brown et al., 2005; Tunceli et al., 2005; Vijan et al., 2004; Bastida & Pagán, 2002).

[4]The two types of diabetes have fundamentally different pathogenesis and expected impact on educational and labor market outcomes. Type 2 diabetes often develops late in life and its onset is driven by lifestyle factors, placing great demands on the data to control for confounding.

2 The contribution of the papers in this thesis

This thesis expands on previous results on type 1 diabetes and adult outcomes by (1) exploring onset at different ages, (2) accounting for changes in diabetes-related consequences over time, (3) considering selection on time-invariant individual-specific unobservable factors, (4) allowing for heterogeneity across socioeconomic groups, (5) studying siblings of individuals with type 1 diabetes, and (6) introducing an alternative outcome, namely the choice of educational field.

2.1 Type 1 diabetes onset in children and young adults

By focusing on onset at different ages I can explore different aspects of the many consequences of type 1 diabetes. Compared to children, young adults have wider responsibilities for their health and education, as parents take on a more advisory (and less governing) role as their children grow older. Therefore, the link between young-adulthood health and educational or labor market outcomes reflects young adults' own decision-making processes, while the link between childhood health and adult outcomes mirrors, in part, parental involvement (Currie & Almond, 2011). Furthermore, early education is important for subsequent academic achievements, and education, per se, affects labor market outcomes.

Studying labor market earnings, Paper 1 focuses on onset at ages when education is already finalized (28 to 34) to minimize any influence on earnings that otherwise may come from health interacting with education and skill formation during upbringing. Paper 3 focuses on childhood onset (ages six to 15) and captures both the direct effect of type 1 diabetes on earnings and the effect via education. Studying education, Paper 2 focuses on onset when individuals are facing the choice of entering the labor market or continuing onto higher education (ages 17 to 20) to ensure that the differences between the studied individuals relates to diabetes and not to other prerequisites for higher education. Paper 4 explores how type 1 diabetes relates to specific education and subsequent careers by studying the choice of educational field.

2.2 Changes over time

Type 1 diabetes involves both acute and more long-term complications (Daneman, 2006). At onset, individuals generally go through a period of severe illness while facing the fact that they have been diagnosed with a chronic disease. Parents of children developing diabetes generally describe onset as a time of crisis, as it is a great challenge for the whole family to learn the child's daily management routines and to accommodate the family's everyday routines within the require-

ments imposed by the unexpected and acute onset of the disease (Wennick et al., 2009; Wennick & Hallström, 2006). The most common acute complication, hypoglycemia, occurs when there is a mismatch between insulin, food, and exercise. Severe episodes of hypoglycemia can lead to permanent brain damage, less severe to transient effects on attention and memory. The cumulative incidence of hypoglycemic episodes and the risk of developing long-term complications increase over time (Daneman, 2006; Groupa et al., 1994). Therefore, only studying average diabetes effects, at a specific point in time, might mask significant heterogeneity throughout individuals' working lives. The studies on earnings account for changes in potential earnings penalties over time and allow the estimates to vary by time from onset (Paper 1) or by age (Paper 3).

2.3 Time-invariant individual-specific effects

Researchers argue that abilities (e.g., IQ, motivation, and other such personal traits and characteristics) may confound child health and adult outcomes as estimated effects are reduced when controlling for cognitive and non-cognitive ability (e.g., Lundborg, Nystedt, & Rooth (2014), and Case & Paxson (2008)). If ability affects type 1 onset (so that individuals with diabetes, as a group, have lower abilities), then the diabetes estimates will absorb the negative influence on earnings from having lower abilities when not controlled for. Such confounding seems reasonable for many health conditions driven by individuals' behavior and lifestyle. However, there is no support for confounding for type 1 diabetes in medical or epidemiological literature (e.g., lifestyle factors appear unrelated to onset (American Diabetes Association, 2008)). Nor can I find any indication in the data of ability differences between individuals that will develop type 1 diabetes and their peers: no descriptive differences in level of education before onset or in parental level of education and no significant predictor of type 1 diabetes (apart from being born outside of the Nordic countries) when I do regressions on the probability of being in the diabetes group.

Moreover, ongoing research on the genetics of cognition and social science debates the existence of associations of specific genes with psychological and behavioral traits such as cognitive ability. Findings are mixed, suggesting that the genetics of complex behavioral traits is far more ambiguous than that of complex physical traits and any effects are small (e.g., Rietveld et al. (2013), and Chabris et al. (2012)). Thus, the risk of potential bias due to genetic factors ought to be low given the low heredity of type 1 diabetes. Also, findings by Lundborg, Nilsson, & Rooth (2014) contradicts confounding, showing that men's earning penalties from diabetes (at age 18) is robust to sibling fixed effects and unobserved factors at a family level. Still, the complex (and partly unknown) etiology of type 1 diabetes implies that we cannot completely rule out endogeneity concerns due to unobservable factors, which may correlate with both type 1 diabetes onset

and adult outcomes.

To assess the potential influence of such personal traits and abilities, the two earnings studies (Papers 1 and 3) compare results from ordinary least square regression models and fixed effect model specifications that add controls for time-invariant individual heterogeneity. When adding the controls for individual fixed effects, the estimated coefficients increase, rather than decrease as we would expect the estimates to if such individual factors confound the studied relationships. Therefore, confounding is unlikely to be the reason behind the increasing estimates. More likely, the increase has something to do with the notion that type 1 diabetes might impact directly on earnings and also via its impact on health. Given that personal traits and abilities are likely to determine both successful disease management (Wennick et al., 2011; Goldman & Smith, 2002) and labor market outcomes (Heckman, 2007; Cunha et al., 2006), the influence of type 1 diabetes on both health and earnings is also likely to vary across personal traits and abilities. Consequently, high-ability individuals, as a group, are more likely to successfully manage the disease and consequently have better health. When we do not control for ability, high-ability individuals may compensate, within the diabetes group, for negative diabetes impact. But when conditioning on abilities, high-ability individuals are compared only with each other and can no longer compensate for lower-ability peers. Therefore, differences due to type 1 diabetes will no longer be masked by the positive influence of ability on both health and earnings.

Some researchers argue that child or adolescent ability is influenced by health and therefore potentially mediates the impact of health (Salm & Schunk, 2012; Currie et al., 2010; Heckman, 2007). Are the increasing estimates then, a sign of mediation? If so, diabetes estimates conditioned on individual fixed effects would not capture the casual impact of type 1 diabetes. However, most abilities have been found to stabilize early in life. For example, IQ generally manifests at age ten, while non-cognitive abilities such as motivation, self-discipline, and time preferences appear more changeable at later ages (Cunha et al., 2006). Type 1 diabetes is therefore more likely to affect abilities (causing mediation) the younger the individuals are at onset. Yet, the increase in the estimates when adding controls for fixed effects is about the same in both earnings papers, in spite of differing onset ages (ages two to five in Paper 3, compared to ages 28 to 34 in Paper 1). If a certain ability is to change during the time-span in which I measure earnings (ages>18, Paper 3 and ages>27, Paper 1), it will no longer be captured by the individual fixed effects as they only control for factors that are constant over time.

Consequently, mediation is not really a cause for concern. Abilities often stabilize early in life, meaning they are unlikely to change much after onset of type 1 diabetes for most individuals in this thesis. Even if, in later life, some diabetes-induced changes in ability still might occur, mediators will not be controlled for,

as the individual fixed effect specifications only capture time-invariant factors. Finally, potential mediation via childhood abilities is, if present, likely to operate in two opposite ways. On the one hand, we generally expect poor health to adversely affect ability formation (Cunha & Heckman, 2008; Heckman, 2007; Cunha et al., 2006). On the other, it seems reasonable to expect that diabetes management will contribute to ability formation via learning of skills such as responsibility and long-sightedness, which are also favorable in the labor market.

The differences found between the two models estimated with and without fixed effects could be attributed, in part, to them capturing somewhat different aspects of the diabetes-earnings relationship, besides the additional controls for individual-specific factors. The individual fixed effects model relies on variation within individuals across time, thereby failing to identify any diabetes-induced differences between individuals with and without type 1 diabetes that do not vary over time. Instead, the fixed effects estimates are adjusted for such differences, which will be captured by the individual fixed effects. Consequently, the fixed effects model only captures changes in earnings over time, while the ordinary least square model (using variations both within and between individuals) captures overall differences in earnings between individuals with and without type 1 diabetes.

In summary, to make sure that individual factors contributing to higher earnings, more successful diabetes management, and better health are not masking actual diabetes-related earnings consequences, this thesis suggests that individual fixed effects should be considered where possible. Still, the ordinary least square model is also informative as it captures overall differences in earnings, while the fixed effects model only captures changes in earnings over time.

2.4 Heterogeneity across socioeconomic groups

I allow for heterogeneity across socioeconomic groups (measured by own education in Paper 1 and parental education in Paper 2). The associations of socioeconomic characteristics, health, and health-related behavior are well known (see, e.g., Smith (1998)) and the degree and severity of diabetes-related complications are, therefore, also likely to vary with socioeconomic background. The adoption and effective use of new drugs and medical technologies relates to traits associated with schooling (Heckman, 2007; Goldman & Smith, 2011, 2002). Education is also positively associated with other health behaviors (e.g. regular exercise and non-smoking[5]) (Goldman & Smith, 2002) that are crucial for the severity and timing of diabetes-related complications (Möllsten et al., 2010; Daneman, 2006; Tesfaye et al., 2005; Miao et al., 2005; Ivers et al., 2001).

As in the intergenerational transition of human capital (Chevalier, 2004;

[5] Smoking is a risk factor of microvascular complications and may shorten life expectancy (Donaghue et al., 2007).

Black et al., 2003; Mulligan, 1999; Dearden et al., 1997), having better educated parents is likely to be positive for health-related behavior, including disease-coping strategies when disease management is as complex as it is with type 1 diabetes (Centers for Disease Control and Prevention, 2005). Having different networks and prior experience, people across the socioeconomic strata may be more or less likely to assimilate the long-term consequences of type 1 diabetes for both health and work. Long-sightedness is crucial as many diabetes-related complications first appear several years after onset, but their severity and timing are influenced by current lifestyle choices. In addition, current educational choices may impact on one's future work situation and ability to incorporate health impairments into everyday life. Such potential differences in diabetes self-management and assimilation of long-term diabetes-related consequences suggest that individuals of different socioeconomic backgrounds may respond differently to the onset of type 1 diabetes. Yet, it is important to remember that education is a crude measure of socioeconomic factors. Other influential heterogeneities might counteract educational differences if, for example, parents with high socioeconomic status work more and spend less time taking part in their children's diabetes management and schooling.

2.5 Siblings of individuals with type 1 diabetes

Paper 3 studies siblings of individuals with type 1 diabetes. Extensive research confirms the link between child health and adult outcomes, but has yet to establish whether poor child health also has long-term consequences for siblings growing up with a sick brother or sister. Health and skill formation during upbringing are closely interrelated (see, e.g., Heckman (2007)), partly because parents contribute to both their children's health and skill formation (Currie, 2009; Cunha & Heckman, 2008; Becker & Tomes, 1976; Behrman et al., 1982). When children experience health problems, their parents' caregiving role intensifies. This increased caregiving need, in turn, may affect engagement in their children's schooling and other family activities. Clearly, poor child health may therefore affect not only the sick child but also his or her siblings: if, for example, parents spend less time helping out with homework, both the sick child and his or her siblings' accumulation of new skills and abilities may suffer. Or, if caring for a sick child makes parents more health- and family-oriented, their children may learn skills (e.g., responsibility) that favor future labor market outcomes. Consequently, the impact on adult outcomes for children with type 1 diabetes and their siblings can be both positive and negative. Also, parents' responses might either compensate or reinforce the impact of type 1 diabetes, equally for all children or differently across siblings (Currie & Almond, 2011).

2.6 Choice of educational field — disease experience as an asset for a health care career

Paper 4 assesses the hypothesis that type 1 diabetes and its management provides individuals with experiences and capabilities that influence their educational choices. While disease onset can influence people in many ways, the impact of such experiences on educational choices has received little attention. Traditionally, the economic literature has explored human capital in the form of years of schooling or attainment of a degree to assess its impact on lifetime earnings (e.g., Card (1999)). Alongside formal education, abilities and skills created from life experiences can give the individual comparative advantages for specific careers (Paglin & Rufolo, 1990). Such comparative advantages could be decisive for paths of formal education and choice of profession (Gemici & Wiswall, 2014; Arcidiacono et al., 2012; Lee, 2005; Montmarquette et al., 2002). A health shock early in life may reduce overall incentives for educational investment (Currie et al., 2010; Currie, 2009; Case et al., 2005), but could also incentivize choosing an educational field where the experience of disease and its treatment could be an asset. Following this argument, own experience of disease would create a comparative advantage in health and medical professions from a qualitative perspective.

3 Data, methods, and validity

This thesis is based on two diabetes registers, covering nearly all individuals with type 1 diabetes in Sweden, combined with national population registers from Statistics Sweden. Papers 1 and 2 are based on the Diabetes Incidence Study in Sweden (Econ-DISS), which registers all incidents of diabetes in the age group 15 to 34 since 1983 (Östman et al., 1986, 2008). Papers 3 and 4 are based on the Swedish Childhood Diabetes Registry (SCDR), which registers incidents up to age 15 since 1977 (Nyström et al., 1990). The thesis applies the toolbox often used for studying child health and adult outcomes, such as different probability models (logit and probit models) and ordinary least squares. In addition, first, earnings Papers 1 and 3 apply individual fixed effects models to account for personal traits and abilities, as an alternative to sibling fixed effects and test scores that have been used previously (e.g., Lundborg, Nilsson, & Rooth (2014) for diabetes and the review by Currie (2009) for other measures of child health). Second, Paper 3 uses a matching method (the entropy balancing technique) that builds on the well-known propensity score matching method, to create the most comparable control groups before applying the individual fixed effect regression models. This matching strategy is especially important when analyzing siblings of individuals with type 1 diabetes, because the control group is originally designed to fit the individuals with type 1 diabetes and not their siblings. Third, Paper 4, which

focuses on choice of educational field, uses multinomial logit models to model the educational decisions as an unsorted series of binary choices.

Before continuing with a brief summary of each paper, the following paragraphs discuss the validity of the data and methods used in this thesis. Both diabetes registers follow the national incidence of type 1 diabetes with a high coverage[6]. The high coverage, together with universal social insurance coverage in Sweden (with publicly funded education and low care costs), ensures high representativeness, which is often troublesome in survey or non-mandatory insurance data. Yet, the magnitude of the link between type 1 diabetes and adult outcomes is likely contextual and affected by the Swedish setting. Sweden, being a country with a high incidence of type 1 diabetes, might complicate generalizations to low-incidence countries. The high prevalence of type 1 diabetes might raise employers' awareness of the disease and possibly (1) lower the risk of statistical discrimination, or (2) make employers more vigilant of the problems associated with type 1 diabetes. Moreover, inference to other less well-defined health shocks, with less clear impact on day-to-day activities, might be troublesome. Consequently, these findings may not be representative for people with less demanding diseases, such as moderate asthma and allergy. However, milder diseases or diseases with fewer demands on daily management are arguably of less concern regarding potential impacts on educational and labor market outcomes.

The high coverage data limits the threats of selection bias, because nearly all cases of type 1 diabetes in Sweden are included in the study. The entropy balancing method, which makes the controls as similar as possible to the individuals with type 1 diabetes with regards to important pre-onset background factors (including parental education and parental year of birth), further limits selection bias (Paper 3). In addition, the individual fixed effects specifications control (at least partly) for selection into employment and all analysis conditioned on employment are assessed in the sensitivity analysis. The type 1 diabetes is physician-assessed and the impact on daily life and health related consequences are well-described (Sparud-Lundin et al., 2010; Wennick et al., 2009), leaving little room for measurement bias and none for potential mix-ups with type 2 diabetes, which differs in etiology and key disease consequences. Additionally, outcomes are taken from national population registers where, for example, annual labor earnings are reported by employers (Statistics Sweden, 2011). The threat of attrition bias in the studied panels also ought to be low, given the number of observations per year is comparable across the studied groups, suggesting that individuals with type 1 diabetes and controls are equally represented throughout the studied years.

A standard concern regarding the validity of causal inferences is confounding.

[6]The SCDR has an estimated coverage of 96 to 99% (Nyström et al., 1990). The Econ-DISS has an estimated coverage of 86 to 91% (Östman et al., 2008)

I discussed this earlier in connection with individual fixed effects and the entropy balancing method. Still, I would like to add that individual fixed effects only considers time-invariant heterogeneity, and potential time-variant third factors affecting both type 1 diabetes onset and adult outcomes could still be at play. However, no known confounders exist and the entropy balancing method offers a way to alleviate the concern of unknown confounding: by tweaking the control group to make them more similar to the individuals with type 1 diabetes, both groups are likely to be equally affected by time-variant, as well as time-invariant, unobservable factors. Also, sensitivity analyzes indicate that the results are robust to other time-variant group-specific characteristics besides type 1 diabetes.

4 The papers

4.1 Onset of type 1 diabetes in young adults and long-term consequences for annual earnings

The traditional view that health problems obstruct work and everyday life is becoming less obvious in today's knowledge-based society with the rapid development of medical technologies. Technological enhancements (the introduction of take-home blood glucose measures, long-acting insulin, and insulin pumps) have enabled diabetes patients to integrate their diabetes management even further into everyday life. Thus, individuals themselves (rather than the medical profession) are responsible for daily disease management and future health (Wennick et al., 2009), making type 1 diabetes an interesting case to study.

Using Swedish longitudinal register data, this paper investigates how onset of type 1 diabetes in the age group 28 to 34 relates to adult annual labor earnings over 22 years. First, by focusing on health in young adults (who are generally in the early stages of their careers, but have already made educational choices), I minimize any influence on earnings that otherwise may come from health interacting with education and skill formation during upbringing. Second, by controlling for individual fixed effects, I minimize any influence from time-invariant unobservable factors, such as cognitive and non-cognitive abilities, which have been found to be rather constant throughout adulthood (Cunha et al., 2006). The results show statistically and quantitatively significant negative links between type 1 diabetes and earnings. Beyond the immediate impact at onset, the negative link increases over time, especially for women's earnings. Women's sensitivity appears across all levels of education, while, among men, those with upper secondary education appear most vulnerable. These gender-related differences may partly be down to absenteeism due to sickness, disability, and parental leave, as women with diabetes, but not men, are shown to differ in their uptake from the Swedish welfare system, compared to population controls.

4.2 Onset of type 1 diabetes in young adults and university education

This paper investigates the interrelationships of young-adulthood health, university education, and family formation. Generally, young adults face the choice of entering the labor market or continuing to university education to increase their future employability and labor earnings. This decision relates to other choices in life. University education has, for example, been found to delay family formation (Boschini et al., 2011; Lundin et al., 2008; Björklund, 2006) as both university education and family formation require investments in time and effort (as well as monetary costs). An unexpected health shock, such as the sudden onset of a lifelong disease, also requires the investment of time and effort to restore and maintain health. Such a change in life constraints can cause young adults to reevaluate previously set university aspirations and other life choices. Family formation and university education are substitutes for women (Boschini et al., 2011; Lundin et al., 2008; Björklund, 2006) and type 1 diabetes reduces fertility and amplifies the risk of severe pregnancy-related complications for both the mother and child (Jonasson et al., 2007; Casson et al., 1997). Thereby, type 1 diabetes adds to the risks all young women face when delaying childbearing, and even more so with increasing age. Such risks may contribute to some women choosing university education over family formation after onset, while the elevated risks may hasten other women to start a family and maybe forgo their academic career.

Using longitudinal register data on individuals with type 1 diabetes onset in the age group 17 to 20 and population controls, we illustrate how an unexpected health shock (imposing changed life-constraints, increased health investments, and higher uncertainty about future outcomes) may affect subsequent university education. We find that type 1 diabetes among women negatively links to university education and motherhood. Comparing only the university educated, women with diabetes become mothers to a lesser extent than other women. Taken together, these results indicate that type 1 diabetes affects both the decision to enter university and to start a family, suggesting that type 1 diabetes sharpens the tradeoff between university education and motherhood: i.e., diabetes might make it more difficult to have both a university education and children. Socioeconomic background also seems to be important: women belonging to different socioeconomic groups appear to respond differently to onset of type 1 diabetes, in terms of both university education and family formation. For men, we find no association between onset and university education.

4.3 Labor market consequences of growing up with type 1 diabetes

Exploring the long-term labor market consequences of growing up with type 1 diabetes, this paper investigates how childhood onset of type 1 diabetes (6 to 15 years old) influences adult labor market outcomes, both for children developing the disease and their siblings. The results indicate a negative impact on labor market outcomes (throughout ages 19 to 48) for those who develop type 1 diabetes as children. Both women and men with diabetes have a lower likelihood of employment and lower annual labor earnings than controls. The decrease in women's likelihood of employment is roughly twice that for men, whereas the increasingly negative link to earnings appears more profound for men. For siblings of individuals with type 1 diabetes, sisters' outcomes appear unaffected, while brothers' outcomes show, on the one hand, a higher likelihood of being employed, but, on the other hand, lower earnings reminiscent of the earnings decrease for individuals with type 1 diabetes themselves. These novel findings for brothers of individuals with type 1 diabetes support actions that consider broader family impact, both when initiating further research and when designing children's diabetes management programs.

4.4 Early onset of type 1 diabetes and educational field at upper secondary and university level: is own experience an asset for a health care career?

Previous evidence shows that ill health in early life has a significant negative impact on school grades, grade repetition, educational level, and labor market outcomes. But are all aspects of a health shock in childhood or adolescence necessarily bad, or could it also create comparative advantages and experiences that could have professional value? We analyze this question using the Swedish Childhood Diabetes Register, the National Educational Register, and other population registers in Sweden. More specifically, we investigate the relationship between onset of type 1 diabetes (up to age 15) and the probability of choosing (and completing) a health-oriented path at upper secondary and university level. By modeling the educational decisions as an unsorted series of binary choices, we shed light on the more qualitative aspects of schooling and assess a potential mechanism linking early life health to adult outcomes. Our results reject the hypothesis of no systematic differences in choice of educational field between people with and without type 1 diabetes. The results are robust to selection on ability proxies and across sensitivity analysis. We conclude that disease onset in childhood and adolescence may generate experiences and comparative advantages for choosing and completing a health-oriented program of education.

References

Almond, D., & Currie, J. (2011). Killing me softly: The fetal origins hypothesis. *Journal of Economic Perspectives*, *25*(3), 153–172.

American Diabetes Association. (2008). Diagnosis and classification of diabetes mellitus. *Diabetes Care*, *31*(Supplement 1), 55–60.

Arcidiacono, P., Hotz, V. J., & Kang, S. (2012). Modeling college major choices using elicited measures of expectations and counterfactuals. *Journal of Econometrics*, *166*(1), 3–16.

Atkinson, M. A., & Eisenbarth, G. S. (2001). Type 1 diabetes: new perspectives on disease pathogenesis and treatment. *The Lancet*, *358*(9277), 221–229.

Bastida, E., & Pagán, J. A. (2002). The impact of diabetes on adult employment and earnings of Mexican Americans: Findings from a community based study. *Health Economics*, *11*(5), 403–413.

Becker, G., & Tomes, N. (1976). Child endowments and the quantity and quality of children. *Journal of Political Economy*, *84*, 143–162.

Behrman, J. R., Pollak, R., & Taubman, P. (1982). Parental preferences and provision for progeny. *Journal of Political Economy*, 52–73.

Behrman, J. R., & Rosenzweig, M. R. (2004). Returns to birthweight. *The Review of Economics and statistics*, *86*(2), 586–601.

Björklund, A. (2006). Does family policy affect fertility? *Journal of Population Economics*, *19*(1), 3–24.

Black, S. E., Devereux, P. J., & Salvanes, K. G. (2003). *Why the apple doesn't fall far: Understanding intergenerational transmission of human capital* (Working Paper No. 10066). National Bureau of Economic Research.

Black, S. E., Devereux, P. J., & Salvanes, K. G. (2007). From the cradle to the labor market? The effect of birth weight on adult outcomes. *The Quarterly Journal of Economics*, *122*(1), 409-439.

Boschini, A., Håkanson, C., Rosén, Å., & Sjögren, A. (2011). *Trading off or having it all? Completed fertility and mid-career earnings of Swedish men and women* (Working Paper, IFAU - Institute for Labour Market Policy Evaluation No. 15).

Brown, H. S., Pagán, J. A., & Bastida, E. (2005). The impact of diabetes on employment: genetic IVs in a bivariate probit. *Health Economics*, *14*(5), 537–544.

Brown, H. S., Perez, A., Yarnell, L. M., Pagan, J. A., Hanis, C. L., Fischer-Hoch, S., & McCormick, J. B. (2010). Diabetes and employment productivity: does diabetes management matter? *The American Journal of Managed Care*, *17*(8), 569–576.

Card, D. (1999). The causal effect of education on earnings. *Handbook of Labor Economics*, *3*, 1801–1863.

Case, A., Fertig, A., & Paxson, C. (2005). The lasting impact of childhood health and circumstance. *Journal of Health Economics*, *24*(2), 365–389.

Case, A., & Paxson, C. (2008). Stature and status: Height, ability, and labor market outcomes. *Journal of Political Economy*, *116*(3).

Casson, I., Clarke, C., Howard, C., Mc-Kendrick, O., Pennycook, S., Pharoah, P., ... Walkinshaw, S. (1997). Outcomes of pregnancy in insulin dependent diabetic women: results of a five year population cohort study. *BMJ*, *315*, 275–278.

Centers for Disease Control and Prevention. (2005). *National diabetes fact sheet*. `http://www.cdc.gov/diabetes/pubs/pdf/ndfs_2005.pdf`. (20 Mars 2012)

Chabris, C. F., Hebert, B. M., Benjamin, D. J., Beauchamp, J., Cesarini, D., van der Loos, M., ... others (2012). Most reported genetic associations with general intelligence are probably false positives. *Psychological Science*, *23*(11), 1314–1323.

Chevalier, A. (2004). *Parental education and child's education : A natural experiment* (IZA Discussion paper series No. 1153).

Cunha, F., & Heckman, J. (2008). Formulating, identifying and estimating the technology of cognitive and noncognitive skill formation. *Journal of Human Resources*, *43*, 738–782.

Cunha, F., Heckman, J., Lochner, L., & Masterov, D. (2006). Interpreting the evidence on life cycle skill formation. *Handbook of the Economics of Education*, *1*, 697–812.

Currie, J. (2009). Healthy, wealthy, and wise: Socioeconomic status, poor health in childhood, and human capital development. *Journal of Economic Literature*, *47*, 87–122.

Currie, J., & Almond, D. (2011). Human capital development before age five. *Handbook of Labor Economics*, *4*, 1315–1486.

Currie, J., & Madrian, B. C. (1999). Health, health insurance and the labor market. *Handbook of Labor Economics*, *3*, 3309–3416.

Currie, J., Stabile, M., Manivong, P., & Roos, L. L. (2010). Child health and young adult outcomes. *Journal of Human Resources*, *45*(3), 517–548.

Cutler, D. M., Glaeser, E. L., & Shapiro, J. M. (2003). Why have Americans become more obese? *Journal of Economic Perspectives*, *17*, 93–118.

Dahlquist, G., Källén, B., & Swedish Childhood Diabetes Study Group. (2007). School performance in children with type 1 diabetes–a population-based register study. *Diabetologia*, *50*(5), 957–64.

Dahlquist, G., & Mustonen, L. (2000). Analysis of 20 years of prospective registration of childhood onset diabetes time trends and birth cohort effects. Swedish Childhood Diabetes Study Group. *Acta Paediatrica*, *89*(10), 1231–1237.

Dahlquist, G., Nyström, L., & Patterson, C. C. (2011). Incidence of type 1 diabetes in Sweden among individuals aged 0–34 years, 1983–2007 an analysis of time trends. *Diabetes Care*, *34*(8), 1754–1759.

Dahlquist, G., Pundziūtė-Lyckå, A., Nyström, L., et al. (2005). Birthweight and risk of type 1 diabetes in children and young adults: a population-based register study. *Diabetologia*, *48*(6), 1114–1117.

Daneman, D. (2006). Type 1 diabetes. *The Lancet*, *367*(9513), 847–858.

Dearden, L., Machin, S., & Reed, H. (1997). Intergenerational mobility in britain. *Economic Journal*, *107*, 47–66.

Devaux, M., & Sassi, F. (2013). Social inequalities in obesity and overweight in 11 OECD countries. *The European Journal of Public Health*, *23*(3), 464–469.

Donaghue, K., Chiarelli, F., Trotta, D., Allgrove, J., & Dahl-Jorgensen, K. (2007). International society for pediatric and adolescent diabetes. ispad clinical practice consensus guidelines 2006–2007. microvascular and macrovascular complications. *Pediatric Diabetes*, *8*(3), 163–170.

Figlio, D. N., Guryan, J., Karbownik, K., & Roth, J. (2013). *The effects of poor neonatal health on children's cognitive development* (Tech. Rep.). National Bureau of Economic Research.

Fletcher, J. M., & Richards, M. R. (2012). Diabetes's 'health shock' to schooling and earnings: Increased dropout rates and lower wages and employment in young adults. *Health Affairs*, *31*(1), 27–34.

Gemici, A., & Wiswall, M. (2014). Evolution of gender differences in post-secondary human capital investments: College majors. *International Economic Review*, *55*(1), 23–56.

Goldman, D., & Smith, J. P. (2002). Can patient self-management help explain the SES health gradient? *Proceedings of the National Academy of Sciences*, *99*(16), 10929–10934.

Goldman, D., & Smith, J. P. (2011). The increasing value of education to health. *Social Science & Medicine*, *72*(10), 1728–1737.

Grossman, M., & Kaestner, R. (1997). Effects of education on health. In J. R. Behrman & N. Stacey (Eds.), *The social benefits of education* (pp. 475–492). Ann Arbor: University of Michigan Press.

Groupa, C. T. R., et al. (1994). Effect of intensive diabetes treatment on the development and progression of long-term complications in adolescents with insulin-dependent diabetes mellitus: Diabetes control and complications trial. *The Journal of Pediatrics*, *125*(2), 177–188.

Haas, S. A., Glymour, M. M., & Berkman, L. F. (2011). Childhood health and labor market inequality over the life course. *Journal of Health and Social Behavior*, *52*(3), 298–313.

Harris, A. (2008). *Chronic disease and labour force participation in Australia: an endogenous multivariate probit analysis of clinical prevalence data* (Tech. Rep.).

Heckman, J. (2007). The economics, technology, and neuroscience of human capability formation. *Proceedings of the National Academy of Sciences*, *104*(33), 13250–13255.

Ivers, R. Q., Cumming, R. G., Mitchell, P., & Peduto, A. J. (2001). Diabetes and risk of fracture the blue mountains eye study. *Diabetes Care*, *24*(7), 1198–1203.

Johnson, R. C., & Schoeni, R. F. (2011). The influence of early-life events on human capital, health status, and labor market outcomes over the life course. *The BE Journal of Economic Analysis & Policy*, *11*(3).

Jonasson, J. M., Brismar, K., Sparén, P., Lambe, M., Nyrén, O., Östenson, C.-G., & Ye, W. (2007). Fertility in women with type 1 diabetes: A population-based cohort study in Sweden. *Diabetes Care*, *30*(9), 2271–2276.

Lahmann, P. H., Lissner, L., Gullberg, B., & Berglund, G. (2000). Sociodemographic factors associated with long-term weight gain, current body fatness and central adiposity in Swedish women. *International Journal of Obesity & Related Metabolic Disorders*, *24*, 685.

Latif, E. (2009). The impact of diabetes on employment in Canada. *Health Economics*, *18*(5), 577–589.

Lee, D. (2005). An estimable dynamic general equilibrium model of work, schooling, and occupational choice*. *International Economic Review*, *46*(1), 1–34.

Lindeboom, M., Llena-Nozal, A., & van der Klaauw, B. (2006). *Disability and work: The role of health shocks and childhood circumstances* (IZA Discussion Papers). Institute for the Study of Labor (IZA).

Lissner, L., Johansson, S., Qvist, J., Rössner, S., & Wolk, A. (2000). Social mapping of the obesity epidemic in Sweden. *International Journal of Obesity & Related Metabolic Disorders*, *24*, 801–5.

Lundborg, P., Nilsson, A., & Rooth, D.-O. (2014). Adolescent health and adult labor market outcomes. *Journal of Health Economics*, *37*, 25–40.

Lundborg, P., Nystedt, P., & Rooth, D.-O. (2014). Height and earnings: The role of cognitive and noncognitive skills. *Journal of Human Resources*, *49*(1), 141–166.

Lundin, D., Mörk, E., & Öckert, B. (2008). How far can reduced childcare prices push female labour supply? *Labour Economics*, *15*(4), 647–659.

Maluccio, J. A., Hoddinott, J., Behrman, J. R., Martorell, R., Quisumbing, A. R., & Stein, A. D. (2009). The impact of improving nutrition during early childhood on education among guatemalan adults*. *The Economic Journal*, *119*(537), 734–763.

Maslow, G. R., Haydon, A., McRee, A.-L., Ford, C. A., & Halpern, C. T. (2011). Growing up with a chronic illness: Social success, educational/vocational distress. *Journal of Adolescent Health*, *49*(2), 206–212.

Miao, J., Brismar, K., Nyrén, O., Ugarph-Morawski, A., & Ye, W. (2005). Elevated hip fracture risk in type 1 diabetic patients: A population-based cohort study in Sweden. *Diabetes Care*, *28*(12), 2850–2855.

Milton, B., Holland, P., & Whitehead, M. (2006). The social and economic consequences of childhood-onset type 1 diabetes mellitus across the lifecourse: a systematic review. *Diabetic Medicine*, *23*, 821–829.

Minor, T. (2011). The effect of diabetes on female labor force decisions: new evidence from the national health interview survey. *Health Economics*, *20*(12), 1468–1486.

Minor, T. (2013). An investigation into the effect of type I and type II diabetes duration on employment and wages. *Economics & Human Biology*, *11*(4), 534–544.

Molarius, A. (2003). The contribution of lifestyle factors to socioeconomic differences in obesity in men and women — a population-based study in Sweden. *European Journal of Epidemiology*, *18*, 227–234.

Molarius, A., Seidell, J. C., Sans, S., Tuomilehto, J., & Kuulasmaa, K. (2000). Educational level, relative body weight, and changes in their association over 10 years: an international perspective from the WHO MONICA project. *American Journal of Public Health*, *90*, 1260–1268.

Möllsten, A., Svensson, M., Waernbaum, I., Berhan, Y., Schön, S., Nyström, L., … Dahlquist, G. (2010). Cumulative risk, age at onset, and sex-specific differences for developing end-stage renal disease in young patients with type 1 diabetes a nationwide population-based cohort study. *Diabetes*, *59*(7), 1803–1808.

Montmarquette, C., Cannings, K., & Mahseredjian, S. (2002). How do young people choose college majors? *Economics of Education Review*, *21*(6), 543–556.

Mulligan, C. B. (1999). Galton versus the human capital approach to inheritance. *Journal of Political Economy*, *107*(6), 184–224.

Nyström, L., Dahlquist, G., Rewers, M., & Wall, S. (1990). The Swedish childhood diabetes study. An analysis of the temporal variation in diabetes incidence 1978–1987. *International Journal of Epidemiology*, *19*(1), 141–146.

Oreopoulos, P., Stabile, M., Walld, R., & Roos, L. L. (2008). Short-, medium-, and long-term consequences of poor infant health an analysis using siblings and twins. *Journal of Human Resources*, *43*(1), 88–138.

Östman, J., Arnqvist, H., Blohmé, G., Lithner, F., Littorin, B., Nyström, L., … Wibell, L. (1986). Epidemiology of diabetes mellitus in Sweden. Results of the first year of a prospective study in the population age group 15–34 years. *Acta Medica Scandinavica*, *220*(5), 437–445.

Östman, J., Lönnberg, G., Arnqvist, H. J., Blohmé, G., Bolinder, J., Ekbom Schnell, A., … Nyström, L. (2008). Gender differences and temporal variation in the incidence of type 1 diabetes: results of 8012 cases in the nationwide Diabetes Incidence Study in Sweden 1983–2002. *Journal of Internal Medicine*, *263*(4), 386–394.

Paglin, M., & Rufolo, A. M. (1990). Heterogeneous human capital, occupational choice, and male-female earnings differences. *Journal of Labor Economics*, 123–144.

Persson, S., Dahlquist, G., Gerdtham, U., & Steen Carlsson, K. (2013). Impact of childhood-onset type 1 diabetes on schooling: a population-based register study. *Diabetologia*, *56*(6), 1254–1262.

Ploug, U. J. (2013). The cost of diabetes-related complications: Registry-based analysis of days absent from work. *Economics Research International*, *2013*.

Pundziute-Lyckå, A., Dahlquist, G., Nyström, L., Arnqvist, H., Björk, E., Blohme, G., … others (2002). The incidence of type I diabetes has not increased but shifted to a younger age at diagnosis in the 0–34 years group in Sweden 1983 to 1998. *Diabetologia*, *45*(6), 783–791.

Rietveld, C. A., Medland, S. E., Derringer, J., Yang, J., Esko, T., Martin, N. W., … others (2013). GWAS of 126,559 individuals identifies genetic variants associated with educational attainment. *Science*, *340*(6139), 1467–1471.

Royer, H. (2009). Separated at girth: Us twin estimates of the effects of birth weight. *American Economic Journal: Applied Economics*, *1*(1), 49–85.

Salm, M., & Schunk, D. (2012). The relationship between child health, developmental gaps, and parental education: Evidence from administrative data. *Journal of the European Economic Association*, *10*(6), 1425–1449.

Smith, J. P. (1998). Socioeconomic status and health. *American Economic Review*, 192–196.

Smith, J. P. (2007). The impact of socioeconomic status on health over the life-course. *Journal of Human Resources*, *42*(4), 739–764.

Smith, J. P. (2009). The impact of childhood health on adult labor market outcomes. *The Review of Economics and Statistics*, *91*(3), 478–489.

Soltesz, G., Patterson, C., & Dahlquist, G. (2007). Worldwide childhood type 1 diabetes incidence–what can we learn from epidemiology? *Pediatric Diabetes*, *8*(s6), 6–14.

Sparud-Lundin, C., Öhrn, I., & Danielson, E. (2010). Redefining relationships and identity in young adults with type 1 diabetes. *Journal of Advanced Nursing*, *66*(1), 128–138.

Statistics Sweden. (2011). *Longitudinal integration database for health insurance and labour market studies (LISA by Swedish acronym)*. `http://www.scb.se/Pages/List____257743.aspx`. (6 June 2012)

Steen Carlsson, K., Landin-Olsson, M., Nyström, L., Arnqvist, H. J., Bolinder, J., Östman, J., & Gudbjörnsdottir, S. (2010). Long-term detrimental consequences of the onset of type 1 diabetes on annual earnings-evidence from annual registry data in 1990–2005. *Diabetologia*, *53*(6), 1084–92.

Tesfaye, S., Chaturvedi, N., Eaton, S. E., Ward, J. D., Manes, C., Ionescu-Tirgoviste, C., … Fuller, J. H. (2005). Vascular risk factors and diabetic neuropathy. *New England Journal of Medicine*, *352*(4), 341–350.

The TEDDY Study Group. (2007). The environmental determinants of diabetes in the young (TEDDY) study: study design. *Pediatric Diabetes*, *8*(5), 286–298.

Tunceli, K., Bradley, C. J., Nerenz, D., Williams, L. K., Pladevall, M., & Lafata, J. E. (2005). The impact of diabetes on employment and work productivity. *Diabetes Care*, *28*(11), 2662–2667.

Vijan, S., Hayward, R. A., & Langa, K. M. (2004). The impact of diabetes on workforce participation: Results from a national household sample. *Health Services Research*, *39*(6), 1653–1670.

Wennick, A., & Hallström, I. (2006). Swedish families' lived experience when a child is first diagnosed as having insulin-dependent diabetes mellitus an ongoing learning process. *Journal of Family Nursing*, *12*(4), 368–389.

Wennick, A., Hallström, I., Lindgren, B., & Bolin, K. (2011). Attained education and self-assessed health later in life when diagnosed with diabetes in childhood: a population-based study. *Pediatric Diabetes*, *12*(7), 619–626.

Wennick, A., & Huus, K. (2012). What it is like being a sibling of a child newly diagnosed with type 1 diabetes: an interview study. *European Diabetes Nursing*, *9*(3), 88–92.

Wennick, A., Lundqvist, A., & Hallström, I. (2009). Everyday experience of families three years after diagnosis of type 1 diabetes in children: A research paper. *Journal of Pediatric Nursing*, *24*(3), 222–230.

Zhang, X., Zhao, X., & Harris, A. (2009). Chronic diseases and labour force participation in Australia. *Journal of Health Economics*, *28*(1), 91–108.

Paper I

Onset of type 1 diabetes in young adults and long-term consequences for annual earnings

Abstract

The traditional view that health problems obstruct work and everyday life is becoming less obvious in today's knowledge-based society with the rapid development of medical technologies. Technological enhancements (the introduction of take-home blood glucose measures, long-acting insulin, and insulin pumps) have enabled diabetes patients to integrate their diabetes management even further into everyday life. Thus, individuals themselves (rather than the medical profession) are responsible for daily disease management and future health, making type 1 diabetes an interesting case to study. Using Swedish longitudinal register data, this paper investigates how onset of type 1 diabetes in the age group 28 to 34 relates to adult annual labor earnings over 22 years. The results show statistically and quantitatively significant negative links between type 1 diabetes and earnings. Beyond the immediate impact at onset, the negative link increases over time, especially for women's earnings. Women's sensitivity appears across all levels of education, while, among men, those with upper secondary education appear most vulnerable. These gender-related differences may partly be down to absenteeism due to sickness, disability, and parental leave, as women with diabetes, but not men, are shown to differ in their uptake from the Swedish welfare system, compared to population controls.

Keywords: Health, earnings, type 1 diabetes
JEL Classification: I10, I12, J24, J31

1 Introduction

The association between early life health and adult outcomes is well documented in the economic literature and a growing number of studies concern the causal impact of health (for reviews, see e.g., Currie & Almond (2011), and Currie (2009)). However, most evidence regards very early measures of health (e.g., birthweight[1]) or broad measures (e.g., overall health[2] or height[3]) rather than specific diagnoses. One exception is presented by Case et al. (2005), who investigate whether chronic conditions (at age 7 and 16) link to educational and labor market outcomes. A second exception is presented by Smith & Smith (2010), who focus on mental disorders (before age 17) such as substance abuse and depression. They suggest that the lower earnings for individuals with mental disorders arise from less time worked and a high probability of not working at all. Third, Currie et al. (2010) assess the impact of different health problems (before age 18), including mental conditions, asthma, and injuries, on educational and financial outcomes among young adults. Fourth, Lundborg, Nilsson, & Rooth (2014) perform a similar study, but on a broader range of health problems (including diabetes at age 18) and labor market outcomes, such as earnings.

Given the complex interplay between childhood skill formation, schooling, and health, a general concern is that the relationships between childhood health and adult outcomes capture the effects of human capabilities (i.e., cognitive and non-cognitive abilities and skills) (Haas et al., 2011; Cunha et al., 2006). In this study, I therefore focus on health in young adults. I investigate how onset of type 1 diabetes in the age group 28–34 relates to adult annual labor earnings, throughout 22 years following onset. I provide estimates, based on Swedish longitudinal register data, that are robust against selection into low education and selection on time-invariant unobservable factors. First, by focusing on health in young adults (who are generally in their early careers, but have already made their educational choices), I minimize any influence on earnings that otherwise might come from health interacting with education and skill formation during upbringing. Second, by controlling for individual fixed effects, I minimize any influence from time-invariant unobservable factors, such as cognitive and non-cognitive abilities, which have been found rather constant throughout adulthood.[4]

The traditional view that health problems obstruct work and everyday life is becoming less obvious in today's knowledge-based society with the rapid development of medical technologies. Technological enhancements (the introduction

[1] See, e.g., Figlio et al. (2013), Almond & Currie (2011), Royer (2009), Black et al. (2007), Behrman & Rosenzweig (2004).

[2] See e.g., Haas et al. (2011), Smith (2009), Smith (2007).

[3] See, e.g., Lundborg, Nystedt, & Rooth (2014), and Case & Paxson (2008).

[4] See e.g., Cunha et al. (2006) for an extensive discussion of human capabilities and critical periods for ability formation. IQ generally manifests at age 10, while non-cognitive abilities such as motivation, self-discipline, and time preference are more changeable at later ages.

of take-home blood glucose measures, long-acting insulin, and insulin pumps) have enabled diabetes patients to integrate their diabetes management even further into everyday life. Thus, individuals themselves (rather than the medical profession) are responsible for their daily diabetes management and future health (Wennick et al., 2009), making type 1 diabetes an interesting case to study. Diabetes management involves daily regular blood glucose monitoring to assess when to inject insulin and the dosage needed to keep the blood glucose levels within a target range (Daneman, 2006). Given that the disease is well defined and its impact on everyday life is well-documented (Sparud-Lundin et al., 2010; Wennick et al., 2009), I can explain, in part, the pathways through which type 1 diabetes might affect earnings, via data from the social welfare system.

Another interesting implication of type 1 diabetes is that its development appears independent of lifestyle factors (American Diabetes Association, 2008), but subsequent diabetes-related complications are not (American Diabetes Association, 2008; Daneman, 2006).[5] Knowing that lifestyle factors correlate with socioeconomic factors (Devaux & Sassi, 2013; Cutler et al., 2003; Molarius, 2003), I provide estimates stratified by educational level to allow for heterogeneity in the diabetes-earnings link across socioeconomic groups. The adoption and effective use of new drugs and medical technologies relate to traits associated with schooling (Goldman & Smith, 2011, 2002; Heckman, 2007). Education is also positively associated with other health behaviors (e.g., regular exercise and non-smoking) (Goldman & Smith, 2002), which are crucial for the severity and timing of diabetes-related complications (Möllsten et al., 2010; Daneman, 2006; Miao et al., 2005; Tesfaye et al., 2005; Ivers et al., 2001). Additionally, theoretical insights show that (1) education is a productivity shifter in the production of health (Grossman, 1972a,b), and (2) socioeconomic factors correlate with experiences, preferences, and beliefs (Fuchs, 1993), suggesting that individuals with higher socioeconomic status might have lower discount rates and therefore invest more in future health.

Type 1 diabetes is generally modeled as an exogenous health shock (Persson et al., 2013; Minor, 2011; Steen Carlsson et al., 2010), because (1) the lifetime risk of onset is uncorrelated with lifestyle factors (i.e., obesity and lack of exercise) (American Diabetes Association, 2008) and (2) type 1 diabetes has a sudden and rapid onset, which the individual is unable to anticipate or influence beforehand (Daneman, 2006). Lundborg, Nilsson, & Rooth (2014) confirm that men's earnings penalty from diabetes is relatively robust to sibling fixed effects and unobserved factors at the family level. Yet, the exact combination of genetic and environmental factors, together with the chain of events to trigger onset of type 1 diabetes, is still unknown (Daneman, 2006). Given this complexity, we cannot rule out endogeneity concerns due to unobservable factors, which may correlate

[5] Diabetes-related complications are diseases affecting, e.g., the heart, eyes, kidneys, and nervous system.

with both type 1 diabetes onset and labor market outcomes. To alleviate this concern, I use a fixed effect models specification to control for time-invariant individual heterogeneity.

This study aims, first, to compare the progression of annual earnings of men and women with type 1 diabetes to the earnings profiles of population controls during the years 1990–2005. Second, to allow for socioeconomic heterogeneity in the earnings differences, I stratify the analysis by level of education. Third, to assess potential mechanisms through which type 1 diabetes may affect earnings, I compare differences in the usage of the Swedish social welfare system between the diabetes groups and the control groups. The results show statistically and quantitatively significant negative links between type 1 diabetes and earnings. Beyond the immediate impact at onset, the negative link increases over time, especially for women's earnings. The structure of the paper is as follows. Section 2 reviews the previous literature on diabetes and earnings. Section 3 describes the etiology of type 1 diabetes and its implications for the interpretation of the estimated coefficients. Section 4 presents the data and descriptive statistics. Section 5 details my econometric strategy. Section 6 presents the main results, and Section 7 the sensitivity analyses. Section 8 concludes.

2 Previous research on diabetes and earnings

Previous economic research reports negative associations between diabetes and labor market outcomes such as employment, hours worked, and earnings (Ploug, 2013; Fletcher & Richards, 2012; Maslow et al., 2011; Brown et al., 2010; Latif, 2009; Zhang et al., 2009; Harris, 2008; Brown et al., 2005; Tunceli et al., 2005; Vijan et al., 2004; Bastida & Pagán, 2002). However, these studies are limited by (1) reporting and recall bias from survey data, and (2) the inability to distinguish between type 1 and type 2 diabetes. The two types of diabetes ought to have different impact on labor market outcomes, as the timing of onset (as well as the factors causing the diseases) differs.

Minor (2013, 2011) and Steen Carlsson et al. (2010) explicitly study type 1 diabetes,[6] but only Steen Carlsson et al. (2010) have register data where the different types of diabetes are classified according to current clinical diabetes criteria (the same data as in this study). Lundborg, Nilsson, & Rooth (2014) use enlistment data[7] for men at age 18, but do not state the type of diabetes that they study. Using survey data, Minor (2013, 2011) assumes that individuals have type 1 diabetes if they reported having diabetes before age 20. Although both

[6] Early results on type 1 diabetes and educational or labor market outcomes are reviewed by Milton et al. (2006), who conclude that most studies use small sample surveys and are likely to be subject to omitted variables bias.

[7] All diagnoses were recorded by a physician, based on an interview and a take-home health declaration form.

reasonable and well justified, his assumption implies that the population in my study (and the larger part of the population in Steen Carlsson et al. (2010)) would have type 2 rather than type 1 diabetes.

Lundborg, Nilsson, & Rooth (2014) report negative earnings penalties due to diabetes (-20.8% when controlling for sibling fixed effects and -24.3% without). Steen Carlsson et al. (2010) report lower earnings for both women (-8%) and men (-4%) after onset of type 1 diabetes in the age group 15–34: they use pooled longitudinal data and a case-control study design. Minor (2013, 2011) studies both type 1 and type 2 diabetes and concludes that the associations between diabetes and labor market outcomes are driven by type 2 diabetes, while he finds no significant associations for type 1 diabetes.[8]

This paper expands on previous results by: (1) considering selection into low education and selection on time-invariant individual-specific unobservable factors; (2) allowing for heterogeneity across socioeconomic groups and over time; and (3) employing high-coverage register data of incidences of type 1 diabetes (since 1983), where the type of diabetes is classified by the reporting physician.

3 Type 1 diabetes—etiology and implications

Type 1 diabetes is a multifactorial disease with a complex genetic component that involves multiple genetic loci contributing to susceptibility to the disease. Although the heritability of the disease is low,[9] this increases with the number of genes shared with a family member with type 1 diabetes (highest in mono-zygotic twins followed by first and second degree relatives).[10] The average prevalence risk in siblings is 6% compared to 0.4% in the general population.[11] (Karvonen et al., 2000) In addition to the inherited susceptibility, something must set off the immune system to turn it against itself and trigger the development of type 1 diabetes. Despite much research, the responsible trigger (or triggers) remains unknown.[12]

[8] Minor (2011) includes only women. Minor (2013) reports a positive association for women's wages for one specification, but he disregards this finding because it is based on an extremely low number of observations.

[9] Dahlquist & Mustonen (2000) report that 90% of all newly diagnosed children in Sweden have no first degree relative with type 1 diabetes.

[10] A person's first-degree relative is a parent, sibling, or child, sharing about half of their genes with the person. A second-degree relative is an uncle, aunt, nephew, niece, grandparent, grandchild, or half-sibling, sharing about one quarter of their genes with the person.

[11] The corresponding relative risk is a 15 times higher risk of getting type 1 diabetes for a person who has a sibling with type 1 diabetes, compared to someone in the general population.

[12] Etiologists have investigated climatic influences and environmental factors such as viral infections, early infant diet, and-or toxins (Dahlquist et al., 2011; Soltesz et al., 2007; The TEDDY Study Group, 2007; Dahlquist et al., 2005; Atkinson & Eisenbarth, 2001).

The complexity of the disease raises two concerns. First, some third factor, linked to the genetics or a disease-modifier, might affect not only the development of type 1 diabetes but also subsequent labor market outcomes. However, I use a fixed effect approach, allowing type 1 diabetes to be correlated with the individual-specific and time-invariant component of earnings, to handle the endogeneity concerns related to unobservable factors that are constant over time. In addition, sensitivity analysis indicates robustness to unobservables that do change over time, and a previous study by Lundborg, Nilsson, & Rooth (2014) shows robustness to sibling fixed effects and a shared family environment.

Second, individuals may hypothetically respond to a perceived risk of developing type 1 diabetes and have already changed their (children's) behavior (e.g., education, lifestyle, and family formation) before onset, with consequent changes in earnings. If so, we get estimates comprising not only effects of type 1 diabetes but also effects of selection through, e.g., changes in education or family formation. However, education, family formation, and labor market decisions are complex, and many factors besides the health history of one's family are likely to influence the final decision. Moreover, even if individuals are responding to a perceived risk of contracting type 1 diabetes, they will not necessarily develop the disease. They need to be exposed to a trigger factor (or factors) within a certain time-frame to develop the disease (Daneman, 2006). Or, they might be a carrier of an inherited genetic factor that influences resistance to type 1 diabetes (Pociot & McDermott, 2002). Consequently, only a few (and we cannot foresee whom) out of those individuals who potentially have changed their behavior will actually develop the disease. Therefore, such hypothetical behavioral changes will exist, if they exist, among both individuals *with* and *without* type 1 diabetes.

4 Data and descriptive analysis

This study uses the Econ-DISS database, which combines the national Diabetes Incidence Study in Sweden (DISS) with national population registers. Since 1983, DISS has registered all incidents of diabetes in the age group 15–34 (Östman et al., 1986, 2008). The type of diabetes was classified by the reporting physician according to current clinical diabetes criteria (1983–91: (WHO, 1980, 1985); 1992 onwards: (CDC The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus, 1997)). To each person with diabetes, Statistics Sweden added four control persons matched by age, gender, and municipality of residence at the time of diabetes diagnosis. Then, they added yearly data on demographic, socioeconomic, and employer-related variables from the LISA database (Statistics Sweden, 2011) for the period 1990–2005.[13]

[13] For details, see Steen Carlsson et al. (2010). The research program was approved by the Regional Ethical Review Board in Lund, Sweden (dnr 393/2005).

I select individuals diagnosed with type 1 diabetes at ages 28–34 years old (n = 1,717) during the years 1983–2000 (and their controls, n = 6,877) and follow yearly labor earnings over the period 1990–2005.[14] The earnings variable sums all (gross) earnings from employment and self-employment, including subsistence allowance and compensation for the first 14 days of a sickness absence.[15] The lower age limit (28 years old) takes into account that the median age when graduating from a Swedish university was, depending on the year, 26 or 27 years old during the studied period (OECD, 2008). To get the longest follow-up period

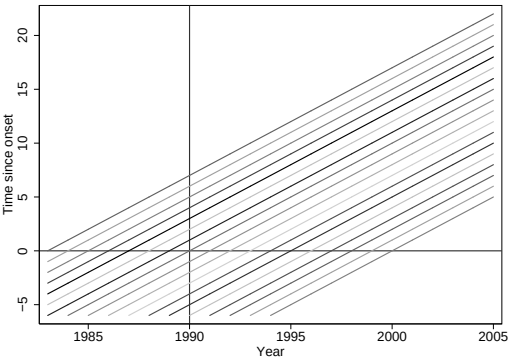

Figure 1: Number of years before and after type 1 diabetes onset (time=0 is the year of onset) for individuals grouped by year of onset. Each line represents a year of onset: onset in year 1983 is the top line, onset in 1984 is the next, ..., onset in year 2000 is the bottom line. The black vertical line at year 1990 marks the first year with earnings data.

possible for the regression analysis, I follow individuals either (1) from year 1990 (the first year with earnings data) and onwards if onset occurs before 1990, or (2) from two years prior to onset and onwards (and correspondingly, given the year of matching, for the controls). The two years prior to onset serve as reference years. The data set is an unbalanced panel following earnings and covariates in the years 1990–2005, with an average of 15.6 observations per individual. The available years with earnings data and the DISS registering onsets since 1983 results in a follow-up period reaching 22 years after onset. Figure 1 illustrates how onsets in each year (1983 to 2000) contribute to the follow-up period. In

[14] The study population consists of more men than women, because type 1 diabetes is more common in men (Pundziute-Lyckå et al., 2002).

[15] Using this definition of labor earnings, I will underestimate any effect of type 1 diabetes on earnings if individuals with type 1 diabetes have more sickness absences shorter than 14 days than the controls.

Econ-DISS, individuals with earnings data at 22 years after diabetes onset (those with onset in 1983) have no earnings data for their first six years with diabetes, while individuals with earnings data also before onset (those with onset in 1991 or later) have at most 14 years of follow up. Moreover, the individuals are born in 1948–1972 and together they cover the age-span 26–57 during the years 1990–2005. Figure 2 shows the ages at onset and the ages with earnings data across the different cohorts in Econ-DISS. For example, in 1990 (2005) the youngest cohort, born 1972, are 18 (33) years old and the oldest cohort, born 1948, are 42 (57) years. It is apparent from Figure 2 how the different cohorts contribute to the potential onset period. Given that onset occurs in ages 28–34 during the years 1983–2000, individuals in the oldest cohort, born 1948, have onset only at age 34, while individuals in the youngest cohort, born 1972, have onset only at age 28.

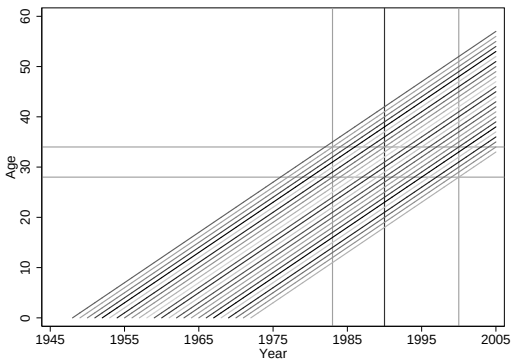

Figure 2: The age of each cohort (born 1948–1972) from birth up to year 2005. Each line represents a cohort. The cohort born 1948 is the top line, the cohort born 1949 is the next, ..., the cohort born 1972 is the bottom line. The gray horizontal lines at ages 28 and 34 mark the lower and upper bounds for onset ages. The gray vertical lines at years 1983 and 2006 mark the bounds for year of diagnosis. The resulting rectangular area marks the potential onset period. The black vertical line at year 1990 marks the first year with earnings data.

As background to the regression analysis, I graph annual earnings by time from type 1 diabetes onset. Figure 3 shows the development of mean earnings (at 2005 prices) for women and men in the type 1 diabetes group (and correspondingly for the controls). Compared to controls, Figure 3 indicates moderate developments for both women and men with type 1 diabetes. Notably, women with diabetes have higher earnings in the period preceding onset. This difference is alarming if it relates to systematic differences already existing between

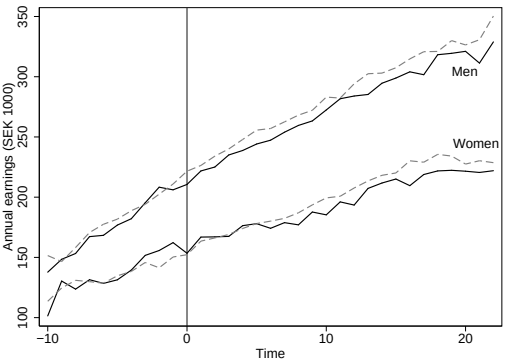

Figure 3: Mean annual earnings (in 2005 prices) before and after type 1 diabetes onset (time=0 is the year of onset/inclusion) for women and men with type 1 diabetes (black solid line) and controls (gray dashed line).

the groups before onset.[16] However, it seems unlikely that the higher earnings preceding onset represent a higher long-term earnings profile that is cut off by onset. Rather, this earnings difference could be attributed to chance or some third factor. Reassuringly, this difference is insignificant three years prior to onset (not shown) and only significant two years prior to onset (p= 0.0041, Table A.1 in Appendix A). The descriptive statistics, Table A.1 in Appendix A, suggest that the higher earnings of women in the diabetes group two years prior to onset (1) are unrelated to education, but (2) might be explained by more market work as women with type 1 diabetes have, on average, fewer small children (p=0.004) and less parental leave (p=0.005).

Previous evidence reports that women's earnings are negatively affected by having children (e.g., Budig & England (2001)), and type 1 diabetes intensifies pregnancy-related risks (Jonasson et al., 2007; Casson et al., 1997). If women with type 1 diabetes, as a group, have children to a lesser extent than controls throughout the studied period, then we can expect any negative earnings differences due to diabetes to be offset by higher earnings, due to fewer career interruptions from childbearing and childrearing. Figure 4 (describing the proportion of women with one or more children) suggests, however, that type 1 diabetes mainly involves a delayed (rather than abstained) family formation.[17]

[16] If women who will develop diabetes are already more sick before onset, their earnings ought to be lower rather than higher.

[17] The differences before onset in Figure 4 are significant only for the two years preceding onset. Figure B.2 in Appendix B, showing the ratio of average number of children in the diabetes group to the average number in the control group, indicates a delay in family formation and fewer children

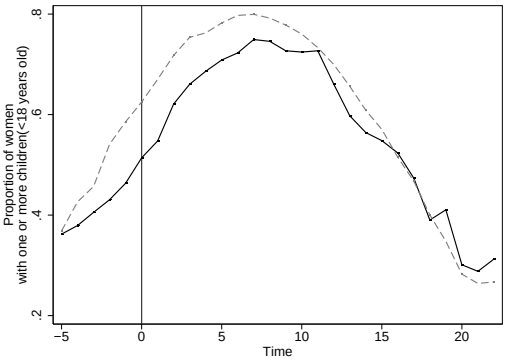

Figure 4: The percentage of women with one or more children (that are younger than 18) by the time since diabetes onset (time=0 is the year of onset/inclusion) for women with type 1 diabetes (black solid line) and controls (gray dashed line).

5 Econometric Strategy

To assess the lasting influence of onset, I model difference in the progression of annual labor earnings between individuals with and without type 1 diabetes. The chosen specification models time-specific differences conditional on individual fixed effects, capturing that onset of diabetes might impact directly on earnings and via its impact on health. Diabetes complications are likely to develop over time (Möllsten et al., 2010; Daneman, 2006) and personal traits and abilities are likely to determine both successful disease management and labor market outcomes (Wennick et al., 2011; Heckman, 2007; Cunha et al., 2006; Goldman & Smith, 2002). I use the following individual fixed effect specification of annual earnings for individual i in year t:

$$y_{it} = \alpha + \beta D_i + \sum_{time} \gamma_{time} TIME_{it} + \sum_{time} \delta_{time} D_i * TIME_{it} + \theta X_{it} + \lambda_t + \mu_i + \epsilon_{it} \quad (1)$$

The dependent variable y_{it} is the logarithm of annual labor earnings for individual i in each year t. D_i is a dummy variable indicating whether the individual has or will develop type 1 diabetes. $TIME_{it}$ is a vector of dummy variables representing time since onset in two-year intervals: 1–2 years before, the year of onset, 1–2, 3–4, ..., and 21–22 years after onset (and correspondingly time past inclusion for the controls). Two years prior to onset is the reference category for time. The interaction terms, $D_i * TIME_{it}$, capture time-specific earnings

for women with type 1 diabetes. Also, men with type 1 diabetes have fewer children on average.

differences between individuals with and without diabetes.[18] The coefficient on the tth interaction term shows how much the average annual earnings in the diabetes group deviate (in percentage points) from the average annual earnings of their controls during these two years, whereas the coefficient of the main variable D_i shows differences between the groups in the two reference years before onset. Note that the fixed effect estimator can only identify coefficients for time-varying regressors: that is, the variables that do not change over time, such as D_i, will be estimated embedded in the individual fixed effects.

X_{it} is a vector of earnings determinants, which holds only *age squared* in this specification.[19] λ_t is a vector of calendar time-fixed effects (i.e., dummies for each year 1990–2005) that control for aggregate changes in the economy over time. μ_i is a vector of individual-fixed effects and ϵ_{it} is an idiosyncratic error term. Given the large number of individuals, I remove μ_i from the estimation problem by using within-transformed data: i.e., removing individual-level averages from each side of Equation 1) instead of including a parameter for every individual (Baltagi, 2008). The fixed effect estimator is thereby unable to identify coefficients on time-invariant regressors (since e.g. $D_i = \bar{D}_i$).

I condition the analysis on years in which individuals are *lastingly* employed by excluding observations for each year in which an individual has annual earnings less than one Price Basic Amount (PBA) (between 29,700 SEK≈€2,970 and 39,400 SEK≈€3,940 depending on the year).[20] An average monthly salary is a multiple of one PBA. Excluding years with labor earnings below this threshold (19.9% of the observations for individuals with diabetes and 18.0% for controls) provides estimates conditional on working, which ought to be a conservative estimate of the full effect of type 1 diabetes. Nevertheless, I test the implication of the employment condition and the threshold chosen by presenting unconditional estimates and estimates conditional on having earnings>100,000 SEK.[21]

The individual fixed effects model relies on variation within individuals across time, thereby failing to identify any diabetes-induced differences between individuals with and without type 1 diabetes that do not vary over time. Instead, the fixed effect estimates are adjusted for such differences, which will be captured by the individual-specific effects. Another implication of the fixed effect model's use only of variation within individuals is that it will not matter if an individual has a

[18] I choose two-year intervals to allow for flexibility and, at the same time, limit the number of variables.

[19] Using individual fixed effects, I cannot control for (linear) age and time simultaneously, because age is a function of time. The year dummies will pick up cohort effects and age squared reflects the income effect associated with increasing age.

[20] The PBA follows the price trend in the country year after year and is set by the government. The PBA is calculated based on changes in the general price level one year at a time. The measure is used, for example, to ensure that sickness benefits, student grants, etc., do not decline in value because of an increase in the general price level.

[21] Studying the returns for education in Sweden, Antelius & Björklund (2000) report that the results when excluding earnings<100,000 SEK are similar to the results for hourly wage.

very high-level or a very low-level earnings profile, because it is only the within-individual variation that will show up as explanatory power. Consequently, the fixed effects model captures only changes in earnings over time, while an ordinary least square (OLS) model (using both within- and between-individual variations) captures overall differences in earnings between individuals with and without type 1 diabetes. Thus, the two models are capturing somewhat different aspects of the diabetes-earnings relationship, besides the controls for individual-specific factors.

Testing whether it is necessary to control for individual-specific heterogeneity, F tests indicate that there are significant individual effects in all my specifications, implying that fixed effects specifications are favorable to pooled OLS.[22] Still, to assess the influence of time-invariant unobservable factors and variation both within and between individuals, I use pooled data and estimate also OLS specifications without the individual fixed effects. To control, at least partly, for the time-invariant observable component of earnings also in the OLS specifications, I add *age, years of education at onset, parental level of education,* and *having a non-native parent*, in addition to the earnings determinant in X_{it}. Thus, we could attribute most of the remaining differences between the fixed effects and the OLS estimates to time-invariant factors that are unobservable.

Controlling for individual fixed effects, I control not only for time-invariant factors that are truly unobservable (e.g., permanent ability), but also for any factor at the individual level that does not change over time. Onset of type 1 diabetes occurred in young adulthood (age 28–34), when most education has been completed; therefore, education should not change much over time. However, I do not control for further education, retraining, and other mediators (e.g., marital status, children, occupation, and comorbidities), through which onset of type 1 diabetes may affect earnings. Consequently, I allow the potential earnings effect of diabetes to go through such variables. If controlled for, mediators may absorb some of the effect related to diabetes. Therefore, the coefficients on the mediators would not truly capture their actual effects and, even more importantly, including these variables may bias the estimate for type 1 diabetes. There are studies arguing that abilities might mediate the relationship between child health and later outcomes, because child abilities are influenced by child health (see, e.g., Salm & Schunk (2012), Currie et al. (2010), and Heckman (2007)). However, onset ought to have little impact on abilities, given that it occurs at ages when IQ and other personal characteristics have mostly stabilized,[23] suggesting that abilities should be controlled for.

I repeat the analysis of Equation 1 stratified by level of education (compulsory, upper secondary, and university) to allow for socioeconomic heterogeneity in

[22] I test the suitability of the fixed effects model over the random effects model with a Hausman test. Results from the F test and Hausman test are available on request.

[23] See, e.g., Cunha et al. (2006) for an extensive discussion on human capabilities and critical periods for ability formation.

the estimates of type 1 diabetes due to the education-health interplay (Goldman & Smith, 2011, 2002; Heckman, 2007; Grossman, 1972a,b). Socioeconomic background translates into own socioeconomic status, as Case et al. (2005), for instance, show that the impact of parent's education is absorbed by own socioeconomic status at ages 23 and 33. Further, successful treatment of type 1 diabetes demands regular self-monitoring (Daneman, 2006), and different educational levels may open up the way to different occupations and workplaces that affect individuals' capacity to self-monitor their condition.

Earnings differentials could be interpreted in terms of productivity or labor supply differences, but the Econ-DISS data are not conclusive as to whether changes in earnings are due to changes in sickness absence, hours worked, and/or wages. However, the data comprise information on the usage of the social welfare system. To test these potential mechanisms through which type 1 diabetes may affect earnings, I repeat the analysis of Equation 1, but with the probability of holding unemployment benefit, student grant and/or loan, parental leave allowance, sickness absence (exceeding 14 days), and disability pension as outcome variables. In addition, I test whether type 1 diabetes affects the probability of having earnings exceeding one Price Basic Amount, i.e., the labor supply condition I use in the earnings analysis.

Before continuing, the following paragraphs will raise some robustness concerns, which I will return to more thoroughly in the sensitivity analysis in Section 7. Not only time-invariant but also time-variant unobservable factors, which are not controlled for by the fixed effect approach, may be important. Reassuringly, Lundborg, Nilsson, & Rooth (2014) show that sibling fixed effects and the influence of a shared childhood environment appear less significant for diabetes than for many other diagnoses and more general measures of health. They also conclude that genetic influences are limited for the health-earnings relationship, when comparing findings for different types of twins. Still, a disease trigger might affect the development of type 1 diabetes and could, at the same time, possibly trigger other diseases that affect earnings. To test whether my estimates might be biased by some other time-variant group-specific characteristic besides type 1 diabetes, Section 7.1 presents placebo estimates where I have moved the time of onset two years back in time. This analysis is important also to exclude third factors related to the pre-onset differences shown descriptively for women in Section 4.

Earnings differentials could be driven by selection into employment. To correct for such selection, it is common to use some form of two-part model.[24] Such models are however sensitive to the use of proper exclusion restrictions (Puhani, 2000), and rely on variables acting as instruments that affect the selection process into employment, but not the earnings equation in question. Instead, I use the fixed effect approach and limit my analysis to observations of earnings

[24] Minor (2011) uses the correction for selection bias proposed by Heckman (1976).

exceeding one PBA. Using a fixed effect model, I implicitly control for selection, as controlling for fixed effects also controls for some of the unobservable factors that might lead to self-selection into employment. Section 7.2 looks further into the issue of selection.

6 Results

6.1 Type 1 diabetes and ln(earnings)

This section presents the estimation results for the interaction terms in Equation 1. These estimates capture time-specific average earnings differences between individuals with and without type 1 diabetes throughout 22 years following onset. In Table 1, columns (1) and (2) report for women, and columns (3) and (4) report for men. Columns (1) and (3) contain fixed effect (FE) estimates conditional on age, individual-fixed and year-fixed effects, while columns (2) and (4) contain OLS estimates conditional on age, year-fixed effects, and time-invariant covariates: years of education at onset, parental education, and non-native parent.

The results show significant negative estimates of type 1 diabetes.[25] For women and men alike, the estimates increase over time, although women appear affected in a more substantial and pervasive manner. The FE estimates indicate that type 1 diabetes reduces women's average annual earnings by 7.45 percentage points in the year of onset and by as much as 23.5 percentage points in the period 21–22 years following onset. These estimates are sizable and can be compared to the average difference in wages between blue and white collar workers in Sweden. From 1990 to 2005, white collar workers' wages increased in general with 42% and blue collar workers' with 16%, i.e., the increase in wages differed with 26 percentage points between white and blue collar workers (Ekonomifakta, 2015). The FE estimates for men are weaker than those for women, in terms of both magnitude and significance. Onset of type 1 diabetes first appears to affect men's earnings after 5 years with the disease, whereas women's earnings appear affected already in the year of onset. With significant FE estimates ranging from -4.12 percentage points (5–6 year period) to -11.5 percentage points (21–22 year period), the increase over time is also more modest for men.

Comparing the FE and OLS estimates, the OLS estimates are smaller for both women and men. Without controls for individual-specific effects (OLS, using both within and between individual variation), the diabetes-time interactions capture both the potential negative impact of diabetes and the potential positive impact of individual-specific factors (e.g., abilities) favoring higher earnings and more successful diabetes management. Consequently, individuals with these

[25] The results are robust to splitting the sample by onset before and after year 1990 to account for the fact that cohorts contribute differently to the potential onset period (see Figures 1–2). Results are available on request.

Table 1: Type 1 diabetes and ln(Earnings) — Results for the FE and the OLS estimator.

	Women		Men	
	(1)	(2)	(3)	(4)
	FE	OLS	FE	OLS
year of onset	-0.0745**	-0.0677**	-0.0137	-0.0329
	(0.0333)	(0.0343)	(0.0186)	(0.0204)
1–2 years past	-0.0720**	-0.0757**	-0.0166	-0.0319
	(0.0349)	(0.0333)	(0.0200)	(0.0213)
3–4 years past	-0.0865**	-0.0894**	-0.0209	-0.0414*
	(0.0367)	(0.0356)	(0.0210)	(0.0225)
5–6 years past	-0.104***	-0.104***	-0.0412*	-0.0581**
	(0.0350)	(0.0344)	(0.0215)	(0.0226)
7–8 years past	-0.131***	-0.123***	-0.0313	-0.0428*
	(0.0369)	(0.0355)	(0.0225)	(0.0243)
9–10 years past	-0.148***	-0.131***	-0.0456**	-0.0445*
	(0.0395)	(0.0381)	(0.0229)	(0.0257)
11–12 years past	-0.170***	-0.129***	-0.0361	-0.0332
	(0.0412)	(0.0392)	(0.0239)	(0.0276)
13–14 years past	-0.142***	-0.0983**	-0.0761***	-0.0703**
	(0.0417)	(0.0401)	(0.0258)	(0.0303)
15–16 years past	-0.176***	-0.135***	-0.0479*	-0.0479
	(0.0447)	(0.0433)	(0.0278)	(0.0341)
17–18 years past	-0.203***	-0.150***	-0.0368	-0.0465
	(0.0496)	(0.0502)	(0.0307)	(0.0366)
19–20 years past	-0.186***	-0.121**	-0.0638*	-0.0471
	(0.0530)	(0.0537)	(0.0364)	(0.0446)
21–22 years past	-0.235***	-0.155*	-0.115**	-0.0507
	(0.0722)	(0.0807)	(0.0464)	(0.0623)
Diabetes		0.0964***		0.0156
		(0.0308)		(0.0214)
Observations	30266	30237	66732	66588
Individuals	2699		5525	
R2	0.307	0.250	0.299	0.249

Robust clustered standard errors in parentheses. *** p<0.01, ** p<0.05. * p<0.1. Age and year-fixed effects included in all models. OLS include also years of education at onset, parental education, and non-Native parent.

factors may compensate for some of the negative impact that the disease has on others in the diabetes group. When conditioning on individual-specific effects (FE, using only within-individual variation), the interactions capture only the impact of diabetes, and individuals with type 1 diabetes will only be compared to others with the same individual-specific factors, and can no longer compensate, within the diabetes group, for those with less successful diabetes management and larger drops in earnings. Thereby, differences in health will no longer be masked by the positive influence from abilities on both health and earnings.

6.2 Type 1 diabetes and ln(earnings) by level of education

Repeating the analyses of Equation 1 by level of education, Table 2 presents the results for women in columns (1)–(3) and men in columns (4)–(6). Columns (1) and (4) hold the results for individuals with only compulsory education, columns (2) and (5) for individuals with upper secondary education, and columns (3) and (6) for university education. The negative estimates for type 1 diabetes on women's earnings persist throughout all levels of education. Earnings for women with only compulsory education, followed by women with university education, appear to be most affected. The sizable estimates for university-educated women may appear striking. Given evidence linking high ability levels to higher education, better health behaviors, and better health (Heckman, 2007; Cunha et al., 2006), we may expect the more educated to have better health, with consequent lower estimates on the diabetes-earnings relationship. Still, the sizable estimates for women throughout all levels of education suggest that this relationship is mediated by some third factor. For example, we know that having children (Budig & England, 2001) and the amount of stress a women faces in her daily life (Hamermesh & Lee, 2007) might affect earnings. Possibly, such factors might also affect the potential health impact across socioeconomic groups.

The delay in family formation for women with type 1 diabetes, shown in Figure 4, appears to be driven by university-educated women (see Figure B.3 in Appendix B), while women with only compulsory education, with and without diabetes, have children to the same extent (see Figure B.4 in Appendix B). Given that university study delays family formation (Boschini et al., 2011; Lundin et al., 2008; Björklund, 2006) and that type 1 diabetes amplifies the risk of pregnancy-related problems when delaying fertility (Jonasson et al., 2007; Casson et al., 1997), this socioeconomic heterogeneity might relate to an enhanced tradeoff between a career and motherhood for women with type 1 diabetes. Such an enhanced tradeoff may, in part, explain the sizable estimates for university-educated women with diabetes. Adding a (linear) control for having an additional child to Equation 1 strengthens the negative diabetes estimates for university-educated women, while adding a (dummy) control for having ones first child after

Table 2: Type 1 diabetes and ln(Earnings) — Results for compulsory, upper secondary, and university education.

	Women			Men		
	(1)	(2)	(3)	(4)	(5)	(6)
	Comp.	Upp. sec.	Uni.	Comp.	Upp. sec.	Uni.
year of onset	-0.229**	-0.0824**	-0.0305	-0.0595	0.00725	-0.0302
	(0.113)	(0.0389)	(0.0620)	(0.0455)	(0.0222)	(0.0382)
1–2 years past	-0.0586	-0.0534	-0.122*	-0.00577	-0.0323	0.0134
	(0.109)	(0.0404)	(0.0676)	(0.0438)	(0.0241)	(0.0401)
3–4 years past	-0.0930	-0.0987**	-0.0943	0.00911	-0.0519**	0.0302
	(0.107)	(0.0428)	(0.0703)	(0.0446)	(0.0245)	(0.0446)
5–6 years past	-0.168	-0.0825**	-0.132*	-0.0283	-0.0742***	0.0319
	(0.104)	(0.0406)	(0.0690)	(0.0427)	(0.0254)	(0.0449)
7–8 years past	-0.257**	-0.0989**	-0.162**	-0.0333	-0.0496*	0.0378
	(0.111)	(0.0434)	(0.0723)	(0.0458)	(0.0267)	(0.0486)
9–10 years past	-0.324***	-0.101**	-0.155**	-0.0333	-0.0564**	0.0121
	(0.122)	(0.0485)	(0.0738)	(0.0464)	(0.0270)	(0.0515)
11–12 years past	-0.243**	-0.126**	-0.202***	-0.0366	-0.0352	0.0174
	(0.119)	(0.0535)	(0.0750)	(0.0476)	(0.0280)	(0.0525)
13–14 years past	-0.286**	-0.0891*	-0.182**	-0.0610	-0.0831***	-0.0215
	(0.122)	(0.0526)	(0.0777)	(0.0524)	(0.0317)	(0.0548)
15–16 years past	-0.378***	-0.115*	-0.200**	-0.0415	-0.0420	0.00265
	(0.129)	(0.0589)	(0.0797)	(0.0614)	(0.0331)	(0.0602)
17–18 years past	-0.324**	-0.166**	-0.226***	-0.0665	-0.0604	0.0950
	(0.139)	(0.0672)	(0.0864)	(0.0606)	(0.0394)	(0.0640)
19–20 years past	-0.306**	-0.158**	-0.236***	-0.0490	-0.107**	0.0614
	(0.144)	(0.0763)	(0.0910)	(0.0624)	(0.0472)	(0.0844)
21–22 years past	-0.411*	-0.217**	-0.241**	-0.150*	-0.102*	0.000839
	(0.218)	(0.104)	(0.103)	(0.0895)	(0.0616)	(0.0892)
Observations	3667	16232	10312	12235	34587	19682
Individuals	414	1593	987	1124	3080	1662
R2	0.254	0.275	0.358	0.236	0.264	0.402

Robust clustered standard errors in parentheses.

*** p<0.01, ** p<0.05. * p<0.1.

Age, individual-fixed, and year-fixed effects included in all models.

onset (onset occurring at ages 28–34) weakens the negative diabetes estimates.[26] We might expect that other life constraints, such as having an additional child (i.e., adding the linear control), intensify the earnings penalty of diabetes, while it might appear strange that starting a family (i.e., adding the dummy control) alleviates the earnings penalty of diabetes. However, this finding of a weaker diabetes estimate when adding the dummy control is likely to be a consequence of a career-family tradeoff, as only the healthiest women with diabetes are likely to start a family this late in life. Moreover, this pattern does not show when adding the same controls for women with only compulsory education, who appear to have, on average, children earlier and to the same extent as their peers without diabetes (see Figure B.4 in Appendix B).[27]

Table 2 for men shows that men with upper secondary education appear to be particularly vulnerable to the long-term consequences of type 1 diabetes. In contrast, university-educated men have positive estimates throughout most years following onset. Note that these estimates are insignificant and small in size. Nonetheless, they might indicate positive selection if the healthiest and most able men within the diabetes group are over represented among those men who actually work. I will return to this potential selection issue in Section 7.2.[28] In summary, repeating the main analysis by level of education reports sizable and significant estimates for women at all levels of education. As in the main analysis, the estimates for men are weaker; however, men with upper secondary education appear to be particularly vulnerable.

6.3 Mechanisms linking type 1 diabetes and earnings

To test potential mechanisms through which type 1 diabetes may affect earnings, I repeat the analysis of Equation 1 using a linear fixed effect probability model and the following outcome variables: having earnings>1PBA, unemployment benefit, student grant and/or loan, parental leave allowance, sickness absence (a period exceeding 14 days), and disability pension. For all outcomes, except for earnings>1PBA, I continue to condition the analysis on earnings exceeding one PBA. Table 3 (women) and Table 4 (men) present the results.

Comparing column (1) of Tables 3 and 4, women's probability of having earnings exceeding one PBA is significantly lower than for controls at the later periods after onset, whereas men's probability appears unaffected, except at the year of onset when men's probability of having earnings exceeding one PBA is, instead, significantly higher. Overall, Table 3 indicates that the shown negative

[26] Results are available on request.

[27] Compared to the university level analysis, the change in estimates for the *compulsory* analysis are more modest, and the direction of the change alters both for linear children and the dummy for child. Results are available on request.

[28] Having children does not appear to be a mediator for men, as the diabetes estimates are robust to additional controls.

Table 3: Potential mechanisms through which type 1 diabetes may affect women's earnings — Fixed effect results for the probability of having earnings>1PBA, unemployment benefit, student grant, parental leave allowance, sickness absence, and disability pension.

	(1)	(2)	(3)	(4)	(5)	(6)
	1PBA	Unemp.	Student	Parent	Sick	Pension
year of onset	-0.0203	-0.0239	0.0136	0.000133	0.446***	0.000333
	(0.0239)	(0.0261)	(0.0201)	(0.0306)	(0.0404)	(0.00392)
1–2 years past	-0.0287	-0.0449*	-0.0147	0.0165	0.182***	0.00467
	(0.0244)	(0.0262)	(0.0211)	(0.0320)	(0.0340)	(0.00498)
3–4 years past	-0.0260	-0.0184	-0.0241	0.0451	0.0998***	0.0104**
	(0.0261)	(0.0282)	(0.0207)	(0.0360)	(0.0312)	(0.00495)
5–6 years past	-0.0171	-0.0144	-0.0342*	0.0535	0.104***	0.0142*
	(0.0271)	(0.0288)	(0.0203)	(0.0390)	(0.0318)	(0.00751)
7–8 years past	-0.0398	-0.0282	-0.0603***	0.0548	0.165***	0.0152*
	(0.0293)	(0.0294)	(0.0199)	(0.0419)	(0.0341)	(0.00852)
9–10 years past	-0.0354	-0.0364	-0.0365*	0.0805*	0.140***	0.0213**
	(0.0310)	(0.0303)	(0.0208)	(0.0434)	(0.0367)	(0.0106)
11–12 years past	-0.0419	-0.0179	-0.0309	0.0890**	0.182***	0.0173
	(0.0311)	(0.0319)	(0.0209)	(0.0445)	(0.0387)	(0.0107)
13–14 years past	-0.0523	-0.0139	-0.0470**	0.0712	0.161***	0.0332**
	(0.0332)	(0.0324)	(0.0218)	(0.0466)	(0.0408)	(0.0148)
15–16 years past	-0.0308	0.00238	-0.0533**	0.0767	0.168***	0.0299*
	(0.0344)	(0.0338)	(0.0212)	(0.0469)	(0.0432)	(0.0156)
17–18 years past	-0.0716*	0.0136	-0.0490**	0.102**	0.192***	0.0329*
	(0.0369)	(0.0348)	(0.0218)	(0.0488)	(0.0478)	(0.0184)
19–20 years past	-0.0970**	0.0175	-0.0428*	0.0931*	0.254***	0.0444*
	(0.0429)	(0.0380)	(0.0219)	(0.0529)	(0.0541)	(0.0257)
21–22 years past	-0.0978**	0.0140	-0.0471**	0.0868	0.135**	0.101**
	(0.0479)	(0.0357)	(0.0222)	(0.0606)	(0.0626)	(0.0438)
Observations	38666	30266	30266	30266	26286	30266
Individuals	2843	2699	2699	2699	2663	2699
R2	0.0167	0.0230	0.00979	0.0978	0.0196	0.0361

Robust clustered standard errors in parentheses.

*** p<0.01, ** p<0.05. * p<0.1.

Age, individual-fixed, and year-fixed effects included in all models.

Table 4: Potential mechanisms through which type 1 diabetes may affect men's earnings — Fixed effect results for the probability of having earning>1PBA, unemployment benefit, student grant, parental leave allowance, sickness absence, and disability pension.

	(1)	(2)	(3)	(4)	(5)	(6)
	1PBA	Unemp.	Student	Parent	Sick	Pension
year of onset	0.0346**	-0.0309**	-0.00946	-0.0185	0.311***	-0.000581
	(0.0139)	(0.0154)	(0.0114)	(0.0194)	(0.0287)	(0.00252)
1–2 years past	0.0271*	-0.00942	-0.0144	0.0135	0.0353**	0.000809
	(0.0144)	(0.0148)	(0.0113)	(0.0208)	(0.0176)	(0.00312)
3–4 years past	0.0194	-0.0113	-0.00933	0.0189	0.00297	0.000674
	(0.0154)	(0.0156)	(0.0115)	(0.0228)	(0.0167)	(0.00381)
5–6 years past	0.0201	-0.0101	-0.00518	-0.00338	0.00134	0.000440
	(0.0160)	(0.0157)	(0.0114)	(0.0244)	(0.0177)	(0.00434)
7–8 years past	0.0146	-0.0131	-0.0129	0.0191	0.00611	0.00414
	(0.0163)	(0.0162)	(0.0109)	(0.0255)	(0.0181)	(0.00480)
9–10 years past	0.00982	-0.00365	-0.00688	0.0113	-0.0143	0.00840
	(0.0172)	(0.0173)	(0.0111)	(0.0269)	(0.0195)	(0.00545)
11–12 years past	0.00320	-0.0269	-0.00787	0.0155	-0.0386*	0.00918*
	(0.0180)	(0.0175)	(0.0115)	(0.0278)	(0.0199)	(0.00552)
13–14 years past	-0.000919	0.00277	0.000252	0.0145	-0.0258	0.00808
	(0.0188)	(0.0183)	(0.0116)	(0.0285)	(0.0218)	(0.00570)
15–16 years past	-0.00237	-0.0238	-0.0120	0.0226	-0.0142	0.00750
	(0.0206)	(0.0192)	(0.0115)	(0.0296)	(0.0249)	(0.00668)
17–18 years past	0.00128	-0.0268	-0.00914	0.00830	-0.00292	0.0137
	(0.0231)	(0.0213)	(0.0116)	(0.0308)	(0.0268)	(0.0102)
19–20 years past	0.00993	-0.0208	-0.00418	0.0350	-0.00861	0.0189
	(0.0257)	(0.0228)	(0.0116)	(0.0334)	(0.0316)	(0.0126)
21–22 years past	-0.0426	-0.0556*	-0.00391	0.00682	-0.0199	0.0202
	(0.0358)	(0.0295)	(0.0120)	(0.0368)	(0.0410)	(0.0175)
Observations	78839	66732	66732	66732	57328	66732
Individuals	5751	5525	5525	5525	5438	5525
R2	0.00946	0.0222	0.0109	0.0649	0.0138	0.0132

Robust clustered standard errors in parentheses.

*** p<0.01, ** p<0.05. * p<0.1.

Age, individual-fixed, and year-fixed effects included in all models.

link between type 1 diabetes and women's earnings may go through absenteeism due to sickness, disability, and parental leave. Surprisingly, type 1 diabetes does not appear to increase men's probability of sickness absence after the two-year period following onset: instead, type 1 diabetes (although insignificantly on the whole) appears to decrease men's probability at the later years following onset. This finding could indicate that the healthiest men within the diabetes group are selected into this study, thereby subjecting the estimates to a downward bias. I discuss this possibility in the sensitivity analysis in Section 7.2.

To provide a more nuanced picture of the absenteeism due to bad health, Figure B.1 in Appendix B, displays differences in sickness absence using a ratio consisting of the mean number of periods of sickness (exceeding 14 days) in the type 1 diabetes group divided by the mean number in the control group. Sickness absence increases dramatically at the time of onset. Compared to women, men have a faster return after the peak, as men's ratio falls back in a steeper manner, although neither men nor women fall back to the absence levels held by their peers (i.e., to a ratio of one).

7 Additional Evidence and Sensitivity Analysis

7.1 Placebo Effects

Testing the robustness of my results, I present placebo estimates where I have moved the time of onset two years back in time (i.e., before onset of type 1 diabetes actually took place). This placebo test is indicative of whether the found earnings differentials are indeed related to type 1 diabetes onset, or if some other time-variant group-specific characteristic is influencing both the probability of onset and a decline in labor earnings. Supporting my empirical strategy and confirming my main conclusions, Table 5 shows no such significance of the FE pre-onset placebo effects.

7.2 Selection

Excluding observations for the years in which individuals have earnings less than one PBA, I do not account for what their earnings would have been if they had been included. Thereby, I might underestimate the full effect of type 1 diabetes on earnings, as the excluded individuals are likely to be those in worst health.[29] However, I reduce potential bias stemming from self-selection into employment

[29] Observations for women in the type 1 diabetes group are comparatively more often excluded, as shown by women's negative long-term probabilities of having earnings>1PBA (column 1) in Table 3. Also, Figure B.5 in Appendix B, showing a ratio between excluded observations in the type 1 diabetes group and the control group, indicates that women with type 1 diabetes are more often excluded than are men with type 1 diabetes.

Table 5: Type 1 diabetes and ln(Earnings) — Fixed effect results for placebo test, artificially moving back onset of type 1 diabetes two years in time.

	(1)	(2)
	Women	Men
1–2 years prior	0.0544	0.00906
	(0.0372)	(0.0213)
year of onset	-0.0173	-0.00358
	(0.0434)	(0.0262)
1–2 years past	-0.0122	-0.00505
	(0.0413)	(0.0263)
3–4 years past	-0.0268	-0.00911
	(0.0432)	(0.0271)
Observations	31850	70285
Individuals	2718	5545
R2	0.309	0.315

Robust clustered standard errors in parentheses.

*** p<0.01, ** p<0.05. * p<0.1.

Age, indivudual-fixed, and year-fixed effects included in all models.

as the individual fixed effects control for the time-invariant factors that might lead to selection. If selection is an important issue, it ought to be particularly important for individuals diagnosed with type 1 diabetes when entering the labor market. The Swedish labor market is characterized by high job security and strong unions. We could, therefore, expect it to be easier to remain employed than to gain new employment while facing the onset of a chronic disease. Being diagnosed early in life, individuals are less likely to be established in the labor market at the time of onset and, thereby, are more likely to be exposed to selection. Therefore, I test the sensitivity of the results to the inclusion of individuals with type 1 diabetes onset before age 30.

When excluding individuals who ought to be the most sensitive to selection (i.e., individuals contributing to an underestimation of the full effect of type 1 diabetes), we could expect the estimates in the restricted sample (with onset ages 31–34) to increase in the presence of selection compared to estimates of the full sample (with onset ages 28–34). However, when we compare columns (1) and (2) of Table 6 for women, and columns (3) and (4) for men, no dramatic changes appear after restricting the sample. The changes that do appear are not statistically significant and are probably more likely to be due to changes in the composition of the samples than to selection into employment.

Table 6: Type 1 diabetes and ln(Earnings) — Fixed effect results for a sub sample excluding individuals with onset before age 30.

	Women		Men	
	(1)	(2)	(3)	(4)
	all	sub sample	all	sub sample
year of onset	-0.0745**	-0.0804**	-0.0137	-0.00236
	(0.0333)	(0.0398)	(0.0186)	(0.0212)
1–2 years past	-0.0720**	-0.0638	-0.0166	-0.0126
	(0.0349)	(0.0405)	(0.0200)	(0.0227)
3–4 years past	-0.0865**	-0.0868**	-0.0209	-0.0190
	(0.0367)	(0.0431)	(0.0210)	(0.0241)
5–6 years past	-0.104***	-0.105**	-0.0412*	-0.0526**
	(0.0350)	(0.0413)	(0.0215)	(0.0250)
7–8 years past	-0.131***	-0.105**	-0.0313	-0.0420
	(0.0369)	(0.0433)	(0.0225)	(0.0260)
9–10 years past	-0.148***	-0.121***	-0.0456**	-0.0449*
	(0.0395)	(0.0459)	(0.0229)	(0.0264)
11–12 years past	-0.170***	-0.115**	-0.0361	-0.0369
	(0.0412)	(0.0482)	(0.0239)	(0.0276)
13–14 years past	-0.142***	-0.121**	-0.0761***	-0.0879***
	(0.0417)	(0.0492)	(0.0258)	(0.0300)
15–16 years past	-0.176***	-0.138***	-0.0479*	-0.0480
	(0.0447)	(0.0510)	(0.0278)	(0.0323)
17–18 years past	-0.203***	-0.182***	-0.0368	-0.0448
	(0.0496)	(0.0590)	(0.0307)	(0.0362)
19–20 years past	-0.186***	-0.207***	-0.0638*	-0.0713*
	(0.0530)	(0.0662)	(0.0364)	(0.0411)
21–22 years past	-0.235***	-0.233**	-0.115**	-0.133**
	(0.0722)	(0.0909)	(0.0464)	(0.0537)
Observations	30266	21008	66732	47641
Individuals	2699	1844	5525	3982
R2	0.307	0.328	0.299	0.284

Robust clustered standard errors in parentheses.

*** p<0.01, ** p<0.05. * p<0.1.

Age, individual-fixed, and year-fixed effects included in all models.

7.3 Alternative thresholds for the employment condition

Testing the implication of the employment condition and the chosen threshold of having earnings>1 PBA applied in the earnings analysis, Table 7 reports unconditional estimates (columns 1 and 3), and estimates conditional on having earnings>100,000 SEK (columns 2 and 4). Confirming the estimates to be conservative in the main analyses, the unconditional estimates increase greatly compared to the estimates conditional on a threshold value of either 1 PBA (as in the main analysis) or 100,000 SEK. Also confirmative of the main results, conditioning on the larger threshold of 100,000 SEK continues to give significant estimates, but reduces the estimates throughout for women. For men, the higher threshold introduces more significant time periods and the estimates maintain their size. Therefore, we need to keep in mind that the estimates are conditional on earnings>1 PBA when interpreting the results, as the size of the estimates is sensitive to the chosen threshold, suggesting that diabetes influences earnings via both wages and labor supply.

7.4 Average Effects

Lundborg, Nilsson, & Rooth (2014), Minor (2013, 2011), and Steen Carlsson et al. (2010) have previously looked at average effects of type 1 diabetes at specific points in time. For comparability, I too provide average effects by replacing the different type 1 diabetes dummies in Equation 1 with a single dummy variable that changes from zero to one at the year of onset. Testing the results sensitivity to the fixed effect estimator, I also run a difference-in-difference model specification that looks at differences between individuals with and without type 1 diabetes before and after onset, while controlling for unobservable variables.

Irrespective of which estimator I use, the results (Table 8) confirm my previous findings and indicate significant negative average effects of type 1 diabetes that are particularly sizable for women: about -11.5% (women) and -3.9% (men). These results are in line with previous results from Swedish settings. Lundborg, Nilsson, & Rooth (2014) find a 19.8% earnings penalty of diabetes for men when controlling for schooling and sibling fixed effects. Steen Carlsson et al. (2010) report associations that are -8.1% for women and -4.2% for men.[30] Minor (2013, 2011) presents contradicting findings from a US setting. While both studies report insignificant average associations, Minor (2013) adds insignificant associations for (linear) duration of diabetes. However, our results are not fully comparable. Besides comprising populations with different duration of type 1 diabetes and different ages at onset, the US and the Swedish labor markets are likely to offer different incentives and possibilities.

[30] These estimates are reported for individuals who have lived ten or more years with type 1 diabetes and have upper secondary education only.

Table 7: Type 1 diabetes and ln(Earnings) — Fixed effect results with alternative employment thresholds.

	Women		Men	
	(1)	(2)	(3)	(4)
	if all	if earnings>100'	if all	if earnings>100'
year of onset	-0.362*	-0.0739***	0.176	-0.0242**
	(0.191)	(0.0194)	(0.128)	(0.0123)
1–2 years past	-0.384*	-0.0323	0.213	-0.0149
	(0.210)	(0.0197)	(0.133)	(0.0124)
3–4 years past	-0.264	-0.0357*	0.191	-0.0163
	(0.228)	(0.0216)	(0.150)	(0.0138)
5–6 years past	-0.340	-0.0718***	0.0918	-0.0192
	(0.239)	(0.0236)	(0.156)	(0.0147)
7–8 years past	-0.449*	-0.0831***	0.123	-0.0255
	(0.257)	(0.0239)	(0.163)	(0.0156)
9–10 years past	-0.361	-0.0862***	-0.0608	-0.0372**
	(0.278)	(0.0263)	(0.175)	(0.0166)
11–12 years past	-0.556*	-0.104***	-0.158	-0.0380**
	(0.288)	(0.0274)	(0.183)	(0.0175)
13–14 years past	-0.557*	-0.109***	-0.254	-0.0569***
	(0.303)	(0.0293)	(0.192)	(0.0187)
15–16 years past	-0.415	-0.124***	-0.298	-0.0456**
	(0.320)	(0.0309)	(0.211)	(0.0203)
17–18 years past	-0.754**	-0.136***	-0.124	-0.0257
	(0.340)	(0.0340)	(0.242)	(0.0231)
19–20 years past	-1.259***	-0.146***	-0.293	-0.0327
	(0.414)	(0.0382)	(0.280)	(0.0277)
21–22 years past	-1.350***	-0.129***	-0.489	-0.0979***
	(0.482)	(0.0458)	(0.379)	(0.0363)
Observations	38666	24518	78839	60575
Individuals	2843	2569	5751	5328
R2	0.0202	0.467	0.0103	0.460

Robust clustered standard errors in parentheses.

*** p<0.01, ** p<0.05. * p<0.1.

Age, individual-fixed, and year-fixed effects included in all models.

Table 8: Type 1 diabetes and ln(Earnings) — Results for average effects.

	Women		Men	
	(1)	(2)	(3)	(4)
	FE	DiD	FE	DiD
Type 1 DM	-0.119***	-0.111***	-0.0319*	-0.0454**
	(0.0284)	(0.0266)	(0.0167)	(0.0177)
Observations	30266	30237	66732	66588
Individuals	2699		5525	
R2	0.305	0.249	0.299	0.250

Robust clustered standard errors in parentheses.

*** p<0.01, ** p<0.05. * p<0.1.

Age, individual-fixed, and year-fixed effects included in all models.

8 Discussion

This study indicates that onset of type 1 diabetes in young adulthood (28–34 years old) has statistically and quantitatively significant negative consequences for annual labor earnings. Beyond the immediate impact at onset, the negative link increases over time, especially for women's earnings. I provide estimates based on Swedish longitudinal register data that are robust against selection into low education and selection on time-invariant unobservable factors. First, by focusing on health in young adults (who generally are in their early careers but have already made their educational choices), I minimize any influence on earnings that otherwise may come from health interacting with education and skill formation during upbringing. Second, by controlling for individual fixed effects, I minimize any influence from time-invariant unobservable factors, such as cognitive and non-cognitive abilities, which have been found rather constant throughout adulthood (Cunha et al., 2006).

Previous studies (e.g., Lundborg, Nystedt, & Rooth (2014); Case & Paxson (2008)) show that the impact of health on adult outcomes is reduced when accounting for measures of cognitive ability and unobservable factors at the family level. These findings suggest that such factors are confounding the link between child health and adult outcomes, contributing to both child health problems and adverse adult outcomes. This seems reasonable for many health conditions driven by individuals' health behaviors. However, from an econometric point of view, type 1 diabetes has characteristics mimicking an unexpected health shock (Persson et al., 2013; Minor, 2011; Steen Carlsson et al., 2010). In addition, there is no support for cofounding in the medical or epidemiological literature (e.g., lifestyle factors appear unrelated to onset) (American Diabetes Association, 2008). Nor can I find any indication in the data of ability differences between individuals

with and without type 1 diabetes, as I find no descriptive differences in education before onset or in parental level of education. Moreover, Lundborg, Nilsson, & Rooth (2014) confirm that men's earnings penalty from diabetes is robust to selection on unobserved factors at the family level, while the earnings penalties decrease substantially for most other conditions studied (asthma, personality disorder, alcoholism and drug dependence, etc.) when introducing sibling fixed effects. Consequently, my finding of increasing (instead of decreasing) estimates when conditioning on individual-specific effects suggests that unobservables are more likely to be determinants of the consequences of diabetes on both health and earnings, rather than to confound the studied relationship. Given that personal traits and abilities have been found to determine both successful disease management (Wennick et al., 2011; Goldman & Smith, 2002) and labor market outcomes (Heckman, 2007; Cunha et al., 2006), the influence of type 1 diabetes on both health and earnings is also likely to vary across personal traits and abilities.

This study confirms previous findings on type 1 diabetes and earnings based on Swedish data (Lundborg, Nilsson, & Rooth, 2014; Steen Carlsson et al., 2010). For example, Lundborg, Nilsson, & Rooth (2014) report negative effects on men's earnings and employment that appear unrelated to a more extensive usage of the social welfare system. They find the impact of diabetes to be larger than for having any diagnosis (irrespective of which), but smaller than for mental conditions, and alcohol or drug dependence (see also Currie et al. (2010) or Smith & Smith (2010) for similar results on mental conditions). The earnings penalties of diabetes diagnosed sometime before age 18 in Lundborg, Nilsson, & Rooth (2014) are much larger than my corresponding results for men diagnosed in ages 28–34, suggesting that disease duration and/or age at onset matters.

Contrasting the overall findings for women and men, women's earnings appear more sensitive to the onset of type 1 diabetes. This sensitivity may, in part, go through absenteeism due to sickness, disability, and parental leave, as women, but not men, are shown to differ in uptake from the Swedish welfare system compared to population controls. Moreover, the findings for earnings larger than SEK 100,000 suggest that men's earnings are sensitive to changes in wages, while women's earnings appear to respond mainly through changes in labor supply. Additionally, the larger estimates for women are driven mainly by women with university and (only) compulsory education, whereas men with upper secondary education appear most vulnerable among the men. Possibly, a diabetes-induced delay in women's family formation and an intensified tradeoff between family and work contributes to these differences between women and men. I hope that scholars will draw on these findings to explore further the gender differences in the mechanisms driving the relationship between type 1 diabetes and labor market outcomes.

Acknowledgments

Katarina Steen Carlsson, Lund University, Sweden, in collaboration with the Diabetes Incidence Study in Sweden (DISS) steering group, designed and constructed the Econ-DISS database for research on labor market outcomes for young adults with diabetes onset. Steen Carlsson's project was funded by research grants from the Foundation for Research at the Swedish Society for Diabetes (DIA 2005–005, DIA 2006–049, DIA 2007–065), and by an unrestricted researcher-initiated grant from Novo Nordisk A/S. DISS was supported by grants from the Juvenile Diabetes Foundation–Wallenberg Diabetes Research Program (K 98–99 JD–128 13), the Swedish Diabetes Association, and the Swedish Medical Research Council (7X–14531). I thank members of the network of diabetologists in the six healthcare regions of Sweden, who have been responsible for data collection for DISS since 1983. The LISA database is administered by Statistics Sweden and the Social Insurance Agency; the Swedish Agency for Innovative Systems participated in its construction. I also thank Carl Hampus Lyttkens, Katarina Steen Carlsson, and Petter Lundborg for valuable support and insightful comments. Seminar participants at the 33st Nordic Health Economists Study Group meeting in Kuopio, Finland, provided helpful comments and suggestions.

References

Almond, D., & Currie, J. (2011). Killing me softly: The fetal origins hypothesis. *Journal of Economic Perspectives, 25*(3), 153–172.

American Diabetes Association. (2008). Diagnosis and classification of diabetes mellitus. *Diabetes Care, 31*(Supplement 1), 55–60.

Antelius, J., & Björklund, A. (2000). How reliable are register data for studies of the return on schooling? An examination of Swedish data. *Scandinavian Journal of Educational Research, 44*(4), 341–355.

Atkinson, M. A., & Eisenbarth, G. S. (2001). Type 1 diabetes: new perspectives on disease pathogenesis and treatment. *The Lancet, 358*(9277), 221–229.

Baltagi, B. (2008). *Econometric analysis of panel data* (Vol. 1). John Wiley & Sons.

Bastida, E., & Pagán, J. A. (2002). The impact of diabetes on adult employment and earnings of Mexican Americans: Findings from a community based study. *Health Economics, 11*(5), 403–413.

Behrman, J. R., & Rosenzweig, M. R. (2004). Returns to birthweight. *The Review of Economics and Statistics, 86*(2), 586–601.

Björklund, A. (2006). Does family policy affect fertility? *Journal of Population Economics, 19*(1), 3–24.

Black, S. E., Devereux, P. J., & Salvanes, K. G. (2007). From the cradle to the labor market? The effect of birth weight on adult outcomes. *The Quarterly Journal of Economics, 122*(1), 409-439.

Boschini, A., Håkanson, C., Rosén, Å., & Sjögren, A. (2011). *Trading off or having it all? Completed fertility and mid-career earnings of Swedish men and women* (Working Paper, IFAU - Institute for Labour Market Policy Evaluation No. 15).

Brown, H. S., Pagán, J. A., & Bastida, E. (2005). The impact of diabetes on employment: genetic IVs in a bivariate probit. *Health Economics, 14*(5), 537–544.

Brown, H. S., Perez, A., Yarnell, L. M., Pagan, J. A., Hanis, C. L., Fischer-Hoch, S., & McCormick, J. B. (2010). Diabetes and employment productivity: does diabetes management matter? *The American Journal of Managed Care, 17*(8), 569–576.

Budig, M. J., & England, P. (2001). The wage penalty for motherhood. *American Sociological Review*, 204–225.

Case, A., Fertig, A., & Paxson, C. (2005). The lasting impact of childhood health and circumstance. *Journal of Health Economics*, *24*(2), 365–389.

Case, A., & Paxson, C. (2008). Stature and status: Height, ability, and labor market outcomes. *Journal of Political Economy*, *116*(3).

Casson, I., Clarke, C., Howard, C., Mc-Kendrick, O., Pennycook, S., Pharoah, P., ... Walkinshaw, S. (1997). Outcomes of pregnancy in insulin dependent diabetic women: results of a five year population cohort study. *BMJ*, *315*, 275–278.

CDC The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. (1997). Report of the expert committee on the diagnosis and classification of diabetes mellitus. *Diabetes Care*, *20*, 1183–1197.

Cunha, F., Heckman, J., Lochner, L., & Masterov, D. (2006). Interpreting the evidence on life cycle skill formation. *Handbook of the Economics of Education*, *1*, 697–812.

Currie, J. (2009). Healthy, wealthy, and wise: Socioeconomic status, poor health in childhood, and human capital development. *Journal of Economic Literature*, *47*, 87–122.

Currie, J., & Almond, D. (2011). Human capital development before age five. *Handbook of Labor Economics*, *4*, 1315–1486.

Currie, J., Stabile, M., Manivong, P., & Roos, L. L. (2010). Child health and young adult outcomes. *Journal of Human Resources*, *45*(3), 517–548.

Cutler, D. M., Glaeser, E. L., & Shapiro, J. M. (2003). Why have Americans become more obese? *Journal of Economic Perspectives*, *17*, 93–118.

Dahlquist, G., & Mustonen, L. (2000). Analysis of 20 years of prospective registration of childhood onset diabetes time trends and birth cohort effects. Swedish Childhood Diabetes Study Group. *Acta Paediatrica*, *89*(10), 1231–1237.

Dahlquist, G., Nyström, L., & Patterson, C. C. (2011). Incidence of type 1 diabetes in Sweden among individuals aged 0–34 years, 1983–2007: an analysis of time trends. *Diabetes Care*, *34*, 1754–1759.

Dahlquist, G., Pundziūtė-Lyckå, A., Nyström, L., et al. (2005). Birthweight and risk of type 1 diabetes in children and young adults: a population-based register study. *Diabetologia*, *48*(6), 1114–1117.

Daneman, D. (2006). Type 1 diabetes. *The Lancet*, *367*(9513), 847–858.

Devaux, M., & Sassi, F. (2013). Social inequalities in obesity and overweight in 11 OECD countries. *The European Journal of Public Health*, *23*(3), 464–469.

Ekonomifakta. (2015). *Wage trends in Sweden since 1970.* `www.ekonomifakta.se/sv/Fakta/Arbetsmarknad/Loner/Loneutveckling-i-Sverige/`. (3 June 2015)

Figlio, D. N., Guryan, J., Karbownik, K., & Roth, J. (2013). *The effects of poor neonatal health on children's cognitive development* (Tech. Rep.). National Bureau of Economic Research.

Fletcher, J. M., & Richards, M. R. (2012). Diabetes's 'health shock'to schooling and earnings: Increased dropout rates and lower wages and employment in young adults. *Health Affairs*, *31*(1), 27–34.

Fuchs, V. R. (1993). Poverty and health: Asking the right questions. In E. G. David E. Rogers (Ed.), *Medical care and the health of the poor.* Boulder, CO: Westview Press.

Goldman, D., & Smith, J. P. (2002). Can patient self-management help explain the ses health gradient? *Proceedings of the National Academy of Sciences*, *99*(16), 10929–10934.

Goldman, D., & Smith, J. P. (2011). The increasing value of education to health. *Social Science & Medicine*, *72*(10), 1728–1737.

Grossman, M. (1972a). The demand for health: a theoretical and empirical investigation. In (Vol. 119). Columbia University Press for the National Bureau of Economic Research, Inc.

Grossman, M. (1972b). On the concept of health capital and the demand for health. *Journal of Political Economy*, *80*, 223–255.

Haas, S. A., Glymour, M. M., & Berkman, L. F. (2011). Childhood health and labor market inequality over the life course. *Journal of Health and Social Behavior*, *52*(3), 298–313.

Hamermesh, D. S., & Lee, J. (2007). Stressed out on four continents: Time crunch or yuppie kvetch? *The Review of Economics and Statistics*, *89*(2), 374–383.

Harris, A. (2008). *Chronic disease and labour force participation in Australia: an endogenous multivariate probit analysis of clinical prevalence data* (Tech. Rep.).

Heckman, J. (1976). The common structure of statistical models of truncation, sample selection and limited dependent variables and a simple estimator for such models. In *Annals of economic and social measurement, volume 5, number 4* (pp. 475–492). National Bureau of Economic Research, Inc.

Heckman, J. (2007). The economics, technology, and neuroscience of human capability formation. *Proceedings of the National Academy of Sciences*, *104*(33), 13250–13255.

Ivers, R. Q., Cumming, R. G., Mitchell, P., & Peduto, A. J. (2001). Diabetes and risk of fracture the blue mountains eye study. *Diabetes Care*, *24*(7), 1198–1203.

Jonasson, J. M., Brismar, K., Sparén, P., Lambe, M., Nyrén, O., Östenson, C.-G., & Ye, W. (2007). Fertility in women with type 1 diabetes: A population-based cohort study in Sweden. *Diabetes Care*, *30*(9), 2271–2276.

Karvonen, M., Viik-Kajander, M., Moltchanova, E., Libman, I., LaPorte, R., & Tuomilehto, J. (2000). Incidence of childhood type 1 diabetes worldwide. Diabetes Mondiale (DiaMond) project group. *Diabetes Care*, *23*(10), 1516–1526.

Latif, E. (2009). The impact of diabetes on employment in Canada. *Health Economics*, *18*(5), 577–589.

Lundborg, P., Nilsson, A., & Rooth, D.-O. (2014). Adolescent health and adult labor market outcomes. *Journal of Health Economics*, *37*, 25–40.

Lundborg, P., Nystedt, P., & Rooth, D.-O. (2014). Height and earnings: The role of cognitive and noncognitive skills. *Journal of Human Resources*, *49*(1), 141–166.

Lundin, D., Mörk, E., & Öckert, B. (2008). How far can reduced childcare prices push female labour supply? *Labour Economics*, *15*(4), 647–659.

Maslow, G. R., Haydon, A., McRee, A.-L., Ford, C. A., & Halpern, C. T. (2011). Growing up with a chronic illness: Social success, educational/vocational distress. *Journal of Adolescent Health*, *49*(2), 206–212.

Miao, J., Brismar, K., Nyrén, O., Ugarph-Morawski, A., & Ye, W. (2005). Elevated hip fracture risk in type 1 diabetic patients a population-based cohort study in Sweden. *Diabetes Care*, *28*(12), 2850–2855.

Milton, B., Holland, P., & Whitehead, M. (2006). The social and economic consequences of childhood-onset type 1 diabetes mellitus across the lifecourse: a systematic review. *Diabetic Medicine*, *23*, 821–829.

Minor, T. (2011). The effect of diabetes on female labor force decisions: new evidence from the national health interview survey. *Health Economics*, *20*(12), 1468–1486.

Minor, T. (2013). An investigation into the effect of type I and type II diabetes duration on employment and wages. *Economics & Human Biology*, *11*(4), 534–544.

Molarius, A. (2003). The contribution of lifestyle factors to socioeconomic differences in obesity in men and women — a population-based study in Sweden. *European Journal of Epidemiology*, *18*, 227–234.

Möllsten, A., Svensson, M., Waernbaum, I., Berhan, Y., Schön, S., Nyström, L., … Dahlquist, G. (2010). Cumulative risk, age at onset, and sex-specific differences for developing end-stage renal disease in young patients with type 1 diabetes a nationwide population-based cohort study. *Diabetes*, *59*(7), 1803–1808.

OECD. (2008). *Education at a glance 2008. OECD briefing note for Sweden* (Tech. Rep.). Paris. Retrieved from `http://www.oecd.org/dataoecd/23/46/41284038.pdf` (6 June 2012)

Östman, J., Arnqvist, H., Blohmé, G., Lithner, F., Littorin, B., Nyström, L., … Wibell, L. (1986). Epidemiology of diabetes mellitus in Sweden. Results of the first year of a prospective study in the population age group 15–34 years. *Acta Medica Scandinavica*, *220*(5), 437–445.

Östman, J., Lönnberg, G., Arnqvist, H. J., Blohmé, G., Bolinder, J., Ekbom Schnell, A., … Nyström, L. (2008). Gender differences and temporal variation in the incidence of type 1 diabetes: results of 8012 cases in the nationwide Diabetes Incidence Study in Sweden 1983-2002. *Journal of Internal Medicine*, *263*(4), 386–394.

Persson, S., Dahlquist, G., Gerdtham, U., & Steen Carlsson, K. (2013). Impact of childhood-onset type 1 diabetes on schooling: a population-based register study. *Diabetologia*, *56*(6), 1254–1262.

Ploug, U. J. (2013). The cost of diabetes-related complications: Registry-based analysis of days absent from work. *Economics Research International*, *2013*.

Pociot, F., & McDermott, M. F. (2002). Genetics of type 1 diabetes mellitus. *Genes and Immunity*, *3*(5), 235–249.

Puhani, P. (2000). The heckman correction for sample selection and its critique. *Journal of Economic Surveys*, *14*(1), 53–68.

Pundziute-Lyckå, A., Dahlquist, G., Nyström, L., Arnqvist, H., Björk, E., Blohme, G., ... others (2002). The incidence of type I diabetes has not increased but shifted to a younger age at diagnosis in the 0–34 years group in Sweden 1983 to 1998. *Diabetologia*, *45*(6), 783–791.

Royer, H. (2009). Separated at girth: US twin estimates of the effects of birth weight. *American Economic Journal: Applied Economics*, *1*(1), 49–85.

Salm, M., & Schunk, D. (2012). The relationship between child health, developmental gaps, and parental education: Evidence from administrative data. *Journal of the European Economic Association*, *10*(6), 1425–1449.

Smith, J. P. (2007). The impact of socioeconomic status on health over the life-course. *Journal of Human Resources*, *42*(4), 739–764.

Smith, J. P. (2009). The impact of childhood health on adult labor market outcomes. *The Review of Economics and Statistics*, *91*(3), 478–489.

Smith, J. P., & Smith, G. C. (2010). Long-term economic costs of psychological problems during childhood. *Social Science & Medicine*, *71*(1), 110–115.

Soltesz, G., Patterson, C., & Dahlquist, G. (2007). Worldwide childhood type 1 diabetes incidence–what can we learn from epidemiology? *Pediatric Diabetes*, *8*(s6), 6–14.

Sparud-Lundin, C., Öhrn, I., & Danielson, E. (2010). Redefining relationships and identity in young adults with type 1 diabetes. *Journal of Advanced Nursing*, *66*(1), 128–138.

Statistics Sweden. (2011). *Longitudinal integration database for health insurance and labour market studies (LISA by Swedish acronym)*. http://www.scb.se/Pages/List____257743.aspx. (6 June 2012)

Steen Carlsson, K., Landin-Olsson, M., Nyström, L., Arnqvist, H. J., Bolinder, J., Östman, J., & Gudbjörnsdóttir, S. (2010). Long-term detrimental consequences of the onset of type 1 diabetes on annual earnings-evidence from annual registry data in 1990–2005. *Diabetologia*, *53*(6), 1084–92.

Tesfaye, S., Chaturvedi, N., Eaton, S. E., Ward, J. D., Manes, C., Ionescu-Tirgoviste, C., … Fuller, J. H. (2005). Vascular risk factors and diabetic neuropathy. *New England Journal of Medicine*, *352*(4), 341–350.

The TEDDY Study Group. (2007). The environmental determinants of diabetes in the young (TEDDY) study: study design. *Pediatric Diabetes*, *8*(5), 286–298.

Tunceli, K., Bradley, C. J., Nerenz, D., Williams, L. K., Pladevall, M., & Lafata, J. E. (2005). The impact of diabetes on employment and work productivity. *Diabetes Care*, *28*(11), 2662–2667.

Vijan, S., Hayward, R. A., & Langa, K. M. (2004). The impact of diabetes on workforce participation: Results from a national household sample. *Health Services Research*, *39*(6), 1653–1670.

Wennick, A., Hallström, I., Lindgren, B., & Bolin, K. (2011). Attained education and self-assessed health later in life when diagnosed with diabetes in childhood: a population-based study. *Pediatric Diabetes*, *12*(7), 619–626.

Wennick, A., Lundqvist, A., & Hallström, I. (2009). Everyday experience of families three years after diagnosis of type 1 diabetes in children: A research paper. *Journal of Pediatric Nursing*, *24*(3), 222–230.

WHO. (1980). *WHO expert committee on diabetes mellitus. Second report. World health organization technical report series* (Tech. Rep.). Geneva. Retrieved from `http://whqlibdoc.who.int/trs/WHO_TRS_646.pdf` (6 June 2012)

WHO. (1985). *Diabetes mellitus. Report of a WHO study group. Technical report series.* (Tech. Rep.). Geneva. Retrieved from `http://whqlibdoc.who.int/trs/WHO_TRS_727.pdf` (6 June 2012)

Zhang, X., Zhao, X., & Harris, A. (2009). Chronic diseases and labour force participation in australia. *Journal of Health Economics*, *28*(1), 91–108.

Appendices

A Descriptive statistics

Table A.1 (women) and Table A.2 (men) show sample statistics for yearly labor earnings and background characteristics two years prior to and fifteen years after type 1 diabetes onset (or year of inclusion for controls). Table A.1 indicates significant (on at least the 10% level) differences for women already two years prior to onset. Compared to controls, women who will later be diagnosed with type 1 diabetes have, on average, higher earnings, fewer small children, less time spent on parental leave, and their mothers are university-educated to a lesser extent. Possibly, their higher earnings might be explained by their having spent more time on market work, as they do not have to devote time to bearing and caring for small children. Except for some missing data discrepancies, men in the type 1 diabetes group appear to be similar to their controls two years before onset, while they deviate significantly (on at least the 10% level) fifteen years after onset (Table A.2). Then, they are on average married and have children to a lesser extent. Moreover, after fifteen years with type 1 diabetes, both women and men with diabetes have more sickness absence (periods exceeding 14 days) than the controls.

Table A.1: Descriptive statistics of background characteristics for women two years before to and 15 years after onset (inclusion for controls).

	2 years before onset		15 years after onset	
	Diabetes %	Controls %	Diabetes %	Controls %
Annual Earnings[a]				
Earnings (mean)	155,724	141,392	215,074	220,113
(sd)	(61,053)	(63,750)	(87,063)	(117,495)
Demographics				
Age (mean)	29	29	46	46
Year of birth (mean)	1965	1965	1955	1955
Non-native parent	14.36	20.66	15.23	16.93
Married	32.67	36.38	56.85	58.40
Divorced	2.48	4.31	15.23	19.51
Child 0–3	21.78	37.90	3.05	1.60
Child 4–6	19.80	27.38	4.57	5.15
Child 7–10	15.35	14.58	14.72	15.21
Child 11–15	6.44	3.30	34.01	36.44
Education				
Compulsory	12.38	11.15	15.23	12.52
Upper secondary	57.43	59.70	47.21	52.76
University	29.70	28.64	37.56	34.72
Mothers' education				
Compulsory	44.55	38.78	45.69	49.33
Upper secondary	36.63	35.61	16.24	21.84
University	9.90	16.10	8.12	7.48
Missing data	8.91	9.51	29.95	21.35
Fathers' education				
Compulsory	33.66	35.87	36.04	36.32
Upper secondary	38.12	34.09	15.74	18.65
University	13.86	14.32	6.60	7.61
Missing data	14.36	15.72	41.62	37.42
Usage of social welfare				
Student grant	5.94	6.72	1.52	3.68
Parental leave	32.18	42.97	19.29	19.63
Unemployment	22.77	20.79	9.64	10.31
Sickness absence	11.19	16.38	25.38	16.56
Disability pension	0.00	0.25	5.08	3.31
Individuals	202	789	197	815

[a] SEK 2005 prices (10 SEK≈€1).

Table A.2: Descriptive statistics of background characteristics for men two years before to and 15 years after onset (inclusion for controls).

	2 years before onset		15 years after onset	
	Diabetes	Controls	Diabetes	Controls
	%	%	%	%
Annual Earnings[a]				
Earnings (mean)	208,293	202,503	298,911	307,443
(sd)	(98,008)	(89,846)	(174,682)	(169,645)
Demographics				
Age (mean)	29	29	46	46
Year of birth (mean)	1965	1965	1955	1955
Non-native parent	15.56	18.74	13.60	17.00
Married	28.15	26.58	51.39	57.92
Divorced	1.83	2.07	20.40	14.38
Child 0–3	31.81	33.28	4.03	4.60
Child 4–6	14.65	16.18	6.80	7.74
Child 7–10	7.09	7.14	15.62	19.44
Child 11–15	1.14	2.02	31.23	37.14
Education				
Compulsory	17.39	15.80	21.41	20.14
Upper secondary	58.58	57.90	50.13	48.08
University	23.34	25.60	28.21	31.72
Mothers' education				
Compulsory	44.16	42.65	48.36	50.29
Upper secondary	34.55	32.84	22.17	23.11
University	13.96	13.78	7.05	6.34
Missing data	7.32	10.73	22.42	20.26
Fathers' education				
Compulsory	40.05	38.89	34.76	36.96
Upper secondary	32.27	28.92	18.14	17.93
University	11.21	13.73	8.82	7.68
Missing data	16.48	18.46	38.29	37.43
Usage of social welfare				
Student grant	5.95	4.58	0.50	0.58
Parental leave	24.94	23.31	12.85	14.26
Unemployment	11.44	14.32	7.81	6.58
Sickness absence	7.84	8.04	15.11	11.58
Disability pension	0.23	0.49	2.52	1.51
Individuals	437	1,836	397	1,718

[a] SEK 2005 prices (10 SEK≈€1).

B Sickness absence, children, and excluded observations

As a background to the empirical analysis, I look graphically at sickness absence (periods exceeding fourteen days), number of children, and percentage of excluded observations.

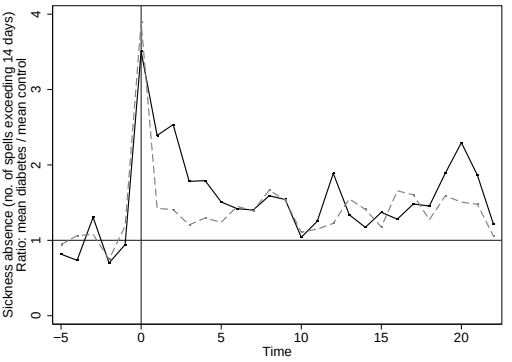

Figure B.1: Sickness absence (no. of periods in a year that exceed 14 days) before and after type 1 diabetes onset (time=0 is the year of onset). Ratio of mean number of periods in the type 1 diabetes group to the mean number in the control group for women (black solid line) and men (gray dashed line) respectively.

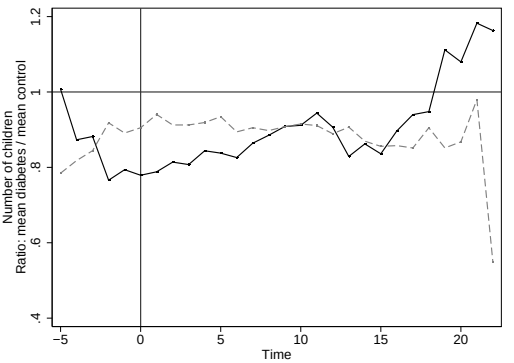

Figure B.2: Number of children (younger than 18) before and after type 1 diabetes onset (time=0 is the year of onset). Ratio of mean number of children in the type 1 diabetes group to the mean number in the control group for women (black solid line) and men (gray dashed line) respectively.

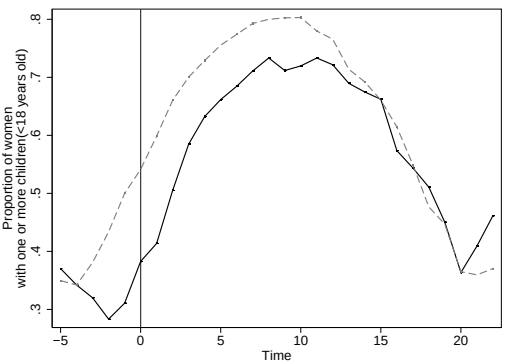

Figure B.3: Child(ren)(younger than 18) before and after type 1 diabetes onset (time=0 is the year of onset). Percentage of *university-educated* women with one or more children for women in the type 1 diabetes group (black solid line), and correspondingly for the controls (gray dashed line).

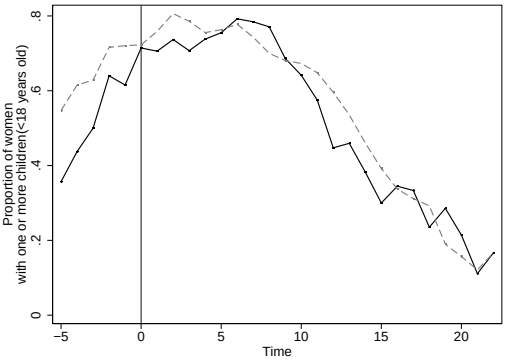

Figure B.4: Child(ren)(younger than 18) before and after type 1 diabetes onset (time=0 is the year of onset). Percentage of *compulsory educated* women with one or more children for women in the type 1 diabetes group (black solid line), and correspondingly for the controls (gray dashed line).

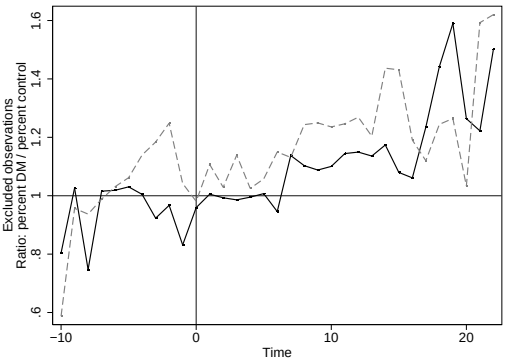

Figure B.5: Excluded observations (i.e., observations where earnings<1PBA) before and after type 1 diabetes onset (time=0 is the year of onset). Ratio of the percentage of exclusions in the type 1 diabetes group to the percentage in the control group for women (black solid line) and men (gray dashed line) respectively.

Paper II

Onset of type 1 diabetes in young adults and university education

with Sofie Gustafsson[i], Lennarth Nyström[ii], and Mona Landin-Olsson[iii]

Abstract

This paper investigates the interrelationships of young-adulthood health, university education, and family formation. Generally, young adults face the choice of entering the labor market or continuing to university education to increase their future employability and labor earnings. This decision relates to other choices in life. University education has, for example, been found to delay family formation as both university education and family formation require investments in time and effort (as well as monetary costs). An unexpected health shock, such as the sudden onset of a lifelong disease, also requires the investment of time and effort to restore and maintain health. Such a change in life constraints can cause young adults to reevaluate previously set university aspirations and other life choices. Using longitudinal register data on individuals with type 1 diabetes onset in the age group 17 to 20 and population controls, we illustrate how an unexpected health shock (imposing changed life-constraints, increased health investments, and higher uncertainty about future outcomes) may affect subsequent university education. We find that type 1 diabetes among women negatively links to university education and motherhood. Comparing only the university educated, women with diabetes become mothers to a lesser extent than other women. Taken together, these results indicate that type 1 diabetes affects both the decision to enter university and to start a family, suggesting that type 1 diabetes sharpens the tradeoff between university education and motherhood: i.e., diabetes might make it more difficult to have both a university education and children. Socioeconomic background also seems to be important: women belonging to different socioeconomic groups appear to respond differently to onset of type 1 diabetes, in terms of both university education and family formation. For men, we find no association between onset and university education.

Keywords: Health, education, diabetes, family formation
JEL Classification: I10, I21, J13, J24

[i]Department of Economics, Lund University, Lund, Sweden.
[ii]Department of Public Health and Clinical Medicine, Division of Epidemiology and Global Health, Umeå University, Umeå, Sweden.
[iii]Department of Medicine and Clinical Science, Helsingborg Hospital, Lund University, Lund, Sweden.

1 Introduction

Growing evidence shows that health in early life and childhood is important for adult outcomes such as academic achievements (see, e.g., Almond & Currie (2011), and Rees & Sabia (2011)). Evidence also establishes that parents contribute to their children's skill formation (Currie, 2009; Cunha & Heckman, 2008; Behrman et al., 1982; Becker & Tomes, 1976). When young children experience health problems, their parents' caregiving role, which is an essential part of parenting, intensifies and may become crucial for the children's long-term health consequences. The increased caregiving need, in turn, may affect parents' possibilities of engaging in their children's schooling and other family activities. If so, the link between university education and childhood health will partly reflect the degree of parental involvement, as early education is important for subsequent academic achievements. This paper focuses on the less explored link between young-adulthood health, measured as type 1 diabetes, and subsequent university education. Young adults are themselves responsible for their health behavior and their academic aspirations, while parents' roles are more advisory. Accordingly, the link reflects how type 1 diabetes influences university education when (1) ruling out the influence of earlier academic achievements and (2) minimizing parental involvement.

Generally, young adults face the choice of entering the labor market or continuing to university education to increase their future employability and labor earnings. This decision relates to other choices in life. University education has, for example, been found to delay family formation[1] (Boschini et al., 2011; Lundin et al., 2008; Björklund, 2006), as both university education and family formation require investment in time and effort (apart from monetary costs). An unexpected health shock, such as the sudden onset of a lifelong disease, also requires time and effort invested to restore and maintain health. Such a change in life constraints can cause young adults to reevaluate previously set university aspirations and other choices in life.

Using detailed register data for individuals diagnosed with type 1 diabetes in the age group 17–20 and population controls in Sweden, this paper analyses the link between young-adulthood health and university education at age 30. By focusing on onset in the age group 17–20, when the young adults have already made their choice of upper secondary education, we minimize the influences that onset might have on the young adults' eligibility for university education.[2] We account for covariates including, for example, socioeconomic background and gender by controlling for parental education, and by conducting separate

[1] Delays in family formation may be an unconscious consequence or a joint decision incorporated into the university decision.

[2] For the time studied, children generally enter upper secondary education at age 15 or 16 and choose either a theoretical program specifically preparing for further studies or a vocational program preparing for labor market entrance.

analyses for men and women, and different socioeconomic groups. We also explore whether family formation mediates the link between type 1 diabetes and university education.

The results show no difference in university education between men with and without type 1 diabetes. In contrast, women with type 1 diabetes are less likely to have a university education than women without diabetes. Persisting at age 35, this gap represents a permanent drop in university education rather than educational delay. Additionally, we find that women with type 1 diabetes are less likely to have children than are controls. Comparing only the university educated, women with diabetes become mothers to a lesser extent than other women. Taken together, these results indicate that type 1 diabetes affects both the decision to enter university and to start a family, suggesting that type 1 diabetes sharpens the tradeoff between university education and motherhood: i.e., diabetes might make it more difficult to have both a university education and children. Socioeconomic background also seems to be important: women belonging to different socioeconomic groups appear to respond differently to onset of type 1 diabetes, in terms of both university education and family formation.

The paper is structured as follows. Section 2 gives a short background. Section 3 reviews the previous literature on type 1 diabetes and economic outcomes. Section 4 describes the conceptual framework. Section 5 presents the data and descriptive statistics. Section 6 details the econometric strategy. Section 7 contains the results, and Section 8 the sensitivity analyses. Section 9 discusses the results.

2 Background

The individual's perceived tradeoff between university education and other choices in life is likely to depend on personal characteristics such as socioeconomic background and gender. Preferences that relate to such characteristics may, therefore, contribute to the observed link between parents' and descendants' educational level (Chevalier, 2004; Black & Devereux, 2011; Mulligan, 1999; Dearden et al., 1997) and educational differences by gender (Boschini et al., 2011; Lundin et al., 2008; Björklund, 2006). Similarly to the intergenerational transition of human capital (Chevalier, 2004; Black & Devereux, 2011; Mulligan, 1999; Dearden et al., 1997), having better-off parents is also likely to be positive for health-related behaviors, including disease-coping strategies when disease management is as complex as it is with type 1 diabetes (Centers for Disease Control and Prevention, 2005).[3][4] The degree and severity of subsequent diabetes-related

[3] Type 1 diabetes management involves regular glucose controls, several daily insulin injections, a healthy diet, and physical exercise.

[4] The associations of socioeconomic characteristics, health and health-related behavior are well known (see, e.g., Smith et al. (1979)). Studies show, for instance, that, in comparison with individuals

complications[5] are, therefore, also likely to vary with socioeconomic background.

Furthermore, having different networks and prior experience, people across the socioeconomic strata may be more or less likely to assimilate the long-term consequences of type 1 diabetes for both health and work. Long-sightedness is crucial, as many diabetes-related complications first appear several years after onset, but their severity and timing are influenced by current lifestyle choices. In addition, current educational choices may affect one's future work situation and ability to incorporate health impairments into everyday life. Such potential differences in diabetes self-management and assimilation of long-term diabetes-related consequences suggest that individuals of different socioeconomic background may respond differently to onset of type 1 diabetes. Adding to this socioeconomic heterogeneity, young adults with high-income parents are more likely than peers with low-income parents to receive financial support from their parents after onset, as better-off parents give, on average, more financial in vivo transfers to adult children (see, e.g., Henretta et al. (2002), and Grundy (2005)).

Differences between men and women might exist because (1) family formation and university education are complements for men, but substitutes for women (see, e.g., Boschini et al. (2011), Lundin et al. (2008), and Björklund (2006)) and (2) type 1 diabetes reduces fertility and amplifies the risk of severe pregnancy-related complications for both the mother and child (Jonasson et al., 2007; Casson et al., 1997). Thus, type 1 diabetes adds to the risks all young women face when delaying childbearing, and even more so with increasing age. Such risks may contribute to some women choosing university education over family formation after onset, while the elevated risks may cause other women to hasten to start a family and perhaps forgo their academic career. Regardless of whether socially and/or biologically induced, these differences imply that young-adulthood onset of type 1 diabetes will affect men's and women's education differently.

Type 1 diabetes typically has a rapid onset without prior symptoms, mimicking a 'before and after' treatment study design. Despite extensive research, the exact combination of environmental and genetic factors, together with the chain of events, triggering type 1 diabetes onset remains unclear. Lifestyle factors (i.e., obesity and physical inactivity) that are associated with low education (Devaux & Sassi, 2013; Cutler et al., 2003; Molarius, 2003; Molarius et al., 2000; Lissner et al., 2000; Lahmann et al., 2000) do not appear to affect the lifetime risk of onset (American Diabetes Association, 2008; Daneman, 2006). It is more likely that

of lower socioeconomic status, socioeconomically advantaged individuals (1) have higher survival rates when it comes to cancer and cardiovascular illness (Schrijvers & Mackenbach, 1994; Smith et al., 1979; Peltonen et al., 2000), (2) adhere better to complex self-management treatments of HIV and diabetes (Goldman & Smith, 2002), and (3) are earlier adopters of new medical technologies (Rosvall et al., 2008; Glied & Lleras-Muney, 2008).

[5] Long-term complications involve, e.g., blindness, kidney failure, heart disease, stroke, nerve damage, and foot amputation.

factors outside the individuals' control such as genetics, cold climate, and virus infection in early life, are at play (Dahlquist et al., 2011, 2005; Soltesz et al., 2007; The TEDDY Study Group, 2007; Atkinson & Eisenbarth, 2001). Due to this complexity and the sudden onset, type 1 diabetes is generally seen as a health shock, which the individual is unable to anticipate or influence beforehand (Persson et al., 2013; Minor, 2011; Steen Carlsson et al., 2010).

However, an association between type 1 diabetes and university education does not necessarily imply a causal relationship: association may appear due to third factors that affect the probability of both onset and university education (i.e., innate ability or socioeconomic characteristics). The presence of such third factors is likely to involve additional systematic group-level differences. Socioeconomics, for example, could affect educational decisions and lifestyle factors that, in turn, may increase the risk of disease development. However, we recall that lifestyle factors do not appear to impact on the lifetime risk of type 1 diabetes. Further, none of the available variables (including socioeconomics) measured pre-onset are associated with type 1 diabetes, rejecting the existence of group-level differences before onset.[6] Still, we have no means for testing if, for example, the genetics of type 1 diabetes is associated with education. However, first, epidemiologic studies have shown that despite the heredity of type 1 diabetes, 90 percent of all newly diagnosed children with type 1 diabetes in Sweden have no close family member with type 1 diabetes (Dahlquist & Mustonen, 2000). Second, Lundborg et al. (2014) show that sibling fixed effects and the influence of shared genes and childhood environment appear less significant for diabetes than for many other diagnoses and more general measures of health. They also conclude that genetic influences are limited for the health-earnings relationship when comparing findings for different types of twins.

3 Previous research on type 1 diabetes

Previous research, using detailed incidence registers for individuals with type 1 diabetes in Sweden, reveals educational and labor market difficulties after onset (Persson et al., 2013; Steen Carlsson et al., 2010; Dahlquist et al., 2007). Compared to controls without diabetes, children with onset in the age group 0–14 have lower grades from compulsory education (Persson et al., 2013; Dahlquist et al., 2007) and from theoretical upper secondary programs preparing for university (Persson et al., 2013), and have a higher risk of unemployment later in life (Persson et al., 2013). Adults with onset in the age group 14–34 have higher risk of unemployment and lower annual earnings (Steen Carlsson et al., 2010).

[6] We estimate probit regression models to test whether any background variable available in our data predicts type 1 diabetes onset and test for differences in means between individuals with and without type 1 diabetes for all of these variables. These analyses are detailed in Section 6.

Using Swedish enlistment data, and data for onset of diabetes before age 18, Lundborg et al. (2014) report negative earnings penalties for men. In another Swedish study, Wennick et al. (2011) use population-based survey data and find that childhood diabetes is associated with lower levels of education and self-assessed health. Using US survey data, Minor (2011, 2013) studies both type 1 and type 2 diabetes, and concludes that the associations between diabetes and labor market outcomes are driven by type 2 diabetes. These studies, however, do not reveal if individuals change their university aspirations after onset, or if it is only their prerequisites for higher education that change.

Most other previous studies on diabetes and economic outcomes depend on small-sample surveys (Milton et al., 2006), or cannot discriminate between type 1 and type 2 diabetes (Ploug, 2013; Fletcher & Richards, 2012; Maslow et al., 2011; Brown et al., 2010; Latif, 2009; Zhang et al., 2009; Harris, 2008; Brown et al., 2005; Tunceli et al., 2005; Vijan et al., 2004; Bastida & Pagán, 2002). The two types of diabetes have fundamentally different pathogenesis and expected impact on university education. For instance, old age (when education is finalized) and life-style factors (i.e., obesity and physical inactivity that are associated with low education), substantially increase the risk of developing type 2 diabetes (Stumvoll et al., 2005; Clausen et al., 1996; Prentice & Jebb, 1995), while the risk of type 1 diabetes depends on other factors (Dahlquist et al., 2011, 2005; Soltesz et al., 2007; The TEDDY Study Group, 2007; Atkinson & Eisenbarth, 2001).

4 Conceptual framework

University education is a human-capital investment generating welfare or utility in terms of greater labor market returns such as employability, career-track, wage-rate, and working conditions (e.g., time and work-hour flexibility, and workplace flexibility, fostering safety and health). Becker's human capital model (Becker, 1962) proposes that individuals, subject to own preferences and resources, opt for the educational level that maximizes lifetime utility. Individuals balance perceived forgone welfare from investing in education against future (time-discounted) university welfare returns. 'Forgone welfare' refers to the fact that resources allocated to education have alternative uses: i.e., individuals could allocate time and effort to earning labor income, rearing children, or to any other welfare-generating activity.

Becker's household time allocation model (Becker, 1965) and more recent life-cycle family models (see, e.g., Greenwood et al. (2003)) illustrate that household labor-division implies that women's career and university decision may conflict with family formation. Moreover, empirical evidence shows that university-educated women postpone motherhood (Boschini et al., 2011; Gustafsson & Adriaan, 2006; Adsera, 2011) and have fewer children or are more often childless (Boschini et al., 2011) than less educated women. University-educated men also

postpone fatherhood (Adsera, 2011; Boschini et al., 2011; Gustafsson & Adriaan, 2006), but have more children and are more seldom childless (Boschini et al., 2011) than less educated men.

Conceptually, the university decision is a process in which young adults weigh the perceived educational costs again benefits, and only invest if the benefits outweigh the costs. The individuals' perception of costs and benefits is governed by time-preference (i.e., willingness to forgo immediate utility for forthcoming payoffs) and preferences for university education as against other objectives in life, such as family formation. In short, myopic time-preferences (e.g., present-time orientation) or family preferences (e.g., aversion to childlessness) decrease university aspirations. As parents' socioeconomic status affects their investment in their children's time preferences (see, e.g., Becker & Mulligan (1997)), the intergenerational transmissions of human capital may partly operate via time preferences. The sudden onset of type 1 diabetes changes life-constraints and imposes greater uncertainty about future health, which may, for instance, form more myopic time preferences. Alternatively, the diabetes-evoked up-shift in health investments may affect resource constraints, increasing the perceived value of the remaining time available for other activities. Consequently, young adults may modify their university decision and other prior-set life goals after onset.

Assuming that type 1 diabetes *affects time-preferences* (due to increased morbidity and mortality risks), university investments (1) increase if, e.g., diabetes management fosters future-oriented time preferences, making young adults more willing to forgo present utility for future educational returns, or (2) decrease if, e.g., the higher risk of adverse health outcomes forms myopic time preferences, making individuals more present-oriented. Assuming that type 1 diabetes *affects women's cost of university education*(due to the amplified risk of pregnancy-related problems when postponing motherhood), university investments (1) increase if type 1 diabetes, by suppressing family aspirations, lowers women's forgone cost for university enrollment, or (2) decrease if type 1 diabetes lowers the transit age for motherhood to minimize the risk of diabetes-related fertility problems, and raises the alternative cost of university enrollment.

5 Data

We use the Econ-DISS database, which combines the national Diabetes Incidence Study in Sweden (DISS) with national population registers. Since 1983, DISS has registered all diagnosed diabetes cases in the age group 15–34 in Sweden (Östman et al., 1986, 2008). The reporting physician classifies the diabetes type according to current clinical diabetes criteria (1983–91 (WHO, 1980, 1985); 1992 onwards (CDC The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus, 1997)). Using a case-control framework, Statistics Sweden

matches each individual in DISS to four controls by age, gender, and municipality of residence at the time of diagnosis, and identifies the parents of all individuals from the Multi-Generation Register (Statistics Sweden, 2009). Statistics Sweden then adds yearly data on demographic and socioeconomic variables from the LISA database (Statistics Sweden, 2011) for the period 1990–2005.[7]

Ideally, we should study individuals with onset just after completing upper secondary education, but this would result in samples that are too small, given the low incidence of type 1 diabetes. Therefore, we select individuals diagnosed with type 1 diabetes in the age group 17–20 (in years 1983–1995; n = 1,034) and their matched controls (n = 4,136). These individuals were born in 1963–1975 and we study outcomes at age 30 (in years 1993–2005).[8] At age seventeen, most young people in Sweden are about to complete upper secondary education. Consequently, the lower age limit rules out that type 1 diabetes affects (1) educational achievements at the compulsory level and (2) the choice between theoretical and vocational programs for upper secondary education. Thereby, we minimize potential disparities in prerequisites for university education between individuals with and without type 1 diabetes. For the studied period, the median age for first-time university enrollment was 22 and the median age when earning a degree was 27–28 (Statistics Sweden, 2008). Therefore, at the age of twenty (the upper age limit), many young people still face the choice of university education.

To exclude supplementary training and retraining following from unemployment later in life, we measure university education at age 30 (age 35 in the sensitive analysis). Due to data restrictions, we exclude individuals with missing data on own education (3 with diabetes, 14 controls), individuals born outside Sweden or if their parent(s) were born outside Sweden (145 with diabetes, 779 controls). After exclusions, the sample consists of 886 individuals with type 1 diabetes and 3,343 controls.

5.1 Dependent variable

The dependent variable *University education* is a dummy variable indicating if the individual has university education at age 30. *University education* is defined as having credits from a Swedish university or university college corresponding to at least 20 weeks of full-time studies.[9] Table 1 shows that 31.7% of the women with type 1 diabetes, compared to 40.1% of the women without diabetes, have

[7] For details see Steen Carlsson et al. (2010). The research program was approved by the Regional Ethical Review Board in Lund, Sweden (dnr 393/2005).

[8] The study population consists of more men than women because type 1 diabetes is more common in men (Pundziute-Lyckå et al., 2002).

[9] We use a rather crude definition of university education as the Swedish educational system changed during the studied period. Certain types of education have become longer and we are unable to track these changes in our data. Still, our results are robust to defining university education as having credits corresponding to more than two or four years of full-time studies (see Section 6).

a university education at age 30 (significantly different at the 5% level),[10] while there are no such differences between men with and without type 1 diabetes (30.2% vs. 33.7%).

5.2 Control variables

The main variable of interest is a dummy indicating individuals with type 1 diabetes onset at age 17–20. The case-control design of the Econ-DISS data (controls matched by age, gender, and municipality of residence at the time of diagnosis) introduces implicit control variables included in all analyzes. *Year of birth* is a proxy for cohort effects that controls for differences in composition and size of each cohort. *Municipality of residence at the time of diagnosis (inclusion for controls)* controls for access to universities and to the local labor market at the time of onset (inclusion). We control also for *year-dummies*, *socioeconomic background* (SEB), and *family* related variables (all measured at age 30). Socioeconomic background captures circumstances pre-onset, while starting a family (generally) is post-onset.

The year dummies (1990–2005) control for factors such as education policies, and economic and technology changes that may influence educational attainment.[11] We use the parents' level of education (compulsory, upper secondary, and university) to indicate SEB in two ways.[12] First, we use mother's and father's levels of education separately as control variables in the regression analysis. Second, we combine mother's and father's education into *low*, *middle*, and *high* SEB for the graphical analysis and the stratified regression analysis. We define SEB as *low* if the highest level of education of either parent is compulsory, *middle* if the highest level of either parent is upper secondary, and *high* if the highest level of either parent is university. Socioeconomic background is frequently measured by parent's level of education (see, e.g., Currie (2009)).[13] Better-educated parents, on average, have a larger income and are therefore more likely to live in areas providing high-quality schooling. The better-educated parents may also have higher (acquired and/or innate) ability (see, e.g., Cunha et al. (2006)), enabling them to better support their children's learning by, for instance, helping with homework.

If SEB is associated with onset, parental level of education will differ between

[10] These differences for women originate at onset (ages 17–20) and there are no differences before onset. See Figure A.1 in Appendix A showing years of education by age.

[11] Studying outcomes at age 30, the year dummies coincide with year of birth.

[12] Ideally, we would measure SEB in childhood, but instead we use the first observation available and measure SEB in 1990 (when the individuals are 16–27 years old). Still, SEB is mainly predetermined and generally does not change much over time.

[13] Parental income is another frequently used measure. However, parental income in Econ-DISS is only available from year 1990 and forward. Parental income is therefore likely to be affected by the child's diabetes and is not an appropriate measure of childhood circumstances.

Table 1: Descriptive statistics at age 30

	Women		Men	
	Diabetes	Controls	Diabetes	Controls
Variables[a]	%	%	%	%
Education				
Compulsory	12.9	8.9	14.0	11.8
Upper secondary	55.4	51.0	55.8	54.5
University	31.7	40.1	30.2	33.7
Family status				
Married	31.3	32.5	22.6	21.9
Divorced	3.8	2.9	2.2	1.6
No. of children (mean)	0.89	1.10	0.66	0.65
Socioeconomic background				
Mothers' education				
Compulsory	33.8	33.0	35.1	34.8
Upper secondary	43.3	41.7	42.8	41.3
University	18.8	22.0	18.4	21.2
Missing data	4.2	3.3	3.7	2.7
Fathers' education				
Compulsory	36.7	35.8	39.1	38.2
Upper secondary	36.7	38.1	34.2	37.2
University	17.5	20.1	17.7	18.8
Missing data	9.2	6.0	9.1	5.9
Individuals	240	911	407	1,577

[a] Significant differences between women with and without type 1 diabetes for the following variables: (tested with t-test) *Compulsory* (p=0.0613), *University* (p=0.0172), *No. of children* (p=0.0046), *Fathers' education missing* (p=0.0844), and (tested with chi-2 test) *Education* (p=0.025). For men: (tested with t-test) *Fathers' education missing* (p=0.0203).

individuals with and without type 1 diabetes, but no such difference (tested with chi-2 test) appears in the descriptive statistics (Table 1). Nevertheless, we account for SEB either by controlling for parental level of education in the regression analysis, or by stratifying parts of our analysis according to low, middle, and high SEB. Figure 1 shows the proportion of women and men with university education for individuals with and without type 1 diabetes, stratified by SEB. Figure 1 indicates that (1) a lower proportion of individuals with type 1 diabetes have university education and (2) these differences in university education are particularly pronounced among women with low SEB or high SEB.

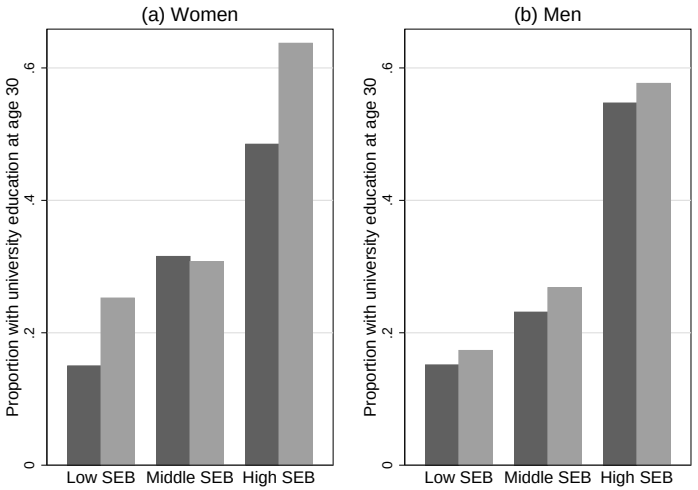

Figure 1: Proportion of women (a) and men (b) with university education for individuals with type 1 diabetes (black bars) and controls (gray bars) by socioeconomic background (SEB). *Low* if highest level of education of either parent is compulsory, *middle* if highest level of either parent is upper secondary, and *high* if highest level of either parent is university.

We control for own family status (measured as number of children, and dummies indicating married and divorced at age 30) as a potential mediator in the link between type 1 diabetes and university education. The family status of men with type 1 diabetes is no different when compared to that of men without diabetes, while women with type 1 diabetes have fewer children than women without diabetes (significant at the 1% level, Table 1). Figure A.2 in Appendix A, showing the proportion of women with one or more children, indicates that these differences are concentrated to women with middle SEB.

6 Empirical method

We estimate the relationship between type 1 diabetes and the probability of having university education, separately for women and men, using a probit model specification. We introduce control variables stepwise to evaluate if any background factor correlates with the type 1 diabetes dummy in our main analysis. First, we control for year dummies and the implicit matching variables, and then we add controls for SEB. Any differences in the estimated diabetes coefficients in the two specifications (i.e., indicating correlation) could mean that SEB is a confounder relating to both type 1 diabetes onset and education. Lastly, we add controls for family status to determine whether these variables are potential mediators through which type 1 diabetes might affect education. We do not assess any other potential mediator such as comorbidities and own income. If controlled for, mediators may absorb some of the effect related to diabetes. Therefore, the coefficients on the mediators would not truly capture their actual effects and, even more importantly, including these variables could bias the estimate for type 1 diabetes. To allow for socioeconomic heterogeneity, we repeat the main analysis stratified into low, middle, and high SEB. To explore the interplay between type 1 diabetes, education, and motherhood, we estimate (1) differences in women's probabilities of having one or more children and (2) differences in women's probabilities of having both university education and one or more children.[14]

To assess the robustness of the results, we perform several sensitivity analyses. First, we test if the groups differ in observable background characteristics by running a regression on the probability of being in the diabetes group. Second, we perform a placebo test to test if any other group level differences, apart from onset of type 1 diabetes, appear to influence the results. This test repeats the main analysis for individuals with onset (inclusion) at the age of 24–26, by which time they ought to have made their choice of university education. Third, we test the sensitivity of the results to how we define university education by redefining it as having more than two or four years of full-time university studies. Similar estimates across definitions indicate that the differences between the groups (diabetes and controls) are related not only to enrollment but also to continuing studies. Fourth, we test if the results are driven by the youngest at the time of onset and exclude individuals with onset (inclusion) at age 17. Fifth, we test for educational delays by estimating the link between type 1 diabetes and education at age 35 instead of 30. Sixth, we test for later family formation by estimating the link between type 1 diabetes and motherhood at age 35. Additionally, we test alternative empirical models (e.g., (generalised) ordered probit and OLS) and an

[14] We explore the corresponding interplay for fathers, but these estimates are all insignificant. Results are available on request.

Table 2: Probit (average marginal effects) estimations of university education at age 30

Dependent variable	Women			Men		
University	(1)	(2)	(3)	(4)	(5)	(6)
Diabetes	-0.090***	-0.083**	-0.10***	-0.037	-0.028	-0.027
	(0.034)	(0.036)	(0.035)	(0.026)	(0.026)	(0.026)
Covariates	Yes	Yes	Yes	Yes	Yes	Yes
SEB	No	Yes	Yes	No	Yes	Yes
Family status	No	No	Yes	No	No	Yes
Observations	1151	1151	1151	1984	1984	1984

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Covariates indicates controls for year, cohort, and municipality of residence.

SEB indicates controls for parental level of education.

Family status indicates controls for marital status and number of children.

alternative definition of SEB.[15][16]

7 Results

7.1 University education

Table 2 shows the main results for women (columns 1–3) and men (columns 4–6). Appendix B reports the average marginal effects for the control variables. The estimated average marginal effects (columns 1 and 4) represents the average difference in the likelihood of having university education between individuals with and without type 1 diabetes. The remaining columns show the results when adding controls for SEB (columns 2 and 5) and also family status (columns 3 and 6).

Women with type 1 diabetes are 8.9 percentage points less likely to have university education than women without diabetes. This significant relationship remains when controlling for SEB (the difference in likelihood decreases somewhat to 8.3 percentage points) and family status (the difference in likelihood increases to 10.4 percentage points). These estimates are sizable given that the overall probability of having a university education in our data is 40% (women)

[15] These results are in line with the overall findings of this study and are available on request.

[16] The alternative definition of SEB is defined as *low* SEB if highest educational level of either parent is compulsory, *middle* SEB if the highest level of either parent is upper secondary, or the highest level of one parent is university and the highest level of the other parent is upper secondary, and *high* SEB if the highest level of both parents is university.

Table 3: Probit (average marginal effects) estimations of university education at age 30 by *low*, *middle*, and *high* socioeconomic background (SEB)

	Women			Men		
Dependent variable	(1)	(2)	(3)	(4)	(5)	(6)
University	Low	Middle	High	Low	Middle	High
Diabetes	-0.10*	0.0064	-0.17**	-0.024	-0.040	-0.040
	(0.056)	(0.050)	(0.070)	(0.039)	(0.035)	(0.052)
Covariates	Yes	Yes	Yes	Yes	Yes	Yes
Observations	262	524	350	451	928	580

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Covariates indicates controls for year, cohort, and municipality of residence.

and 34% (men) for the controls. The increase in the estimate of type 1 diabetes when including family status indicates that type 1 diabetes correlates with both family status and university education. Still, this change in estimates is only marginal, and the relationship between type 1 diabetes and university education appears robust to both SEB and family status.

Table 3 shows the results when stratifying the sample by SEB for women (columns 1–3) and men (columns 4–6). Columns (1) and (4) presents the estimates for individuals with low SEB, columns (2) and (5) for individuals with middle SEB, and columns (3) and (6) for individuals with high SEB. The results show a negative link between education and type 1 diabetes for women with low or high SEB. Women with type 1 diabetes and high (low) SEB are on average 17.2 percentage points (10.4 percentage points) less likely to have university education than women without diabetes and high (low) SEB. In contrast, the link between education and type 1 diabetes is small and insignificant for women with middle SEB. For men, there is still no relationship between type 1 diabetes and university education, regardless of SEB.

7.2 Children

Table 4 presents estimates of women's probabilities of having one or more children when stratifying the sample by SEB. Compared to women without diabetes and middle SEB, women with type 1 diabetes and middle SEB are, on average, 17.3 percentage points less likely to be mothers. For women with low or high SEB, the marginal effect is small and insignificant. Combining these results (Table 4) with the stratified results for university education (Table 3), suggests that type 1 diabetes affects both the decision to enter university and to start a family. Women with type 1 diabetes and low or high SEB have a lower likelihood of higher

Table 4: Probit (average marginal effects) estimations of having one or more children at age 30 by socioeconomic background (SEB) for women

Dep. var.	(1)	(2)	(3)
Child(ren)	Low SEB	Middle SEB	High SEB
Diabetes	-0.036	-0.17***	-0.064
	(0.075)	(0.053)	(0.068)
Covariates	Yes	Yes	Yes
Observations	262	524	350

Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1.
Covariates indicates controls for year, cohort, and municipality of residence.

Table 5: Probit (average marginal effects) estimations of having *both* university education and one or more children at age 30 for women

Dependent variable		
Uni&Child(ren)	(1)	(2)
Diabetes	-0.087***	-0.081***
	(0.025)	(0.025)
Covariates	Yes	Yes
SEB	No	Yes
Observations	1151	1151

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.
Covariates indicates controls for year, cohort, and municipality of residence.
SEB indicates controls for parental level of education.

education, but their likelihood of motherhood appears unaffected, while women with type 1 diabetes and middle SEB have a lower likelihood of motherhood, but their likelihood of higher education appears unaffected.

Table 6: Probit (average marginal effects) estimations of being in the type 1 diabetes group

Dep. var.	(1)	(2)
Diabetes	Women	Men
Compulsory M	-0.0045	-0.0094
	(0.029)	(0.021)
University M	-0.027	-0.027
	(0.032)	(0.025)
Missing M	0.013	0.022
	(0.071)	(0.060)
Compulsory F	0.0080	0.016
	(0.029)	(0.022)
University F	-0.011	0.011
	(0.035)	(0.028)
Missing F	0.078	0.090*
	(0.059)	(0.046)
Covariates	Yes	Yes
Observations	1151	1984

Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1.
Covariates indicates controls for year, cohort, and municipality of residence.

Table 5 presents the estimated differences in women's probabilities of having both university education and one or more children. These results show a negative association, in line with the suggestion that type 1 diabetes might sharpen the tradeoff between university education and motherhood. The marginal effect of type 1 diabetes is significant and sizable, and remains so when controlling for SEB. The negative association is driven by women with low and high SEB (see Table C.1 in Appendix C).

8 Sensitivity analysis

Testing if background variables are associated with the probability of being in the diabetes group (Table 6), the results show no association, suggesting that neither the individuals' nor the parents' behavior influences onset.[17] The placebo test

[17] There is a positive association between missing data on the father's level of education and type 1 diabetes. As only a few individuals have missing data on the father's level of education, the association is likely to be of lower importance.

Table 7: Probit (average marginal effects) estimations of university education at age 30 for onset (inclusion) ages 24–26

Dependent variable	Women			Men		
University	(1)	(2)	(3)	(4)	(5)	(6)
Diabetes	-0.021	-0.0063	-0.022	0.016	0.0093	0.0010
	(0.036)	(0.038)	(0.038)	(0.026)	(0.027)	(0.026)
Covariates	Yes	Yes	Yes	Yes	Yes	Yes
SEB	No	Yes	Yes	No	Yes	Yes
Family status	No	No	Yes	No	No	Yes
Observations	1069	1069	1069	1855	1855	1855

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Covariates indicates controls for year, cohort, and municipality of residence.

SEB indicates controls for parental level of education.

Family status indicates controls for marital status and number of children.

(Table 7), repeats the main analysis for onset at the age of 24–26. Showing no association, this test rejects that the negative link between type 1 diabetes and university education is driven by any (observable or unobservable) group level difference other than type 1 diabetes. The estimates for women only decreases slightly when redefining the outcome variable as having two or more years of university education (Table 8), indicating that the differences between women with and without type 1 diabetes persist past enrollment. The results persist when using university programs that are four years or longer as the outcome variable. These results are available on request.

The results are robust for omitting individuals with type 1 diabetes onset at age 17 (and controls) (Table 9), ensuring that the youngest individuals are not the ones driving the result. Repeating the main analysis for university education at age 35 (Table 10) generates similar results as at age 30, indicating a permanent shortfall in education rather than educational delay. Similarly, the results persist also for having one or more children at age 35 (Table 11).

9 Discussion

Using Swedish nationwide register data on individuals with young-adulthood onset of type 1 diabetes (ages 17–20), this paper shows that women are less likely to have a university education (at age 30), while no such difference appears for men. The negative link for women remains after controlling for covariates such as SEB, family status, year dummies, and the case-control matching on age,

Table 8: Probit (average marginal effects) estimations of having more than two years of university education at age 30

Dep. var.	Women			Men		
Uni 2+	(1)	(2)	(3)	(4)	(5)	(6)
Diabetes	-0.061*	-0.053*	-0.068**	-0.010	-0.0037	-0.0031
	(0.032)	(0.032)	(0.031)	(0.023)	(0.023)	(0.023)
Covariates	Yes	Yes	Yes	Yes	Yes	Yes
SEB	No	Yes	Yes	No	Yes	Yes
Family status	No	No	Yes	No	No	Yes
Observations	1151	1151	1151	1984	1984	1984

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.
Covariates indicates controls for year, cohort, and municipality of residence.
SEB indicates controls for parental level of education.
Family status indicates controls for marital status and number of children.

Table 9: Probit (average marginal effects) estimations of university education at age 30 excluding those with onset (inclusion) at age 17

Dependent variable	Women			Men		
University	(1)	(2)	(3)	(4)	(5)	(6)
Diabetes	-0.075*	-0.073*	-0.089**	-0.032	-0.022	-0.022
	(0.039)	(0.041)	(0.041)	(0.029)	(0.029)	(0.030)
Covariates	Yes	Yes	Yes	Yes	Yes	Yes
SEB	No	Yes	Yes	No	Yes	Yes
Family status	No	No	Yes	No	No	Yes
Observations	896	896	896	1537	1537	1537

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.
Covariates indicates controls for year, cohort, and municipality of residence.
SEB indicates controls for parental level of education.
Family status indicates controls for marital status and number of children.

Table 10: Probit (average marginal effects) estimations of university education at age 35

Dependent variable	Women			Men		
University	(1)	(2)	(3)	(4)	(5)	(6)
Diabetes	-0.098**	-0.084*	-0.086*	-0.038	-0.033	-0.033
	(0.045)	(0.046)	(0.047)	(0.033)	(0.034)	(0.034)
Covariates	Yes	Yes	Yes	Yes	Yes	Yes
SEB	No	Yes	Yes	No	Yes	Yes
Family status	No	No	Yes	No	No	Yes
Observations	653	653	653	1124	1124	1124

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.
Covariates indicates controls for year, cohort, and municipality of residence.
SEB indicates controls for parental level of education.
Family status indicates controls for marital status and number of children.

Table 11: Probit (average marginal effects) estimations of having one or more children at age 35 by socioeconomic background (SEB) for women

Dep. var.	(1)	(2)	(3)
Child(ren)	Low SEB	Middle SEB	High SEB
Diabetes	-0.13	-0.24***	-0.083
	(0.080)	(0.074)	(0.093)
Covariates	Yes	Yes	Yes
Observations	183	247	184

Robust standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1.
Covariates indicates controls for year, cohort, and municipality of residence.

gender, and municipality of residence. Persisting at age 35, this link appears to represent a permanent shortfall in education rather than educational delay.

Although the underlying mechanisms remain unknown, differing fertility constraints offer one explanation for why type 1 diabetes affects women but not men. Given that university studies delay family formation (Boschini et al., 2011; Gustafsson & Adriaan, 2006; Adsera, 2011) and that type 1 diabetes amplifies the risk of pregnancy-related problems when delaying fertility (Jonasson et al., 2007; Casson et al., 1997), it is not surprising that our results indicate that type 1 diabetes affects both the decision to enter university and to start a family. Combined, our findings (together with the recollection of amplified pregnancy-related problems) suggest an enhanced tradeoff between university education and motherhood after type 1 diabetes onset: women with type 1 diabetes and a high SEB have a lower likelihood of university education than other women with a high SEB, but women with diabetes have the same likelihood of being mothers, in spite of their higher risk of pregnancy-related complications. We find the same pattern among women with a low SEB, although the drop in university education is somewhat smaller for women with type 1 diabetes and a low SEB. In contrast, women with type 1 diabetes and a middle SEB do not show a drop in university education, but they are less likely to be mothers, compared to other women with a middle SEB.

Intuitively, it may appear striking that women with high SEB are the ones with the largest reduction in their likelihood of having a university education after onset. Possibly, the reasons for not attending university could vary across the socioeconomic strata. Type 1 diabetes increases the uncertainty about future health, and therefore jeopardizes educational returns. Women with different SEB might respond differently to these uncertainties as SEB is likely (through, e.g., prior experience and differing networks) to relate to access to diabetes-specific information. If women with a high SEB are more likely than other women to assimilate the knowledge that diabetes induces pregnancy-related complications (and that these complications increase with age) then women with a high SEB who wish to become mothers may be less inclined than women with a lower SEB to postpone motherhood for university education. Moreover, as individuals with higher SEB receive, on average, more financial support from their parents throughout life (see, e.g., Henretta et al. (2002), and Grundy (2005)), women with type 1 diabetes and a high SEB may be more willing than other women with type 1 diabetes to forgo future earnings premiums from university education, and still have the financial means to start a family.

From an econometric point of view, type 1 diabetes has characteristics mimicking an unexpected health shock (Persson et al., 2013; Minor, 2011; Steen Carlsson et al., 2010). Lundborg et al. (2014) confirm that men's earnings penalty from diabetes is robust to selection on unobserved factors at the family level. In addition, there is no support for confounding in the medical or epidemiological

literature (e.g., lifestyle factors appear unrelated to onset (American Diabetes Association, 2008). Nor can we find any indication in the data of pre-onset differences between individuals with and without type 1 diabetes, as (1) a placebo test rejects any group level differences apart from type 1 diabetes, (2) none of the observable factors available in the data predicts onset, and (3) no descriptive differences in education or in parental level of education exist in the data before onset. Consequently, we find no indication that potential third factors are influencing the studied relationship.

The magnitude of the link between type 1 diabetes and university education is likely to be contextual, and affected by the education and health care systems. In Sweden, university education and health care are primarily publicly funded and the pharmaceutical reimbursement system fully subsidizes insulin for individuals with type 1 diabetes. As diabetes management is costly, one would expect a larger negative link between type 1 diabetes and university education in privately funded health care settings with substantial out-of-pocket payments for pharmaceutical and medical care. The reason is that present health care expenditure (and inadequate financial markets for borrowing) would be likely to increase the demand for present labor market earnings.

To conclude, this study indicates that type 1 diabetes affects women's decisions on both university education and family formation. Taken together, the results suggest that type 1 diabetes intensifies the conflict between motherhood and university education, and that women of different socioeconomic backgrounds may respond differently to such a conflict. Still, the remaining uncertainty in the exact mechanisms that govern women's educational decisions calls for prudence in policy interventions and encourages further research.

Acknowledgments

Katarina Steen Carlsson, Lund University, Sweden, in collaboration with the Diabetes Incidence Study in Sweden (DISS) steering group, designed and constructed the Econ-DISS database for research on labor market outcomes for young adults with diabetes onset. Steen Carlsson's project was funded by research grants from the Foundation for Research at the Swedish Society for Diabetes (DIA 2005–005, DIA 2006–049, DIA 2007–065), and by an unrestricted researcher-initiated grant from Novo Nordisk A/S. DISS was supported by grants from the Juvenile Diabetes Foundation–Wallenberg Diabetes Research Program (K 98–99 JD–128 13), the Swedish Diabetes Association, and the Swedish Medical Research Council (7X–14531). We thank members of the network of diabetologists in the six health-care regions of Sweden, who have been responsible for data collection for DISS since 1983. The LISA database is administered by Statistics Sweden and the Social Insurance Agency, and the Swedish Agency for Innovative Systems participated in its construction. We also thank Elvira Andersson, Kristian

Bolin, David Granlund, Soffia Gudbjörnsdottir, Björn Lindgren, Carl Hampus Lyttkens, Olov Rolandsson, Katarina Steen Carlsson, Therese Nilsson and Jan Östman for valuable support and insightful comments. Seminar participants at the 8th European Conference on Health Economics in Helsinki, Finland, and the 31st Nordic Health Economists Study Group meeting in Umeå, Sweden, provided helpful comments and suggestions.

References

Adsera, A. (2011). Where are the babies? labor market conditions and fertility in europe. *European Journal of Population*, *27*(1), 1-32.

Almond, D., & Currie, J. (2011). Killing me softly: The fetal origins hypothesis. *Journal of Economic Perspectives*, *25*(3), 153–172.

American Diabetes Association. (2008). Diagnosis and classification of diabetes mellitus. *Diabetes Care*, *31*(Supplement 1), 55–60.

Atkinson, M. A., & Eisenbarth, G. S. (2001). Type 1 diabetes: new perspectives on disease pathogenesis and treatment. *The Lancet*, *358*(9277), 221–229.

Bastida, E., & Pagán, J. A. (2002). The impact of diabetes on adult employment and earnings of mexican americans: Findings from a community based study. *Health Economics*, *11*(5), 403–413.

Becker, G. S. (1962). Investment in human capital: A theoretical analysis. *Journal of Political Economy*, *70*, 9–49.

Becker, G. S. (1965). A theory of the allocation of time. *The Economic Journal*, *72*(299), 493–517.

Becker, G. S., & Mulligan, C. B. (1997). The endogenous determination of time preference. *The Quarterly Journal of Economics*, *112*(3), 729–758.

Becker, G. S., & Tomes, N. (1976). Child endowments and the quantity and quality of children. *Journal of Political Economy*, *84*, 143–162.

Behrman, J., Pollak, R., & Taubman, P. (1982). Parental preferences and provision for progeny. *Journal of Political Economy*, 52–73.

Björklund, A. (2006). Does family policy affect fertility? *Journal of Population Economics*, *19*(1), 3–24.

Black, S. E., & Devereux, P. J. (2011). Recent developments in intergenerational mobility. *Handbook of Labor Economics*, *4*, 1487–1541.

Boschini, A., Håkanson, C., Rosén, Å., & Sjögren, A. (2011). *Trading off or having it all? completed fertility and mid-career earnings of Swedish men and women* (Working Paper, IFAU - Institute for Labour Market Policy Evaluation No. 15).

Brown, H. S., Pagán, J. A., & Bastida, E. (2005). The impact of diabetes on employment: genetic IVs in a bivariate probit. *Health Economics*, *14*(5), 537–544.

Brown, H. S., Perez, A., Yarnell, L. M., Pagan, J. A., Hanis, C. L., Fischer-Hoch, S., & McCormick, J. B. (2010). Diabetes and employment productivity: does diabetes management matter? *The American journal of managed care*, *17*(8), 569–576.

Casson, I., Clarke, C., Howard, C., Mc-Kendrick, O., Pennycook, S., Pharoah, P., ... Walkinshaw, S. (1997). Outcomes of pregnancy in insulin dependent diabetic women: results of a five year population cohort study. *BMJ*, *315*, 275–278.

CDC The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. (1997). Report of the expert committee on the diagnosis and classification of diabetes mellitus. *Diabetes Care*, *20*, 1183–1197.

Centers for Disease Control and Prevention. (2005). *National diabetes fact sheet*. `http://www.cdc.gov/diabetes/pubs/pdf/ndfs_2005.pdf`. (20 Mars 2012)

Chevalier, A. (2004). *Parental education and child's education : A natural experiment* (IZA Discussion paper series No. 1153).

Clausen, J. O., Borch-Johnsen, K., Ibsen, H., Bergman, R. N., Hougaard, P., Winther, K., & Pedersen, O. (1996). Insulin sensitivity index, acute insulin response, and glucose effectiveness in a population-based sample of 380 young healthy Caucasians. Analysis of the impact of gender, body fat, physical fitness, and life-style factors. *The Journal of Clinical Investigation*, *98*.

Cunha, F., & Heckman, J. (2008). Formulating, identifying and estimating the technology of cognitive and noncognitive skill formation. *Journal of Human Resources*, *43*, 738–782.

Cunha, F., Heckman, J., Lochner, L., & Masterov, D. (2006). Interpreting the evidence on life cycle skill formation. *Handbook of the Economics of Education*, *1*, 697–812.

Currie, J. (2009). Healthy, wealthy, and wise: Socioeconomic status, poor health in childhood, and human capital development. *Journal of Economic Literature*, *47*, 87–122.

Cutler, D. M., Glaeser, E. L., & Shapiro, J. M. (2003). Why have americans become more obese? *Journal of Economic Perspectives, 17*, 93–118.

Dahlquist, G., Källén, B., & Swedish Childhood Diabetes Study Group. (2007). School performance in children with type 1 diabetes–a population-based register study. *Diabetologia, 50*(5), 957–64.

Dahlquist, G., & Mustonen, L. (2000). Analysis of 20 years of prospective registration of childhood onset diabetes time trends and birth cohort effects. Swedish Childhood Diabetes Study Group. *Acta Paediatrica, 89*(10), 1231–1237.

Dahlquist, G., Nyström, L., Patterson, C. C., et al. (2011). Incidence of type 1 diabetes in Sweden among individuals aged 0–34 years, 1983–2007. An analysis of time trends. *Diabetes Care, 34*(8), 1754–1759.

Dahlquist, G., Pundziūtė-Lyckå, A., Nyström, L., et al. (2005). Birthweight and risk of type 1 diabetes in children and young adults: a population-based register study. *Diabetologia, 48*(6), 1114–1117.

Daneman, D. (2006). Type 1 diabetes. *The Lancet, 367*(9513), 847–858.

Dearden, L., Machin, S., & Reed, H. (1997). Intergenerational mobility in britain. *Economic Journal, 107*, 47–66.

Devaux, M., & Sassi, F. (2013). Social inequalities in obesity and overweight in 11 OECD countries. *The European Journal of Public Health, 23*(3), 464–469.

Fletcher, J. M., & Richards, M. R. (2012). Diabetes's 'health shock'to schooling and earnings: Increased dropout rates and lower wages and employment in young adults. *Health Affairs, 31*(1), 27–34.

Glied, S., & Lleras-Muney, A. (2008). Technological innovation and inequality in health. *Demography, 45*(3), 741–761.

Goldman, D., & Smith, J. P. (2002). Can patient self-management help explain the ses health gradient? *Proceedings of the National Academy of Sciences of the United States of America, 99*(16), 10929–10934.

Greenwood, J., Guner, N., & Knowles, J. A. (2003). More on marriage, fertility, and the distribution of income. *International Economic Review, 44*(3), 827–862.

Grundy, E. (2005). Reciprocity in relationships: socio-economic and health influences on intergenerational exchanges between third age parents and their adult children in Great Britain. *The British Journal of Sociology*, *56*(2), 233–255.

Gustafsson, S., & Adriaan, K. (2006). Fertility decisions : economic theory, empirical analysis, and policy relevance. In (pp. 31–64). Springer.

Harris, A. (2008). *Chronic disease and labour force participation in australia: an endogenous multivariate probit analysis of clinical prevalence data* (Tech. Rep.).

Henretta, J., Grundy, E., & Harris, S. (2002). The influence of socio-economic and health differences on parents' provision of help to adult children: A British–United States comparison. *Ageing and Society*, *22*(4), 41–58.

Jonasson, J. M., Brismar, K., Sparén, P., Lambe, M., Nyrén, O., Östenson, C.-G., & Ye, W. (2007). Fertility in women with type 1 diabetes: A population-based cohort study in Sweden. *Diabetes Care*, *30*(9), 2271–2276.

Lahmann, P. H., Lissner, L., Gullberg, B., & Berglund, G. (2000). Sociodemographic factors associated with long-term weight gain, current body fatness and central adiposity in Swedish women. *International Journal of Obesity & Related Metabolic Disorders*, *24*, 685.

Latif, E. (2009). The impact of diabetes on employment in Canada. *Health Economics*, *18*(5), 577–589.

Lissner, L., Johansson, S., Qvist, J., Rössner, S., & Wolk, A. (2000). Social mapping of the obesity epidemic in Sweden. *International Journal of Obesity & Related Metabolic Disorders*, *24*, 801–5.

Lundborg, P., Nilsson, A., & Rooth, D.-O. (2014). Adolescent health and adult labor market outcomes. *Journal of Health Economics*, *37*, 25–40.

Lundin, D., Mörk, E., & Öckert, B. (2008). How far can reduced childcare prices push female labour supply? *Labour Economics*, *15*(4), 647–659.

Maslow, G. R., Haydon, A., McRee, A.-L., Ford, C. A., & Halpern, C. T. (2011). Growing up with a chronic illness: Social success, educational/vocational distress. *Journal of Adolescent Health*, *49*(2), 206–212.

Milton, B., Holland, P., & Whitehead, M. (2006). The social and economic consequences of childhood-onset type 1 diabetes mellitus across the lifecourse: a systematic review. *Diabetic Medicine*, *23*, 821–829.

Minor, T. (2011). The effect of diabetes on female labor force decisions: new evidence from the national health interview survey. *Health Economics*, *20*(12), 1468–1486.

Minor, T. (2013). An investigation into the effect of type I and type II diabetes duration on employment and wages. *Economics & Human Biology*, *11*(4), 534–544.

Molarius, A. (2003). The contribution of lifestyle factors to socioeconomic differences in obesity in men and women — a population-based study in Sweden. *European Journal of Epidemiology*, *18*, 227–234.

Molarius, A., Seidell, J. C., Sans, S., Tuomilehto, J., & Kuulasmaa, K. (2000). Educational level, relative body weight, and changes in their association over 10 years: an international perspective from the who monica project. *American Journal of Public Health*, *90*, 1260–1268.

Mulligan, C. B. (1999). Galton versus the human capital approach to inheritance. *Journal of Political Economy*, *107*(6), 184–224.

Östman, J., Arnqvist, H., Blohmé, G., Lithner, F., Littorin, B., Nyström, L., … Wibell, L. (1986). Epidemiology of diabetes mellitus in Sweden. results of the first year of a prospective study in the population age group 15–34 years. *Acta Medica Scandinavica*, *220*(5), 437–445.

Östman, J., Lönnberg, G., Arnqvist, H. J., Blohmé, G., Bolinder, J., Ekbom Schnell, A., … Nyström, L. (2008). Gender differences and temporal variation in the incidence of type 1 diabetes: results of 8012 cases in the nationwide Diabetes Incidence Study in Sweden 1983–2002. *Journal of Internal Medicine*, *263*(4), 386–394.

Peltonen, M., Rosen, M., & Asplund, V. L. K. (2000). Social patterning of myocardial infarction and stroke in Sweden: Incidence and survival. *American Journal of Epidemiology*, *3*(151), 283–92.

Persson, S., Dahlquist, G., Gerdtham, U., & Steen Carlsson, K. (2013). Impact of childhood-onset type 1 diabetes on schooling: a population-based register study. *Diabetologia*, *56*(6), 1254–1262.

Ploug, U. J. (2013). The cost of diabetes-related complications: Registry-based analysis of days absent from work. *Economics Research International*, *2013*.

Prentice, A. M., & Jebb, S. A. (1995). Obesity in britain: gluttony or sloth? *BMJ*, *311*, 437–439.

Pundziute-Lyckå, A., Dahlquist, G., Nyström, L., Arnqvist, H., Björk, E., Blohme, G., ... others (2002). The incidence of type I diabetes has not increased but shifted to a younger age at diagnosis in the 0–34 years group in Sweden 1983 to 1998. *Diabetologia*, *45*(6), 783–791.

Rees, D., & Sabia, J. (2011). The effect of migraine headache on educational. *Journal of Human Resources*, *46*(2), 317–332.

Rosvall, M., Chaix, B., Lynch, J., Lindström, M., & Merlo, J. (2008). The association between socioeconomic position, use of revascularization procedures and five-year survival after recovery from acute myocardial infarction. *BMC Public Health*, *8*(1), 44.

Schrijvers, C. T., & Mackenbach, J. P. (1994). Cancer patient survival by socioeconomic status in seven countries: a review for six common cancer sites [corrected]. *Journal of Epidemiology and Community Health*, *48*(16), 441–446.

Smith, G. D., Hart, C., Watt, G., Hole, D., & Hawthorne, V. (1979). Individual social class, area-based deprivation, cardiovascular disease risk factors, and mortality: The renfrew and paisley study. *Journal of Epidemiology and Community Health*, *52*(6).

Soltesz, G., Patterson, C., & Dahlquist, G. (2007). Worldwide childhood type 1 diabetes incidence–what can we learn from epidemiology? *Pediatric Diabetes*, *8*(s6), 6–14.

Statistics Sweden. (2008). *Higher education. students and graduated students in undergraduate education 2006/07*. `http://www.scb.se/statistik/UF/UF0205/2008A01/UF0205_2008A01_SM_UF20SM0801.pdf`. (20 Februari 2014)

Statistics Sweden. (2009). *Multi-generation register*. `www.scb.se/Pages/List____257501`. (3 October 2012)

Statistics Sweden. (2011). *Longitudinal integration database for health insurance and labour market studies (LISA by Swedish acronym)*. http://www.scb.se/Pages/List____257743.aspx. (6 June 2012)

Steen Carlsson, K., Landin-Olsson, M., Nyström, L., Arnqvist, H. J., Bolinder, J., Östman, J., & Gudbjörnsdottir, S. (2010). Long-term detrimental consequences of the onset of type 1 diabetes on annual earnings-evidence from annual registry data in 1990–2005. *Diabetologia*, *53*(6), 1084–92.

Stumvoll, M., Goldstein, B. J., & van Haeften, T. W. (2005). Type 2 diabetes: principles of pathogenesis and therapy. *The Lancet*, *365*(9467), 1333–1346.

The TEDDY Study Group. (2007). The environmental determinants of diabetes in the young (TEDDY) study: study design. *Pediatric Diabetes*, *8*(5), 286–298.

Tunceli, K., Bradley, C. J., Nerenz, D., Williams, L. K., Pladevall, M., & Lafata, J. E. (2005). The impact of diabetes on employment and work productivity. *Diabetes Care*, *28*(11), 2662–2667.

Vijan, S., Hayward, R. A., & Langa, K. M. (2004). The impact of diabetes on workforce participation: Results from a national household sample. *Health Services Research*, *39*(6), 1653–1670.

Wennick, A., Hallström, I., Lindgren, B., & Bolin, K. (2011). Attained education and self-assessed health later in life when diagnosed with diabetes in childhood: a population-based study. *Pediatric diabetes*, *12*(7), 619–626.

WHO. (1980). *WHO expert committee on diabetes mellitus. Second report. World health organization technical report series*. http://whqlibdoc.who.int/trs/WHO_TRS_646.pdf. Geneva. (6 June 2012)

WHO. (1985). *Diabetes mellitus. Report of a WHO study group. Technical report series*. http://whqlibdoc.who.int/trs/WHO_TRS_727.pdf. Geneva. (6 June 2012)

Zhang, X., Zhao, X., & Harris, A. (2009). Chronic diseases and labour force participation in australia. *Journal of Health Economics*, *28*(1), 91–108.

Appendices

A Figure of years of education by age

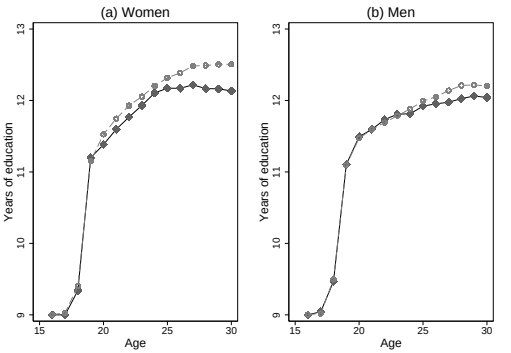

Figure A.1: Years of education by age for women (a) and men (b) with type 1 diabetes (black line with black diamonds) and controls (gray line with hollow diamonds). Secondary education is often registered with a lag; therefore, the data points at age 16–18 should be interpreted cautiously.

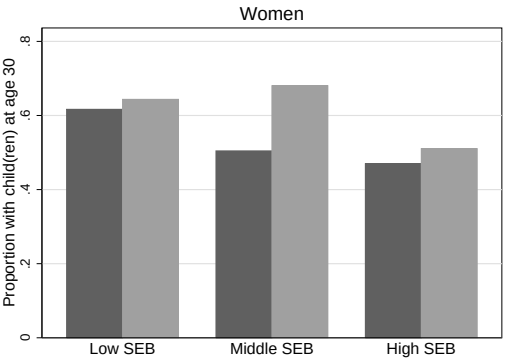

Figure A.2: Proportion of women with one or more children among women with type 1 diabetes (black bars) and controls (gray bars) by socioeconomic background (SEB). *Low* if highest level of education of either parent is compulsory, *middle* if highest level of either parent is upper secondary, and *high* if highest level of either parent is university.

B Extended results

Table B.1: Probit (average marginal effects) estimation of university education at age 30

Dep. var.	Women			Men		
University	(1)	(2)	(3)	(4)	(5)	(6)
Diabetes	-0.0898***	-0.0827**	-0.104***	-0.0368	-0.0283	-0.0274
	(0.0342)	(0.0357)	(0.0353)	(0.0256)	(0.0261)	(0.0262)
Compulsory M		-0.0233	-0.0198		-0.0577**	-0.0536**
		(0.0362)	(0.0368)		(0.0253)	(0.0254)
Upper secondary M		ref.	ref.		ref.	ref.
University M		0.261***	0.234***		0.169***	0.160***
		(0.0412)	(0.0425)		(0.0322)	(0.0322)
Missing M		-0.0310	-0.0458		-0.158***	-0.158***
		(0.0831)	(0.0841)		(0.0524)	(0.0531)
Compulsory F		-0.0815**	-0.0847**		-0.0906***	-0.0923***
		(0.0352)	(0.0354)		(0.0251)	(0.0251)
Upper secondary F		ref.	ref.		ref.	ref.
University F		0.153***	0.122***		0.227***	0.217***
		(0.0443)	(0.0451)		(0.0340)	(0.0342)
Missing F		-0.00644	-0.0319		-0.0182	-0.0122
		(0.0639)	(0.0631)		(0.0462)	(0.0474)
Children			-0.113***			-0.0775***
			(0.0169)			(0.0144)
Married			0.140***			0.120***
			(0.0372)			(0.0319)
Divorced			-0.149			-0.0707
			(0.0951)			(0.0834)
Covariates	yes	yes	yes	yes	yes	yes
Observations	1151	1151	1151	1984	1984	1984

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Covariates indicates controls for year, cohort, and municipality of residence.

C Table 5 stratified by SEB

Table C.1: Probit (average marginal effects) estimation of having *both* university education and one or more children at age 30 by socioeconomic background (SEB) for women

Dependent variable	(1)	(2)	(3)
Uni&Child(ren)	Low SEB	Middle SEB	High SEB
Diabetes	-0.0910***	-0.0512	-0.140***
	(0.0337)	(0.0365)	(0.0532)
Covariates	Yes	Yes	Yes
Observations	237	524	350

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.
Covariates indicates controls for year, cohort, and municipality of residence.

Paper III

Labor market consequences of growing up with type 1 diabetes

Abstract

Exploring the long-term labor market consequences of growing up with type 1 diabetes, this paper investigates how childhood onset of type 1 diabetes (6 to 15 years old) influences adult labor market outcomes, both for children developing the disease and their siblings. The results indicate a negative impact on labor market outcomes (throughout ages 19 to 48) for those who develop type 1 diabetes as children. Both women and men with diabetes have a lower likelihood of employment and lower annual labor earnings than controls. The decrease in women's likelihood of employment is roughly twice that for men, whereas the increasingly negative link to earnings appears more profound for men. For siblings of individuals with type 1 diabetes, sisters' outcomes appear unaffected, while brothers' outcomes show, on the one hand, a higher likelihood of being employed, but, on the other hand, lower earnings reminiscent of the earnings decrease for individuals with type 1 diabetes themselves. These novel findings for brothers of individuals with type 1 diabetes support actions that consider broader family impact, both when initiating further research and when designing children's diabetes management programs.

Keywords: Health, earnings, diabetes, siblings
JEL Classification: D13, I10, I12, J24, J13, J31

1 Introduction

The existing evidence suggests that early life conditions and childhood health affect adult labor market outcomes (for reviews, see e.g., Currie & Almond (2011), and Currie (2009)). First, poor child health may affect future health (see, e.g., the literature on fetal origins as described in Almond & Currie (2011)), which in turn can affect labor supply and productivity (see, e.g., Lundborg et al. (2014), Johnson & Schoeni (2011), Smith (2009), Lindeboom et al. (2006), Case et al. (2005), and Currie & Madrian (1999)). Second, poor child health may affect cognition and impair children's ability to learn new skills (see, e.g., Johnson & Schoeni (2011), Maluccio et al. (2009), and Cunha & Heckman (2008)). Third, poor child health may operate via absenteeism in school, thereby affecting educational outcomes (see, e.g., Grossman & Kaestner (1997), Oreopoulos et al. (2008), and Case et al. (2005)). Extensive research confirms the link between child health and adult outcomes for many aspects of health (e.g., conditions in utero, low birth weight, chronic illnesses, and acute conditions), but it remains unknown whether poor child health also has long-term consequences for siblings growing up with a sick brother or sister.

Spending time and effort to restore and maintain health, families with a sick child are likely to have fewer resources available for other activities.[1] Clearly, poor child health may therefore affect not only the sick child but also his or her siblings: if, for example, parents spend less time helping with homework, both the sick child and his or her siblings may suffer negative consequences in relation to their accumulation of new skills and abilities. Alternatively, if, in caring for a sick child, parents become more health- and family-oriented, their children may learn skills (such as responsibility) that favor future labor market outcomes. Consequently, parents' responses might either compensate for or reinforce the impact of type 1 diabetes, equally for all children or differently across siblings.[2]

This study examines the progression of annual labor earnings after childhood onset of type 1 diabetes (age 6–15) both for adults with type 1 diabetes and for their adult siblings (throughout ages 19–48). Type 1 diabetes typically develops with a rapid onset without prior symptoms, mimicking a 'before and after' treatment study design. Despite extensive research, the exact combination of environmental and genetic factors, together with the chain of events triggering type 1 diabetes onset, remains unclear. Lifestyle factors (i.e., obesity and physical

[1] This reasoning builds on the work of (1) Bolin et al. (2003, 2002), and Jacobson (2000), who model the family as the health-producing unit and assume that the health of all family members is interrelated, and (2) Cunha & Heckman (2007), and Heckman et al. (2006), who argue that human capabilities (i.e., health, cognitive skills and noncognitive skills) are closely related and are formed throughout the life cycle of a child.

[2] Currie & Almond (2011) provide a structural framework and discuss how parents influence their children's human capital accumulation in response to early childhood conditions. They conclude that there is presently little support for any systematic reinforcement or compensation for early childhood events in developed countries.

inactivity) that are associated with low education (Devaux & Sassi, 2013; Cutler et al., 2003; Molarius, 2003; Lahmann et al., 2000; Molarius et al., 2000; Lissner et al., 2000) do not appear to affect lifetime risk for onset (American Diabetes Association, 2008). More likely, factors outside the individual's control, such as genetics, cold climate, and virus infection in early life, seem to be at play (Dahlquist et al., 2011, 2005; Soltesz et al., 2007; The TEDDY Study Group, 2007; Atkinson & Eisenbarth, 2001). Due to this complexity and the sudden onset, type 1 diabetes is generally seen as a health shock, which the individual is unable to anticipate or influence beforehand (Persson et al., 2013; Minor, 2011; Steen Carlsson et al., 2010).

Still, we cannot rule out that third factors are influencing the studied relationship. I, therefore, use a fixed effect approach to control for time-invariant factors (observable and unobservable), at the individual level, that may be associated with both onset and labor market outcomes. However, the fixed effect approach relies on variation within individuals, but onset of type 1 diabetes occurs before the individual enters the labor market and will not vary over time. To get the variation needed to identify diabetes-related changes in labor market outcomes, I estimate age-specific earnings differences. This strategy allows the influence of diabetes to vary over time, capturing variations in health due to diabetes-related complications (Daneman, 2006; Möllsten et al., 2010).

Furthermore, the data of this study builds on a case-control design with four population controls matched, to each individual with diabetes, by year of birth and municipality of residence at the time of diabetes diagnosis. Given this matching strategy, the controls are not randomly assigned and are not optimal for studying siblings of the individuals with diabetes (even if the siblings are likely to have had the same municipality of residence as their brother or sister and I only include siblings from the same cohorts as the controls). To correct, at least in part, for the fact that the control group is designed to fit the diabetes group and not their siblings, and to improve the match also for individuals with diabetes, I perform the entropy balancing method proposed by Hainmueller (2012).[3] By reweighting the data before applying the fixed effect approach, I tweak the control groups to control for further influences from potential factors during upbringing beyond the individual fixed effects. Building on the well-known propensity score matching (PSM) technique, the entropy balancing method achieves balance by constructing a weight for *each control observation* such that the sample moments of covariates are identical between the treated (diabetes and siblings respectively) and the weighted controls. In practice, the weights are chosen to make the weighted control groups match the treatment groups in terms of observable pre-onset characteristics, including, e.g., year of birth, parental education, and parental year of birth.

[3] The novel entropy balancing method has previously been used by, e.g., Huang & Yeh (2014), Marcus (2013), and Krishnan & Krutikova (2013).

Using Swedish longitudinal register data for the years 1990–2010, I study differences in labor market outcomes (throughout ages 19–48) between individuals with type 1 diabetes and weighted population controls. I also compare siblings of the individuals with type 1 diabetes to weighted population controls (i.e., the same controls as for the diabetes group but with different weights). The results indicate a negative impact on labor market outcomes (throughout ages 19–48) for those who developed type 1 diabetes as children (aged 6–15). Both women and men with diabetes have a lower likelihood of employment and lower annual labor earnings than controls. The decrease in women's likelihood of employment is about twice the size of that of men, whereas the increasingly negative link to earnings appears more profound for men. For siblings of individuals with type 1 diabetes, sisters' outcomes appear unaffected, while brothers' report, on the one hand, a higher likelihood of being employed, but, on the other hand, lower earnings, reminiscent of the earnings decrease for individuals with type 1 diabetes themselves.

The structure of the paper is as follows. Section 2 reports previous research on type 1 diabetes and economic outcomes, and insights from research within psychology on siblings of chronically ill children. Section 3 discusses a conceptual framework. Section 4 presents the data. Section 5 details my econometric strategy and describes the entropy balancing technique. Section 6 presents the results, and Section 7 the sensitivity analyses. Section 8 concludes.

2 Previous research

Previous economic research indicates that children with type 1 diabetes grow up to have adverse educational and labor market outcomes. Based on Swedish register data, children with onset of type 1 diabetes at ages 0–14 have lower grades from compulsory education (Persson et al., 2013; Dahlquist et al., 2007) and from theoretical upper secondary programs preparing for university (Persson et al., 2013), and have higher risk of unemployment later in life (Persson et al., 2013). Also, young adults with onset of type 1 diabetes at ages 14–34 have higher risk of unemployment and lower annual labor earnings (Steen Carlsson et al., 2010). Using Swedish enlistment data and data for onset of diabetes before age 18, Lundborg et al. (2014) report negative earnings penalties for men. Using US survey data, Minor (2011, 2013) studied both type 1 and type 2 diabetes, and concludes that the associations between diabetes and labor market outcomes are driven by type 2 diabetes, while he finds no significant associations for type 1 diabetes.[4] Probably because of data limitations, most other previous studies on diabetes and economic outcomes depend on small-sample surveys (Milton et al.,

[4]The study by Minor (2011) includes only women. Minor (2013) reports a positive association for women's wages for one specification, but he disregards this finding because it is based on an extremely low number of observations.

2006) or cannot discriminate between type 1 and type 2 diabetes. [5] (Ploug, 2013; Fletcher & Richards, 2012; Maslow et al., 2011; Brown et al., 2010; Latif, 2009; Zhang et al., 2009; Harris, 2008; Brown et al., 2005; Tunceli et al., 2005; Vijan et al., 2004; Bastida & Pagán, 2002). None of these studies considers individual fixed effects and influences from third factors.

To date, economic research has mainly focused on the long-term labor market consequences of having impaired health as a child, and not, to the best of my knowledge, assessed spillover effects for siblings. The literature within psychology suggests that siblings of chronically ill children often have contradictory feelings towards their sick brother or sister: a strong sense of responsibility (e.g., acting as protector and caregiver); resentment (e.g., being jealous of the sick sibling receiving extra attention); exaggerated sibling rivalry (e.g., fighting for parents' attention); and social and emotional isolation (e.g., being afraid to increase their parents' worries and to evoke their parents' anger if they have negative feelings for, or fail to protect, their sick sibling)(Wennick & Huus, 2012). To have such feelings may interfere with psychological growth and contribute to feelings of low self-esteem, anxiety and/or depressive and psychosomatic symptoms (Hollidge, 2001).

Siblings of children with type 1 diabetes, however, have not been thoroughly studied and the existing findings are inconsistent (Sleeman et al., 2010; Barlow & Ellard, 2006): some studies find increased risk of maladjustment (Adams et al., 1991), while others find that siblings of children with type 1 diabetes function as well, or even better, than siblings of children without diabetes (Sleeman et al., 2010; Jackson et al., 2008; Hollidge, 2001; Lavigne et al., 1982). Even if reporting conflicting results, the psychological literature suggests that boys and girls may respond differently when their sibling falls ill (ÓBrien et al., 2009; Hollidge, 2001; Lavigne et al., 1982). Girls tend to show more internalizing symptoms (e.g., depression, withdrawal), while boys show more externalizing ones (e.g., hyperactivity, aggression). Within this literature, also adjustment studies on children and adolescents with type 1 diabetes show inconsistency (Luyckx et al., 2010; Gendelman et al., 2009), and suggest similar differences between boys and girls (Gendelman et al., 2009; Hood et al., 2006; Naar-King et al., 2006). Possibly, we could expect gender difference also in the studied labor market responses to growing up with type 1 diabetes, as externalizing behaviors have been connected to adverse educational (McLeod & Kaiser, 2004; Miech et al., 1999) and labor market (Gregg & Machin, 2000) outcomes, whereas internalizing strategies appear unimportant for future outcomes (McLeod & Kaiser, 2004; Miech et al., 1999). Contributing to these gender differences, type 1 diabetes amplifies the risk of severe pregnancy-related complications for both the mother and child, and even more so with increasing age (Jonasson et al., 2007; Casson et al., 1997).

[5] The two types of diabetes have fundamentally different pathogenesis and expected impact on labor market outcomes.

3 Conceptual framework

Type 1 diabetes is a lifelong chronic disease that generally develops early in life (Daneman, 2006), when children are still highly dependent on their parents. Acknowledging that parents care for the wellbeing of their children and that the wellbeing of all family members is interrelated, extensions of Grossman's (1972a; 1972b) traditional demand-for-health model view the family as a health producer, in the sense that each member can, and does, influence his or her own and other members' health (Bolin et al., 2003, 2002; Jacobson, 2000). The family utility function for a husband, h, a wife, w, and a child, c, can be written as:

$$U(H_t^h, H_t^w, H_t^c, Z_t), \tag{1}$$

where $H_t^i (i = h, w, c)$ is health capital and Z_t is other commodities. Apart from own health and other commodities for consumption and investments, as in the traditional model, each family member derives utility also from the health of other family members. Health gives utility per se (the consumption aspect of health) and healthy time that the family can spend on activities other than being sick (the investment aspect of health). Theoretically, health capital depreciates with time, but parents can invest in their child's health to offset this depreciation. Health is produced by choosing a lifestyle that makes different health outcomes more or less likely, and by using market goods, such as health care services, pharmaceuticals, and treatments, to improve health. Combining market goods and own time, parents invest to produce child health, given each parents' level of productivity (i.e., their human capital).

Parents allocate their time to work and to the production of (their own, their spouse's and their child's) health and other commodities. Healthy time (i.e., time available for productive use) is determined, in part, by the health of all family members and, therefore, market income is dependent on both parental and child health. To adjust from an actual to a desired level of health involves a cost and the marginal cost of net investment (after depreciation) is assumed to be a positive function of the amount invested. Parents will invest in their child's health, not only because parents care about their child's wellbeing, but also because healthier children will decrease the time needed to care for sick children. That time can instead be spent on market work to raise family income and, consequently, also increase consumption and investments for all family members.

More recent research (summarized in, e.g., Heckman (2007), and Cunha et al. (2006)) challenges Grossman's (1972a; 1972b) traditional model and its sharp distinction between acquired skills and genetically determined cognitive ability. Instead, the new evidence shows that behaviors, abilities, and skills have both a genetic and an acquired character. Measured abilities are the outcome of investments and gene-environment interactions. Abilities are cognitive (e.g., IQ)

or noncognitive (e.g., patience, motivation, time preferences, and self-control) and affect learning, health behaviors, and health. Thus, health, cognitive skills and noncognitive skills (known as human capabilities) are closely related.

Human capabilities are formed throughout the life cycle, which consists of $2T$ years representing both childhood and adulthood (when individuals becomes parents themselves) (Cunha & Heckman, 2007; Heckman et al., 2006). Children are born with some initial endowment of skills and abilities, θ_1, that develop throughout their upbringing according to the production function:

$$\theta_{t+1} = f_t(h, \theta_t, I_t), \tag{2}$$

where h represents parental characteristics and I parents' investments in their child. The model implies that, first, skills are both persistent and self-reinforcing as higher skills in one period lead to higher skills in the next period and, second, previously acquired skills make further investments more productive. Consequently, even small childhood health shocks might snowball into adverse adult health and labor market outcomes.

If a child develops type 1 diabetes, the family may face lasting consequences for both health and everyday life, even when investing in managing the disease by regular monitoring of blood glucose levels, insulin injections, and a healthy lifestyle (Daneman, 2006). Diabetes management is time consuming and the time available for market work is likely to decrease. When family income decreases, it might not cover the investment needed to compensate for the family members' health depreciation. In addition, type 1 diabetes increases the child's depreciation rate and it becomes more costly to resume health. Child health might decrease as the cost of health capital increases (the income effect), and also because it becomes relatively more costly to invest in a child with type 1 diabetes than in a healthier family member (the substitution effect).

Moreover, caring for a child with type 1 diabetes imposes a host of long-term stressors (for instance, fear of hypoglycemia and increased insecurities about future health) in addition to the everyday activities of caring for the child and management of the child's diabetes (Wennick et al., 2009; Wennick & Hallström, 2006). Apart from caring for their child with diabetes, parents with other children might have additional strain on the hours and resources they have available for each child.[6] Possibly, having a sibling might worsen a child's prerequisites for accumulating skills, thereby worsening the adverse labor market effects of type 1 diabetes. Still, siblings could act as role models or inspire each other to progress

[6]This argument builds on the literature on the quantity and quality of children introduced by Becker & Lewis (1974). The empirical support for this literature from a Nordic context is weak. Using Swedish data and twin births as an instrument of family size, Åslund & Grönqvist (2010) studied effects on children's educational outcomes and found small and insignificant effects. Using Norwegian military enlistment data, Black et al. (2010) report that one additional child decreases cognitive ability by 0.008 of a standard deviation.

at school or in other areas.

To sum up, the family needs to shift resources towards restoring and maintaining health when a child develops type 1 diabetes; thus, the family is likely to redefine its preferences towards health as well as other aspects of life. These changes may in turn affect the child's skill formation and labor market performance later in life. Clearly, not only the sick child, but also the siblings without diabetes, could be affected by strained family resources. Conversely, siblings might benefit from a potential diabetes-evoked shift towards parental preferences favoring family and health.

4 Data

The Swedish Childhood Diabetes Register (SCDR) has recorded incident cases of type 1 diabetes in children aged 0–14.9 years in Sweden since 1 July 1977 (Dahlquist et al., 1982) to enable epidemiological studies on etiology, incidence trends, and complications of diabetes. Data for the SCDR are collected according to the Declaration of Helsinki, and informed consent was given by all parents of registered children. To study socioeconomic effects of having childhood onset diabetes, the Swedish Childhood Diabetes Study Group has added data to the SCDR as follows: for each individual in the SCDR, Statistics Sweden identified parents and siblings from the Multi-Generation Register (Statistics Sweden, 2009) and matched four non-diabetic control persons from the Total Population Register to each individual with diabetes by year of birth and municipality of residence at the time of diabetes diagnosis. Statistics Sweden identified parents also for the population controls. Covering the period 1990–2010, Statistics Sweden then added yearly education and earnings data for each individual with type 1 diabetes, siblings, population controls and parents from the national population registers the Longitudinal Integration Database for Health Insurance and Labour Market Studies (LISA) (Statistics Sweden, 2011) and the Swedish Register of Education (Statistics Sweden, 2006).[7]

I retrieve data on 1,404 individuals born in 1962–1971 and diagnosed with type 1 diabetes at ages 6–15 (during the years 1977–86), together with data on 2,685 siblings to individuals with diabetes and 5,616 matched population controls.[8] To prevent differing age distributions affecting the results, I include only siblings from the same cohorts as the controls: that is, those born in the years 1962–1971 (527 siblings born in the years 1935–61 and 846 siblings born in the years 1963–95 are excluded). I exclude siblings older than 15 at the time their brother or sister was diagnosed (414 siblings), as siblings older than 15 only

[7] All data provided to the research group are anonymized by coding performed by Statistics Sweden.

[8] The study population consists of more men than women because type 1 diabetes is more common in men (Pundziute-Lyckå et al., 2002).

share a relatively short period of their upbringing with a sick brother or sister. In addition, I exclude all members of families with more than one sibling with type 1 diabetes (16 individuals with diabetes and 29 siblings), as I cannot distinguish whether the observed effect comes from having type 1 diabetes or from having a sibling with diabetes. Then I exclude, first, controls who are also a sibling to an individual with diabetes (5 controls), second, individuals with missing LISA-data (23 individuals with diabetes, 95 siblings, and 57 controls), and third, individuals with a register of education that is decreasing in level (11 individuals with diabetes, 10 siblings, and 48 controls).

The data set is an unbalanced panel following earnings and covariates in the years 1990–2010, for 1,354 individuals with type 1 diabetes, 764 siblings, and 5,506 controls: 155,876 observations with an average of 20.4 observations per individual. The individuals are born in 1948–1972 and together they cover the age span 19–48 during the years 1990–2010. Figure 1 illustrates the ages at onset and the ages with earnings data across the different cohorts. For example, in 1990 (2010) the youngest cohort, born 1971, are 19 (39) years old and the oldest cohort, born 1962, are 28 (48) years. It is apparent from Figure 1 how the different cohorts contribute to the potential onset period. Given that onset occurs at ages 6–15 during the years 1977–1986, individuals in the oldest cohort, born 1962, have onset only at age 15, while individuals in the youngest cohort, born 1971, have onset at all ages throughout the age span 6–15. The study was approved by the Regional Research Ethics Board in Umeå (Dnr 07- 169M).

5 Method

As the data build on a case-control design for studying individuals with type 1 diabetes, the controls are not randomly assigned to the siblings of the individuals with type 1 diabetes. Therefore, only to study average earnings differentials between siblings and controls will not capture the causal effect of growing up with a brother or sister with type 1 diabetes. The matching, on year of birth and municipality of residence at the time of diabetes diagnosis, is equally unlikely to capture all systematic (observable and unobservable) pre-onset differences between the individuals with diabetes and the controls. Consequently, I pre-process the data, using a reweighting technique, the entropy balancing method for causal effects proposed by Hainmueller (2012), to balance the samples. This method constructs a weight for each control observation to create covariate balance between the treated groups (diabetes and siblings respectively) and the control groups.

Then, I estimate fixed effects (FE) regressions for individuals with type 1 diabetes or siblings and their respective weighted controls throughout ages 19–48. Using the FE approach, instead of a more simple comparison of raw earnings differentials between individuals growing up with type 1 diabetes and their

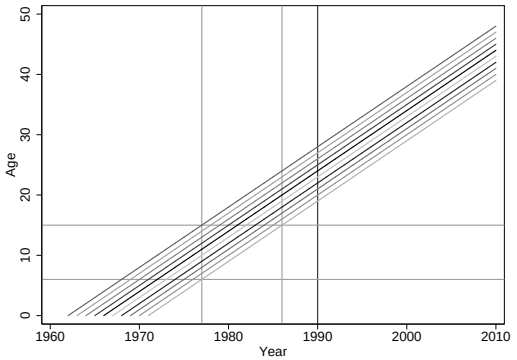

Figure 1: The age of each cohort (born 1962–1971) from birth up to year 2010. Each line represents a cohort: The cohort born 1962 is the top line, the cohort born 1963 is the next, ..., the cohort born 1971 is the bottom line. The gray horizontal lines at ages 6 and 15 mark the lower and upper bounds for onset ages. The gray vertical lines at years 1977 and 1986 mark the bounds for year of diagnosis. The resulting rectangular area marks the potential onset period. The black vertical line at year 1990 marks the first year with earnings data.

weighted controls, (1) I can explicitly control for time-invariant unobservable factors, and (2) I can include controls for education and family status to test if the earnings differentials are driven by post-onset differences in observables. To estimate fixed effects without first reweighting the data might not be enough when studying outcomes over such a long period of time (ages 19–48), as time variant unobservable factors might also be important. By tweaking the control group, making the controls more similar to the treated, the two groups will also be more likely to be affected equally by time-variant, as well as time-invariant, unobservable factors.

5.1 Entropy balancing

The entropy balancing method (Hainmueller & Xu, 2013; Hainmueller, 2012) reweights data to obtain covariate balance, making the treated group (the diabetes group or the siblings group) as equal as possible to the weighted controls, so that the treatment variable gains independence from background characteristics. Among the possible sets of weights that achieve covariate balance, entropy balancing chooses the set of weights that deviates as little as possible from uni-

form weights.[9] The entropy balancing technique builds on the (more often used) propensity score method (PSM) while remedying some of the shortcomings of PSM (Hainmueller, 2012). PSM implies a repeated process of propensity score estimation, matching, and balance checking, to try to identify the propensity scores that provide the most balanced covariate distribution. Often, this time-consuming process succeeds only in improving the balance on one covariate at the cost of that of another (Hainmueller, 2012; Stuart, 2010; Ho et al., 2007). Entropy balancing directly secures balance by calculating weights so that the treatment and weighted controls satisfy pre-specified balancing conditions (i.e., balance can be archived on the first (mean), second (variance), and third (skewness) moments of the covariate distributions). The non-parametric weighting procedure calibrates weights to be as similar as possible to uniform base weights, optimizing the goals of improving balance in covariate distribution and maximum retention of information. In creating a weighted control group with characteristics mimicking the treatment group, more weight is given to the under-represented groups and less weight to over-represented groups.

For women and men separately, and for each group studied (i.e., the treated), I balance the data on an extensive set of background characteristics[10] to calibrate the weights creating the best control groups possible given the available data (e.g., year of birth, parents' years of birth, mother's age at child's birth, and indicator variables for parental level of education, parents' born in a non-Nordic country). I apply the same balancing conditions across all samples and these are listed in Appendix C. By including interaction terms, covariates will be balanced across subsample groups. In practice, the entropy balancing method computes the values of the specified moments in each treatment group and seeks a set of entropy weights to adjust the control sample to match the treated.

Figure 2 for men with type 1 diabetes, and Figure 3 for brothers of individuals with type 1 diabetes, present standardized differences in means of the covariates used as balancing conditions before and after entropy balancing.[11] After entropy balancing, differences between the treated sample and the weighted control sample, for all moment conditions for all variables, are reduced to zero. As

[9] I estimate treatment effects on the treated, i.e., the diabetes-induced change in earnings of those individuals who are actually affected by type 1 diabetes.

[10] A summary of the background factors before entropy balancing can be found in Tables B.1 and B.2. Ideally, background data should be measured during the years preceding onset, but year 1990, when individuals are 19–28 years old, is the first year available in this data set. Still, these background factors either are fixed over time or are predetermined and generally do not change much over time. It is noteworthy that parents of women and men in the diabetes and siblings groups appear more educated than parents of controls. Reassuringly, we would expect controls, and not individuals with diabetes or siblings, to have parents with higher education if socioeconomic factors, such as low education, contribute to the development of type 1 diabetes.

[11] Appendix C holds a list of the abbreviated variable names. All variables but year of birth and mother's age at child's birth are indicator variables. For indicator variables, balancing on the first moment is sufficient to get balance also on higher moments. Higher order moments of the continuous variables are dropped because of co-linearity.

we would expect, given that the control group is originally designed to fit the diabetes population, the differences before entropy balancing are larger for brothers of individuals with type 1 diabetes.[12] Entropy balancing is performed by the ebalance package for Stata (Hainmueller & Xu, 2013).

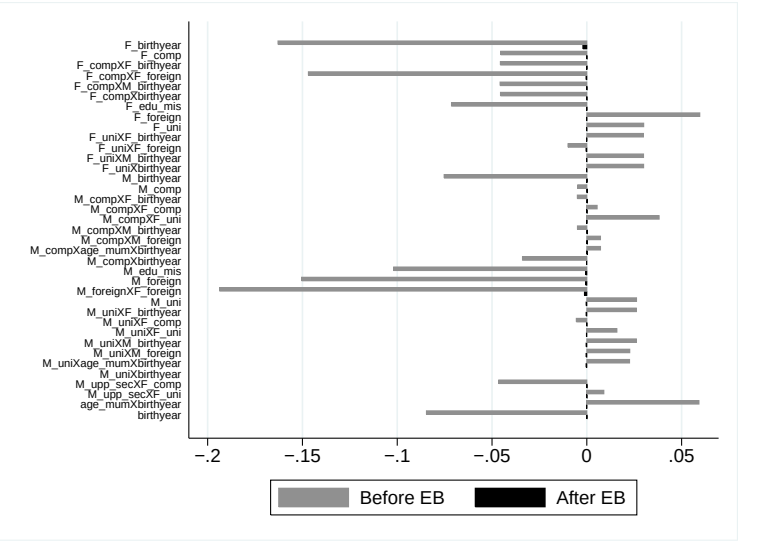

Figure 2: Covariate balance for all moment conditions before and after entropy balance (EB) weighting for men with type 1 diabetes and controls.

5.2 Fixed effects estimations

To investigate the lasting influence of type 1 diabetes, the chosen specification assesses age-specific differences conditional on individual fixed effects, capturing that onset of diabetes might impact directly on earnings and indirectly via its impact on health: diabetes complications are likely to develop over time (Möllsten et al., 2010; Daneman, 2006) and personal traits and abilities are likely to determine both successful disease management and labor market outcomes (Wennick et al., 2011; Heckman, 2007; Cunha et al., 2006; Goldman & Smith, 2002).[13] I model differences between, first, individuals with type 1 diabetes and their weighted controls and, second, siblings of individuals with type 1 diabetes

[12] The entropy balancing produces similar results also for women. Figures presenting the standardized difference in means for women are available on request.

[13] Both cognitive and non-cognitive abilities are rather constant throughout adulthood: for example, IQ is manifest at around the age of 10 and does not change much later on (Cunha et al., 2006).

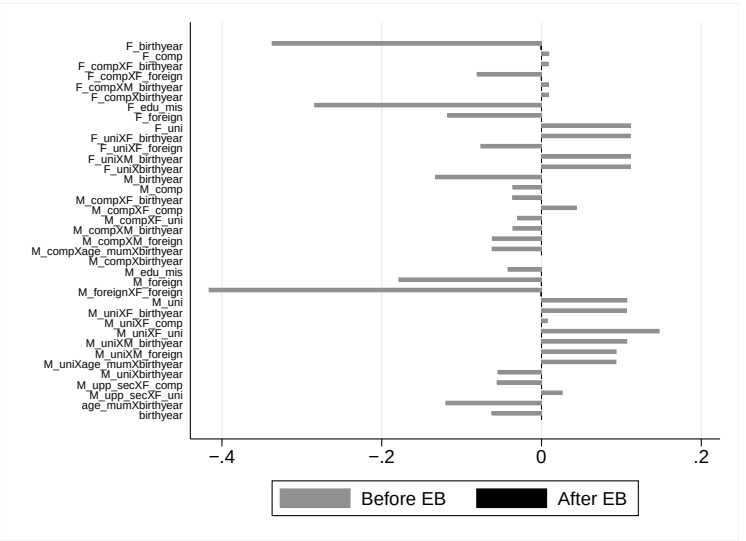

Figure 3: Covariate balance for all moment conditions before and after entropy balance (EB) weighting for brothers of individuals with type 1 diabetes and controls.

and their weighted controls. I use the following fixed effect model specification for individual i in year t:

$$y_{it} = \alpha + \beta D_i + \sum_{Age} \gamma_{age} AGE_{it} + \sum_{Age} \delta_{age} D_i * AGE_{it} + \theta X_{it} + \lambda_t + \mu_i + \epsilon_{it} \quad (3)$$

The dependent variable, y_{it}, is the natural logarithm of annual labor earnings each year t for individual i, conditional on earnings being larger than one Price Basic Amount (PBA).[14] The earnings variable sums all (gross) earnings from employment and self-employment, including compensation for the first 14 days of a period of sickness.[15] D_i is a dummy variable indicating treatment (diabetes or siblings). AGE_{it} is a vector of dummy variables representing the age categories: 19–25, 26–30, 31–35, 41–45, and 46–48 years of age. The age category 19–25 serves as the reference category for age.

Given that diabetes complications develop over time, the influence of diabetes on both health and earnings is also likely to vary. The interaction terms, $D_i * AGE_{it}$, capture age-specific differences in average annual earnings between the treated and weighted controls. For example, the coefficient on the second interaction term shows how much the average annual earnings in the treatment group deviate (in percentage points) from average annual earnings of weighted controls aged 26–30, whereas the coefficient of the main variable, D_i, shows differences between the groups in the age category 19–25. Note that the fixed effect estimator can only identify coefficients for time-varying regressors, meaning that the variables that do not change over time, such as D_i, will be estimated embedded in the individual fixed effects, and the first observable coefficient on treatment is for the age category 26–30 years old.

X_{it} is a vector of earnings determinants such as education and family status. λ_t is a vector of calendar time-fixed effects (i.e., dummies for each year 1990–2010) that control for aggregate changes in the economy over time. μ_i is a vector of individual-fixed effects and ϵ_{it} is an idiosyncratic error term. Given the large number of individuals, I remove μ_i from the estimation problem by using within-transformed data (i.e., removing individual-level averages from each side of Equation 3) instead of including a parameter for every individual (Baltagi, 2008).

I run Equation 3, first, for women and men with type 1 diabetes and their weighted controls, and, second, for sisters and brothers of individuals with type 1 diabetes and their weighted controls. I restrict the analyses to the years individuals are *lastingly* employed by excluding observations for each year an

[14] The PBA follows the price trend in the country year after year and is set by the government. The PBA is calculated based on changes in the general price level one year at a time. The measure is used, for instance, to ensure that sickness benefits, student grants, etc., do not decline in value because of an increase in the general price level.

[15] Using this definition of labor earnings, I will underestimate any effect of type 1 diabetes on earnings if individuals with type 1 diabetes or their siblings have more periods of sickness that are shorter than 14 days than do the population controls.

individual has annual earnings of less than one PBA (between 29,700 SEK≈€2,970 and 42,400 SEK≈€4,240 depending on the year). An average monthly salary is a multiple of one PBA. Excluding years with labor earnings below this threshold (22.2% of the observations for individuals with diabetes, 18.1% for siblings, and 20.0% for controls) provides estimates conditional on working, which ought to be a conservative estimate of the full effect of type 1 diabetes. Overviewing consequences of this employment condition on the studied panels, Appendix A presents histograms of the number of observations per year during the studied period, for the full sample (Figure A.1) and the sample conditional on having earnings exceeding one PBA (Figure A.2). The histograms display comparable patterns, suggesting that individuals with type 1 diabetes, siblings, and controls are equally represented among those with earnings exceeding one PBA.[16]

The individual fixed effect model relies on variation within individuals across time, thereby failing to identify diabetes-induced differences between treated and controls that do not vary with age. Instead, the FE estimates are adjusted for such differences, which will be captured by the individual-specific effects. Consequently, the impact of potential differences in the reference ages (19–25 years old), which might have arisen already before labor market entrance, will not be identified, but only changes in earnings over time. Another implication of the FE model exploiting only variation within individuals is that it will not matter if an individual has a very high-level or a very low-level earnings profile, because it is only changes in earnings over time that will show up as explanatory power. To assess the total average earnings gap, comprising differences in both entry wages and earnings trends, I (1) report "raw" differences in the mean progression of unconditional earnings by age and (2) run Equation 3 without controls for fixed effects: that is, pooled ordinary least square models (OLS). Testing whether it is necessary to control for individual-specific heterogeneity, F tests indicate that there are significant individual effects in all my specifications. Thus, FE specifications are favorable to pooled OLS.[17] Still, both models are informative as they are capturing somewhat different aspects of the diabetes-earnings relationship, besides the controls for individual-specific factors.

The study comprises individuals with onset of type 1 diabetes at ages 6–15 years old; consequently, education is also likely to have been affected by onset. I therefore add controls for education at three levels: compulsory, upper secondary, and university.[18] In contrast to the estimates unconditional on edu-

[16] Any over-representation among one or more groups as a consequence of the imposed earnings threshold could imply attrition bias. The sensitivity analysis in Section 7 applies alternative thresholds.

[17] I test the suitability of the fixed effects model over the random effects model with a Hausman test. Results from the F test and Hausman test are available on request.

[18] Following individuals from age 19 and onwards, education is generally not finalized and the variable has sufficient within-individual variation to be estimated. At the time-period studied, the median age when earning a degree from a Swedish university or university college was 27–28

cation, changing diabetes estimates in the conditional specification indicates that the potential treatment effects on labor market outcomes operate partly through education. Besides education, I control also for family-related mediators by adding indicator variables for marital status and having children.[19]

5.3 Sensitivity analysis

Controlling for individual fixed effects, I control not only for time-invariant factors that are truly unobservable (e.g., permanent ability) but also for any factor at the individual level that does not change over time (e.g., parental education). Thereby, I account for any time-invariant influence that parents and childhood circumstances may have had on the labor market effects for the adult growing up with type 1 diabetes. Still, we would like to control also for time-varying unobservable factors. This is, however, not possible with the available data, but I perform a placebo test, redoing the main analysis on siblings older than 15 when their brother or sister was diagnosed with type 1 diabetes. Such a test could point to any group level differences that affect earnings other than growing up with type 1 diabetes in the family. It is also possible that siblings in this age group might be sensitive to their brother or sister developing a life-long disease, but that they will have any long-term earnings consequences appears more unlikely.

Returning to the fixed effects, these implicitly control for selection into employment, as controlling for fixed effects also controls for some of the unobservable factors that might lead to self-selection into employment. Alternatively, scholars often suggest some form of two-part model, such as the one proposed by Heckman (1976), to correct for selection bias. However, these models are sensitive to the use of proper exclusion restrictions, because they rely on variables acting as instruments that affect the selection process into employment but not the earnings equation in question (Puhan, 2000). Nevertheless, I test the implication of the employment condition and the threshold chosen by presenting unconditional estimates and estimates conditional on having earnings>100,000 SEK.[20]

Lastly, I present alternative estimates using Propensity Scores (PS) instead of entropy balancing to generate the weights and also the estimates, without first pre-processing the data to check the robustness of the results to different empirical strategies. Furthermore, this test could be informative as to whether entropy balancing offers any correction for the fact that the control group is designed to fit the diabetes group and not the siblings, and whether it improves the matching also for individuals with diabetes. Calculating the propensity

(Statistics Sweden, 2008).

[19] I do not control for other potential mediators, such as comorbidities, occupation, tenure, and industry; consequently, I allow the effect of type 1 diabetes to go through such variables.

[20] Studying the returns on education in Sweden, Antelius & Björklund (2000) report that the results when excluding earnings<100,000 SEK are similar to the results for hourly wage.

scores, I condition on the same balancing constraints as I use for the entropy balancing weighting, but to get the (probit) estimations of the propensity scores to converge, I have to use a more restricted set of constraints, excluding most of the interactions, e.g., between (own, mother's, or father's) year of birth and parental level of education.

6 Results

6.1 Unconditional earnings and the probability of employment

To visualize "raw" differences in the mean progression of unconditional earnings throughout the age categories, Figure 4 presents the mean growth of annual earnings since the reference ages 19–25 for treated and their weighted controls, without conditioning on earnings larger than one PBA and without adjusting for control variables.

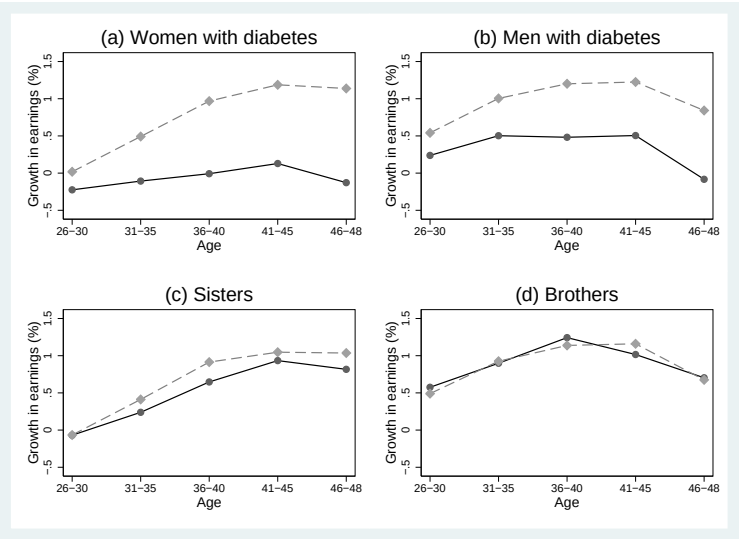

Figure 4: Mean progression (% change since age 19–25) in unconditional earnings for treated (diabetes or siblings, black solid lines) and their weighted controls (gray dashed lines) for each age category (26–30, 31–35, 36–40, 41–45, 46–48).

While the mean unconditional earnings for both men and women with type 1 diabetes appear to grow at a slower rate compared to the mean unconditional earnings of weighted controls, women's earnings appear to progress particularly

Table 1: Probit estimations (average marginal effects) of the probability of employment for women and men with type 1 diabetes and their siblings

	Diabetes		Siblings	
Dependent variable	(1)	(2)	(3)	(4)
Earnings> 0	Women	Men	Women	Men
Treatment	-0.24***	-0.10**	-0.048	0.15**
	(0.045)	(0.047)	(0.060)	(0.066)
Age	-0.043	0.12***	0.016	0.21***
	(0.037)	(0.034)	(0.045)	(0.038)
Age squared	0.00042	-0.0019***	-0.00014	-0.0034***
	(0.00057)	(0.00054)	(0.00069)	(0.00064)
Year FE	Yes	Yes	Yes	Yes
Education	Yes	Yes	Yes	Yes
Observations	67793	72478	62522	65713

Robust (clustered) standard errors in parentheses.

*** p<0.01, ** p<0.05, * p<0.1.

Year FE indicates controls for year fixed effects.

Education indicates controls for level of education.

slowly. The pattern for siblings is not as obvious. For most ages, brothers and sisters appear to have the same as, or somewhat lower growth rate than, their weighted controls: perhaps siblings are not systematically different from peers. Or, the ambiguity for siblings in Figure 4 might be a consequence of the fact that we can interpret the shown mean progression in unconditional earnings in terms of both productivity and labor supply differences. The data are not conclusive as to whether changes in earnings are due to changes in hours worked, wages, and/or sickness absence. Consequently, actual differences (if they exist) between siblings and weighted controls might not show if, for instance, the siblings, as a group, are less educated and, therefore, enter the labor market earlier than weighted controls. Then the siblings' probability of being employed is likely to be higher (at least initially), while their wages are likely to lag behind the wages of the more educated weighted controls.

To get a crude estimate of how treatment links to employment, I define employment as having any earnings registered during the year (earnings> 0) and estimate probit models of the probability of employment. Table 1 hold the results for diabetes in columns (1)–(2) and siblings in columns (3)–(4), and for women in columns (1) and (3) and men in columns (2) and (4). Both women and men with type 1 diabetes have lower probabilities of employment than weighted controls, but those for women are about twice those for men (-24 percentage

points for women compared to about -10 points for men). These estimates are large, especially for women, given that the overall probability of not being employed in the data is only 10.5% for women and 8.9% for men.

Contrastingly, among siblings, brothers have a higher probability of employment than weighted controls (15 percentage points), while the estimate for sisters is insignificant. This finding, which indicates a difference between brothers and sisters, may (1) strengthen the suspicion that there are no systematic differences between sisters and weighted controls, and (2) underlines the question of whether brothers are less educated than weighted controls. If the treated (diabetes or siblings) are selected into low education, then we would expect the estimates to change when redoing the probit analysis excluding the controls for education, but the estimates remain practically unchanged among all groups.[21] This robustness indicates that shown differences in employment are not driven by differences in education.

Taken together, the probit analyses suggest that the differences in the mean progression of unconditional earnings between the treated and their weighted controls comprise differences also in the probability of employment. In particular, women with type 1 diabetes deviate much from weighted controls, in terms of both employment and mean progression of unconditional earnings. As mentioned in Section 5.3, it is difficult to estimate the *full* treatment effect on earnings, as it requires that we adjust for selection into employment. The next section presents (conservative) estimates conditional on (1) being lastingly employed (earnings >1 PBA), and (2) time-invariant individual fixed effects that are likely to be important in the selection process.

6.2 Fixed effects estimations

This section presents the fixed effects (FE) estimations of Equation 3 on samples including either (1) individuals with type 1 diabetes and weighted controls (Table 2 women, Table 3 men) or (2) siblings of individuals with type 1 diabetes and weighted controls (Table 4 women, Table 5 men). Column (1) shows OLS estimates serving as a reference. Column (2) shows the age-specific average earnings differences (in percentage points) between the treated (diabetes or siblings) and the weighted controls, conditional only on individual- and year-specific effects, while columns (3)–(4) show the results conditional also on covariates. Entering the covariates stepwise, column (3) adds education at three levels, and column (4) adds indicators for marital status and having children.[22]

[21] Results are available on request. Showing differences in university education at age 20, 30, and 40, Figure B.2 in Appendix B, reveals that, compared to unweighted controls, (1) a lower proportion of men with diabetes have university education at age 30 (p=0.0808), and (2) a higher proportion of brothers have university education, but these differences are all insignificant.

[22] See Appendix D for results for covariates.

Table 2: Age-specific diabetes estimates for women with type 1 diabetes

Dependent variable	OLS	FE		
log(*Earnings*)	(1)	(2)	(3)	(4)
diabetes	0.0112			
	(0.0170)			
*diabetes*26–30*	-0.0252	-0.00573	-0.00374	-0.0123
	(0.0219)	(0.0230)	(0.0228)	(0.0225)
*diabetes*31–35*	-0.0346	-0.0264	-0.0211	-0.0331
	(0.0265)	(0.0278)	(0.0277)	(0.0272)
*diabetes*36–40*	-0.0247	-0.0308	-0.0246	-0.0387
	(0.0267)	(0.0276)	(0.0276)	(0.0274)
*diabetes*41–45*	-0.129***	-0.0860**	-0.0818**	-0.0995***
	(0.0368)	(0.0338)	(0.0339)	(0.0341)
*diabetes*46–48*	-0.152*	-0.150*	-0.149*	-0.178**
	(0.0925)	(0.0903)	(0.0898)	(0.0855)
Year FE	Yes	Yes	Yes	Yes
Education	No	No	Yes	Yes
Family	No	No	No	Yes
Observations	51022	51022	51022	51022
Individuals		3218	3218	3218
R2	0.279	0.349	0.351	0.359

Robust (clustered) standard errors in parentheses.

*** p<0.01, ** p<0.05, * p<0.1.

Year FE indicates controls for year fixed effects.

Education indicates controls for level of education.

Family indicates controls for marital status and child(ren).

Table 3: Age-specific diabetes estimates for men with type 1 diabetes

Dependent variable	OLS	FE		
log(*Earnings*)	(1)	(2)	(3)	(4)
diabetes	0.0344**			
	(0.0167)			
*diabetes*26–30*	-0.0593***	-0.0607***	-0.0574***	-0.0567***
	(0.0182)	(0.0184)	(0.0183)	(0.0182)
*diabetes*31–35*	-0.0813***	-0.0886***	-0.0829***	-0.0807***
	(0.0216)	(0.0214)	(0.0212)	(0.0211)
*diabetes*36–40*	-0.115***	-0.127***	-0.121***	-0.117***
	(0.0235)	(0.0229)	(0.0228)	(0.0227)
*diabetes*41–45*	-0.135***	-0.164***	-0.157***	-0.151***
	(0.0318)	(0.0266)	(0.0263)	(0.0262)
*diabetes*46–48*	-0.217**	-0.141*	-0.128	-0.120
	(0.104)	(0.0804)	(0.0803)	(0.0815)
Year FE	Yes	Yes	Yes	Yes
Education	No	No	Yes	Yes
Family	No	No	No	Yes
Observations	60586	60586	60586	60586
Individuals		3479	3479	3479
R2	0.362	0.491	0.493	0.495

Robust (clustered) standard errors in parentheses.

*** p<0.01, ** p<0.05, * p<0.1.

Year FE indicates controls for year fixed effects.

Education indicates controls for level of education.

Family indicates controls for marital status and child(ren).

The results for individuals with type 1 diabetes (Tables 2–3) show lower earnings compared to controls, and this drop increases with age (although the FE results for men at ages 46–48 deviate somewhat as there are few men in this category). The estimates are significant and sizable throughout most age categories for men, while women's earnings appear to be affected, at first, after age 40, and then the impact is smaller than for men. When controlling for year- and individual-specific effects (column 2), the earnings of men (women) with type 1 diabetes is 16.4 (8.6) percentage points lower than for weighted controls in the age category 41–45. These estimates are sizable and can be compared to the average difference in wages between blue and white collar workers in Sweden. From 1990 to 2010, white collar workers' wages increased in general with 55% and blue collar workers' with 25%, i.e., the increase in wages differed with 30 percentage points between white and blue collar workers (Ekonomifakta, 2015).

Turning to siblings of individuals with type 1 diabetes (Tables 4–5), only brothers' outcomes appear affected. Brothers' estimates are significantly lower for all categories, except for ages 46–48 (again, there are few men in this category). Comparing findings for brothers and men with type 1 diabetes at age 26–30, the estimate for brothers is 0.048 and it is 0.061 for men with type 1 diabetes. The negative estimates for brothers' earnings are reminiscent of the estimates for individuals with type 1 diabetes themselves. The estimates are (1) significantly lower than for weighted controls for all ages (apart from ages 46–48) as for men with type 1 diabetes, and (2) about the same size as for women with type 1 diabetes.

The results correspond to findings from other studies of childhood health and adult outcomes. For example, Johnson & Schoeni (2011) find that low birth weight lowers annual earnings for adults (ages 18–52) by roughly 15%. Smith (2009), studying overall measures of health, reports 12% higher earnings among those with excellent or very good childhood health compared to those with poor health. In relation to other studies of type 1 diabetes, my results are in line with the findings of Lundborg et al. (2014), Persson et al. (2013) and Steen Carlsson et al. (2010) using Swedish data. For example, Lundborg et al. (2014) report negative earnings penalties due to diabetes: that is, -20.8% when controlling for sibling fixed effects and -24.3% without. Steen Carlsson et al. (2010) report lower earnings for both women (-8%) and men (-4%) after onset of type 1 diabetes in the age group 15–34. In contrast, Minor (2013) and Minor (2011) find no association between type 1 diabetes and earnings, but Minor (2011) finds a negative association for women's hours worked. Possibly, the contradiction might relate to differences between the US and the Swedish labor markets, and to differing study designs.

My finding of increasing estimates over time supports the notion from the human capabilities literature that the effects of early health insults accumulate over time (Cunha & Heckman, 2007; Heckman et al., 2006) rather than, as pre-

Table 4: Age-specific sibling estimates for sisters of individuals with type 1 diabetes

Dependent variable	OLS	FE		
log(*Earnings*)	(1)	(2)	(3)	(4)
sibling	-0.0130			
	(0.0238)			
*sibling*26–30*	-0.00164	-0.00622	-0.00475	-0.000436
	(0.0283)	(0.0291)	(0.0289)	(0.0286)
*sibling*31–35*	0.0275	0.0180	0.0204	0.0163
	(0.0331)	(0.0331)	(0.0328)	(0.0324)
*sibling*36–40*	0.0232	-0.00173	0.00331	-0.00285
	(0.0351)	(0.0344)	(0.0343)	(0.0343)
*sibling*41–45*	0.0246	-0.00517	-0.000831	-0.00602
	(0.0397)	(0.0366)	(0.0364)	(0.0365)
*sibling*46–48*	0.0358	-0.00377	-0.000354	-0.00445
	(0.0763)	(0.0681)	(0.0677)	(0.0657)
Year FE	Yes	Yes	Yes	Yes
Education	No	No	Yes	Yes
Family	No	No	No	Yes
Observations	47577	47577	47577	47577
Individuals		2974	2974	2974
R2	0.293	0.387	0.388	0.396

Robust (clustered) standard errors in parentheses.

*** p<0.01, ** p<0.05, * p<0.1.

Year FE indicates controls for year fixed effects.

Education indicates controls for level of education.

Family indicates controls for marital status and child(ren).

Table 5: Age-specific sibling estimates for brothers of individuals with type 1 diabetes

Dependent variable	OLS	FE		
log(*Earnings*)	(1)	(2)	(3)	(4)
sibling	0.0283			
	(0.0232)			
*sibling*26–30*	-0.0403	-0.0480*	-0.0475*	-0.0466*
	(0.0250)	(0.0257)	(0.0252)	(0.0251)
*sibling*31–35*	-0.0312	-0.0515*	-0.0519*	-0.0507*
	(0.0302)	(0.0310)	(0.0306)	(0.0304)
*sibling*36–40*	-0.0525	-0.0638*	-0.0634*	-0.0602*
	(0.0337)	(0.0333)	(0.0327)	(0.0325)
*sibling*41–45*	-0.0313	-0.0760**	-0.0760**	-0.0716**
	(0.0410)	(0.0365)	(0.0359)	(0.0356)
*sibling*46–48*	0.0575	-0.106	-0.103	-0.0957
	(0.105)	(0.0778)	(0.0774)	(0.0784)
Year FE	Yes	Yes	Yes	Yes
Education	No	No	Yes	Yes
Family	No	No	No	Yes
Observations	55319	55319	55319	55319
Individuals		3154	3154	3154
R2	0.349	0.490	0.493	0.495

Robust (clustered) standard errors in parentheses.

*** p<0.01, ** p<0.05, * p<0.1.

Year FE indicates controls for year fixed effects.

Education indicates controls for level of education.

Family indicates controls for marital status and child(ren).

dicted by Grossman (1972a,b), diminish in importance. The increasing estimates could also be explained by childhood onset affecting future outcomes largely because onset affects future health, and not only because of a direct link between childhood onset and earnings. However, it seems unlikely that subsequent health alone would explain the results: woman and men ought to have had more similar results as glycemic control and the frequency of diabetic complications, for example, are comparable between the sexes (Kautzky-Willer et al., 2013).

6.2.1 FE vs. OLS estimates

Comparing the FE and OLS specifications (column 1 vs. column 2 of Tables 2–5), the OLS estimates intuitively ought to be larger than the FE estimates, as the FE specification controls for time-invariant individual-specific effects that are omitted in the OLS specification. Moreover, the FE specification captures only changes over time, and controls for any potential onset-induced differences that do vary with age.

However, the FE estimates are larger in size (as well as significant for brothers) for both men with type 1 diabetes and brothers of individuals with type 1 diabetes. This finding might stem from the fact that the FE estimates are conditioned on individual specific effects, while the OLS estimates are not. The OLS estimates will be biased downward if some factors in the individual-specific effect (e.g., permanent ability) contribute to both higher earnings and more successful diabetes management (with consequent better health). Without controls for individual-specific effects (OLS), the diabetes-age interactions will capture both the potential negative impact of diabetes and the positive impact of these factors favoring higher earnings and more successful diabetes management. Consequently, individuals with these factors may compensate for some of the negative impact that the disease has on others in the diabetes group. With controls for individual-specific effects (FE), the interactions will capture only the impact of diabetes, and individuals with type 1 diabetes will only be compared to others with the same individual-specific factors, and can no longer compensate, within the group, for those with less successful diabetes management and larger drops in earnings. Possibly, the factors favoring higher earnings and more successful diabetes management are valuable also for siblings, and how they handled growing up with a sick brother or sister.

But why are the FE estimates not larger than the OLS estimates also for women with type 1 diabetes? This might be related to differing wage profiles. Remember that the FE estimates capture only changes in earnings, while the OLS estimates capture overall differences in earnings between the treated and weighted controls. Men with type 1 diabetes appear to have a much flatter earnings profile than weighted controls: initially, earnings are higher for men with type 1 diabetes but this head start is then replaced by an increasingly negative earnings gap (Table 3 column 1). Changes over time therefore appear very influ-

ential in the diabetes-earnings relationship for men. For women, changes over time appear less dramatic, as women with and without diabetes start from more equal initial earnings (Table 2 column 1).

6.2.2 Mediator variables

The estimates in columns (1) and (2) of Tables 2–5 do not condition on mediators through which 'diabetes' (or 'sibling') may affect labor market outcomes. Consequently, I allow the link to go through such variables. When controlled for, as in columns (3)–(4), the mediator variables absorb some of the treatment effect. Therefore, we must interpret the estimates in these specifications with care. The estimated coefficients of the mediator variables may not truly capture their actual effects and, even more importantly, including these variables could bias the treatment estimates. Still, the estimates appear robust across all specifications with only small deviations in size. It is noteworthy that, as in the previous analysis of the probability of having employment (earnings>0) (Table 1), the link does not appear to operate via education.[23] The effect of childhood health on adult earnings also remains after controlling for education for Smith (2009), who examines effects of retrospective reports of self-assessed health in childhood, and for Johnson & Schoeni (2011), who study effects of low birth weight. Controlling for family status has little effect on the estimates for men with type 1 diabetes or siblings, while it may be a more important mediator for women with type 1 diabetes, as their estimates increase somewhat when controls are added for family status.

7 Sensitivity analysis

7.1 Placebo test for siblings older than 15 when their brother or sister was diagnosed with type 1 diabetes

Table 6 shows the results of the placebo test, redoing parts of the main analysis on siblings more than 15 years old when their brother or sister was diagnosed with type 1 diabetes.[24] Both the OLS (columns 1 and 3) and the FE estimates (columns 2 and 4) are small and insignificant. Thereby, they reject that any group level

[23] Even if the variable has enough within-individual variation to be estimated, individuals generally do not change their level of education very much after age 19. Therefore, education could also be controlled for, in part, in column (2) via the individual-specific effect. However, when adding controls for education also to the OLS-specification in column (1), the estimates do not change much. Results are available on request.

[24] I redo the entropy balancing, calculating new weights using the same balancing conditions as in the main analysis.

Table 6: Age-specific sibling estimates for siblings older than 15 at the time their brother or sister was diagnosed with type 1 diabetes

	Women		Men	
Dependent variable	(1)	(2)	(3)	(4)
log(*Earnings*)	OLS	FE	OLS	FE
sibling	0.0156		0.0397	
	(0.0628)		(0.0435)	
*sibling*26–30*	-0.0291	-0.0432	-0.0147	0.00341
	(0.0669)	(0.0649)	(0.0533)	(0.0668)
*sibling*31–35*	-0.0631	-0.0816	0.0198	0.0223
	(0.0679)	(0.0670)	(0.0627)	(0.0731)
*sibling*36–40*	-0.0417	-0.0676	-0.0115	-0.00395
	(0.0763)	(0.0716)	(0.0672)	(0.0739)
*sibling*41–45*	0.00673	0.00758	0.0367	0.0148
	(0.0797)	(0.0746)	(0.0809)	(0.0773)
*sibling*46–48*	0.0837	0.0439	0.0568	-0.0326
	(0.0963)	(0.0870)	(0.0985)	(0.0924)
Year FE	Yes	Yes	Yes	Yes
Observations	44724	44724	52283	52283
Individuals		2800		2979
R2	0.300	0.427	0.260	0.449

Robust (clustered) standard errors in parentheses.

*** p<0.01, ** p<0.05, * p<0.1.

Year FE indicates controls for year fixed effects.

differences other than growing up with a brother of sister with type 1 diabetes is affecting earnings.

7.2 Alternative thresholds for the employment condition

Testing the implication of the employment condition, and the application of the chosen threshold of earnings>1 PBA to the earnings analysis, Table 7 (diabetes) and Table 8 (siblings) report unconditional estimates (columns 1 and 3) and estimates conditional on having earnings>100,000 SEK (columns 2 and 4).

Confirming the estimates to be conservative in the main analyses, the unconditional estimates increase greatly compared to the estimates conditional on a threshold value of either one PBA (as in the main analysis) or 100,000 SEK for all but brothers of individuals with type 1 diabetes. On the contrary, the brothers' unconditional estimates have decreased and are no longer significant as a consequence of their having an increased likelihood of employment (recall the probit

Table 7: Age-specific diabetes estimates (FE) for individuals with type 1 diabetes using different employment thresholds

	Women		Men	
Dependent variable	(1)	(2)	(3)	(4)
log(*Earnings*)	if all	if earnins>100'	if all	if earnins>100'
*diabetes*26–30*	-0.115	0.00536	-0.263***	-0.0150
	(0.0796)	(0.0138)	(0.0777)	(0.0111)
*diabetes*31–35*	-0.400***	0.00681	-0.430***	-0.0208
	(0.101)	(0.0171)	(0.0958)	(0.0138)
*diabetes*36–40*	-0.707***	-0.0165	-0.613***	-0.0562***
	(0.115)	(0.0190)	(0.106)	(0.0158)
*diabetes*41–45*	-0.672***	-0.0525**	-0.714***	-0.0881***
	(0.146)	(0.0241)	(0.126)	(0.0202)
*diabetes*46–48*	-1.152***	-0.0928	-0.532*	-0.0798
	(0.402)	(0.0672)	(0.303)	(0.0551)
Year FE	Yes	Yes	Yes	Yes
Observations	67793	39403	72478	53499
Individuals	3309	3114	3551	3408
R2	0.0427	0.499	0.0614	0.600

Robust standard errors in parentheses.

*** p<0.01, ** p<0.05, * p<0.1.

Year FE indicates controls for year fixed effects.

analysis in Table 1). Also confirmative of the main results, adopting the larger threshold of 100,000 SEK as a condition continues to give significant estimates, but reduces the estimates throughout. Therefore, we need to keep in mind that the estimates are conditional on earnings>1 PBA when interpreting the results, as the size of the estimates is sensitive to the chosen threshold, suggesting that diabetes influences earnings via both wages and labor supply.

7.3 Propensity Scores: weighted and unweighted estimates

Table 9 for men with type 1 diabetes and Table 10 for brothers present alternative estimates using Propensity Scores (PS), instead of entropy balancing, to generate the weights (columns 1–2), and the estimates without first pre-processing the data (columns 3–4).[25]

The discrepancy between the unweighted and the weighted estimates is larger for brothers than for men with type 1 diabetes, as we would expect given

[25] I perform the same analyses for women and the resulting estimates appear robust across the different empirical strategies. Results are available on request.

Table 8: Age-specific sibling estimates (FE) for siblings of individuals with type 1 diabetes using different employment thresholds

	Women		Men	
Dependent variable	(1)	(2)	(3)	(4)
log(*Earnings*)	if all	if earnins>100'	if all	if earnins>100'
*sibling*26–30*	-0.0522	-0.00250	0.0984	-0.0264*
	(0.104)	(0.0183)	(0.0982)	(0.0157)
*sibling*31–35*	-0.176	0.000167	0.0271	-0.0373*
	(0.124)	(0.0206)	(0.114)	(0.0191)
*sibling*36–40*	-0.295**	-0.00388	0.136	-0.0299
	(0.130)	(0.0223)	(0.114)	(0.0211)
*sibling*41–45*	-0.234	-0.00252	-0.0119	-0.0565**
	(0.154)	(0.0257)	(0.146)	(0.0247)
*sibling*46–48*	-0.302	-0.0493	-0.0572	-0.0579
	(0.414)	(0.0486)	(0.286)	(0.0491)
Year FE	Yes	Yes	Yes	Yes
Observations	62522	36876	65713	48857
Individuals	3054	2884	3216	3098
R2	0.0613	0.535	0.0950	0.604

Robust standard errors in parentheses.

*** p<0.01, ** p<0.05, * p<0.1.

Year FE indicates controls for year fixed effects.

Table 9: Age-specific diabetes estimates for men with type 1 diabetes using Propensity score weighting or unweighted controls

	PS weights		No weights	
Dependent variable	(1)	(2)	(3)	(4)
log(*Earnings*)	OLS	FE	OLS	FE
diabetes	0.0313*		0.0424**	
	(0.0171)		(0.0167)	
*diabetes*26–30*	-0.0633***	-0.0615***	-0.0609***	-0.0613***
	(0.0184)	(0.0187)	(0.0181)	(0.0183)
*diabetes*31–35*	-0.0848***	-0.0896***	-0.0846***	-0.0910***
	(0.0217)	(0.0219)	(0.0214)	(0.0212)
*diabetes*36–40*	-0.115***	-0.124***	-0.115***	-0.127***
	(0.0235)	(0.0234)	(0.0232)	(0.0227)
*diabetes*41–45*	-0.137***	-0.164***	-0.134***	-0.166***
	(0.0316)	(0.0265)	(0.0312)	(0.0263)
*diabetes*46–48*	-0.247**	-0.178**	-0.217**	-0.153**
	(0.106)	(0.0804)	(0.101)	(0.0763)
Year FE	Yes	Yes	Yes	Yes
Observations	60586	60586	60586	60586
Individuals		3479		3479
R2	0.364	0.491	0.374	0.506

Robust (clustered) standard errors in parentheses.

*** p<0.01, ** p<0.05, * p<0.1.

Year FE indicates controls for year fixed effects.

the design of the data. Confirming the main results, the different strategies yield about the same results for men with type 1 diabetes, while the weighting seems to (at least partly) correct for some of the design-related bias in the sibling sample, as the estimates using either entropy balancing (Table 5) or PS are smaller in both size and significance compared to the estimates using unweighted data. The entropy balancing method produces somewhat more conservative estimates in terms of size, although the PS method documents fewer age categories with significant differences. Still, the estimates for brothers appear robust across the two weighting schemes. We cannot expect the results to be exactly the same, as I use fewer balancing constraints when estimating the PS weights.

8 Discussion

Besides confirming the message from previous research on type 1 diabetes, this study underlines that brothers of children with type 1 diabetes also face later

Table 10: Age-specific sibling estimates for brothers of individuals with type 1 diabetes using Propensity score weighting or unweighted controls

	PS weights		No weights	
Dependent variable	(1)	(2)	(3)	(4)
log(*Earnings*)	OLS	FE	OLS	FE
sibling	0.0177		0.0617***	
	(0.0266)		(0.0232)	
*sibling*26–30*	-0.0254	-0.0260	-0.0565**	-0.0570**
	(0.0302)	(0.0317)	(0.0247)	(0.0254)
*sibling*31–35*	-0.0253	-0.0371	-0.0542*	-0.0649**
	(0.0338)	(0.0357)	(0.0296)	(0.0307)
*sibling*36–40*	-0.0791**	-0.0843***	-0.0747**	-0.0791**
	(0.0318)	(0.0313)	(0.0332)	(0.0329)
*sibling*41–45*	-0.0409	-0.0843**	-0.0596	-0.0957***
	(0.0396)	(0.0356)	(0.0393)	(0.0358)
*sibling*46–48*	0.0791	-0.0960	0.0623	-0.101
	(0.0884)	(0.0671)	(0.0986)	(0.0736)
Year FE	Yes	Yes	Yes	Yes
Observations	55319	55319	55319	55319
Individuals		3154		3154
R2	0.364	0.497	0.375	0.510

Robust (clustered) standard errors in parentheses.

*** p<0.01, ** p<0.05, * p<0.1.

Year FE indicates controls for year fixed effects.

consequences on labor market outcomes. The results indicate a negative impact on labor market outcomes (throughout ages 19–48) for those who developed type 1 diabetes as children (aged 6–15). Both women and men with diabetes have a lower likelihood of employment and lower annual labor earnings than controls. The size of the decrease in women's likelihood of employment is about twice that for men, whereas the increasingly negative link to earnings appears more profound for men. For siblings of individuals with type 1 diabetes, sisters' outcomes appear unaffected, while brothers' report, on the one hand, a higher likelihood of being employed, but, on the other hand, lower earnings, reminiscent of the earnings decrease for individuals with type 1 diabetes themselves.

These results are based on detailed longitudinal register data comprising nearly all incident cases of type 1 diabetes in Sweden in ages 6–15 during the years 1977–86. The estimates are robust against selection on both time-invariant individual-specific factors and parental background factors. When ignoring such factors, we may expect the estimates to be biased. First, onset of type 1 diabetes might impact directly on earnings and also via its impact on health. Given that personal traits and abilities are likely to determine both successful disease management (Wennick et al., 2011; Goldman & Smith, 2002) and labor market outcomes (Heckman, 2007; Cunha et al., 2006), the influence of type 1 diabetes on both health and earnings is also likely to vary across personal traits and abilities. Second, in studying child health and circumstances during upbringing (when children are highly dependent on their parents), we cannot rule out the possibility that the estimates (diabetes or siblings) will capture also effects of parental influences, if parental background factors are not controlled for.

The fixed effects approach controls for time-invariant abilities and other time-invariant individual-specific factors. Additionally, the entropy balancing pre-processing technique makes the controls more similar to the treated by increasing independence between treatment and influential factors during upbringing. Thus, the two groups will be more likely to be affected equally by *time-variant* as well as *time-invariant* unobservable factors.[26] Even though we cannot test this expectation, the sensitivity analysis confirms the robustness of the results. For example, entropy balancing appears to correct for some of the potential design-related bias in the sibling sample, and a placebo test rejects the presence of any group level differences that affect earnings, other than growing up with type 1 diabetes.

Some researchers argue that child or adolescent abilities are influenced by health and therefore potentially mediate the impact of health (Salm & Schunk, 2012; Currie et al., 2010; Heckman, 2007). If so, diabetes estimates conditioned on ability measures would not capture the casual impact of type 1 diabetes.

[26] This approach also corrects, at least in part, for the fact that the control group is designed to fit the diabetes group and not their siblings.

However, most abilities have been found to stabilize early in life. For example, IQ generally manifests around age 10, while non-cognitive abilities such as motivation, self-discipline, and time preferences appear more changeable at later ages (Cunha et al., 2006). Type 1 diabetes will therefore be more likely to affect abilities (causing mediation) the younger the individuals are at onset. Even if some diabetes-induced changes in abilities might occur during the time-span in which I measure earnings (age 18–48), such mediators will not be controlled for as the individual fixed effect specifications only captures time-invariant factors. In addition, potential mediation via childhood abilities is, if present, likely to operate in two opposite ways. On the one hand, we generally expect poor health to have adverse effects on ability formation (Cunha & Heckman, 2008; Heckman, 2007; Cunha et al., 2006). On the other hand, it seems reasonable to expect that diabetes management will contribute to ability formation via learning of skills such as responsibility and long-sightedness that are favorable also on the labor market. Consequently, mediation is not likely to be a cause of concern and individual fixed effect specifications ensure that individual factors (contributing to higher earnings, more successful diabetes management, and better health) are not masking actual diabetes-related consequences on earnings. Still, the ordinary least square model is also informative as the fixed effects model captures only changes in earnings over time, while the ordinary least square model captures overall differences in earnings.

Why do the results show such large differences between women and men? These differences could, perhaps, relate to differences in childhood adjustment strategies. The psychological literature suggests that boys more often adopt externalizing behaviors, which have been linked to adverse educational and labor market outcomes (Gregg & Machin, 2000; McLeod & Kaiser, 2004; Miech et al., 1999), while girls more often show internalizing symptoms, which have been reported as unimportant for future outcomes (McLeod & Kaiser, 2004; Miech et al., 1999). Differences in adjustment strategies could thereby explain why women's earnings (both for women with diabetes and sisters) appear less affected than men's, but it cannot explain why women with type 1 diabetes have a two times lower probability of employment than men with type 1 diabetes. Nor can it explain why brothers instead have a higher probability of employment compared to weighted controls.

Even though long-term labor market consequences from childhood adjustment symptoms might be important, physical diabetes-related deteriorations in health, intuitively, appear to be even more important for the labor market outcomes of men and women with type 1 diabetes. In addition, the diabetes-induced risk of pregnancy-related complications (Jonasson et al., 2007; Casson et al., 1997) might contribute to women's relatively large impact on employment, as women might prioritize differently between work and family formation. Having to prioritize, in turn, might induce positive selection for earnings, as the

healthiest women are the ones less likely to experience any complications during pregnancy and most likely to work. In support of this reasoning, family formation is shown to be a mediator for women, while the diabetes effect remains unchanged when adding controls for family formation for men.

Continuing with why brothers' have a higher probability of employment but lower annual earnings, one would suggest, intuitively, that brothers are less educated and, therefore, have a head start entering the labor market but lower productivity and wages. However, no such differences in level of education appear to exist in the data. Selection into different fields of education or occupations might still be important and, also, to differentiate between productivity and labor supply with more nuanced earnings data.[27] Theoretically, the probit estimations of the probability of employment will be biased by omitted time-invariant individual fixed effects if any such factors exist that contribute to both employment and being a brother of an individual with type 1 diabetes. Still, it seems unlikely that any such factor would impact differently on brothers than it would on sisters or individuals with type 1 diabetes themselves.

This study underlines the importance of considering all family members when studying the consequences of childhood onset of chronic illness. These novel findings for brothers of individuals with type 1 diabetes support actions that have regard to broader family impact, both when initiating further research and when designing children's diabetes management programs.

Acknowledgments

I thank the Swedish Childhood Diabetes Study Group that developed the Swedish Childhood Diabetes Database, sponsored by the Swedish Research Council project no 07531, Västerbotten County Council, The Swedish Diabetes Foundation, the Swedish Council for Working Life and Social Research (FAS) (2009-0768), and Diabetesfonden (2009-065). The Health Economics Program (HEP) at Lund University also receives core funding from FAS (Dnr 2006-1660), the Government Grant for Clinical Research ("ALF") and Region Skåne. I also thank Gisela Dahlquist, Jens Dietrichson, Petter Lundborg, Carl Hampus Lyttkens, and Katarina Steen Carlsson for valuable support and insightful comments.

[27] I have no information on wages or hours worked but rely on annual earnings alone.

References

Adams, R., Peveler, R. C., Stein, A., & Dunger, D. B. (1991). Siblings of children with diabetes: Involvement, understanding and adaptation. *Diabetic Medicine*, *8*(9), 855–859.

Almond, D., & Currie, J. (2011). Killing me softly: The fetal origins hypothesis. *Journal of Economic Perspectives*, *25*(3), 153–172.

American Diabetes Association. (2008). Diagnosis and classification of diabetes mellitus. *Diabetes Care*, *31*(Supplement 1), 55–60.

Antelius, J., & Björklund, A. (2000). How reliable are register data for studies of the return on schooling? An examination of Swedish data. *Scandinavian Journal of Educational Research*, *44*(4), 341–355.

Åslund, O., & Grönqvist, H. (2010). Family size and child outcomes: Is there really no trade-off? *Labour Economics*, *17*(1), 130–139.

Atkinson, M. A., & Eisenbarth, G. S. (2001). Type 1 diabetes: new perspectives on disease pathogenesis and treatment. *The Lancet*, *358*(9277), 221–229.

Baltagi, B. (2008). *Econometric analysis of panel data* (Vol. 1). John Wiley & Sons.

Barlow, J. H., & Ellard, D. R. (2006). The psychosocial well-being of children with chronic disease, their parents and siblings: an overview of the research evidence base. *Child: care, health and development*, *32*(1), 19–31.

Bastida, E., & Pagán, J. A. (2002). The impact of diabetes on adult employment and earnings of Mexican Americans: Findings from a community based study. *Health Economics*, *11*(5), 403–413.

Becker, G. S., & Lewis, H. G. (1974). Interaction between quantity and quality of children. In *Economics of the family: Marriage, children, and human capital* (pp. 81–90). UMI.

Black, S. E., Devereux, P. J., & Salvanes, K. G. (2010). Small family, smart family? Family size and the IQ scores of young men. *Journal of Human Resources*, *45*(1), 33–58.

Bolin, K., Jacobson, L., & Lindgren, B. (2002). The family as the health producer—when spouses act strategically. *Journal of Health Economics*, *21*, 475–495.

Bolin, K., Lindgren, B., Lindström, M., & Nystedt, P. (2003). Investments in social capital—implications of sicial interactions for the production of health. *Social Science & Medicine, 56*, 2379–2390.

Brown, H. S., Pagán, J. A., & Bastida, E. (2005). The impact of diabetes on employment: genetic IVs in a bivariate probit. *Health Economics, 14*(5), 537–544.

Brown, H. S., Perez, A., Yarnell, L. M., Pagan, J. A., Hanis, C. L., Fischer-Hoch, S., & McCormick, J. B. (2010). Diabetes and employment productivity: does diabetes management matter? *The American Journal of Managed Care, 17*(8), 569–576.

Case, A., Fertig, A., & Paxson, C. (2005). The lasting impact of childhood health and circumstance. *Journal of Health Economics, 24*(2), 365–389.

Casson, I., Clarke, C., Howard, C., Mc-Kendrick, O., Pennycook, S., Pharoah, P., ... Walkinshaw, S. (1997). Outcomes of pregnancy in insulin dependent diabetic women: results of a five year population cohort study. *BMJ, 315*, 275–278.

Cunha, F., & Heckman, J. (2007). *The technology of skill formation* (Tech. Rep.). National Bureau of Economic Research.

Cunha, F., & Heckman, J. (2008). Formulating, identifying and estimating the technology of cognitive and noncognitive skill formation. *Journal of Human Resources, 43*, 738–782.

Cunha, F., Heckman, J., Lochner, L., & Masterov, D. (2006). Interpreting the evidence on life cycle skill formation. *Handbook of the Economics of Education, 1*, 697–812.

Currie, J. (2009). Healthy, wealthy, and wise: Socioeconomic status, poor health in childhood, and human capital development. *Journal of Economic Literature, 47*, 87–122.

Currie, J., & Almond, D. (2011). Human capital development before age five. *Handbook of Labor Economics, 4*, 1315–1486.

Currie, J., & Madrian, B. C. (1999). Health, health insurance and the labor market. *Handbook of Labor Economics, 3*, 3309–3416.

Currie, J., Stabile, M., Manivong, P., & Roos, L. L. (2010). Child health and young adult outcomes. *Journal of Human Resources*, *45*(3), 517–548.

Cutler, D. M., Glaeser, E. L., & Shapiro, J. M. (2003). Why have Americans become more obese? *Journal of Economic Perspectives*, *17*, 93–118.

Dahlquist, G., Gustavsson, K. H., Holmgren, G., Hägglöf, B., Larsson, Y., Nilsson, K. O., ... Wall, S. (1982). The incidence of diabetes mellitus in Swedish children 0–14 years of age. A prospective study 1977–1980. *Acta Paediatrica Scandinavica*, *71*(1), 7–14.

Dahlquist, G., Källén, B., & Swedish Childhood Diabetes Study Group. (2007). School performance in children with type 1 diabetes–a population-based register study. *Diabetologia*, *50*(5), 957–64.

Dahlquist, G., Nyström, L., Patterson, C. C., et al. (2011). Incidence of type 1 diabetes in Sweden among individuals aged 0–34 years, 1983–2007 an analysis of time trends. *Diabetes Care*, *34*(8), 1754–1759.

Dahlquist, G., Pundziūtė-Lyckå, A., Nyström, L., et al. (2005). Birthweight and risk of type 1 diabetes in children and young adults: a population-based register study. *Diabetologia*, *48*(6), 1114–1117.

Daneman, D. (2006). Type 1 diabetes. *The Lancet*, *367*(9513), 847–858.

Devaux, M., & Sassi, F. (2013). Social inequalities in obesity and overweight in 11 OECD countries. *The European Journal of Public Health*, *23*(3), 464–469.

Ekonomifakta. (2015). *Wage trends in Sweden since 1970*. `www.ekonomifakta.se/sv/Fakta/Arbetsmarknad/Loner/Loneutveckling-i-Sverige/`. (3 June 2015)

Fletcher, J. M., & Richards, M. R. (2012). Diabetes's 'health shock'to schooling and earnings: Increased dropout rates and lower wages and employment in young adults. *Health Affairs*, *31*(1), 27–34.

Gendelman, N., Snell-Bergeon, J. K., McFann, K., Kinney, G., Wadwa, R. P., Bishop, F., ... Maahs, D. M. (2009). Prevalence and correlates of depression in individuals with and without type 1 diabetes. *Diabetes Care*, *32*(4), 575–579.

Goldman, D. P., & Smith, J. P. (2002). Can patient self-management help explain the ses health gradient? *Proceedings of the National Academy of Sciences*, *99*(16), 10929–10934.

Gregg, P., & Machin, S. (2000). The relationship between childhood experiences, subsequent educational attainment and adult labour market performance. *Child Well Being in Modern Nations: What do we Know*.

Grossman, M. (1972a). The demand for health: a theoretical and empirical investigation. In (Vol. 119). Columbia University Press for the National Bureau of Economic Research, Inc.

Grossman, M. (1972b). On the concept of health capital and the demand for health. *Journal of Political Economy*, *80*, 223–255.

Grossman, M., & Kaestner, R. (1997). Effects of education on health. In J. Behrman & N. Stacey (Eds.), *The social benefits of education* (pp. 475–492). Ann Arbor: University of Michigan Press.

Hainmueller, J. (2012). Entropy balancing for causal effects: A multivariate reweighting method to produce balanced samples in observational studies. *Political Analysis*, *20*(1), 25–46.

Hainmueller, J., & Xu, Y. (2013). ebalance: A stata package for entropy balancing. *Journal of Statistical Software*, *54*(7), 1–18.

Harris, A. (2008). *Chronic disease and labour force participation in australia: an endogenous multivariate probit analysis of clinical prevalence data* (Tech. Rep.).

Heckman, J. (1976, January). The common structure of statistical models of truncation, sample selection and limited dependent variables and a simple estimator for such models. In *Annals of economic and social measurement, volume 5, number 4* (pp. 475–492). National Bureau of Economic Research, Inc.

Heckman, J. (2007). The economics, technology, and neuroscience of human capability formation. *Proceedings of the National Academy of Sciences*, *104*(33), 13250–13255.

Heckman, J., J.Stixrud, & Urzua, S. (2006). The Effects of Cognitive and Noncognitive Abilities on Labor Market Outcomes and Social Behavior. *Journal of Labor Economics*, *24*(3), 411–482.

Ho, D. E., Imai, K., King, G., & Stuart, E. A. (2007). Matching as nonparametric preprocessing for reducing model dependence in parametric causal inference. *Political Analysis*, *15*(3), 199–236.

Hollidge, C. (2001). Psychological adjustment of siblings to a child with diabetes. *Health & Social Work*, *26*(1), 15–25.

Hood, K. K., Huestis, S., Maher, A., Butler, D., Volkening, L., & Laffel, L. M. B. (2006). Depressive symptoms in children and adolescents with type 1 diabetes association with diabetes-specific characteristics. *Diabetes Care*, *29*(6), 1389–1389.

Huang, H.-C., & Yeh, C.-C. (2014). Inflation targeting on unemployment rates: a quantile treatment effect approach. *Applied Economics Letters*, *21*(7), 453–458.

Jackson, C., Richer, J., & Edge, J. A. (2008). Sibling psychological adjustment to type 1 diabetes mellitus. *Pediatric Diabetes*, *9*(4 Pt 1), 308–311.

Jacobson, L. (2000). The family as producer of health — an extended grossman model. *Journal of Health Economics*, *19*, 611–637.

Johnson, R. C., & Schoeni, R. F. (2011). The influence of early-life events on human capital, health status, and labor market outcomes over the life course. *The BE Journal of Economic Analysis & Policy*, *11*(3).

Jonasson, J. M., Brismar, K., Sparén, P., Lambe, M., Nyrén, O., Östenson, C.-G., & Ye, W. (2007). Fertility in women with type 1 diabetes: A population-based cohort study in Sweden. *Diabetes Care*, *30*(9), 2271–2276.

Karvonen, M., Viik-Kajander, M., Moltchanova, E., Libman, I., LaPorte, R., & Tuomilehto, J. (2000). Incidence of childhood type 1 diabetes worldwide. diabetes mondiale (DiaMond) project group. *Diabetes Care*, *23*(10), 1516–1526.

Kautzky-Willer, A., Stich, K., Hintersteiner, J., Kautzky, A., Kamyar, M., Saukel, J., ... Lemmens-Gruber, R. (2013). Sex-specific-differences in cardiometabolic risk in type 1 diabetes: a cross-sectional study. *Cardiovascular Diabetology*, *12*(1), 78.

Krishnan, P., & Krutikova, S. (2013). Non-cognitive skill formation in poor neighbourhoods of urban india. *Labour Economics*, *24*, 68–85.

Lahmann, P. H., Lissner, L., Gullberg, B., & Berglund, G. (2000). Sociodemographic factors associated with long-term weight gain, current body fatness and central adiposity in Swedish women. *International Journal of Obesity & Related Metabolic Disorders*, *24*, 685.

Latif, E. (2009). The impact of diabetes on employment in Canada. *Health Economics*, *18*(5), 577–589.

Lavigne, J. V., Traisman, H. S., Marr, T. J., & Chasnoff, I. J. (1982). Parental perceptions of the psychological adjustment of children with diabetes and their siblings. *Diabetes Care*, *5*(4), 420–426.

Lindeboom, M., Llena-Nozal, A., & van der Klaauw, B. (2006). *Disability and work: The role of health shocks and childhood circumstances* (IZA Discussion Papers). Institute for the Study of Labor (IZA).

Lissner, L., Johansson, S., Qvist, J., Rössner, S., & Wolk, A. (2000). Social mapping of the obesity epidemic in Sweden. *International Journal of Obesity & Related Metabolic Disorders*, *24*, 801–5.

Lundborg, P., Nilsson, A., & Rooth, D.-O. (2014). Adolescent health and adult labor market outcomes. *Journal of Health Economics*, *37*, 25–40.

Luyckx, K., Seiffge-Krenke, I., & Hampson, S. E. (2010). Glycemic control, coping, and internalizing and externalizing symptoms in adolescents with type 1 diabetes. A cross-lagged longitudinal approach. *Diabetes Care*, *33*(7), 1424–1429.

Maluccio, J. A., Hoddinott, J., Behrman, J. R., Martorell, R., Quisumbing, A. R., & Stein, A. D. (2009). The impact of improving nutrition during early childhood on education among guatemalan adults*. *The Economic Journal*, *119*(537), 734–763.

Marcus, J. (2013). The effect of unemployment on the mental health of spouses–evidence from plant closures in germany. *Journal of Health Economics*, *32*(3), 546–558.

Maslow, G. R., Haydon, A., McRee, A.-L., Ford, C. A., & Halpern, C. T. (2011). Growing up with a chronic illness: Social success, educational/vocational distress. *Journal of Adolescent Health*, *49*(2), 206–212.

McLeod, J. D., & Kaiser, K. (2004). Childhood emotional and behavioral problems and educational attainment. *American Sociological Review*, *69*(5), 636–658.

Miech, R. A., Caspi, A., Moffitt, T. E., Wright, B. R. E., & Silva, P. A. (1999). Low socioeconomic status and mental disorders: a longitudinal study of selection and causation during young adulthood. *American Journal of Sociology*, *104*(4), 1096–1131.

Milton, B., Holland, P., & Whitehead, M. (2006). The social and economic consequences of childhood-onset type 1 diabetes mellitus across the lifecourse: a systematic review. *Diabetic Medicine*, *23*, 821–829.

Minor, T. (2011). The effect of diabetes on female labor force decisions: new evidence from the national health interview survey. *Health Economics*, *20*(12), 1468–1486.

Minor, T. (2013). An investigation into the effect of type I and type II diabetes duration on employment and wages. *Economics & Human Biology*, *11*(4), 534–544.

Molarius, A. (2003). The contribution of lifestyle factors to socioeconomic differences in obesity in men and women — a population-based study in Sweden. *European Journal of Epidemiology*, *18*, 227–234.

Molarius, A., Seidell, J. C., Sans, S., Tuomilehto, J., & Kuulasmaa, K. (2000). Educational level, relative body weight, and changes in their association over 10 years: an international perspective from the WHO MONICA Project. *American Journal of Public Health*, *90*, 1260–1268.

Möllsten, A., Svensson, M., Waernbaum, I., Berhan, Y., Schön, S., Nyström, L., ... others (2010). Cumulative risk, age at onset, and sex-specific differences for developing end-stage renal disease in young patients with type 1 diabetes a nationwide population-based cohort study. *Diabetes*, *59*(7), 1803–1808.

Naar-King, S., Idalski, A., Ellis, D., Frey, M., Templin, T., Cunningham, P. B., & Cakan, N. (2006). Gender differences in adherence and metabolic control in urban youth with poorly controlled type 1 diabetes: The mediating role of mental health symptoms. *Journal of Pediatric Psychology*, *31*(8), 793–802.

ÓBrien, I., Duffy, A., & Nicholl, H. (2009). Impact of childhood chronic illnesses on siblings: a literature review. *British Journal of Nursing*, *18*(22), 1358–1365.

Oreopoulos, P., Stabile, M., Walld, R., & Roos, L. L. (2008). Short-, medium-, and long-term consequences of poor infant health an analysis using siblings and twins. *Journal of Human Resources*, *43*(1), 88–138.

Persson, S., Dahlquist, G., Gerdtham, U., & Steen Carlsson, K. (2013). Impact of childhood-onset type 1 diabetes on schooling: a population-based register study. *Diabetologia*, *56*(6), 1254–1262.

Ploug, U. J. (2013). The cost of diabetes-related complications: Registry-based analysis of days absent from work. *Economics Research International*, *2013*.

Puhan, P. (2000). The heckman correction for sample selection and its critique. *Journal of Economic Surveys*, *14*(1), 53—68.

Pundziute-Lyckå, A., Dahlquist, G., Nyström, L., Arnqvist, H., Björk, E., Blohme, G., ... others (2002). The incidence of type I diabetes has not increased but shifted to a younger age at diagnosis in the 0–34 years group in Sweden 1983 to 1998. *Diabetologia*, *45*(6), 783–791.

Salm, M., & Schunk, D. (2012). The relationship between child health, developmental gaps, and parental education: Evidence from administrative data. *Journal of the European Economic Association*, *10*(6), 1425–1449.

Sleeman, F., Northam, E. A., Crouch, W., & Cameron, F. J. (2010). Psychological adjustment of well siblings of children with type 1 diabetes. *Diabetic Medicine*, *27*(9), 1084–1087.

Smith, J. P. (2009). The impact of childhood health on adult labor market outcomes. *The Review of Economics and Statistics*, *91*(3), 478–489.

Soltesz, G., Patterson, C., & Dahlquist, G. (2007). Worldwide childhood type 1 diabetes incidence–what can we learn from epidemiology? *Pediatric Diabetes*, *8*(s6), 6–14.

Statistics Sweden. (2006). *Evaluation of the Swedish Register of Education.* `http://www.scb.se/Pages/PublishingCalendarViewInfo___259924.aspx?PublObjId=3299`. (3 October 2012)

Statistics Sweden. (2008). *Higher education. students and graduated students in undergraduate education 2006/07.* `http://www.scb.se/statistik/UF/UF0205/2008A01/UF0205_2008A01_SM_UF20SM0801.pdf`. (20 Februari 2014)

Statistics Sweden. (2009). *Multi-generation register.* `www.scb.se/Pages/List____257501`. (3 October 2012)

Statistics Sweden. (2011). *Longitudinal integration database for health insurance and labour market studies (LISA by Swedish acronym)*. `http://www.scb.se/Pages/List____257743.aspx`. (6 June 2012)

Steen Carlsson, K., Landin-Olsson, M., Nyström, L., Arnqvist, H. J., Bolinder, J., Östman, J., & Gudbjörnsdottir, S. (2010). Long-term detrimental consequences of the onset of type 1 diabetes on annual earnings-evidence from annual registry data in 1990–2005. *Diabetologia*, *53*(6), 1084–92.

Stuart, E. A. (2010). Matching methods for causal inference: A review and a look forward. *Statistical Science: a review journal of the Institute of Mathematical Statistics*, *25*(1), 1–29.

The TEDDY Study Group. (2007). The environmental determinants of diabetes in the young (TEDDY) study: study design. *Pediatric Diabetes*, *8*(5), 286–298.

Tunceli, K., Bradley, C. J., Nerenz, D., Williams, L. K., Pladevall, M., & Lafata, J. E. (2005). The impact of diabetes on employment and work productivity. *Diabetes Care*, *28*(11), 2662–2667.

Vijan, S., Hayward, R. A., & Langa, K. M. (2004). The impact of diabetes on workforce participation: Results from a national household sample. *Health Services Research*, *39*(6), 1653–1670.

Wennick, A., & Hallström, I. (2006). Swedish families' lived experience when a child is first diagnosed as having insulin-dependent diabetes mellitus an ongoing learning process. *Journal of Family Nursing*, *12*(4), 368–389.

Wennick, A., Hallström, I., Lindgren, B., & Bolin, K. (2011). Attained education and self-assessed health later in life when diagnosed with diabetes in childhood: a population-based study. *Pediatric Diabetes*, *12*(7), 619–626.

Wennick, A., & Huus, K. (2012). What it is like being a sibling of a child newly diagnosed with type 1 diabetes: an interview study. *European Diabetes Nursing*, *9*(3), 88–92.

Wennick, A., Lundqvist, A., & Hallström, I. (2009). Everyday experience of families three years after diagnosis of type 1 diabetes in children: A research paper. *Journal of Pediatric Nursing*, *24*(3), 222–230.

Zhang, X., Zhao, X., & Harris, A. (2009). Chronic diseases and labour force participation in Australia. *Journal of Health Economics*, *28*(1), 91–108.

Appendices

A Histograms of the studied panels

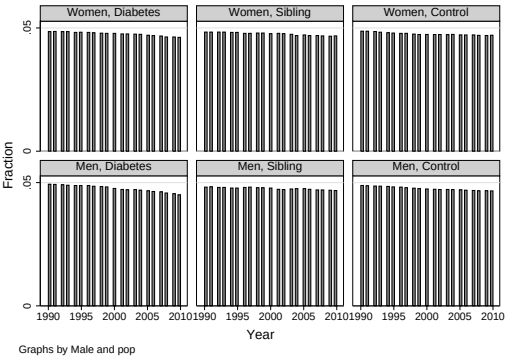

Figure A.1: Histogram of the number of observations per year during the studied period *before* excluding observations exceeding one PBA, for individuals with type 1 diabetes, siblings, and controls, and for women and men respectively

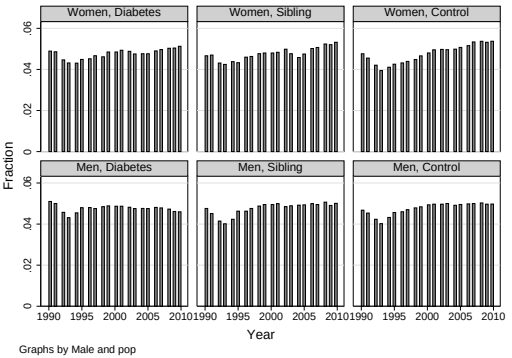

Figure A.2: Histogram of the number of observations per year during the studied period *after* excluding observations exceeding one PBA, for individuals with type 1 diabetes, siblings, and controls, and for women and men respectively

B Descriptive statistics

This section reports summary statistics without conditioning on earnings>1 PBA for individuals with diabetes, siblings, and unweighted controls in year 1990, for women (Table B.1) and men (Table B.2) separately. Economic variables are in 2010 prices. Overall, there are some average differences between the groups (significant on at least the 10 percent level). When including also low-earners, men in the diabetes and siblings groups have higher mean earnings than controls, while women in the diabetes group have lower earnings.

Being somewhat older than controls, siblings are more educated (tested with chi-2 test): more sisters and brothers have university education (tested with t-test) and fewer sisters have only compulsory education (tested with t-test) than controls. Controlling for age, Figures B.1 and B.2 and t-tests indicate instead that controls are the only group with significantly higher university attendance. Compared to individuals with diabetes, the differences are significant at age 20 for women (p=0.0998) and at age 30 for men (p=0.0808), while the differences compared to siblings are significant only at age 40 for women (p=0.0750).

Due to the relatively high prevalence of type 1 diabetes among native Swedes and Finnish people (Karvonen et al., 2000), a higher proportion of controls are born outside of the Nordic countries (tested with t-test). However, the entropy balancing method controls for these differences.

Turning to parental education, women show more group-level differences than men. Compared to controls, there are differences in both mothers' and fathers' level of education for both women in the diabetes group and the sibling group, while there are differences only in fathers' level of education and only for men in the siblings group (tested with chi-2 test). Women with type 1 diabetes and sisters of individuals with type 1 diabetes have university-educated mothers to a greater extent than women controls (tested with t-test). In addition, more women with type 1 diabetes have fathers with university education (tested with t-test), while more sisters have fathers with only compulsory education (tested with t-test). Brothers of individuals with type 1 diabetes have both parents with more university education (tested with t-test). Reassuringly, if socioeconomic factors are associated with the development of type 1 diabetes, we would expect controls, and not individuals with diabetes or siblings, to have parents with higher education. The groups also appear to differ on the amount of missing data for parental education (tested with t-test). However, the entropy balancing method also controls for these differences. Furthermore, compared to controls, siblings have somewhat older fathers and men with type 1 diabetes also have somewhat older mothers (tested with t-test). Women in both the diabetes group and the sibling group had older mothers when they were born than women controls (tested with t-test).

Table B.1: Descriptive statistics of own and parents' background factors for women in year 1990

	Diabetes		Siblings		Controls	
	mean	(sd)	mean	(sd)	mean	(sd)
Own						
Earnings[a]	99,448	(64,505)	106,596	(69,602)	104,537	(68,465)
Age	22	(2.2)	23	(2.4)	22	(2.2)
Birthyear	1968	(2.2)	1967	(2.4)	1968	(2.2)
Mother's age at birth	27	(5.3)	26	(4.9)	26	(5.5)
Non-Nordic (%)	0.5	(6.9)	0.3	(5.2)	2.7	(16.1)
Compulsory (%)	14.1	(34.8)	11.2	(31.6)	15.6	(36.3)
Upper secondary (%)	76.4	(42.5)	74.9	(43.4)	73.6	(44.1)
University (%)	9.5	(29.3)	13.6	(34.3)	10.7	(30.9)
Missing data (%)	0.0	(0.0)	0.3	(5.2)	0.1	(3.4)
Mothers						
Birthyear	1942	(5.8)	1942	(4.9)	1942	(5.9)
Non-Nordic (%)	1.4	(11.9)	2.4	(15.3)	5.0	(21.8)
Compulsory (%)	34.5	(47.6)	36.8	(48.3)	37.4	(48.4)
Upper secondary (%)	41.0	(49.2)	38.4	(48.7)	40.9	(49.2)
University (%)	21.8	(41.3)	23.5	(42.4)	18.0	(38.4)
Missing data (%)	2.7	(16.2)	1.3	(11.5)	3.7	(18.8)
Fathers						
Birthyear	1939	(6.7)	1938	(6.1)	1939	(6.6)
Non-Nordic (%)	4.4	(20.6)	3.7	(19.0)	7.4	(26.2)
Compulsory (%)	41.3	(49.3)	43.5	(49.6)	38.5	(48.7)
Upper secondary (%)	33.4	(47.2)	34.9	(47.7)	36.4	(48.1)
University (%)	19.1	(39.4)	17.9	(38.4)	15.9	(36.5)
Missing data (%)	6.2	(24.1)	3.7	(19.0)	9.2	(28.9)
Individuals	632		375		2,667	

[a] SEK 2010 prices (10 SEK≈€1).

Table B.2: Descriptive statistics of own and parents' background factors for men in year 1990

	Diabetes		Siblings		Controls	
	mean	(sd)	mean	(sd)	mean	(sd)
Own						
Earnings[a]	143,003	(76,093)	148,811	(80,962)	127,712	(79,690)
Age	22	(2.3)	23	(2.4)	22	(2.2)
Birthyear	1968	(2.3)	1967	(2.4)	1968	(2.2)
Mother's age at birth	27	(5.5)	26	(4.8)	26	(5.6)
Non-Nordic (%)	0.3	(5.3)	0.3	(5.1)	1.9	(13.8)
Compulsory (%)	15.5	(36.2)	12.7	(33.3)	15.4	(36.1)
Upper secondary (%)	71.6	(45.1)	70.6	(45.6)	72.7	(44.5)
University (%)	12.8	(33.4)	16.7	(37.3)	11.6	(32.1)
Missing data (%)	0.1	(3.7)	0.0	(0.0)	0.2	(4.6)
Mothers						
Birthyear	1941	(6.0)	1941	(5.0)	1942	(6.1)
Non-Nordic (%)	2.1	(14.3)	0.8	(8.9)	4.5	(20.7)
Compulsory (%)	38.8	(48.8)	37.3	(48.4)	38.5	(48.7)
Upper secondary (%)	40.6	(49.1)	37.0	(48.4)	39.8	(49.0)
University (%)	18.3	(38.7)	23.3	(42.3)	18.3	(38.7)
Missing data (%)	2.2	(14.7)	2.4	(15.3)	3.4	(18.0)
Fathers						
Birthyear	1938	(6.8)	1938	(6.4)	1939	(6.9)
Non-Nordic (%)	4.2	(20.0)	2.9	(16.8)	6.9	(25.4)
Compulsory (%)	38.4	(48.7)	41.0	(49.2)	39.6	(48.9)
Upper secondary (%)	37.9	(48.5)	35.4	(47.9)	35.3	(47.8)
University (%)	16.6	(37.3)	20.4	(40.3)	15.8	(36.5)
Missing data (%)	7.1	(25.7)	3.2	(17.6)	9.2	(29.0)
Individuals	721		378		2,826	

[a] SEK 2010 prices (10 SEK≈€1).

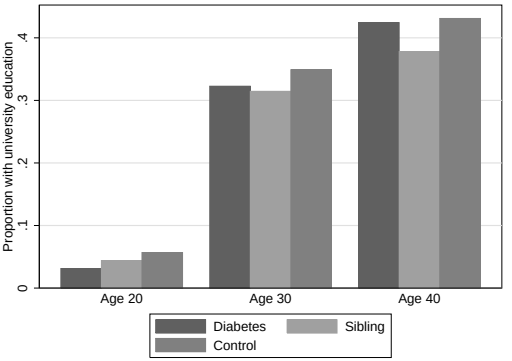

Figure B.1: Proportion of university-educated women at ages 20, 30, and 40 years old

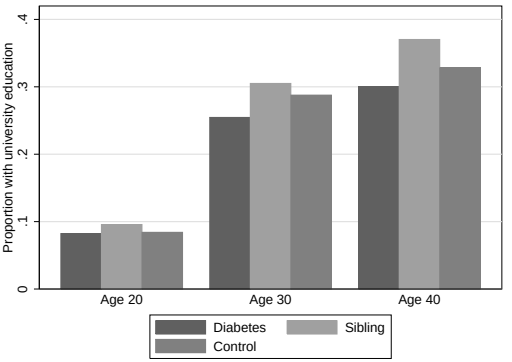

Figure B.2: Proportion of university-educated men at ages 20, 30, and 40 years old

C Variables used as entropy balancing conditions

birthyear	year of birth
M birthyear	mother's year of birth
F birthyear	father's year of birth
M foreign	dummy indicating mothers born in a non-Nordic country
F foreign	dummy indicating fathers born in a non-Nordic country
age mum	mother's age at child's birth
M comp	dummy indicating mothers with only compulsory education
M uni	dummy indicating mothers with university education
M edu mis	dummy indicating mothers with missing data on education
F comp	dummy indicating fathers with only compulsory education
F uni	dummy indicating fathers with university education
F edu mis	dummy indicating fathers with missing data on education
F comp * F birthyear	
F comp * M birthyear	
F comp * birthyear	
F comp * F foreign	
F uni * F birthyear	
F uni * M birthyear	
F uni * birthyear	
F uni * F foreign	
M comp * F birthyear	
M comp * M birthyear	
M comp * birthyear	
M comp * M foreign	
M uni * F birthyear	
M uni * M birthyear	
M uni * birthyear	
M uni * M foreign	
M comp * F comp	
M comp * F uni	
M uni * F uni	
M upp sec * F comp	
M upp sec * F uni	
M foreign * F foreign	
M comp * age mum * birthyear	
M uni * age mum * birthyear	
age mum * birthyear	

D Extended results

Table D.1: Age-specific diabetes estimates (FE) and covariates for individuals with type 1 diabetes

Dependent variable	Women		Men	
log(*Earnings*)	(1)	(2)	(3)	(4)
*diabetes*26–30*	-0.00374	-0.0123	-0.0574***	-0.0567***
	(0.0228)	(0.0225)	(0.0183)	(0.0182)
*diabetes*31–35*	-0.0211	-0.0331	-0.0829***	-0.0807***
	(0.0277)	(0.0272)	(0.0212)	(0.0211)
*diabetes*36–40*	-0.0246	-0.0387	-0.121***	-0.117***
	(0.0276)	(0.0274)	(0.0228)	(0.0227)
*diabetes*41–45*	-0.0818**	-0.0995***	-0.157***	-0.151***
	(0.0339)	(0.0341)	(0.0263)	(0.0262)
*diabetes*46–48*	-0.149*	-0.178**	-0.128	-0.120
	(0.0898)	(0.0855)	(0.0803)	(0.0815)
Age 26–30	-0.0220	-0.00500	0.0893***	0.0868***
	(0.0163)	(0.0162)	(0.0125)	(0.0125)
Age 31–35	-0.0384	-0.000350	0.0764***	0.0699***
	(0.0246)	(0.0244)	(0.0176)	(0.0176)
Age 36–40	-0.0207	0.0193	0.0203	0.0129
	(0.0302)	(0.0297)	(0.0212)	(0.0212)
Age 41–45	-0.0145	-0.00207	-0.0461*	-0.0506**
	(0.0368)	(0.0361)	(0.0257)	(0.0255)
Age 46–48	-0.0398	-0.0748	-0.170***	-0.169***
	(0.0520)	(0.0517)	(0.0513)	(0.0516)
Compulsory	-0.0629	-0.0414	0.0766	0.0689
	(0.0414)	(0.0420)	(0.0517)	(0.0516)
University	0.111***	0.0974***	0.167***	0.163***
	(0.0226)	(0.0230)	(0.0267)	(0.0266)
Married		-0.0345**		0.0674***
		(0.0164)		(0.0124)
Divorced		0.0728**		0.0345
		(0.0321)		(0.0283)
Widow or Widower		-0.138		0.162*
		(0.188)		(0.0849)
Child in household		-0.127***		0.00432
		(0.0125)		(0.00927)
Year FE	Yes	Yes	Yes	Yes
Observations	51022	51022	60586	60586
Individuals	3218	3218	3479	3479
R2	0.351	0.359	0.493	0.495

Robust (clustered) standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1.
Year FE indicates controls for year fixed effects.

Table D.2: Age-specific sibling estimates (FE) and covariates for siblings of individuals with type 1 diabetes

Dependent variable	Women		Men	
log(*Earnings*)	(1)	(2)	(3)	(4)
*sibling*26–30*	-0.00475	-0.000436	-0.0475*	-0.0466*
	(0.0289)	(0.0286)	(0.0252)	(0.0251)
*sibling*31–35*	0.0204	0.0163	-0.0519*	-0.0507*
	(0.0328)	(0.0324)	(0.0306)	(0.0304)
*sibling*36–40*	0.00331	-0.00285	-0.0634*	-0.0602*
	(0.0343)	(0.0343)	(0.0327)	(0.0325)
*sibling*41–45*	-0.000831	-0.00602	-0.0760**	-0.0716**
	(0.0364)	(0.0365)	(0.0359)	(0.0356)
*sibling*46–48*	-0.000354	-0.00445	-0.103	-0.0957
	(0.0677)	(0.0657)	(0.0774)	(0.0784)
Age 26–30	-0.0213	0.000500	0.0719***	0.0680***
	(0.0179)	(0.0178)	(0.0152)	(0.0151)
Age 31–35	-0.0270	0.0190	0.0601***	0.0501**
	(0.0276)	(0.0274)	(0.0225)	(0.0226)
Age 36–40	0.00585	0.0541	0.00714	-0.00378
	(0.0336)	(0.0332)	(0.0274)	(0.0278)
Age 41–45	0.0193	0.0384	-0.0606*	-0.0670**
	(0.0411)	(0.0407)	(0.0335)	(0.0336)
Age 46–48	-0.00721	-0.0277	-0.149**	-0.147**
	(0.0578)	(0.0576)	(0.0590)	(0.0593)
Compulsory	-0.0689	-0.0400	0.191***	0.188***
	(0.0532)	(0.0531)	(0.0511)	(0.0509)
University	0.108***	0.0892***	0.175***	0.175***
	(0.0299)	(0.0298)	(0.0438)	(0.0434)
Married		-0.0335		0.0641***
		(0.0215)		(0.0177)
Divorced		0.0764**		0.0184
		(0.0338)		(0.0345)
Widow or Widower		0.250*		-0.0356
		(0.128)		(0.125)
Child in household		-0.121***		0.0154
		(0.0134)		(0.0117)
Year FE	Yes	Yes	Yes	Yes
Observations	47577	47577	55319	55319
Individuals	2974	2974	3154	3154
R2	0.388	0.396	0.493	0.495

Robust (clustered) standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1.
Year FE indicates controls for year fixed effects.

Paper IV

Early onset of type 1 diabetes and educational field at upper secondary and university level: is own experience an asset for a health care career?

with Katarina Steen Carlsson[i]

Abstract

Previous evidence shows that ill health in early life has a significant negative impact on school grades, grade repetition, educational level, and labor market outcomes. But are all aspects of a health shock in childhood or adolescence necessarily bad, or could it also create comparative advantages and experiences that could have professional value? We analyze this question using the Swedish Childhood Diabetes Register, the National Educational Register, and other population registers in Sweden. More specifically, we investigate the relationship between onset of type 1 diabetes (up to age 15) and the probability of choosing (and completing) a health-oriented path at upper secondary and university level. By modeling the educational decisions as an unsorted series of binary choices, we shed light on the more qualitative aspects of schooling and assess a potential mechanism linking early life health to adult outcomes. Our results reject the hypothesis of no systematic differences in choice of educational field between people with and without type 1 diabetes. The results are robust to selection on ability proxies and across sensitivity analysis. We conclude that disease onset in childhood and adolescence may generate experiences and comparative advantages for choosing and completing a health-oriented program of education.

Keywords: Health, educational choices, diabetes, comparative advantages
JEL Classification: I10, I12, I21, J13, J24

[i]Department of Clinical Sciences, Health Economic Unit Lund, Lund University, Malmö, Sweden. The Swedish Institute for Health Economics, Lund, Sweden

1 Introduction

Earlier work has shown that ill health in early life has a significant negative impact on school grades, grade repetition, educational level, and labor market outcomes (see, e.g., Currie & Almond (2011), and Currie (2009)). But are all aspects of a health shock in childhood or adolescence necessarily bad, or could it also create comparative advantages and experiences that could have a professional value? Childhood and adolescence are formative years and the choices made could have life-long consequences (Cunha & Heckman, 2008). The choice of educational path may be influenced by numerous factors within or beyond the individuals' (or their families) control. Previous research connects cognitive abilities and socioeconomic background to both educational achievements (see, e.g., Currie & Almond (2011), and Cunha et al. (2006)) and labor market outcomes (see, e.g., Cunha et al. (2006), Heckman et al. (2006), and Card (1999)). Besides cognitive abilities, life experiences and preferences are likely drivers of choice. For example, Heckman et al. (2006) conclude that such non-cognitive abilities were equally important as cognitive abilities for an individual's choice of schooling and wage (given the level of education chosen).

Traditionally, the economic literature has explored human capital in the form of years of schooling or attainment of a degree to assess its impact on life-time earnings (see, e.g., Card (1999)). Complementary to formal education, abilities and skills created from life experiences could give the individual comparative advantages for specific careers (Paglin & Rufolo, 1990). Such comparative advantages could be decisive for paths of formal education and the choice of profession (Gemici & Wiswall, 2014; Arcidiacono et al., 2012; Lee, 2005; Montmarquette et al., 2002).

Drawing on the definition by Cunha & Heckman (2008) of human capabilities as health, cognitive abilities, and non-cognitive abilities, we use onset of type 1 diabetes, as a measure of change in human capabilities, to assess consequences for individuals' choice of educational field. Health events early in life, such as the onset of chronic disease, are not in all aspects detrimental, but may also provide useful experiences and new capabilities. Yet, only a few studies in the literature linking child health to educational and labor market outcomes assess the influence of human capabilities. Some researchers state that abilities may confound child health and adult outcomes as estimated effects are reduced when controlling for cognitive and non-cognitive ability (Lundborg, Nystedt, & Rooth, 2014; Case & Paxson, 2008). This evidence seems reasonable for many health conditions driven by individuals' behaviors and lifestyle. However, Salm & Schunk (2012), Currie et al. (2010), and Heckman (2007) argue that child or adolescent abilities are influenced by health and therefore potentially also mediate the impact of health.

While disease onset can be expected to influence people in many dimensions, the impact of such experiences on educational choices has received little

attention. On balance, a health shock early in life may reduce overall incentives for educational investments (Currie et al., 2010; Currie, 2009; Case et al., 2005), but it could also create incentives for choosing an educational field where the experience of disease and its treatment could be an asset. Following this line of argument, own experience of disease would create a comparative advantage in health and medical professions from a qualitative perspective.

We contribute to the literature on child and adolescent health and adult outcomes by investigating whether experience of chronic disease influences educational choices. We analyzed the relationship between onset of type 1 diabetes (up to age 15) and the probability of choosing (and completing) a health-oriented educational program at upper secondary school and university. By modeling the educational decisions as an unsorted series of binary choices, we shed light on the more qualitative aspects of schooling and assessed a potential mechanism linking early life health to adult outcomes.

We use national longitudinal population registers from Statistics Sweden and the national Childhood Diabetes Registry (SCDR), which registers incidences of type 1 diabetes up to age 15 for men and women born 1962–1975. These data bring several advantages. First, the onset of type 1 diabetes is triggered by a complex combination of both genetic and environmental components (Daneman, 2006). Heritability is low and more than 90% of cases occur among individuals without a first-degree relative with the disease (Dahlquist & Mustonen, 2000). Consequently, individuals are unable to influence or anticipate onset beforehand (Dahlquist et al., 1989) and type 1 diabetes has therefore been described as an exogenous health shock (Persson et al., 2013; Minor, 2011; Steen Carlsson et al., 2010). Lundborg, Nilsson, & Rooth (2014) confirm this notion: they show that men's earnings penalty from diabetes (at age 18) is robust to sibling fixed effects and unobserved factors at the family level. The shock-like nature of onset supports the argument that abilities will mediate, rather than confound, the studied health-education relationship.

Second, we can explore different fields of education, aggregated via the official Swedish coding system (SUN, the Swedish Educational Terminology), and assess the influence of different observable ability measures (i.e., parental level of education, maternal ability, and upper secondary grades). Such family background factors are highly correlated with children's cognitive and non-cognitive abilities (Heckman, 2007; Cunha et al., 2006). Third, the SCDR has followed the national incidence of type 1 diabetes since 1977 and has an estimated coverage of 96–99% (Nyström et al., 1990). The high coverage, together with universal social insurance coverage in Sweden (with low cost of care, and pediatric care free), ensures high representativeness, which is often troublesome in survey data or non-mandatory insurance data. Fourth, the health shock is physician-assessed, and the impact on daily life and the health-related consequences are well-described (Sparud-Lundin et al., 2010; Wennick et al., 2009), leaving no

room for potential confusion with type 2 diabetes, which differs in etiology and key disease consequences.

We investigate whether a health shock early in life links with a health-oriented education chosen at age 16 or after age 18. Using multinomial logit regressions, we compare educational choices of people with type 1 diabetes to population controls with the same year of birth and municipality of residence in the year of disease onset. Our results reject the hypothesis of no systematic differences in choice of educational field between people with and without type 1 diabetes. The results were robust to selection on ability proxies and across sensitivity analysis.

The structure of the paper is as follows. Section 2 provides a conceptual framework. Section 3 presents the data and Section 4 details our econometric strategy. Section 5 presents the results. Section 6 discusses the results and concludes.

2 Conceptual framework

In contrast to the traditional human capital literature (Becker, 1962; Grossman, 1972a,b), which distinguishes between acquired skills and genetically determined cognitive ability, Cunha & Heckman (2008), Heckman (2007) and Cunha et al. (2006) argue that behaviors and abilities have both a genetic and an acquired character. Measured abilities are the outcome of investments and gene-environment interactions. Abilities are cognitive (e.g., IQ) or non-cognitive (e.g., patience, motivation, time preferences, and self-control) and affect learning, health behaviors, and health. Thus, the human capabilities (health, cognitive abilities and non-cognitive abilities) are closely related and are formed throughout the life cycle of an individual. The model of human capability formation implies that, first, abilities are both persistent and self-reinforcing as higher abilities in one period lead to higher abilities in the next period and, second, previously acquired abilities make further investments more productive. Consequently, even small childhood health shocks might snowball into adverse adult health and labor market outcomes. Following, Cunha & Heckman (2008), Heckman (2007), and Cunha et al. (2006), experiencing the onset of a chronic disease and daily disease management could translate into preferences and comparative advantages for a health-oriented course of education.

3 Data

The Swedish Childhood Diabetes Register (SCDR) has recorded incident cases of type 1 diabetes in children aged 0–14.9 years in Sweden since 1 July 1977 (Dahlquist et al., 1982) to enable studies on the etiology, incidence trends, and complications of diabetes. Data for the SCDR are collected according to the

Declaration of Helsinki, and informed consent is given by all parents of registered children. To study socioeconomic effects of the onset of type 1 diabetes in childhood and adolescence, the Swedish Childhood Diabetes Study Group has added data to the SCDR as follows: for each individual in the SCDR, Statistics Sweden matched four people without diabetes from the Total Population Register by year of birth and municipality of residence at the time of type 1 diabetes diagnosis. Statistics Sweden identified parents of persons with type 1 diabetes and population controls from the Multi-Generation Register (Statistics Sweden, 2009). Covering the period 1990–2010, Statistics Sweden then added socioeconomic and demographic data for each person in the research database from the national population registers, the Longitudinal Integration Database for Health Insurance and Labour Market Studies (LISA) (Statistics Sweden, 2011b) and the Swedish Register of Education (Statistics Sweden, 2006).[1]

We retrieved data on 2,756 individuals born in 1962–1975 and diagnosed with type 1 diabetes (hereafter referred to as diabetes) in the age group 2–5 (during the years 1977–90) and 11,020 matched population controls. The study was approved by the Regional Research Ethics Board in Umeå (Dnr 07- 169M).

3.1 Dependent variables

Onset occurs before entering upper secondary education as students enroll at age 16 (Statistics Sweden, 2008). We use Statistics Sweden's SUN classification to identify level of education and aggregate programs into the different fields.[2] For university education, we use the last available SUN registration up to year 2010. For upper secondary programs, we use the Swedish Register of Education and classify programs to mimic the SUN-classification.

To capture all types of preferences and abilities irrespective of whether they favor an education that is, e.g., long or short, theoretical or vocational, humanistic or technical, the dependent upper secondary variable is an unordered categorical variable with four categories: *vocational health*, *vocational other*, *theoretical health*, and *theoretical other*. Vocational health includes programs that train for jobs in the health care sector, such as nursing assistant, but also jobs within social services, child care and care for the elderly.[3] Possibly, an interest in caring for other people, rather than an interest in health per se, has motivated some people

[1] All data provided to the research group have been anonymized by Statistics Sweden.

[2] The SUN-classification system is adapted to International Standard Classification of Education ISCED 97 (Statistics Sweden, 2000). Appendix A lists the different educational fields available in SUN.

[3] Vocational health programs, and especially those directed towards child care, were popular during the studied period with more students applying than admitted. For example, 13,613 applicants had vocational health as their first choice and 13,206 students were admitted in 1994 (The Swedish National Agency for Education, 1995). Child care was the third (tenth) most popular upper secondary program among girls (boys)(Broady et al., 2000).

Table 1: Descriptive statistics of educational field at upper secondary school and university

	Women				Men			
	Diabetes		Control		Diabetes		Control	
	N=1292		N=5444		N=1464		N=5576	
	prop	n	prop	n	prop	n	prop	n
Upper secondary								
Attendance[a]	0.895	1156	0.907	4940	0.881	1290	0.882	4917
Type if attending[b]								
Vocational health	0.302	349	0.248	1223	0.083	107	0.052	255
Vocational other	0.318	368	0.321	1588	0.613	791	0.597	2936
Theoretical health	0.061	71	0.048	237	0.006	8	0.013	65
Theoretical other	0.318	368	0.383	1892	0.298	384	0.338	1661
University								
Attendance[a]	0.401	518	0.458	2494	0.300	439	0.350	1954
Type if attending[b]								
Health	0.303	157	0.233	581	0.075	33	0.073	142
Other	0.697	361	0.767	1913	0.925	406	0.927	1812

[a] *Attendance* is tested with t-tests. Diabetes and controls are significantly different at university for both women and men ($p<0.01$).

[b] *Type if attending* is tested with chi2 tests. Diabetes and controls are significantly different at upper secondary for both women and men ($p<0.01$) and at university for women ($p<0.01$).

to choose such programs, and we test the robustness of the results to the chosen classification in a sensitivity analysis. Theoretical programs prepare for all types of university studies and it is not possible to identify a health interest via the chosen program. Instead, we derive information on health orientation during theoretical upper secondary programs from subsequent choices of university education.

For the university level, we define two dichotomous dependent variables. *University education* indicates individuals with credits from a Swedish university corresponding to at least 20 weeks of full-time studies. *Health-oriented* indicates university programs to become a physician, physiotherapist, occupational therapist, pharmacist, biomedical scientist, dentist, social worker, etc.

The distribution of education is shown in Table 1. Comparing individuals with and without diabetes, there are no significant differences in upper secondary attendance, but a lower proportion of students with diabetes attended univer-

sity (women and men p<0.01). Women with diabetes are overrepresented in health-oriented education at all levels (p<0.01), while men with diabetes are overrepresented for vocational health but underrepresented for theoretical health (p<0.01).[4]

3.2 Control variables

Given general gender differences in attainment, grades, and future career choices (Statistics Sweden, 2008), diabetes may influence women's and men's educational choices differently and we present results stratified by gender.[5] We control for year of birth in three categories (1962-65, 1966-1970, and 1971-1975)[6] to capture (1) competition in accessing different educational programs and jobs, and (2) economic trends and changes in the educational system as cohorts generally follow each other through the educational system. We also control for being born in a non-Nordic country and parents with non-Nordic origins, due to the relatively high incidence of diabetes in the Nordic countries (Karvonen et al., 2000) and findings of labor market discrimination (see, e.g., Altonji & Blank (1999)).

Following Salm & Schunk (2012), Currie et al. (2010), and Heckman (2007), we argue that child or adolescent abilities are influenced by health and therefore potentially mediate the impact of health. To the extent that this argument is true, changes in individual characteristics due to diabetes are captured by the diabetes estimates when abilities are not controlled for. We test the robustness to observable background factors by assessing changes in the diabetes estimates when adding variables to our specification. We present a specification that controls only for year of birth and non-Nordic-origin (spec. 1) and another specification with the full set of controls (spec. 2). We test the influence of ability measures and variables that may correlate with socioeconomic status, health, and educational choices. We account for mother's and father's level of education (compulsory, upper secondary, and university) because parents contribute to their child's health, abilities, and schooling (Currie, 2009; Cunha & Heckman, 2008; Cunha et al., 2006; Chevalier, 2004; Black & Devereux, 2011). Better-educated parents, as a group, have been found to have higher (acquired and/or innate) ability,

[4] Only eight men (71 women) with diabetes study a theoretical upper secondary program and then continue studying health at university (theoretical health). Still, 33 men (157 women) with diabetes study health at university, meaning that people also continue to university after vocational upper secondary programs. To be accepted at a university after a vocational upper secondary program generally required supplementary studies regardless of field of education(Broady et al., 2000). We discuss the alternative choices of a health-oriented education when discussing the assumptions of the multinomial logit model in Section 4.1.

[5] To present separate estimates for women and men is standard in labor economics. See, e.g., (Card, 1999).

[6] Using dummy variables for each year of birth introduces problems with perfect predications among some years.

Table 2: Descriptive statistics of own and parents' background factors

	Women		Men	
Variables[a]	Diabetes prop	Control prop	Diabetes prop	Control prop
Born 1962-65	0.056	0.076	0.090	0.072
Born 1966-70	0.336	0.329	0.337	0.344
Born 1971-75	0.608	0.595	0.573	0.584
Non-Nordic	0.009	0.029	0.003	0.024
Mothers				
Compulsory	0.321	0.335	0.351	0.344
Upper secondary	0.444	0.429	0.426	0.424
University	0.209	0.202	0.206	0.203
Edu missing	0.026	0.033	0.017	0.029
Edu in health	0.243	0.221	0.227	0.215
Non-Nordic	0.022	0.048	0.020	0.050
Year of birth (mean)	1944	1945	1944	1944
Age at child's birth (mean)	27	26	27	26
Fathers				
Compulsory	0.379	0.359	0.372	0.363
Upper secondary	0.373	0.380	0.393	0.377
University	0.186	0.186	0.185	0.184
Edu missing	0.062	0.076	0.051	0.075
Edu in health	0.026	0.026	0.025	0.022
Non-Nordic	0.056	0.083	0.055	0.087
N	1292	5444	1464	5576

[a] The following variables are significantly different (on at least the 10% level) between the diabetes group and the control group for both women and men: *birth categories, non-Nordic, mother's years of birth, age at child's birth*; for women: *mother with edu in health*, for men: *mother's level of education*.

earn more income, and live in areas providing high quality schooling. For the university-level analysis, we add a control for upper secondary grades, which might be a stronger ability proxy than parental education, but the grades are likely to be affected by onset. Moreover, parents' choice of occupation might influence the child's choice and we control for parents having a health-related upper secondary or university education (using the same classification as for the dependent variables).

Mother's year of birth captures the increasing trend in women's labor market participation since 1960 in Sweden, contributing to an increasing number of women working after they have had children (Statistics Sweden, 2011a). Mother's age at child's birth takes into account that (1) younger mothers are less likely than older mothers to have had time to educate and establish themselves on the labor market, and (2) late childbearing increases the pregnancy-related risks for both mother and child (Jonasson et al., 2007; Casson et al., 1997).[7] Furthermore, Cunha et al. (2006) show that women with low cognitive ability are more likely to bear children at younger ages.

The descriptive statistics in Table 2 confirm a higher proportion of non-Nordic-born individuals in the control groups than in the diabetes groups. Notably, there is no significant difference in parental level of education between the groups for women. Mothers of women with diabetes are overrepresented in health-oriented education (p=0.096). For men, the difference is not in health-orientation but in level of education (p=0.014). However, this difference is driven by differences in missing educational data, which is more common among those with a non-Nordic origin. Mothers in the diabetes group are on average older (women p=0.076, men p=0.068) and had children at older ages (women p=0.0056, men p=0.070) than mothers of controls. We test if these differences imply confounding by regressing the independent variables on the probability of being in the diabetes group, but only non-Nordic-origin predicts diabetes.[8]

4 Methods

4.1 Upper secondary education

We use a multinomial logit model to determine whether diabetes links to the probability of choosing (and completing) a health-oriented upper secondary education. In this setting, a child i gains utility from choosing an educational field j given his or her individual characteristics x:

$$U_{ij} = \beta' x_i + \epsilon_{ij} \tag{1}$$

[7] We control also for a polynomial of the variable mother's age at child's birth, as we expect the impact of this variable to be non-linear. However, the polynomial is omitted because of collinearity.

[8] Results are available on request.

The child chooses the field that he or she prefers to all others and the probability that the child will choose field j is:

$$Prob(Y_i = j) = \frac{\exp(\beta_j' x_i)}{\sum_{k=1}^{4} \exp(\beta_k' x_i)}, j = 1,2,3,4. \quad (2)$$

Where the choice set j is: 1=vocational other, 2=vocational health, 3=theoretical health, and 4=theoretical other. Multinomial logit models are estimated using maximum likelihood to find the β_j:s that best fit the data. We condition this analysis on having an upper secondary education. Consequently, each student falls into one of the educational fields and the probabilities will sum to one. To ensure model identification, β_1 is set to zero (the reference category) and coefficients are interpreted with respect to vocational other (the largest group). The choice of reference category is important as the estimated coefficients apply in reference to that group.[9] We find no indication that diabetes causes selection into upper secondary education or into vocational other.[10]

The advantage of the multinomial logit over other multinomial models is that the computations are simple and parameter estimates are relatively easy to interpret with marginal effects. The drawback is the restrictiveness of the independence of irrelevant alternatives (IIA) assumption. This assumption implies that the choice between any two pairs of alternatives is not affected if we add another alternative to the choice set or change the characteristics of a third alternative.

The IIA assumption is most likely to hold when the categories of the dependent variables are sufficiently different. It seems plausible to assume that one's interest in health will not depend on the supply of educational programs in other fields. Still, individuals might have more than one career choice and, if the alternative choice becomes more accessible, one might favor this field instead. Moreover, if the two paths to a health-oriented education (vocational or theoretical) can serve as substitutes, then the results of the multinomial logit may not be realistic. However, the categories of our dependent variable appear to be different. Using likelihood-ratio test and Wald test for combining outcome categories, we reject the hypothesis that our independent variable does not differentiate between categories.[11] Moreover, a health-related occupation requiring a university education is arguably not the same as one requiring only upper

[9] Compulsory education might appear a natural comparison group, as most people first decide whether to study and then what to study. However, only about 10% never continue to upper secondary education (Table 1) and this is likely to be a socioeconomically disadvantaged group (Broady et al., 2000).

[10] We test for selection due to diabetes by running a logit model of (1) the likelihood of having upper secondary education and (2) the likelihood of having vocational other. The results are available on request.

[11] Results are available on request.

secondary qualifications. Also, to be accepted to a university after a vocational upper secondary program generally required supplementary studies regardless of field of education (Broady et al., 2000). Nevertheless, to alleviate the IIA concerns, we test the IIA assumption with a suest-based Hausman test and find no indication that the assumption is violated. Additionally, we run a nested logit model that relaxes the IIA assumption[12] and find similar results as when using the multinomial logit. Results are available in Appendices B and C.

4.2 University education

For the university-level analysis, we model diabetes-related differences in (1) the probability of having a university education, and (2) the probability of having a health-oriented education for those with a university-level education. We use the logit model for these analyses as the outcomes are limited to choices between two alternatives.

4.3 Sensitivity analysis

4.3.1 Different definitions of health-oriented education

Lower grades and previous achievements might hinder individuals with diabetes from getting into popular educational programs. In addition, lower grades and attainment could be interpreted in terms of less productive learners or producers of abilities and deter those with diabetes from the more demanding programs. We cannot fully answer to the mechanisms at play, but we make a first attempt when testing the robustness of our results to (1) a more narrow definition of health-oriented education with a strict focus on health care and (2) the longest and most demanding university programs.

4.3.2 Age at onset and duration of diabetes

Evidence shows that children's vulnerability to health shocks differs by age (Currie & Almond, 2011; Cunha et al., 2006). Duration of a disease might also be important, given that it generally takes time to adapt to new life circumstances (Wennick et al., 2009) and for difficulties to manifest as, for example, lower educational achievements (Persson et al., 2013). Crudely testing the results sensitivity to onset ages and consequent duration, we present results from analyses where

[12] We group vocational and theoretical health into one nest and vocational and theoretical other into a second nest: thereby, we allow the errors to be correlated within each nest, while we still assume them to be independent between the nests.

the diabetes variable indicates onset at ages 2–9 or 10–15 (controls are still the reference).[13]

5 Results

5.1 Upper secondary education

Table 3 for women and Table 4 for men show significant average marginal effects (AME) of diabetes on the probability of having upper secondary education in both vocational and theoretical health, using vocational other as the reference category.[14] Relative to vocational other, diabetes is associated with a 5.5 percentage points increase in women's likelihood of vocational health and a 1.3 percentage points increase for theoretical health. Diabetes is also associated with an increase in men's likelihood of vocational health (0.031), but the association is negative for theoretical health (-0.007).

When adding the full set of controls (comparing spec. 1 and spec. 2), the diabetes estimates do not change much, suggesting that results are robust to the influence of, e.g., mother's and father's education and maternal ability. Mother's and father's level of education have the expected signs for both women and men. Mother's year of birth and mother's age at child's birth appear influential for women's likelihood of having a health-oriented education, when simultaneously controlling for parental level of education. To have a mother who is born later is associated with an increasing likelihood of theoretical health, while the likelihood decreases for vocational health. Thus, women's increased labor market participation appears to have a positive net effect on theoretical health, but a negative one for vocational health. Mother's age at child's birth is negatively associated with vocational health, suggesting that the likelihood of vocational health decreases with increasing age of the mother at the time of the child's birth.

In summary, diabetes relates to an increased likelihood of vocational health for both women and men, while the likelihood of theoretical health is increased for women, but decreased for men. Both women (-0.063) and men (-0.039) have a lower likelihood of theoretical other.

[13] Due to the design of the data, individuals with onset in ages 2–9 were born during the years 1968–1975, while individuals with onset in ages 10–15 were born in 1962–1975. All individuals born before 1968 are excluded from this analysis.

[14] A positive (negative) estimate for an educational field implies that diabetes is associated with an increased (decreased) likelihood of that field relative to vocational other. All the following results from the multinomial logit model are to be interpreted in relation to having an education in vocational other.

Table 3: Multinomial logit estimations (average marginal effects) of women's probability of having different fields of upper secondary education using *vocational other* as the reference category

	Spec. 1			Spec. 2		
	Voc. health	Theo. health	Theo. other	Voc. health	Theo. health	Theo. other
Diabetes	0.055***	0.013*	-0.063***	0.053***	0.014*	-0.064***
	(0.015)	(0.0078)	(0.015)	(0.015)	(0.0078)	(0.015)
Year of birth						
1962–1965	0.080***	-0.020**	-0.11***	-0.099***	0.0064	0.074
	(0.024)	(0.0099)	(0.024)	(0.037)	(0.024)	(0.049)
1966–1970	-0.0087	-0.010*	-0.11***	-0.097***	0.0036	-0.015
	(0.012)	(0.0060)	(0.013)	(0.022)	(0.012)	(0.025)
Mothers						
Non-Nordic	-0.093***	0.015	0.10**	-0.10***	0.020	0.095**
	(0.033)	(0.020)	(0.043)	(0.034)	(0.021)	(0.043)
Year of birth				-0.018***	0.0022	0.017***
				(0.0042)	(0.0021)	(0.0045)
Age at child's birth				-0.019***	0.0029	0.022***
				(0.0043)	(0.0022)	(0.0047)
Compulsory				0.024	-0.025***	-0.057***
				(0.015)	(0.0061)	(0.015)
University				-0.072***	0.033***	0.11***
				(0.015)	(0.0095)	(0.018)
Missing				-0.047	-0.052***	-0.0078
				(0.13)	(0.0045)	(0.13)
Health				0.083***	-0.010*	-0.065***
				(0.016)	(0.0062)	(0.015)
Fathers						
Non-Nordic	-0.045*	0.025*	0.033	-0.034	0.025*	0.031
	(0.024)	(0.015)	(0.027)	(0.025)	(0.015)	(0.027)
Compulsory				0.042***	-0.0047	-0.057***
				(0.014)	(0.0068)	(0.015)
University				-0.072***	0.022***	0.12***
				(0.016)	(0.0086)	(0.019)
Missing				-0.031	0.0047	-0.037
				(0.024)	(0.013)	(0.027)
Health				0.026	0.022	-0.029
				(0.040)	(0.017)	(0.036)
Observations	5952	5952	5952	5846	5846	5846

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Year of birth in 1971–1975 and *Upper secondary education* are reference categories.

Non-Nordic predicts failure perfectly and 171 observations are dropped.

Table 4: Multinomial logit estimations (average marginal effects) of men's probability of having different fields of upper secondary education using *vocational other* as the reference category

	Spec. 1			Spec. 2		
	Voc. health	Theo. health	Theo. other	Voc. health	Theo. health	Theo. other
Diabetes	0.031***	-0.0070**	-0.039***	0.032***	-0.0072**	-0.041***
	(0.0083)	(0.0028)	(0.014)	(0.0084)	(0.0028)	(0.014)
Year of birth						
1962–1965	0.046***	-0.011***	-0.019	0.031	-0.0085	0.027
	(0.015)	(0.0030)	(0.024)	(0.031)	(0.0056)	(0.047)
1966–1970	-0.0070	-0.0025	-0.14***	-0.013	0.0019	-0.11***
	(0.0062)	(0.0030)	(0.012)	(0.012)	(0.0063)	(0.023)
Mothers						
Non-Nordic	-0.013	0.0029	0.055	-0.014	0.0069	0.060
	(0.017)	(0.0082)	(0.040)	(0.017)	(0.0099)	(0.038)
Year of birth				-0.0013	0.00050	0.000016
				(0.0023)	(0.0010)	(0.0042)
Age at child's birth				-0.0015	0.00052	0.0057
				(0.0023)	(0.0011)	(0.0043)
Compulsory				0.0071	-0.0061**	-0.087***
				(0.0081)	(0.0027)	(0.014)
University				0.0044	0.012**	0.13***
				(0.0083)	(0.0047)	(0.017)
Missing				0.034	-0.011***	0.015
				(0.087)	(0.0021)	(0.13)
Health				0.018**	0.0016	-0.074***
				(0.0085)	(0.0032)	(0.013)
Fathers						
Non-Nordic	0.015	0.011	-0.0011	0.017	0.011	-0.0018
	(0.015)	(0.0079)	(0.025)	(0.015)	(0.0079)	(0.025)
Compulsory				-0.0068	0.0012	-0.099***
				(0.0073)	(0.0032)	(0.014)
University				0.0020	0.012***	0.17***
				(0.0094)	(0.0043)	(0.018)
Missing				-0.0036	0.0061	-0.086***
				(0.014)	(0.0068)	(0.025)
Health				0.0014	0.020*	-0.055*
				(0.019)	(0.011)	(0.033)
Observations	6100	6100	6100	5997	5997	5997

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Year of birth in 1971–1975 and *Upper secondary education* are reference categories.

Non-Nordic predicts failure perfectly and 140 observations are dropped.

5.2 University education

Tables 5 and 6 presents AME from logit estimations of the probability of having a university education (columns 1–2) and the probability of having a health-oriented education for those with a university education (columns 3–4). We do not present results from the reference specification (controlling only for year of birth and being born in a non-Nordic country) as the results are robust to the influence of additional controls.[15] Without controlling for grades, diabetes is associated with a decreased likelihood of attending university (-0.064 for women, and -0.057 for men). When attending, the likelihood of health orientation is increased for women (0.074) but not for men (the estimate is close to zero and insignificant). When adding controls for grades[16] (column 1 vs. 2), the diabetes estimates on attendance is reduced by approximately one third: the AME of diabetes changes from -0.064 to -0.045 for women and from -0.057 to -0.044 for men. This finding suggests that some of the negative impact of diabetes might be attributed to lower upper secondary grades among students with diabetes.[17] The diabetes estimates on the probability of having a health-oriented education appear robust to the influence of grades. Higher grades are positively associated with attendance, but its link to educational field is weaker (partly because the analysis for health-orientation is conditioned on attendance, and grades affect university attendance).

Mothers with a health-oriented education along with parents' level of education have larger estimates for attendance than for educational field, indicating that they are more strongly linked to attendance. In contrast, having fathers with a health-oriented education is strongly related to health orientation (for men at least), while it appears unrelated to attendance. Possibly, the differing influence of mothers' and fathers' health interest could relate to women and men generally having different types of occupations within the health care sector (Statistics Sweden, 2014). The influence of parents appears less important once we add controls for grades, as both grades and parental level of education are proxies for abilities and skills.

[15] The changes in estimates between this specification and the reference specification (controlling only for year of birth and being born in a non-Nordic country) are marginally larger than for the upper secondary analysis. Results are available on request.

[16] Grades were available from the Swedish Register of Education: only 11.1% of those attending upper secondary education have missing grades.

[17] Grades were set on a scale from 1–5. t-tests indicate significant mean differences between the groups. Mean upper secondary grades for women (men) with diabetes were 3.26 (3.09) compared to 3.32 (3.13) for women (men) controls.

Table 5: Logit estimations (average marginal effects) of women's probability of having a university education *(Uni)* and the probability of having a health-related university education for those with a university education *(Health)*

	Uni		Health	
	(1)	(2)	(3)	(4)
Diabetes	-0.064***	-0.045***	0.074***	0.076***
	(0.015)	(0.014)	(0.022)	(0.022)
Upp sec grades		0.26***		0.034**
		(0.0091)		(0.015)
Grades missing		0.37***		0.22***
		(0.010)		(0.076)
Mothers				
Non-Nordic	0.0071	0.030	0.037	0.035
	(0.036)	(0.034)	(0.050)	(0.049)
Year of birth	0.0013	-0.0016	-0.0096	-0.0097
	(0.0043)	(0.0040)	(0.0059)	(0.0059)
Age at child's birth	0.0096**	0.0023	-0.011*	-0.012*
	(0.0044)	(0.0041)	(0.0061)	(0.0061)
Compulsory	-0.14***	-0.094***	-0.012	-0.011
	(0.015)	(0.014)	(0.022)	(0.022)
University	0.18***	0.12***	-0.032	-0.037*
	(0.017)	(0.016)	(0.019)	(0.019)
Edu missing	-0.13	0.0029	-0.12	-0.13
	(0.088)	(0.089)	(0.13)	(0.12)
Edu in health	-0.061***	-0.031**	0.018	0.020
	(0.014)	(0.013)	(0.020)	(0.020)
Fathers				
Non-Nordic	0.014	0.026	-0.013	-0.016
	(0.026)	(0.023)	(0.033)	(0.033)
Compulsory	-0.052***	-0.031**	0.047**	0.046**
	(0.014)	(0.013)	(0.020)	(0.020)
University	0.14***	0.089***	-0.0060	-0.0080
	(0.018)	(0.017)	(0.020)	(0.020)
Edu missing	-0.082***	-0.032	0.022	0.014
	(0.025)	(0.023)	(0.038)	(0.038)
Edu in health	-0.0099	0.0089	0.078	0.077
	(0.037)	(0.036)	(0.048)	(0.048)
Demographics	Yes	Yes	Yes	Yes
Observations	6508	6508	2946	2946

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.
Demographics indicates controls for *year of birth* and *non-Native*.

Table 6: Logit estimations (average marginal effects) of men's probability of having a university education *(Uni)* and the probability of having a health-related university education for those with a university education *(Health)*

	Uni		Health	
	(1)	(2)	(3)	(4)
Diabetes	-0.057***	-0.044***	0.0046	0.0049
	(0.013)	(0.012)	(0.015)	(0.015)
Upp sec grades		0.20***		0.012
		(0.0079)		(0.0100)
Grades missing		0.34***		0.044
		(0.021)		(0.061)
Mothers				
Non-Nordic	0.0087	-0.0014	0.044	0.042
	(0.033)	(0.031)	(0.040)	(0.039)
Year of birth	0.00093	-0.0021	0.0012	0.0011
	(0.0039)	(0.0037)	(0.0040)	(0.0040)
Age at child's birth	0.0095**	0.0035	-0.00091	-0.0011
	(0.0041)	(0.0038)	(0.0040)	(0.0040)
Compulsory	-0.11***	-0.086***	-0.0040	-0.0041
	(0.013)	(0.013)	(0.014)	(0.014)
University	0.15***	0.096***	0.0046	0.0027
	(0.017)	(0.015)	(0.014)	(0.014)
Edu missing	0.010	0.095		
	(0.099)	(0.093)		
Edu in health	-0.051***	-0.035***	0.011	0.012
	(0.013)	(0.012)	(0.014)	(0.014)
Fathers				
Non-Nordic	-0.0027	0.0039	0.058**	0.057*
	(0.024)	(0.022)	(0.029)	(0.029)
Compulsory	-0.099***	-0.076***	0.052***	0.052***
	(0.013)	(0.012)	(0.017)	(0.017)
University	0.17***	0.13***	0.00074	-0.00072
	(0.018)	(0.016)	(0.013)	(0.013)
Edu missing	-0.10***	-0.053**	0.023	0.022
	(0.022)	(0.022)	(0.026)	(0.026)
Edu in health	-0.0070	-0.019	0.083*	0.082*
	(0.031)	(0.030)	(0.044)	(0.044)
Demographics	Yes	Yes	Yes	Yes
Observations	6854	6854	2313	2313

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.
Demographics indicates controls for *year of birth* and *non-Native*.

5.3 Sensitivity analysis

5.3.1 Different definitions of health-oriented education

We test a more narrow definition of health-oriented education with a strict focus on health care. When we relabel social care, child care and care for the elderly as 'other', rather than 'health', the proportion of vocational health decreases from 15.7% to 7.4% of all upper secondary students. Theoretical health also decreases slightly from 3.11% to 3.05%. Compared to the broader definition in Tables 3 and 4, the estimates in Table 7 decrease in size for vocational health (for both women and men) and theoretical health becomes insignificant. However, the message of the main analysis remains, even though the shift substantially changes the distribution of educational fields: 46.9% of the diabetes group and 55.1% of the control group switched category.

Table 7: Test with a more restrictive definition of health-oriented: Multinomial logit estimations (average marginal effects) of the probability of having different fields of upper secondary education using *vocational other* as the reference category

	Women			Men		
	Voc. health	Theo. health	Theo. other	Voc. health	Theo. health	Theo. other
Diabetes	0.049***	0.010	-0.059***	0.021***	-0.0067**	-0.040***
	(0.012)	(0.0075)	(0.015)	(0.0051)	(0.0028)	(0.014)
Year of birth						
1962–1965	0.011	-0.027***	-0.11***	0.0053	-0.014***	-0.017
	(0.018)	(0.0091)	(0.024)	(0.0061)	(0.0019)	(0.024)
1966–1970	0.016*	-0.012**	-0.11***	0.0084**	-0.0025	-0.14***
	(0.0097)	(0.0060)	(0.013)	(0.0035)	(0.0030)	(0.012)
Mothers						
Non-Nordic	-0.026	0.0087	0.11**	0.0052	0.0029	0.055
	(0.028)	(0.019)	(0.043)	(0.012)	(0.0081)	(0.040)
Fathers						
Non-Nordic	-0.021	0.027*	0.031	0.0014	0.011	-0.00095
	(0.018)	(0.015)	(0.027)	(0.0072)	(0.0078)	(0.025)
Observations	5952	5952	5952	6100	6100	6100

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Year of birth in 1971–1975 is the reference category.

Non-Nordic predicts failure perfectly and 311 observations are dropped.

We recall from Tables 5–6 that both men and women with diabetes have a lower likelihood of attending university. If this is a sign of lower levels of skills

Table 8: Test with the longest university programs: Logit estimations (average marginal effects) of women's probability of having a university education longer than four years *(Uni4+)* and the probability of having a health-related university education longer than 4 years for those with a university education longer than 4 years *(Health4+)*

	Uni4+		Health4+	
	(1)	(2)	(3)	(4)
Diabetes	-0.027***	-0.019**	0.044	0.045
	(0.0090)	(0.0089)	(0.043)	(0.043)
Upp sec grades		0.13***		0.019
		(0.0066)		(0.029)
Grades missing		0.58***		0.066
		(0.025)		(0.14)
Year of birth				
1962-1965	0.020	0.0059	-0.13	-0.14
	(0.033)	(0.029)	(0.084)	(0.084)
1966-1970	0.0056	-0.00075	-0.062	-0.063
	(0.017)	(0.016)	(0.058)	(0.058)
Non-Nordic	0.027	0.038	0.024	0.031
	(0.039)	(0.038)	(0.12)	(0.12)
Mothers	Yes	Yes	Yes	Yes
Fathers	Yes	Yes	Yes	Yes
Observations	6508	6508	730	730

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.
Year of birth in 1971–1975 is the reference category.
Mothers (Fathers) indicates the full set of controls for mother's (father's) background factors.

and abilities among the diabetes population, we would expect an even larger diabetes impact when looking at the longest and most demanding university courses, such as medical programs, which generally demand top-rated upper secondary grades.[18] However, the results in Tables 8 and 9 indicate that diabetes neither deters nor excludes students from longer programs to any greater extent than shorter programs. On the contrary, the negative diabetes estimates of the likelihood of attending university programs that are four years or longer are lower than the estimates from the main analysis.

Among those attending for four or more years, there is no significant difference in health orientation, although the estimate for women maintains its size,

[18] The mean grade requirement was 4.95 (out of 5) for medical programs in the year 2000 (läkarstudent.se, 2015).

Table 9: Test with the longest university programs: Logit estimations (average marginal effects) of men's probability of having a university education longer than four years *(Uni4+)* and the probability of having a health-related university education longer than 4 years for those with a university education longer than 4 years *(Health4+)*

	Uni4+		Health4+*	
	(1)	(2)	(3)	(4)
Diabetes	-0.024***	-0.016**	0.0025	0.0066
	(0.0078)	(0.0075)	(0.028)	(0.029)
Upp sec grades		0.13***		0.036
		(0.0055)		(0.023)
Grades missing		0.58***		0.50
		(0.028)		(0.31)
Year of birth				
1962-1965	-0.0050	-0.012	-0.072*	-0.072*
	(0.025)	(0.023)	(0.039)	(0.039)
1966-1970	-0.0039	-0.00039	-0.014	-0.016
	(0.014)	(0.013)	(0.046)	(0.045)
Non-Nordic	-0.066***	-0.050*		
	(0.020)	(0.029)		
Mothers	Yes	Yes	Yes	Yes
Fathers	Yes	Yes	Yes	Yes
Observations	6854	6854	654	654

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.
**Non-Nordic* predicts failure perfectly and 3 observations are dropped.
Year of birth in 1971–1975 is the reference category.
Mothers (Fathers) indicates the full set of controls for mother's (father's) background factors.

and the power to detect differences decreases as the samples are smaller. Looking descriptively at average representation between women with and without diabetes on programs to become medical doctors, of those women who have a university education that is four years or longer, 7.76% of the women with diabetes, compared to 7.68% of the controls, study to become doctors, although these differences are not significant.[19]

5.3.2 Age at onset and duration of diabetes

Tables 10 and 11 present results from analyses where the diabetes variable indicates onset at ages 2–9 or 10–15 (controls are still the reference). For upper secondary education (Table 10), the diabetes estimates indicate a stronger association for onset in the older age groups than for onset at ages 2–9, although men with early onset appear to be the ones driving the results for theoretical other. For university education (Table 11), the results for the two onset categories are mixed. Overall, these analyses support our main findings, as both early and late onset appear linked to upper secondary and university outcomes.

6 Discussion

We rejected the hypothesis of no systematic differences in the choice of educational field between people with diabetes onset up to age 15 and population controls. The consistent pro-health orientation was found at all levels of education for women and for vocational health programs for men. These findings support the argument that disease onset early in life may generate experiences and comparative advantages for choosing and completing a health-oriented course of education, both at upper secondary and university level. The results were robust in sub-group analysis and remained when controlling for school grades at the previous level.

The educational patterns of women and men in our control group (see Table 1) mirrors the general patterns for these birth cohorts presented by Statistics Sweden. For instance, national data for people aged 25–54 in 2010 showed that, at the upper secondary level, women are five times more likely than men to take a health-oriented examination and this over-representation is threefold for university degrees (Statistics Sweden, 2010). Given our results, women's pro-health orientation in general is further accentuated among women with diabetes. Register-based data cannot disentangle to what extent this finding depends on stronger preferences for a health-oriented education or comparative advantages in achieving such academic degrees, although both explanations

[19] Tested with t-test. Of those men who have a university education that is four years or longer, 4.35% of the men with diabetes compared to 4.68% of the controls study to become medical doctors.

Table 10: Test with two diabetes onset categories: Multinomial logit estimations (average marginal effects) of the probability of having different fields of upper secondary education using *vocational other* as the reference category

	Women			Men		
	Voc. health	Theo. health	Theo. other	Voc. health	Theo. health	Theo. other
Diabetes						
Onset 2–9	0.050**	0.020	-0.042	0.026*	-0.0034	-0.056**
	(0.025)	(0.014)	(0.026)	(0.014)	(0.0060)	(0.025)
Onset 10–15	0.074***	0.0036	-0.093***	0.030***	-0.0089**	-0.042**
	(0.020)	(0.0099)	(0.020)	(0.011)	(0.0035)	(0.019)
1968–1970	-0.029**	-0.016**	-0.11***	-0.013**	-0.00077	-0.15***
	(0.014)	(0.0066)	(0.015)	(0.0067)	(0.0036)	(0.014)
Mothers						
Non-Nordic	-0.093***	-0.0047	0.082*	-0.021	-0.00068	0.084*
	(0.036)	(0.018)	(0.047)	(0.016)	(0.0081)	(0.043)
Fathers						
Non-Nordic	-0.046*	0.035**	0.040	0.0055	0.014	-0.0025
	(0.025)	(0.017)	(0.030)	(0.014)	(0.0095)	(0.028)
Observations	4897	4897	4897	4993	4993	4993

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Year of birth in 1971–1975 is the reference category.

Non-Nordic predicts failure perfectly and 311 observations are dropped.

Table 11: Test with two diabetes onset categories: Logit estimations (average marginal effects) of the probability of having a university education *(Uni)* and the probability of having undertaken a health-related university program for those with a university education *(Health)*

	Women		Men	
	Uni	Health	Uni	Health*
Diabetes				
Onset 2–9	-0.034	0.095**	-0.069***	-0.031
	(0.025)	(0.037)	(0.023)	(0.021)
Onset 10–15	-0.089***	0.074**	-0.042**	0.021
	(0.019)	(0.030)	(0.017)	(0.021)
1968–1970	0.0016	-0.024	0.00066	0.0063
	(0.025)	(0.033)	(0.023)	(0.024)
Non-Nordic	-0.045	-0.075	-0.074	
	(0.057)	(0.070)	(0.057)	
Mothers	Yes	Yes	Yes	Yes
Fathers	Yes	Yes	Yes	Yes
Observations	5368	2477	5609	1952

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.
Year of birth in 1971–1975 is the reference category.
Mothers (Fathers) indicates the full set of controls for mother's (father's) background factors.
**Non-Nordic* predicts failure perfectly and 34 observations are dropped.

agree with our result. For men with diabetes, the only over-representation was seen for vocational health programs at the upper secondary level, while they were less likely to choose theoretical health programs in upper secondary school. At the university level, men and women alike had a lower likelihood of attending university, but with regard to educational field, men with diabetes did not differ from population controls. We note that the positive association for vocational health appears to be larger for women than for men (0.055, Table 3 compared to 0.031, Table 4). Moreover, there is a negative association for theoretical other that seems stronger for women (-0.063, Table 3 compared to -0.039, Table 4). These negative associations for theoretical other might relate to different interests, or the comparative advantages for a health-oriented education might have come to dominate the theoretical upper secondary programs.

We used choice of educational field and completed health-oriented education as the dependent variable. In terms of returns to education and expected monthly salary from a future job, health-oriented programs thus include education from the whole range of occupations: from low-wage occupations (e.g., nurse assistants and dental assistants), through middle-wage (e.g., physiotherapists and dental hygienist), to high-wage (e.g., physicians and dentists). Consequently, preferences for a future job in the health sector could satisfy a wide range of ambitions in terms of future labor earnings. Moreover, working in the health sector is possible for all levels of educational achievement, spanning from vocational upper secondary programs to long university programs.

We used a clearly defined and well-described health shock, with daily lifelong disease management, as an indicator of health to assess the potential impact on preferences and comparative advantages for educational fields. The burden of disease management may trigger an interest for health-related professions, or deter from such professions as one might wish to separate one's private and professional life. Our empirical results are consistent with both tendencies, as men and women showed heterogeneous responses to disease onset.

Using data matched by municipality of residence in the year of onset and controlling for parental education and measures of maternal ability, we account, in part, for unmeasured attributes of the family and the environment in which people are raised. Comparing specifications with and without these controls indicates a robust relationship between diabetes and choice of educational field. Our diabetes estimates decrease by about one third when we add upper secondary grades to the university attendance analysis, indicating that the diabetes impact is mediated by grades. Lundborg, Nilsson, & Rooth (2014) confirm that men's earnings penalty from diabetes is robust to sibling fixed effects and unobserved factors at the family level, while Lundborg, Nystedt, & Rooth (2014), for instance, show large reductions for a height-earnings association when adding controls for: (1) parental education and earnings; (2) test scores from cognitive ability; or

(3) sibling fixed effects.[20]

In line with the literature on intergenerational transmissions of human capital (Currie, 2009; Cunha & Heckman, 2008; Cunha et al., 2006; Chevalier, 2004; Black & Devereux, 2011), our results indicate that parental level of education is positively linked to own education (see Tables 3–6), with an interesting pattern across the upper secondary fields. To have a university-educated mother is associated with a decreased likelihood of vocational health, but an increased likelihood of theoretical health and theoretical other. This pattern is reversed for mothers who have received only compulsory education: the likelihood is decreased for theoretical health and theoretical other but increased for vocational health. The same patterns exist also for fathers, but their influence is weaker, as shown before by Cunha et al. (2006). We are however not aware of any similar precedents for parents' differing influence for different educational fields. Our results offers yet another example: to have a mother with a health-oriented education appears to influence the child's choice in a similar manner to having a mother with only compulsory education (the likelihood of vocational health is increased, but theoretical health is decreased).

Our choice of health indicator is one of the study's strengths, while it could be argued that inference cannot be made with regard to other less well-defined health shocks with less clear impact on day-to-day activities. Consequently, these findings may not be representative for people with less demanding diseases, such as moderate asthma and allergy. However, milder disease or lower demands on daily management are arguably of less concern regarding potential impact on educational and labor market outcomes.[21]

Earlier work from our group has shown that diabetes has a small but significant impact on school grades (Persson et al., 2013; Dahlquist et al., 2007) and labor market outcomes (Persson et al., 2013, 2014). Recent results from Swedish data on enlistment for military services for male cohorts born 1950–1970 supported this negative association with earnings and employment for men with diabetes (at age 18) (Lundborg, Nilsson, & Rooth, 2014). We contribute to these studies on diabetes, as well as to the more general literature on early life health and adult outcomes (described in, e.g., Currie (2009)), by offering an alternative perspective. This study views experiences of disease not only as burden but also as a potential asset driving comparative advantages; thus, differing choices of educational field might explain why controlling for quantitative measures of schooling have had little impact on health effects in many previous studies, including, for example, Lundborg, Nilsson, & Rooth (2014) for diabetes and Smith (2009) for an overall measure of health.

This study introduces an alternative outcome where ranking in terms of

[20] Their estimated height-earnings premium decreases by about one-sixth when adding parental controls and by one third when adding tests scores or sibling fixed effects.

[21] For example, Lundborg, Nilsson, & Rooth (2014) and Currie et al. (2010) find only a weak or insignificant effect of asthma on adult outcomes.

desirability might not be obvious. For example, our results show that people with diabetes were less likely to have a university education. However, women with a university education were on average 7 percentage points more likely to have a health-oriented degree if they had diabetes. Own experience of disease and its treatment may accordingly translate into valuable assets and insights in working life. Still, the gender-related differences indicate a heterogeneous patient group. Society, the health sector in general, and clinicians in diabetes care should not take for granted that patients share this view. We therefore encourage future research to explore further the mechanisms at play.

Acknowledgments

We thank the Swedish Childhood Diabetes Study Group that developed the Swedish Childhood Diabetes Database sponsored by the Swedish Research Council project no 07531, Västerbotten County Council, The Swedish Diabetes Foundation, the Swedish Council for Working Life and Social Research (FAS) (2009-0768), and Diabetesfonden (2009-065). The Health Economics Program (HEP) at Lund University also receives core funding from FAS (Dnr 2006-1660), the Government Grant for Clinical Research ("ALF") and Region Skåne.

References

Altonji, J. G., & Blank, R. M. (1999). Race and gender in the labor market. *Handbook of Labor Economics, 3*, 3143–3259.

Arcidiacono, P., Hotz, V. J., & Kang, S. (2012). Modeling college major choices using elicited measures of expectations and counterfactuals. *Journal of Econometrics*, *166*(1), 3–16.

Becker, G. S. (1962). Investment in human capital: A theoretical analysis. *Journal of Political Economy*, *70*, 9–49.

Black, S. E., & Devereux, P. J. (2011). Recent developments in intergenerational mobility. *Handbook of Labor Economics*, *4*, 1487–1541.

Broady, D., Andersson, M. B., Börjesson, M., Gustafsson, J., Hultqvist, E., & Palme, M. (2000). Skolan under 1990-talet. *Sociology of Education and Culture Research Reports (Sociala förutsättningar och utbildningsstrategier)*, 5–133.

Card, D. (1999). The causal effect of education on earnings. *Handbook of Labor Economics*, *3*, 1801–1863.

Case, A., Fertig, A., & Paxson, C. (2005). The lasting impact of childhood health and circumstance. *Journal of Health Economics*, *24*(2), 365–389.

Case, A., & Paxson, C. (2008). Stature and status: Height, ability, and labor market outcomes. *Journal of Political Economy*, *116*(3).

Casson, I., Clarke, C., Howard, C., Mc-Kendrick, O., Pennycook, S., Pharoah, P., ... Walkinshaw, S. (1997). Outcomes of pregnancy in insulin dependent diabetic women: results of a five year population cohort study. *BMJ*, *315*, 275–278.

Chevalier, A. (2004). *Parental education and child's education : A natural experiment* (IZA Discussion paper series No. 1153).

Cunha, F., & Heckman, J. (2008). Formulating, identifying and estimating the technology of cognitive and noncognitive skill formation. *Journal of Human Resources*, *43*, 738–782.

Cunha, F., Heckman, J., Lochner, L., & Masterov, D. (2006). Interpreting the evidence on life cycle skill formation. *Handbook of the Economics of Education*, *1*, 697–812.

Currie, J. (2009). Healthy, wealthy, and wise: Socioeconomic status, poor health in childhood, and human capital development. *Journal of Economic Literature*, *47*, 87–122.

Currie, J., & Almond, D. (2011). Human capital development before age five. *Handbook of Labor Economics*, *4*, 1315–1486.

Currie, J., Stabile, M., Manivong, P., & Roos, L. L. (2010). Child health and young adult outcomes. *Journal of Human Resources*, *45*(3), 517–548.

Dahlquist, G., Blom, L., Tuvemo, T., Nyström, L., Sandström, A., & Wall, S. (1989). The Swedish childhood diabetes study—results from a nine year case register and a one year case-referent study indicating that type 1 (insulin-dependent) diabetes mellitus is associated with both type 2 (non-insulin-dependent) diabetes mellitus and autoimmune disorders. *Diabetologia*, *32*(1), 2–6.

Dahlquist, G., Gustavsson, K. H., Holmgren, G., Hägglöf, B., Larsson, Y., Nilsson, K. O., … Wall, S. (1982). The incidence of diabetes mellitus in Swedish children 0–14 years of age. A prospective study 1977–1980. *Acta Paediatrica Scandinavica*, *71*(1), 7–14.

Dahlquist, G., Källén, B., & Swedish Childhood Diabetes Study Group. (2007). School performance in children with type 1 diabetes–a population-based register study. *Diabetologia*, *50*(5), 957–64.

Dahlquist, G., & Mustonen, L. (2000). Analysis of 20 years of prospective registration of childhood onset diabetes time trends and birth cohort effects. Swedish Childhood Diabetes Study group. *Acta Paediatrica*, *89*(10), 1231–1237.

Daneman, D. (2006). Type 1 diabetes. *The Lancet*, *367*(9513), 847–858.

Gemici, A., & Wiswall, M. (2014). Evolution of gender differences in post-secondary human capital investments: College majors. *International Economic Review*, *55*(1), 23–56.

Grossman, M. (1972a). The demand for health: a theoretical and empirical investigation. In (Vol. 119). Columbia University Press for the National Bureau of Economic Research, Inc.

Grossman, M. (1972b). On the concept of health capital and the demand for health. *Journal of Political Economy*, *80*, 223–255.

Heckman, J. (2007). The economics, technology, and neuroscience of human capability formation. *Proceedings of the National Academy of Sciences*, *104*(33), 13250–13255.

Heckman, J., Stixrud, J., & Urzua, S. (2006). The Effects of Cognitive and Noncognitive Abilities on Labor Market Outcomes and Social Behavior. *Journal of Labor Economics*, *24*(3), 411–482.

Jonasson, J. M., Brismar, K., Sparén, P., Lambe, M., Nyrén, O., Östenson, C.-G., & Ye, W. (2007). Fertility in women with type 1 diabetes: A population-based cohort study in Sweden. *Diabetes Care*, *30*(9), 2271–2276.

Karvonen, M., Viik-Kajander, M., Moltchanova, E., Libman, I., LaPorte, R., & Tuomilehto, J. (2000). Incidence of childhood type 1 diabetes worldwide. Diabetes Mondiale (DiaMond) project group. *Diabetes Care*, *23*(10), 1516–1526.

läkarstudent.se. (2015). `http://xn--lkarstudent-l8a.se/gamla_kvoter.html`. (11 Mars 2015)

Lee, D. (2005). An estimable dynamic general equilibrium model of work, schooling, and occupational choice*. *International Economic Review*, *46*(1), 1–34.

Lundborg, P., Nilsson, A., & Rooth, D.-O. (2014). Adolescent health and adult labor market outcomes. *Journal of Health Economics*, *37*, 25–40.

Lundborg, P., Nystedt, P., & Rooth, D.-O. (2014). Height and earnings: The role of cognitive and noncognitive skills. *Journal of Human Resources*, *49*(1), 141–166.

Minor, T. (2011). The effect of diabetes on female labor force decisions: new evidence from the national health interview survey. *Health Economics*, *20*(12), 1468–1486.

Montmarquette, C., Cannings, K., & Mahseredjian, S. (2002). How do young people choose college majors? *Economics of Education Review*, *21*(6), 543–556.

Nyström, L., Dahlquist, G., Rewers, M., & Wall, S. (1990). The Swedish childhood diabetes study. an analysis of the temporal variation in diabetes incidence 1978–1987. *International Journal of Epidemiology*, *19*(1), 141–146.

Paglin, M., & Rufolo, A. M. (1990). Heterogeneous human capital, occupational choice, and male-female earnings differences. *Journal of Labor Economics*, 123–144.

Persson, S., Dahlquist, G., Gerdtham, U., & Steen Carlsson, K. (2013). Impact of childhood-onset type 1 diabetes on schooling: a population-based register study. *Diabetologia*, *56*(6), 1254–1262.

Persson, S., Dahlquist, G., Gerdtham, U., & Steen Carlsson, K. (2014). *Childhood health and labor market outcomes in the case of type 1 diabetes* (Tech. Rep.).

Salm, M., & Schunk, D. (2012). The relationship between child health, developmental gaps, and parental education: Evidence from administrative data. *Journal of the European Economic Association*, *10*(6), 1425–1449.

Smith, J. P. (2009). The impact of childhood health on adult labor market outcomes. *The Review of Economics and Statistics*, *91*(3), 478–489.

Sparud-Lundin, C., Öhrn, I., & Danielson, E. (2010). Redefining relationships and identity in young adults with type 1 diabetes. *Journal of Advanced Nursing*, *66*(1), 128–138.

Statistics Sweden. (2000). *Swedish standard classification of education, SUN 2000*. http://www.scb.se/en_/Finding-statistics/Publishing-calendar/Show-detailed-information/?PublObjId=2061. (11 Mars 2015)

Statistics Sweden. (2006). *Evaluation of the Swedish register of education*. http://www.scb.se/Pages/PublishingCalendarViewInfo___259924.aspx?PublObjId=3299. (3 October 2012)

Statistics Sweden. (2008). *Higher education. students and graduated students in undergraduate education 2006/07*. http://www.scb.se/statistik/UF/UF0205/2008A01/UF0205_2008A01_SM_UF20SM0801.pdf. (20 Februari 2014)

Statistics Sweden. (2009). *Multi-generation register*. www.scb.se/Pages/List____257501. (3 October 2012)

Statistics Sweden. (2010). *Online statistical database. educational attainment of the population*. http://www.scb.se. (6 April 2015)

Statistics Sweden. (2011a). *The labour force surveys 50 years. A presentation of four different perspectives from independent researchers*. http://www.scb.se/statistik/_publikationer/AM0401_2011A01_BR_AM76BR1103.pdf. (11 Mars 2015)

Statistics Sweden. (2011b). *Longitudinal integration database for health insurance and labour market studies (LISA by Swedish acronym).* http://www.scb.se/Pages/List____257743.aspx. (6 June 2012)

Statistics Sweden. (2014). *Women and men in Sweden 2014, facts and figures.* http://www.scb.se/Statistik/_Publikationer/LE0201_2013B14_BR_X10BR1401.pdf. (19 April 2015)

Steen Carlsson, K., Landin-Olsson, M., Nyström, L., Arnqvist, H. J., Bolinder, J., Östman, J., & Gudbjörnsdottir, S. (2010). Long-term detrimental consequences of the onset of type 1 diabetes on annual earnings-evidence from annual registry data in 1990–2005. *Diabetologia*, *53*(6), 1084–92.

The Swedish National Agency for Education. (1995). *Statistics for upper secondary educations.* http://www.skolverket.se/statistik-och-utvardering/statistik-i-tabeller/gymnasieskola/skolor-och-elever/skolor-och-elever-i-gymnasieskolan-lasar-1994-95-1.29631. (11 Mars 2015)

Wennick, A., Lundqvist, A., & Hallström, I. (2009). Everyday experience of families three years after diagnosis of type 1 diabetes in children: A research paper. *Journal of Pediatric Nursing*, *24*(3), 222–230.

Appendices

A The SUN-classification system

List of educational programs in the SUN-classification system:

1. Universal educational programs at upper secondary level preparing for higher education, including natural science, and humanistic and social science
2. Pedagogy and pedagogical work
3. Art and culture
4. Social science, law, commerce, administration
5. Natural science, mathematics, computers and network technology
6. Engineering and manufacturing
7. Natural resources and forestry
8. Health, medical, and social care
9. Services
10. Other

Vocational health includes programs from item 8 (health, medical, and social care).

1. Vocational health
   (a) health and medicine
   (b) dental care
   (c) child care
   (d) social work
   (e) care and treatment
2. Vocational health restrictive
   (a) health and medicine
   (b) dental care

Theoretical health includes programs from item 1 (universal educational programs) if they are followed by a university-level program from item 8 (health, medical, and social care).

B Tests of the IIA assumption

Ho: Odds (Outcome-J vs Outcome-K) are independent of other alternatives.

Table B.1: Suest-based Hausman tests of the IIA assumption

Result for women				
Omitted	chi2	df	p>chi2	evidence
Voc. health	5.363	12	0.945	for Ho
Theo. health	15.256	12	0.228	for Ho
Theo. other	6.701	12	0.877	for Ho
Result for men				
Omitted	chi2	df	p>chi2	evidence
Voc. health	3.864	12	0.986	for Ho
Theo. health	5.834	12	0.924	for Ho
Theo. other	5.766	12	0.927	for Ho

C Nested logit models

Table C.1: Nested logit estimations (average marginal effects) of the probability of having different fields of upper secondary education using *vocational other* as the reference category.(Controlling for year of birth and (own and parental) non-Nordic-origin.)

Result for women		
Outcome	Diabetes AME	Std. Dev.
voc. health	0.064	0.016
theo. health	0.005	0.004
theo. other	-0.061	0.007
Result for men		
Outcome	Diabetes AME	Std. Dev.
voc. health	0.031	0.006
theo. health	-0.007	0.002
theo. other	-0.039	0.007