Special Needs Children and Sibling Spillover Effects: Evidence from the National Longitudinal Study of Youth

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Abstract

This paper investigates the spillover effects of siblings growing up with a brother or sister diagnosed with a chronic disease. I focus on the effect of having a sibling diagnosed with low birth weight on outcomes measuring educational attainment and achievement. The empirical analysis draws on data from the 'NLSY79 Child and Youth Survey' and I find evidence of a significant negative effect on the likelihood of attending college at a magnitude of around 12 percentage points. The estimated effects on educational achievement are more ambiguous and non-robust. However, the lions' share of the point estimates is in line with what is found in previous comparable research.

Keywords: Siblings, Peer effects, Parental Investment, NLSY

1 Introduction

Siblings growing up in the same family have a very special relationship and share not only parents' genes and DNA, but also environment, socioeconomic situation and political context. As they grow up together, the ways in which siblings influence each other are many. These impacts and influences can be summarized within a phenomenon referred to as a *peer effect*. In the presence of such peer effects, public policies affecting one sibling may also influence other siblings within the same family. For example, a care taker or assistant that is hired to take care of a disabled sibling when parents are away, might help out with cleaning, cooking or other home related activities, thereby also servicing a non-disabled sibling. If forming a policy where the cost of hiring assistants caring for disabled children is publicly financed, studying sibling spillovers within the family becomes important from a public policy perspective. Failing to account for the potential positive spillover that an assistant might have on a healthy sibling means that the overall effect of the policy will be underestimated, in turn leading to distortions in policy cost-benefit analysis.

Despite the potential importance of sibling spillovers, there is relatively little evidence on them. Possibly because studying these spillovers is challenging. Impact from one sibling to another goes in both directions and it is furthermore hard to disentangle between what is caused by a shared childhood and what is due to a shared genetic heritage. As such, the peer effects between brothers and sisters are hard to investigate scientifically. Since the relationship between brothers and sisters is potentially one of the most long-lasting interpersonal relationships a person will ever have, the existence and nature of peer effects between siblings may be extra relevant from a policy perspective. As brothers and sisters may keep in touch for all their lives, sibling spillover effects may have substantial consequences even if they would be small if exposed to during a short time span. Research wise, not many articles has been published which examines the sibling spillover effects of disabled children. Black et al. [2017] utilize registry data of all children born in Florida between 1994 and 2002, and in Denmark between 1990 and 2001. By looking at families with three or more children, of which the third (or higher order) child has a disability. They exploit differences between the older non-disabled siblings to estimate the effect of being exposed to a disabled sibling on educational outcomes. Relying on the assumption that the oldest sibling has been exposed to the disabled brother or sister for a lower share of his/her lifetime compared to the second oldest, thereby being less exposed compared to his/her younger brother/sister. The estimated results reveals a difference in standardized test scores of about 0.11 standard deviations

in favor of the first-born child compared to the second-born.

In this thesis, I investigate whether or not the health status of a child does have a casual impact on the educational outcomes of his/her siblings. More specifically, my research question is defined as follows: *Are the educational outcomes of a certain individual causally affected by the fact that he or she has been growing up with a disabled sibling?*. The outcome variables I utilize are GPA at last year of high school and college attendance rate. Hence, I am able to capture both educational achievement and attainment, if even at a somewhat crude level. I focus on the case of growing up with younger sibling with a low birth weight (LBW), defined as less than 2,500 grams. LBW children have an significantly higher risk for disabilities such as attention-deficit/hyperactivity disorder, other psychiatric disorders and problems, low IQ, and poor neurocognitive functioning [Whitaker et al., 1997, Johnson and Breslau, 2000]. Studies have found that LBW children have around three times a higher risk of developing a learning disability [Johnson and Breslau, 2000]. This definition of a special needs child follows partly the analysis of Black et al. [2017], as the authors use this definition in one of their specifications.

The terms 'special needs' and 'disability' will be used synonymously throughout this paper regardless of any medical distinction between the two concepts. My intention is to use a language that is neutral and non-stigmatized while still being simple and clear.

My empirical analysis draws on data from the 'National Longitudinal Survey of Youth - Child and Young Adults' Survey, which consists of individual level data of 11,521 children born to the mothers of the nationally representative sample of Americans within the original 'National Longitudinal Survey of Youth' starting in 1979. I utilize a propensity score matching strategy and match siblings that have an equal propensity of being treated with an LBW sibling, but differing in observed treatment status. Thereby hopefully reducing the amount of bias in my estimates. I find a positive and significant result that healthy siblings of LBW children have a reduced probability of attending college at around 12 percentage points. However, I find no clear evidence that sibling spillovers from special needs siblings have any impact on high school GPA. The potential causes behind this difference between outcome variables are discussed further in section 6.

As mentioned, not many papers are published within this subject, meaning that further insights regarding the spillover effects from disabilities ought to be a relatively relevant and pioneering. Especially considering the impact that such insights might have on future policy designs and cost benefit analyzes. Apart from Black et al. [2017], only a few relevant articles exist. Breining [2014] utilize the same type of registry data as Black and coauthors to estimate the sibling spillover effects from ADHD and find

a negative and significant effect that sibling ADHD decreases upper secondary GPA with about 12 percentage points. Parman [2015] investigates sibling spillover effects from children carried in utero during the 1918 influenza pandemic, and finds that health shock tended to reinforce rather than compensate for differences in endowments across children. Nicoletti and Rabe [2014] perform a similar analysis on British registry data and find a small negative spillover effect from disadvantaged siblings.

This paper contributes to the existing literature in two main ways. First, I utilize a matching strategy to control for potentially unobservable characteristics, an empirical strategy that has never before been utilized within this context. Secondly, while most other studies have focused on registry data from small countries or states, I use survey data representing the whole U.S. population.

The remainder of this paper is structured as follows: Section 2 presents a theoretic framework explaining the potential link between sibling disabilities and parental investment. From this theory, a research hypothesis is stated which then serves as an outset for the empirical analysis that follows. Section 3 provides an account of the dataset and describes the imposed restrictions in detail. The empirical framework is then outlined in section 4, followed by a presentation of the estimated results in section 5. Section 6 discuss the validity of these results. Finally, section 7 concludes.

2 Theory

As mentioned earlier, the peer effects between siblings of the same family might work through a variety of different mechanisms and channels which interacts between siblings in both directions. The complex nature of these peer effects in turn implicates that any attempt to formulate and explain the effects of sibling spillovers within a theoretical framework, is likely to fall short. To be able to hold a discussion throughout the paper which is consistent and tangible, I limit my theoretical account to a framework regarding parental investment. Specifically, the theory presented in this section considers the aspect of how the relative health level between siblings of the same family might affect the investment decision of the parents of these children, in deciding how much parental investment/care will be devoted to each individual child. The focus of this account is likely just one individual piece in the various ways that siblings interact and affect each other, which one should bear in mind throughout the remainder of this section.

A theoretical framework regarding how the health status of siblings affects investment decisions of their parents is presented by [Becker and Tomes, 1976]. Consider the following utility function, explaining household utility as a function of the number of children

(n), individual child qualities (w_i) and the aggregated amount of other commodities (y):

$$U = U(y, w_1, ..., w_n) \tag{1}$$

where the quality of the ith child w_i can be stated as a function of (1), the individual initial endowment (e_i).

$$w_i = e_i + q_i. \tag{2}$$

The endowment (e_i) consists of pre-conditions at birth, such as IQ or propensity to develop a certain type of musculature. And the household contribution towards child i (q_i). An example of a household contribution could be that one of the parents spend playing football with the child, thereby increasing the child's level of health.

The household's budget constraint becomes

$$p_y y + \sum_{i=1}^{n} p_{q_i} q_i = I, \tag{3}$$

where I is the household's total income, p_y is the price of one unit y and p_{q_i} is the average price of increasing the quality of the ith child (q_i) one unit.
Maximizing equations 1 and 2, subject to the constraint in equation 3, yields the following solution:

$$\frac{\partial U}{\partial w_i} \Big/ \frac{\partial U}{\partial w_j} = p_{q_i}/p_{q_j} \tag{4}$$

Or rearranged to a more common notation:

$$\frac{\partial U}{\partial w_i} \Big/ p_{q_i} = \frac{\partial U}{\partial w_j} \Big/ p_{q_j} \tag{5}$$

Consequently, if p_{q_1} and p_{q_2} are the identical for siblings i and j, parents would aim to equalize w_i and w_j, so that there is no difference in quality between siblings. In other words, if initial endowment e differs between siblings within the same household (e.g. $e_i < e_j$) parents will adjust the quality investment in each child accordingly so that $e_i + q_i = e_j + q_j$. Thus, $e_i < e_j$ implies $q_i > q_j$.

On the other hand, assuming that prices of quality differ between siblings, parents would invest relatively more in siblings with a comparably low p_{q_i}, as the expected return for an equally big investment is higher for a lower p_{q_i}. If, as Becker and Tomes [1976] suggest, a lower endowment is negatively correlated with the price of q_i, it is

not necessarily the case that parents will make up for an initially lower endowment by increasing the parental investment towards that child. Quite contrary, such scenario would lead to a situation where lower endowed children will be even worse of, as the low endowment also reduces the amount of parental support.

In this context, three potential situations arise regarding the peer effects between different endowed siblings, where one of them is lesser endowed due to, for example, a limiting chronic condition. Firstly, parents may adjust their level of parental investment so that the resulting individual quality is equalized between siblings. If so, a non-disabled child will receive less parental investment if having a disabled sibling, compared to a situation where all siblings are healthy, ceteris paribus. Secondly, if the price of quality is negatively correlated with initial endowment, a child will receive higher levels of parental investment if living in a family with a disabled siblings compared to if not. However, this second prediction holds only if the initial endowment of the lesser endowed child is big enough to fulfill $e_i \geq t$. If, on the other hand, $e_i < t$, parents are required to devote a certain amount of investment (q_i) towards the disabled child i. Consequently, the disposable amount of investment available to the non-disabled sibling j (q_j) is reduced. Depending on the level of investment required by sibling i ($t - e_i$) the non-disabled sibling might be worse of, irrespective of whether $p_{q_i} > p_{q_j}$ or not.

3 Data and Descriptive Statistics

Since the occurrence of disabilities and various special needs is relatively rare in relation to the general population, a big challenge when investigating this type of question empirically is to find sufficient and suitable data on which such analysis can be based. There exist a variety of datasets which contain information on disabled individuals themselves, collected from hospital registers as part of the medical therapy directed towards these individuals. However, such data does seldom include data of the eventual siblings of these individuals, which makes them non-suitable for the research question I investigate here. Moreover, the majority of the population studies available for economic research at an individual level consists of unrelated individuals drawn from some kind of random sample. Consequently, such datasets also lack the required information to analyze peer effects among siblings.

Perhaps, the most intuitive type of data suitable for answering my research question would be national registry data of entire populations; as is used by Black et al. [2017]. Provided that the examined population is large enough and that the information from health registers is detailed enough, population registers should contain a large enough

sample of disabled/special needs individuals, as well as the information required to link these individuals to their siblings and family characteristics. However, the possibility to acquire such data is very limited at my level of academia, due to cost and time saving reasons.

In light of this, I turn to the openly accessible National Longitudinal Survey of Youth 1979 - Children and Young Adults (NLSY79CYA),[1] a survey containing information of a total of 11,521 children, born by the mothers of the original National Longitudinal Survey of Youth (NLSY79); in turn carried out for the first time in 1979. The original NLSY79 was a multi-purpose panel study of a U.S. nationally representative sample of men and women that all were between 14-21 years of age on December 21, 1978. The NLYS79 survey consists of interviews of over 12,000 people of which 6,283 were women.

The Children and Young Adults Survey consists of two separate part of assessments aimed towards different periods in the respondent's life cycle. The child survey, which has been carried out every other year since [...] is answered by the mother of the respondent and concerns different aspects of early life such as pre- and postnatal living conditions, family environment during early childhood etc. Starting from 1994, an additional youth survey has been carried out to all children from age 15 or older. This sub survey is answered by the youth itself and concerns aspects of adolescence such as high school grades, personal impressions, health status etc. Additionally, since this survey follows the respondents also after they move out from home, some information exists also on adulthood, such as employment status and income. As the most part of children are born during the late 80's and early 90's, they have entered the labor market just recently. Consequently, the access and quality of such information within the data set is, as of today, relatively sparse. Moreover, the youth assessment contains quite a large share of missing values, which imposes yet another threat to a dataset which, as discussed before, suffers from a limited amount of usable data.[2] In the light of this, I restrict my analysis to include mostly variables from the child assessments. The main outcome variables of choice are measures of educational attainment and achievement, which are yielded from the youth survey. Choosing outcome variables from the youth survey is a necessary restriction, as my research question is focused on the sibling spillovers to later life outcomes such as education. [3]

[1] See NLSYCYA [1979-2014]

[2] One potential way to address this problem is to replace missing values with imputed values. However, as the process of calculating such imputes relies on strong assumptions, such techniques have been excluded in order to limit the scope of this paper.

[3] Even though the NLSYCYA contains a panel aspect, where the same individuals are followed over time, I construct my dataset so that it uses as much information as possible from the different individuals in the sample, while still treating them as part of a cross sectional dataset. The reason behind this is

Using data from the NLSYCYA, I link observations at an individual level so that individual and family characteristics of one individual is combined with information concerning the health of his/her siblings. The linking of siblings is conducted using the information and algorithms presented in Rodgers et al. [2016], and the process of linking siblings together has been one of the most work intensive parts in writing this thesis.

I will now continue this section with a more detailed account of the actual variables used within the analysis, together with an explanation on why choosing these.The variables used within the scope of my empirical analysis can be divided into three different groups: Treatment, outcome and control variables.

3.1 Treatment Variables

As for the treatment variable, the natural variable one might think of in view of my research question would be an indicator of whether or not an individual has a sibling with special needs. After which such an individual would be considered treated. To my knowledge, there exist no consensual definition of what is to be regarded as a special need, and the severity of the same type of diagnose may vary across individuals. Moreover, disabilities are rare, and early attempts of limiting my analysis to focus only on one specific diagnose, has proven not to be a successful strategy. The amount of treated individuals in such estimation strategy is simply to few to provide enough variation for obtaining reliable estimates. To address this problem, I adopt a rather broad measure of the definition of a 'special needs' child. Following the same procedure as in one of the specifications in Black et al. [2017], I define a child as having special needs if he/she had a weight at birth lower than 5.5 pounds (2,495 grams). The use of this specific threshold relies on two aspects. Firstly, it does come close to the official definition of low birth weight (LBW) from the Wold Health Organization [WHO, 1992], thereby corresponding to the LBW threshold of 2,500 grams that is used in Black et al. [2017]. Moreover, the NLSYCYA does not contain information on exact birth weight, but whether or not the surveyed individuals had a birth weight over or under 5.5 pounds. Consequently, the resulting treatment variable is a dummy taking value one if an individual has a sibling diagnosed with LBW and zero otherwise.

The use of a LWB threshold as the underlying treatment variable somewhat solves the problem of achieving a uniform 'special needs' definition, that is still broad enough to be represented in the relatively small sample. The share of children diagnosed with LBW within the NLSYCYA, constitutes 9.2 percent of the original sample. Other studies

that the definition of a special needs child that I use (low birth weight) is determined at birth and does not vary over time.

have shown that around 6.1 percent of the the total 3.8 million U.S. births during 2011 were diagnosed with LBW [Kowlessar et al., 2013]. This makes LBW a relatively common diagnose, which is especially important concerning that the available number of observations is relatively limited.

Furthermore, as an LBW diagnose is closely related to inhibited growth and cognitive development, as well as to chronic diseases later in life, LBW should serve as a suitable indicator variable for having 'special needs' [Stevens-Simon and Orleans, 1999]. However, the use of LBW as an indicator variable does also comes with a set of problems. Firstly, the health outcomes that arise from a LBW diagnose may vary substantially between individuals and the impact that such repercussions might have on the siblings of these individuals therefore likely also differ. Consequently, the implications of the estimates yielded from using this variable are describing an overall relationship rather than a detailed description of the impact of a specific health state at an individual family level, somewhat limiting the possibilities to draw tangible conclusions and make concrete policy implications. Moreover, the risk of a LBW diagnosis is greatly affected by inheritance and siblings within the same family are therefore often all diagnosed with LBW. As this in turn means that the treatment is likely not random, plain OLS estimates are very likely suffering from bias. For example, an empirical specification that explains the impact of having a sibling with LBW on educational achievement, without taking into account that the individual itself might suffer from LBW, could lead to an over-estimated effect that is caused by the bad health state of the individual herself rather than the health state of her sibling. I address this problem by excluding observations with an LBW diagnosis from my final sample, thereby ensuring that I only focus on individuals with a normal health state, but that have LBW diagnosed siblings.

3.2 Outcome Variables

There are two different outcome variables in the analysis. College attendance and average high school grades during last year of high school (GPA). Three main reasons exists behind the choice of these. Firstly, as I investigate the same main research question as in Black et al. [2017], the choice of GPA as an outcome variable makes my findings somewhat comparable to those of Black et al. Secondly, the availability of reliable outcome variables within the NLSY79CYA is, as mentioned, rather limited. The fact that these outcomes are only available for respondents who have finished high school, and that this information is provided within the scope of the Youth survey, creates a natural limitation of the data so that only children aged 15 or older are included in the

final sample. Consequently, as the latest available wave was carried out during 2014, the sample is limited to include respondents born 1999 or earlier. Thirdly, the two outcomes can be seen as measures of educational *attainment* and *achievement* respectively. By focusing on these two measures, I capture two different aspects of education that are common within microeconomic, educational research.

3.3 Control Variables

In addition to the treatment and outcome variables I include a set of control variables of family and individual characteristics. Namely year of birth, month of birth, gender, race (black, hispanic or neither of the former), maternal education in years and urban/rural region of residence. These all serve to capture such factors that may have a causal impact on both the outcome and treatment variables, and the exclusion of these would hence result in bias in any estimates of the causal relationship between treatment and outcome.

3.4 Summary Statistics

Table 1 presents summary statistics for all variables mentioned above as well as for some previously unmentioned control variables. After dropping observations with missing information for any of the control variables, 4,007 of the original 11,521 observations remain. The variables within this table are defined as follows.

'Sibling LBW' is a dummy taking value one if the respondent has a younger sibling diagnosed with LBW , and zero otherwise. Since the event of getting a special needs child may impact the decision getting additional children, siblings younger that the oldest diagnosed child are excluded from the sample. 'College Attendance' is a dummy taking value one if the respondent has ever attended college and zero otherwise. 'Age' denotes the age in years of the respondent at the time of the most recent answered survey. 'High School GPA' is the average grade at the last year of high school on an 12 grade scale, normalized into mean zero and standard deviation one.[4] 'Year and Month of Birth' denotes the birth date of the individuals in the sample. 'Female' is a dummy taking value one if the respondent is a female and zero otherwise. 'Black' and 'Hispanic' are dummy variables indicating the ethnic origin of respondents, 'Maternal Education' is the education level of the mother measured in years. 'Urban' is a dummy taking value

[4]Contrary to the corresponding variable in the original dataset, where a lower value indicates a better score, this variable is transformed, so that a higher number indicates a better score.

one if the individual was raised[5] in an urban region of residence, and zero if he/she was raised in a rural region.

Table 1: Summary Statistics

Variable	Mean	Std. Dev.	Min.	Max.	N
Sib. LBW	0.052	0.223	0	1	4,007
College Attendance	0.536	0.499	0	1	4,007
GPA	0.062	0.984	-3.89	1.307	3,831
Year of Birth	1986.227	5.809	1970	1999	4,007
Month of Birth	6.621	3.404	1	12	4,007
Female	0.487	0.5	0	1	4,007
Black	0.497	0.5	0	1	4,007
Hispanic	0.309	0.462	0	1	4,007
Maternal Education	13.39	2.485	3	20	4,007
Urban	0.737	0.44	0	1	4,007

4 Methodology

The empirical framework consists of two parts. Firstly, plain OLS regressions with suitable controls are estimated. Secondly, this is followed by a matching procedure that aims to address potential bias in the OLS estimates.

The baseline OLS regression formula looks as follows:

$$O_i = \alpha + \beta d_{jc} + \gamma s_i + \theta f_{ij} + \epsilon_{cij} \tag{6}$$

Where the outcome variable of interest for individual i (O_i) is determined by a dummy of whether or not i's sibling j is diagnosed with LBW. Hence, d_{jc} is a dummy taking value one if is sibling j suffers from LBW and zero otherwise. Additional individual characteristics for i are denoted by the vector s_i which contains information on the month of birth fixed effects, the year of birth and the sex of i.[6] as well as a vector of family specific variables, namely race, maternal education level and region of residence, that are shared by i and j, f_{ij}. ϵ_{cij} denotes the remaining error term.

[5]This variable is calculated so that it takes the value of the first reported value, it does therefore not take into account if an individual moved to another region after that point in time. Assuming that movements occur randomly and that families with special needs children does not move/ not move in a pattern different to other families, this will not give rise to bias the results.

[6]The reason behind including month of bith as levels while year of birth is continuous comes down to the limited number of observations in the sample. When including year of birth in levels, e.g. as year fixed effects, the matrix of the coefficient estimates becomes rank deficient, so that no estimates can be calculated.

This specification relies on the assumption that having a disabled sibling, or being disabled oneself, is determined exogenously, which might not hold. Many chronic conditions are determined both by nature and nurture and as siblings typically share both genes and environment, the validity of this model specification can be questioned. As mentioned in section 3, the risk of LWB is substantially affected by inherited factors and genetics, together with the fact that siblings, by construction, share such inherited factors, means that siblings of disabled children might not be comparable to those that only have non-disabled siblings. In more specific terms, this means that treatment and controls are not necessarily comparable, giving rise to bias in any estimates failing to control for such aspects.

To further address this problem of non-comparable treatment and control groups, I conduct a matching procedure where treated and non-treated individuals are matched on a set of characteristics affecting the likelihood of being treated with a sibling having special needs. The idea behind this strategy being that treated and non-treated individuals that has the same underlying probability of being treated, are more comparable to each other, thereby more suitable as treatment and controls to one another.

I present three different types of matching estimates which all relies on the same estimated probabilities of being treated. Individuals are matched to one another on basis of all the characteristics described as controls in section 3. Specifically, a logistic regression is carried out which explains the probability of treatment as a function of these characteristics, as described by the following equation:

$$Y_{cij} = log(\frac{P_{cij}}{1 - P_{cij}}) = \gamma z_i + \theta g_{ij} + \eta_{cij} \tag{7}$$

Where the probability that i has a sibling j affected by LBW ,P_{ij}, is determined by a vector of individual characteristics of i (z_i) and shared family characteristics of both i and j. The variables within these two vectors follows the same methodology as the linear estimation described in equation 6. The error term is denoted as η_{cij}.

After estimating probabilities P_{cij} for each individual in the sample, I execute a variety of matching algorithms that matches individuals that have an equal probability of being treated, but differs in actual treatment status. Three different types of matching algorithms are used in this part of the analysis, namely nearest neighbor, radius and kernel matching. All three types aims to achieve a sample of matched, comparable groups of treatment and control groups but varies in their definition of which observations that are seen as suitable matches and which are not.

Nearest neighbor matching is a traditional pairwise matching procedure in which

each individual in the treatment group are matched to an individual in the control group which has an estimated propensity of being treated that is as close as possible to the corresponding value for the treated individual. This method provides a narrow definition of matched samples, which are likely to be comparable. However, it falls short in the sense that it fails to utilize all available data in the sample. If, as is the case here, the amount of observations differs between the treatment and control groups, the data that is available for unmatched individuals remains unused. This, in turn, means that nearest neighbor matching is not a very effective strategy in the context of this paper.

Radius matching is more effective than nearest neighbor in the sense that it reuses observations from the group with less observations and match individuals together as long as their estimated propensity of treatment lies within a certain threshold. Therefore, radius matching takes grater advantage of all available data and is hence a relatively more effective matching strategy. However, radius that is too broad suffers from the same problems as the unmatched sample, and on the other hand, a radius that is extremely narrow eventually corresponds to a nearest neighbor match.

Finally, kernel matching creates a non-real matched individual, for each observation in the treatment group, which consists of a weighted average of all individuals in the control group. Consequently, kernel matching is an intelligent use of the available data that tries to simulate observations that are as comparable as possible, based on all the available information in the original data, thereby making it efficient. However, this strategy relies more heavily on a correct specification of matching variables, as they are used not only to calculate propensity scores, but also to create simulated individuals.

Assuming that the estimated probabilities are sufficiently accurate, the matched pairs of treated and non-treated individuals should be more comparable to treatment and controls in a randomized experiment. Consequently, OLS estimates based from the matched sample should theoretically suffer from less bias compared to the original, non-matched sample.

The structural form of the linear regressions estimated within the frame of the matching process follows the same structural form as the estimations described by equation 6, except the fact that it excludes the additional controls used for estimating the propensity of treatment.

One big drawback that arise from applying this matching procedure on data of disabled children is that disabilities in general are relatively rare. Only about five percent of the individuals in my sample has a younger sibling who's defined as having special needs. As matching compares one treated individual to one non-treated, the resulting sample sizes from matching are comparably small. Small sample sizes means big stress

of the data when calculating estimates. Consequently, there is an increased risk of data mining and limited room for conducting robustness checks.

A final remark connects to the description of the empirical framework and concerns the choice on which variables that are included as controls, as well as for calculating the probabilities used for matching. It is always possible to include more control variables, providing a more detailed picture of different pathways which might interact as a common factor related to both the health status of a sibling and educational outcomes for an individual. One example being maternal education which is likely to have a positive relationship with both the health level of ones siblings as well as with the own level of education. A potential extension of the analysis that I conduct would be to include information on a more detailed set of individual characteristics such as personal health status, number of siblings, order of birth etc. However, I choose to limit my analysis due to a reason that is related to the nature of the matching framework. Early attempts that included such variables simply failed to achieved a sample of matched individuals that was balanced enough to make treatment and controls comparable to one another. In other words, inclusion of such variables together with the limited amount of observations created a setup in which the matching algorithms failed to find comparable observations, thereby failing to calculate any estimates of the impact of having a special needs sibling on educational outcomes.

5 Results

This section is divided into three subsections, each devoted to different parts of the empirical framework. The first section presents the results from the OLS estimates, the second one concerns the results and diagnostics of the matching strategy. Section 5.3 provides a set of estimates that investigates heterogeneity and robustness of previously presented results.

5.1 OLS Results

The results from carrying out the OLS regression described in equation 6, are presented in table 2. Three different specifications, differing in the number of included controls, are presented for each of the two outcome variables respectively.

Looking at college attendance, the estimates suggests that there is indeed a negative and significant effect of having a special needs sibling on the likelihood of attending college, e.g. educational attainment. The magnitude of the estimated decrease in likelihood

Table 2: OLS - Estimates

	College Attendance			High School GPA		
	(1)	(2)	(3)	(4)	(5)	(6)
Sib. LBW	-0.194	-0.203	-0.120	-0.0844	-0.173	-0.0484
	(0.0339)	(0.0328)	(0.0320)	(0.0713)	(0.0683)	(0.0690)
Constant	0.546	0.826	0.275	0.0659	0.409	-0.157
	(0.00808)	(0.0400)	(0.0583)	(0.0163)	(0.0852)	(0.126)
Observations	4,007	4,007	4,007	3,831	3,831	3,831
R-squared	0.008	0.125	0.188	0.000	0.099	0.136
Birth date Controls		X	X		X	X
Gender Control		X	X		X	X
Race Controls			X			X
Family Char.			X			X

Robust standard errors in parentheses

Note: Birth date indicates the inclusion of month of birth fixed effects, and year of birth. Gender and race denotes dummies for sex and ethnic origin. Family characteristics indicates the inclusion of maternal education and urban/rural region of residence.

varies at around 12 to 20 percentage points depending on specification and the estimated impact decreases substantially with the inclusion of family characteristic variables, e.g. urban/rural region of residence and maternal level of education. This illustrates the previous discussion regarding maternal education as a common factor related both to the health status of children, as well as to their likelihood of attending higher education. Consequently, it is likely that the higher magnitude of the estimates excluding maternal education as a regressors are likely biased downwards. My belief is that the third specification, with the highest amount of included controls, is most likely to reflect an estimate close to the true value.

Moreover, the estimated effect of being treated with a disabled sibling becomes more ambiguous when looking at high school GPA as the outcome variable. Estimates vary substantially between specifications and I only find significant evidence of a negative effect in the fifth specification of the table. The standard errors are generally large compared to the point estimates and in the fifth specification the standard error is even larger than the point estimate, decreasing the validity of the estimated results for high school GPA. However, the previous indication that the exclusion of maternal education might impose a downwards bias to the estimates, remains also in the context of this

outcome. Note that the non-significant results does not necessarily imply that there is no impact of having an LBW sibling on high school GPA, only that the uncertainty within the estimates are too large to draw any conclusions regarding the size and direction of the impact. Specifically, 95 percent confidence interval varies at a range from -0.307 to +0.868 depending on specification.

5.2 Propensity Score Matching Results

Table 3: Difference in Means

Variable	Treated	Untreated	Difference	P-value of T-test	
College Attendace	0.3896	0.5285	-0.1388	2.2510E-07	***
High School GPA	-0.0492	0.0055	-0.0547	0.3268	
Year of Birth	1987.5	1985.5	1.9384	3.8302E-12	***
Month of Birth	6.4904	6.6118	-0.1214	0.4537	
Female	0.5245	0.4831	0.0415	0.0804	*
Black	0.3497	0.5688	-0.2191	1.1717E-20	***
Hispanic	0.4222	0.2423	0.1799	2.0648E-18	***
Maternal Education	12.4612	13.4565	-0.9953	1.1442E-08	***
Urban	0.7836	0.7457	0.0379	0.1057	

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table 3 shows descriptive statistics comparable to those in table 1, separated by groups of treatment and controls, e.g. treated and untreated individuals, Together with a two sample T-test of the difference in means between the groups. Results from the T-test indicates that the to groups are different with respect to numerous of the observable characteristics. Thus, the test gives some support to the previously discussed concern, that treated and untreated individuals are different in aspects that are unrelated to the birth weight of their siblings, thereby making them unsuitable as comparisons within the context of the research question at hand. In view of the results in table 3, a matching procedure might serve as a suitable way of achieving comparable groups and, in turn, more reliable estimates.

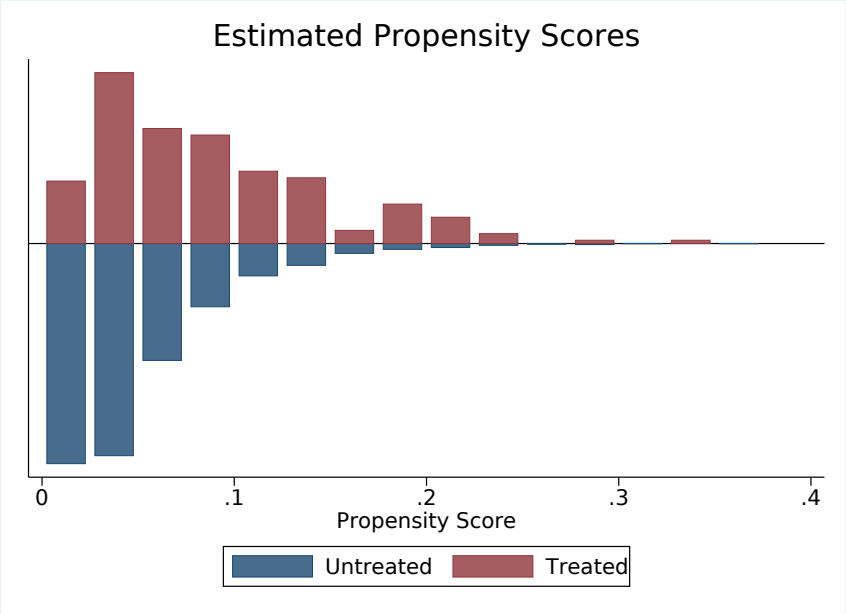

Figure 1: Histogram of estimated propensity scores, separated by groups of untreated and treated observations

Figure 1 shows the the distribution of estimated propensities of treatment, after carrying out the regression described by equation 7. The figure illustrates the region of common support between untreated and treated individuals and illustrates two important aspects regarding the success rate of the matching. First and foremost, range of the region of common support is relatively equal between the two groups, which increases the likelihood of finding comparable individuals. Secondly, there is a clear pattern within the figure that higher propensity scores are more common within the group of actually treated individuals, giving some support that the regressors used to estimate the propensities are actually able to sufficiently estimate a realistic probability of treatment. However, the estimated probabilities are in general quite small, which can be caused by two things. Either, the estimation strategy behind the propensity scores are not able to achieve correct and reliable propensity score estimates, which would threaten the validity of future estimates that build on these. Or, it could simply be a sign of the fact that the occurrence LBW is not easily predictable and can also be caused by unknown, random factors. In which case it does not threaten the validity of the estimation strategy.

Table 4: Difference in Means - Propensity Score Matching

	Unmatch.	Mean			%reduct	T-test	
Variable	Matched	Treated	Control	%bias	—bias—	t	P-value
College Att.	U	0.3897	0.5285	-28.1		-5.18	0
	M	0.3524	0.4667	-23.1	17.7	-2.39	0.017
High School GPA	U	-0.0492	0.0055	-5.5		-0.98	0.327
	M	-0.0185	-0.0414	2.3	58.2	0.24	0.813
Year of Birth	U	1987.5	1985.5	34.2		6.95	0
	M	1988.2	1987.7	8	76.7	0.87	0.387
Month of Birth	U	6.4904	6.6118	-3.5		-0.75	0.454
	M	6.2381	5.9429	8.6	-143.2	0.87	0.385
Female	U	0.5245	0.4831	8.3		1.75	0.08
	M	0.4857	0.4857	0	100	0	1
Black	U	0.3497	0.5688	-45		-9.34	0
	M	0.2667	0.2476	3.9	91.3	0.45	0.656
Hispanic	U	0.4222	0.2423	38.9		8.77	0
	M	0.5095	0.5571	-10.3	73.5	-0.98	0.329
Mat. Education	U	12.4610	13.4570	-39.7		-5.72	0
	M	12.5190	12.5290	-0.4	99	-0.04	0.966
Urban	U	0.7836	0.7457	8.9		1.62	0.106
	M	0.7524	0.7810	-6.7	24.6	-0.69	0.49

* if variance ratio outside [0.83; 1.20] for U and [0.76; 1.31] for M

After matching individuals using either nearest neighbor, radius or kernel matching, the difference in means between untreated and treated individuals is no longer significant in the matched sample. Table 4 presents diagnostics and T-tests of difference in means for the unmatched and matched sample respectively, when the matched sample is constructed using nearest neighbor matching.[7] As shown in the table, the is no significant difference in means between the treatment and control groups after matching. Thus proving that the resulting sample is indeed balanced. The only exception is college attendance, which is still significantly different between treatment groups also in the matched sample. This is not an indication of a failed matching procedure, but rather a preliminary result showing that the likelihood of attending college is indeed lower individuals with a sibling that has special needs, even after treatment groups are matched. The same holds for high school GPA, as the other outcome variable. However, the difference in means is not significant for this outcome. Additionally, the table shows that there is a substantial reduction in potential bias in the matched compared to the unmatched sample, further indicating that the matching strategy has been successful.

Tables 5 and 6 show the result of the estimated impact of having a sibling with special needs, for each outcome variable respectively. The estimates draws on data from the resulting matched sample of each matching strategy. As these samples should be comparable with respect to the matching variables, all specifications presented in these tables are estimated without control variables. For radius, four specifications are presented in each table. These specifications differ only in the width of the radius that is considered an acceptable match. Hence, a radius match with a radius of 0.001 matches untreated and treated individuals with a difference in estimated propensity score less than, or equal to 0.001. Each table preset estimates of average treatment effect on the treated (ATT), average treatment effect on the untreated (ATU) and average treatment effect on the matched sample as a whole (ATE). As the research question at hand focuses on the effect on actually treated individuals, the most relevant measure is the ATT.

Beginning with table 5, the impact of an LBW sibling on the likelihood of attending college, is negative and significant at a five percent level in all specifications. The estimates are robust at around -0.12 percentage points, irrespective of matching algorithm. This, together with the fact that the estimated effect is rather close to the full specifications, 3 and 6, of table 2, gives some degree of validity to the estimates. One exception is the 0.1 caliper radius matching, which estimates a magnitude of the impact of -0.1580 percentage points, substantially lower than the corresponding numbers of the

[7] The corresponding tables for radius and kernel matching specifications give the same overall picture as those shown in table 4, but are excluded here for space saving reasons.

other specifications. The reason behind this stems on what I mention in section 4; the greater the radius, the closer the matched sample gets to the original unmatched sample. Consequently, the same type of downward bias present in the most simple specifications of the OLS estimations, is present also here. The overall picture given by the results in table 5 is that an LBW diagnose of a child does indeed have spillover effects to his/her siblings, with a decreased likelihood of attending college at a magnitude of around 12 ± 7 percentage points.[8] Provided that the matching strategy is correctly specified, these estimates should theoretically provide evidence of a causal effect on college attendance from sibling spillovers.

Table 5: Matching Estimates - College Attendance

	Nearest	Radius				Kernel
	Neighbor	0.1	0.01	0.001	0.0001	
ATT	-0.1151	-0.1580	-0.1021	-0.1197	-0.1199	-0.1234
	(0.0459)	(0.0342)	(0.0353)	(0.0368)	(0.0431)	(0.0346)
ATU	-0.0695	-0.1637	-0.1210	-0.0878	-0.1156	-0.1537
ATE	-0.0747	-0.1634	-0.1200	-0.0897	-0.1162	-0.1521
Treated Observations	210	210	210	210	210	210
Unreated Observations	3,797	3,797	3,797	3,797	3,793	3,793
Total Observations	4,007	4,007	4,007	4,007	4,007	4,007

Robust standard errors in parenthesis

Continuing with the corresponding impact on high school GPA, the results shown in table 6 more or less gives the same overall picture as is given in table 2. The estimated ATT is relatively small at around 2-3 percentages of a standard deviation, specifications 2 and 5 being exceptions that I believe are caused by the broad and narrow calipers respectively. (Matching using a radius too small, will aggravate the process of finding suitable matches, thereby broadening the standard errors.) However, none of the point estimates are positive, which makes my results somewhat comparable to those of Black et al. [2017].

[8] $point\ estimate \cdot S.E \approx 0.12 \pm 0.36 \cdot 1.96 \approx 0.12 \pm 0.7$

Table 6: Matching Estimates - High School GPA

	Nearest	Radius				Kernel
	Neighbor	0.1	0.01	0.001	0.0001	
ATT	-0.0170	-0.0723	-0.0200	-0.0291	0.0000	0.0286
	(0.1006)	(0.0718)	(0.0739)	(0.0783)	(0.0925)	(0.0728)
ATU	0.0754	-0.1091	-0.0840	-0.0446	-0.0297	-0.0973
ATE	0.0706	-0.1072	-0.0807	-0.0437	-0.0252	-0.0937
Treated Observations	195	195	195	195	195	195
Untreated Observations	3,636	3,636	3,636	3,636	3,636	3,636
Total Observations	3,831	3,831	3,831	3,831	3,831	3,831

Robust standard errors in parenthesis

5.3 Heterogeneity and Robustness Checks

The relatively small sample of reliable data within the NLSYCYA, together with the fact that special needs children are not very common in the general population, naturally give rise to the limitation that the estimates that I present in the previous part of this section, are relatively crude. Being born with a birth weight lower that 5.5 pounds does not necessarily imply that one might be a special needs child. Even so, the variety of needs that arise from having a low birth weight at first, might vary substantially between children. To further investigate the implications of certain specific needs, as well as providing some robustness to earlier findings, I extend my original analysis by introducing an alternative measure of disability/ special need. Specifically, I look at the effect of having a sibling diagnosed with one of the following three health limitations: Type 1 diabetes, orthopedic impairment and mental retardation.

The reason behind choosing these three disorders is that they give arise to different types of symptoms, and thereby different parental challenges. Together, they will hopefully account for a representative variety of challenges faced by the parents of these children and the difference in estimates across outcome variables provides insights that illuminates different aspects of the theoretical framework presented in section 2. Furthermore, the importance of inheritance as an underlying cause of these conditions is believed to be generally small[9], meaning that the likelihood of being assigned to a dis-

[9] The discussion regarding the impact of environment versus genetics is heavily debated in medicine. As this paper is focused towards economics, I do not wish to discuss the validity of findings presented

abled sibling is more likely to be considered random. This latter aspects is especially important for causal inference of the estimates.

As the number of siblings affected by any of these health limitations are even more rare than being born with a low birth weight, estimates calculated from these subsamples of observations are suffering even more from the limited amount of observations, resulting in large standard errors and potentially low point estimates. Moreover, the amount of treated individuals makes this hard to execute a matching strategy similar to the one in section 5.2, why only plain OLS estimates are calculated. Consequently, the results given by these alternative specifications should be seen as complements to earlier results rather than conclusive evidence by itself.

in medical papers, even less take part in such debate. Further information on diabetes type 1 is given in Atkinson and Eisenbarth [2001], McDermott and F [2002], Mehers and Gillespie [2008]. Medical information on the importance of genetics in mental retardation in Moser [2004], Chelly et al. [2006]. Finally, medical information on orthopedic impairment is given found in Reddihough and Collins [2003], MacLennan et al. [2015]. The conclusive picture given by these sources is that genetics is at least partly defining the risk of being affected by any of the three conditions. However, these genetic factors are only explaining a small part of the risk and the reminding part is unknown.

Table 7: Alternative Treatment Variables - OLS Estimates

	College Attendance			High School GPA		
	(1)	(2)	(3)	(4)	(5)	(6)
Sib.Diabetes	-0.0706	-0.0484	0.00858	0.153	0.0187	0.113
	(0.122)	(0.115)	(0.109)	(0.278)	(0.282)	(0.282)
Sib. Orth. Imp.	-0.121	-0.149	-0.125	-0.0906	-0.138	-0.114
	(0.0514)	(0.0518)	(0.0496)	(0.107)	(0.110)	(0.109)
Sib. Mental. Ret.	-0.252	-0.218	-0.134	0.0821	-0.0149	0.0846
	(0.0697)	(0.0683)	(0.0604)	(0.172)	(0.157)	(0.160)
Constant	0.523	0.803	0.256	0.0639	0.380	-0.133
	(0.00729)	(0.0364)	(0.0522)	(0.0147)	(0.0766)	(0.114)
Observations	4,824	4,824	4,824	4,610	4,610	4,610
R-squared	0.004	0.124	0.195	0.000	0.094	0.136
Birthdate Controls		X	X		X	X
Gender Control		X	X		X	X
Race Controls			X			X
Family Char.			X			X

Robust standard errors in parentheses

Table 7 shows result comparable to those described by equation 6 and table 2, differing only in the definition of special needs. As expected, the estimated standard errors are very large throughout the table. Consequently, the estimates does not give much room to any useful conclusions. The overall picture given by the results presented in this section points in the same direction as what has been seen earlier, thereby strengthening the validity of my findings. For orthopedic impairment and mental retardation, estimates of the treatment effect are negative and significant in all specifications regarding college attendance. When excluding family characteristics from the regression, the magnitude of the estimates increase in the same way as is seen in the previously presented results. The full specification of column three, shows results that are in line with previous findings, further strengthening the validity of these. Interestingly, The effect of a sibling with diabetes on college attendance seems to be low or even close to zero in the full specification. Connecting this result to the theoretical framework of section 2, one potential explanation of this result lies in the nature of the symptoms of diabetes, and

how these symptoms relate to initial endowment and price of investment. The medical treatment of diabetes, typically through the injection of insulin with a syringe at specific times during the day, is effective but rather time consuming. Consequently, the parents of a child with diabetes are required to put a lot of effort and investment in order to maintain the well being of this child. However, as treatment is effective, children with diabetes can live a rather normal life. As the child grows, she does also learn to take care of the disease by herself. This is not the case for the other two health conditions, as they are typically not treated equally effective. In terms of the theoretical framework, these aspects might show themselves in a way where diabetes only affects the initial endowment of a child. Whereas orthopedic impairment and mental retardation, which cannot be cured effectively, does also affect the price of quality, for parental investments directed towards such child. Thus affecting the investment decision of the parents to a larger extent.

Regarding high school attendance, I likewise find no significant evidence of the type of spillover effects that I investigate. Hence, the results of this alternative specification are similar to the previous findings also in this respect. The main conclusion is obvious; the results are robust even when estimates draws on data from this limited specification.

6 Discussion

The conclusive picture of my results is clear; I find evidence of a potentially causal relationship stating that siblings of disabled children are facing a spillover effect which makes them less likely to attend college. More general, sibling spillover effects of health have a negative impact on educational *attainment* to at least some degree. However, my results are not reliable enough to express anything about the impact on educational *attainment*. There may be many potential causes behind this difference in findings between outcome variables. One could be that my model is misspecified in some way, another that the amount of available data is to limited to find a significant effect where there really is one. However, one potential cause could be that there are financial motives behind the attendance effect, e.g. that the educational performance of siblings to LBW children is not affected, but that the disability itself leads to increased costs for the family. In turn meaning that they cannot afford to pay for a college education to the same extent. I do not have any evidence for such channel, but it could in theory explain my results. I strongly encourage for future research to investigate this matter more closely.

Moreover, my results are neither strengthening nor contradicting the findings pre-

sented in Black et al. [2017]. Nevertheless, my point estimates regarding the spillover effects of a disabled sibling on the average high school grades lies at around 0.03 standard deviations, thereby being quite close to the corresponding estimate of 0.048 standards deviations found in Black et al. [2017]. This is promising, as it somewhat increases the validity of both my own findings, as well as those presented in Black et al. [2017]. Furthermore, it gives some support that my estimation strategy, being different to most previous research investigating this matter, is successful. Consequently, it is likely that my estimates of the special needs sibling impact on *attainment* are indeed close to a true, causal relationship.

Relating my findings to the implications of the theory described in section 2, the most likely way in which parents adjust their investment decision, as a response of getting a disabled child, is that they try to minimize the difference in quality between siblings rather than trying to maximize total sum of quality of all children. As in the latter case, a lesser endowed child with a higher price price of quality would lead to a situation where healthy siblings of this child are actually better off compared to a situation where the endowment and return to investment are equal among siblings. However, the validity of my results as an empirical proof of the legitimacy of the Becker and Tomes [1976] model should not be overrated, as my findings to not identify through which channels the sibling spillover effects actually works. Instead, my results only illustrates the externalities that disabilities impose on other individuals than those actually diagnosed; externalities that might have been overlooked in political decision making and calculations of the societal and individual costs that are caused by various disabilities.

That said, there are several limitations and drawback of my analysis that might have affected the results. I will here discuss three such that I identify as being most relevant.

First of all, a limitation of the strategy, only remarked briefly, is the relatively few amount of included controls and matching variables. There is an imminent risk that the observable characteristics are simply not detailed enough to sufficiently reduce the amount of noise in the estimates, which might lead to the inadequate conclusion of a causal effect even if this is not actually the case. Numerous variables, all available within the NLSYCYA, could provide a more detailed picture which in turn might lead to a more accurate match of observations. As mentioned, inclusion of additional controls caused a situation where the matching algorithms failed to achieve a balanced sample. This could simply be an effect of the limited original sample size, in which case the exclusion of such variables do not necessarily threatens the validity of the yielded results. However, an alternate explanation of the matching failure that occurs when these variables are included, is that they constitute a common factor through which sibling health status

and educational outcomes interacts. Specifically, if this underlying factor determines both treatment and outcome, it is, by construction, impossible to observe to individuals which match with respect to this underlying factor, but differ in terms of their observed treatment status. Consequently, any specification that omits this factor suffers from a bias caused by misspecification. An example of a such variable is the number of siblings within each family. Even if the occurrence of a disability would be determined randomly, the likelihood of having a sibling with such disability increases with the number of siblings within the family. Simultaneously, the average parental investment available to each individual child also decrease with the number of siblings. It is therefore possible that the estimates presented in section 5.2 are reflecting a non-causal relationship between the specific treatment and outcome variables at hand. I have been able to test the presence of some of such factors, among them number of siblings, by including them as regressors within the framework of the plain OLS estimates (not presented here for space saving reasons) Since these specific factors did not substantially affected the estimate of the coefficient of interest, it is unlikely that the exclusion of these specific variables threaten the validity of my results. However the discussion above illustrates an important concept; since I cannot test for all potential pathways, there is still a risk that I have overlooked some important aspect of the mechanisms behind the relationship at hand.

Moreover, my analysis somewhat falls short in the sense that it fails to control for the actual time that siblings share together. It is unlikely that the disability of a specific child is equally influential regardless of the age difference between siblings. Quite the opposite, it is probably realistic to assume that the age difference between siblings is an important determinant of how much influence siblings will have on each other. Therefore, it would make sense to include the age difference between siblings as a measure of the length of exposure to the treatment. Again, the reason for not doing so comes back to the limited amount of observations within the data. Trying to estimate a treatment effect measured in terms of years of exposure would induce a stress to the data which would heavily increase the risk of data mining and reduce the level of external validity in the results.

Concerning the external validity of my results, it is likely to assume that they share the same type of characteristics as other empirical investigations drawn on similar types of data. Thus, it is likely to assume that impacts of similar magnitudes should be found in comparable populations of the western world, with a relatively easy access to advanced health care and effective therapies. Especially if seen in the light of the corresponding estimates in Black et al. [2017] show that there is no substantial difference between estimates from U.S. underlying data, with a heavily privatized health sector,

and those drawn from the danish registers, with the danish health care system that builds on taxes and public funds. However, my results may not necessarily be valid in the context of a developing country, where access to health care is limited and the nature of certain disabilities might cause even larger challenges for children, siblings and families living in more rural parts of the world. Thus, there is a possibility that the sibling spillovers of disabilities might impose even greater externalities in such economies. Thereby increasing the relevance and importance of research within this field.

7 Conclusion

The clear and robust indication of a sibling spillover effect of health status on educational attainment illustrates an important aspect of the social cost of disabilities that has typically been overlooked. Consequently, the policy implication to be drawn from the evidence provided by this paper is that such sibling spillovers should be included in any type of calculation aiming to calculate the social benefits and costs of health limitations and disabilities with accuracy.

Even if the focus of my empirical analysis does not explicitly focus on the impact that special needs children might have on the decision making of parental investment, it is possible to understand some of the mechanisms behind my results by modeling the different aspects of such an economic decision. Naturally, I strongly encourage for further research to look into these mechanism, how they work, which of them that are more relevant than others and naturally, how one could design policies that internalizes these sibling spillover externalities in an effective way.

Furthermore, as the empirical framework that I outline in this paper is limited heavily by the amount of available data, there is a big room for applying the same type of matching procedure on bigger and more accurate samples. This type of data is demonstrably available in registers of entire populations. Hence, such elaborations of my work should be feasible.

Last, but not least, the stress and uncertainty that is experienced by many of the parents responsible for raising a disabled child calls for the need of a deeper understanding of how disabilities affect not only disabled people themselves, but also those that are close to him or her. It is only in the light of increased knowledge that we are able to give support to these families, thereby potentially avoiding unnecessary feelings of distress and impotence. Thereby contributing, if even just a little, in making the world a better place to live in.

References

Mark A Atkinson and George S Eisenbarth. Type 1 diabetes: New perspectives on disease pathogenesis and treatment. In *Lancet*, volume 358, pages 221–229, jul 2001. doi: 10.1016/S0140-6736(01)05415-0. URL http://linkinghub.elsevier.com/retrieve/pii/S0140673601054150.

Gary S. Becker and Nigel Tomes. Child Endowments and the Quantity and Quality of Children. *Journal of Political Economy*, 84(4, Part 2):S143–S162, 1976. ISSN 0022-3808. doi: 10.1086/260536. URL http://www.jstor.org.ludwig.lub.lu.se/stable/1831106http://www.journals.uchicago.edu/doi/10.1086/260536.

Sandra E. Black, Sanni Breining, David N. Figlio, Jonathan Guryan, Krzysztof Karbownik, Helena Skyt Nielsen, Jeffrey Roth, and Marianne Simonsen. Sibling Spillovers. *NBER Working Paper Series*, 23062, 2017. URL http://www.nber.org/papers/w23062.pdf.

Sanni Nørgaard Breining. The presence of ADHD: Spillovers between siblings. *Economics Letters*, 124(3):469–473, 2014. ISSN 01651765. doi: 10.1016/j.econlet.2014.07.010. URL www.elsevier.com/locate/ecolet.

Jamel Chelly, Malik Khelfaoui, Fiona Francis, Beldjord Chérif, and Thierry Bienvenu. Genetics and pathophysiology of mental retardation. *European Journal of Human Genetics*, 14(6):701–713, jun 2006. ISSN 1018-4813. doi: 10.1038/sj.ejhg.5201595. URL http://www.nature.com/doifinder/10.1038/sj.ejhg.5201595.

Eric O Johnson and Naomi Breslau. Increased risk of learning disabilities in low birth weight boys at age 11 years. *Biological Psychiatry*, 47(6):490 – 500, 2000. URL http://www.sciencedirect.com/science/article/pii/S0006322399002231.

Niranjana M Kowlessar, D Ph, H Joanna Jiang, D Ph, and Claudia Steiner. STATISTICAL BRIEF # 163 Hospital Stays for Newborns , 2011. *Healthcare Cost and Utilization Project (HCUP) Statistical Briefs*, pages 1–12, 2013. URL http://www.ncbi.nlm.nih.gov/pubmed/24308074.

Alastair H. MacLennan, Suzanna C. Thompson, and Jozef Gecz. Cerebral palsy: Causes, pathways, and the role of genetic variants, dec 2015. ISSN 10976868. URL http://linkinghub.elsevier.com/retrieve/pii/S0002937815005104.

F Pociot McDermott and M F. Genetics of type 1 diabetes mellitus. *Genes and Immunity*, 3:235–259, feb 2002. doi: 10.1038. URL https://www.nature.com/gene/journal/v3/n5/full/6363875a.html.

Kay L. Mehers and Kathleen M. Gillespie. The genetic basis for type 1 diabetes, dec 2008. ISSN 00071420. URL https://academic.oup.com/bmb/article-lookup/doi/10.1093/bmb/ldn045.

Hugo W. Moser. Genetic causes of mental retardation. In *Annals of the New York Academy of Sciences*, volume 1038, pages 44–48, dec 2004. doi: 10.1196/annals.1315.010. URL http://www.ncbi.nlm.nih.gov/pubmed/15838096http://doi.wiley.com/10.1196/annals.1315.010.

Cheti Nicoletti and Birgitta Rabe. Sibling spillover effects in school achievement. 2014.

NLSYCYA. Children of the NLSY79, 1979-2014. Bureau of Labor Statistics, U.S. Department of Labor and National Institute for Child Health and Human Development. Produced and distributed by the Center for Human Resource Research, The Ohio State University. Columbus, OH:2017.

John Parman. Childhood health and sibling outcomes: Nurture reinforcing nature during the 1918 influenza pandemic. *Explorations in Economic History*, 58:22–43, 2015.

Dinah S Reddihough and Kevin J Collins. The epidemiology and causes of cerebral palsy. *Australian Journal of Physiotherapy*, 49(1):7–12, 2003. ISSN 0004-9514. doi: 10.1111/j.1469-8749.2005.tb01195.x. URL http://linkinghub.elsevier.com/retrieve/pii/S0004951414601835.

Joseph Lee Rodgers, William H. Beasley, David E. Bard, Kelly M. Meredith, Michael D. Hunter, Amber B. Johnson, Maury Buster, Chengchang Li, Kim O. May, S. Mason Garrison, Warren B. Miller, Edwin van den Oord, and David C. Rowe. The NLSY Kinship Links: Using the NLSY79 and NLSY-Children Data to Conduct Genetically-Informed and Family-Oriented Research, 2016. ISSN 15733297.

Catherine Stevens-Simon and Miriam Orleans. Low-birthweight prevention programs: The enigma of failure. *Birth*, 26(3):184–191, sep 1999. ISSN 07307659. doi: 10.1046/j.1523-536x.1999.00184.x. URL http://doi.wiley.com/10.1046/j.1523-536x.1999.00184.x.

Agnes H Whitaker, Ronan Van Rossem, Judith F Feldman, Irvin Sam Schonfeld, Jennifer A Pinto-Martin, Carolyn Torre, David Shaffer, and Nigel Paneth. Psychiatrie

outcomes in low-birth-weight children at age 6 years: Relation to neonatal cranial ultrasound abnormalities. *Archives of general psychiatry*, 54(9):847–856, 1997.

WHO. P07 - Disorders related to short gestation and low birth weight, 1992. URL `http://apps.who.int/classifications/icd10/browse/2010/en{#}/P07`.