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Mortality and Morbidity Risks and Economic Behavior

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Abstract

There are theoretical reasons to expect that high risk of mortality or morbidity during young adulthood decreases investment in human capital. However, investigation of this hypothesis is complicated by a variety of empirical challenges, including difficulties in inferring causation due to omitted variables and reverse causation. For example, to compare two groups with substantially different mortality rates, one typically has to use samples from different countries or time periods, making it difficult to control for other relevant variables. Reverse causation is important because human capital investment can affect mortality and morbidity. To counter these problems, we collected data on human capital investments, fertility decisions, and other economic choices of people at risk for Huntington's disease. Huntington's disease is a fatal genetic disorder that introduces a large and exogenous risk of early mortality and morbidity. We find a strong negative relation between mortality and morbidity risks and human capital investment.

Keywords

Health; Mortality; Human Capital

Introduction

High risk of mortality or morbidity during young adulthood decreases the likelihood that an investment in human capital made during childhood and youth will produce positive returns. As a result, there are theoretical reasons to expect that high mortality and/or morbidity during young adulthood will decrease investment in human capital. However, empirical investigation of this hypothesis is complicated by various problems. These include difficulties in inferring causation, omitted variables, and data problems. For example, if one wants to compare two groups with substantially different mortality rates, one typically has to use samples from different countries or time periods, making it difficult to control for other relevant variables. Inferring a causal effect of health on human capital investment based on observed associations between mortality or morbidity and human capital investment is complicated because people with high levels of human capital tend to have higher incomes and demand higher levels of healthcare, affecting the risk of mortality and morbidity. In order to counter these problems in studying the effect of mortality and morbidity risk on human capital investment, we studied human capital investments of people at risk for

Conflicts of Interest and Research Approval

We declare we have no conflicts of interest. The research project was approved by the appropriate committees in both the University of Chicago and Rush Hospital.

Huntington's disease, a fatal genetic disorder that introduces a large and exogenous risk of early mortality and morbidity.

Huntington's disease is a progressive disorder of motor, cognitive and psychiatric disturbances. The age of onset of symptoms is usually 30 to 45 years. Huntington's disease slowly diminishes the affected individual's ability to walk, think, talk and reason. In the early stages of Huntington's disease, cognitive ability and mobility may be affected. Early symptoms include depression, mood swings, forgetfulness, clumsiness, involuntary twitching and lack of coordination. As the disease progresses, concentration and short-term memory diminish and involuntary movements of the head, trunk and limbs increase. Walking, speaking and swallowing abilities deteriorate. Eventually the person is unable to care for themself. Death follows from complications such as choking, infection or heart failure, typically 15 to 18 years after onset. There is presently no effective treatment or cure.

Huntington's disease is an inherited disorder that affects males and females equally and crosses all ethnic and racial boundaries. A child of a person with Huntington's disease has a 50% probability of inheriting the fatal gene.[1] Everyone who carries the gene will eventually develop the disease. However, because symptoms can develop over a range of ages, people who are at risk due to family history become less likely to be carrying the gene the longer they have lived without showing symptoms.[2]

In 1993, the Huntington's disease gene was isolated and a direct genetic test was developed. The test can accurately determine whether a person carries the Huntington's disease gene, but it cannot predict when symptoms will begin. However, more than ninety percent of individuals who are at risk elect not to be tested.

Because disability usually follows soon after the onset of symptoms, people who are at risk for Huntington's disease have a substantially lower expected rate of return on human capital investments. The higher risks of early mortality and morbidity could also affect investments in health, fertility decisions, risk taking behaviors, and other economic decisions.

In order to examine the relation between mortality and morbidity risks and economic choices empirically, one of us (AS) interviewed people who are (or were) at risk for Huntington's disease. Questions focused on various economic choices such as educational attainment, fertility, and risk taking behaviors. Sixty five interviews were conducted. Out of the sixty five subjects, fifty six were recruited in Rush Hospital, located in Chicago, Illinois. The nine remaining subjects were recruited from an email list related to Huntington's disease. All subjects signed consent forms approved by the University of Chicago Institutional Review Board. Fifty-six interviews were conducted face-to-face, and nine completed over the phone. A typical interview took about thirty minutes. Some of the subjects interviewed at Rush Hospital were already in the early stages of Huntington's disease. Therefore, every person interviewed at Rush Hospital was screened by a neurologist to confirm no substantial cognitive damage that could compromise either the data or that person's ability to sign consent forms. Subjects came to the hospital for various reasons: monitor symptoms, inquire about genetic testing, get prescriptions for symptoms, receive information about filing for disability, or escort affected family members to their appointments.

There are several reasons why studying the economic choices of people who are at risk for Huntington's disease is useful in identifying the effects of morbidity and mortality risks on

[1]This is correct only if just one parent is affected, which is almost always the case due to the rarity of the disease.
[2]For example, the probability that an individual who had 50% risk at birth is carrying the gene is 49.6% at age 20 if no symptoms are present, but only 31.5% at age 50.

economic choices. First, some people who are at risk for Huntington's disease learn of their risk status at an early age, while others only learn of their risk status much later in life, usually upon diagnosis. For economic choices such as the level of educational attainment, the variation in the age at which people first learn of their risk status creates a natural control group, as people who learn about their risk status early in life are able to adjust their educational attainment fully, while people who learn about their risk status much later are not able to adjust. Therefore, a comparison of these two groups could identify the effects of mortality and morbidity risks on economic choices.

Second, self-selection is not a major concern. Since Huntington's disease is purely genetic, individuals cannot select their risk status.[3] This is an important advantage of the sample, as self selection is a common complication in the empirical human capital literature. [4] Third, the additional risk of early mortality and morbidity for individuals who are at risk for Huntington's disease is approximately fifty percentage points, a dramatic increase which is much larger than the possible effect of human capital investments on early mortality and morbidity risks. As a result, confounding of investment decisions by other causes of health is not a major concern, because any changes in mortality or morbidity risks that are a result of the individual's choices will be small compared to the changes in morbidity and mortality risks due to risk for Huntington's disease. Fourth, the age of becoming aware of the risk is often early enough to alter some long-term economic decisions. If the age of onset were very late in life, its effect on choices would be minimal. On the other hand, an early onset of Huntington's disease can result in other problems, such as cognitive changes that decrease the rate of return to schooling, or physical limitations that significantly increase the full cost of attending college. This possibility will have to be taken into account. Fifth, the accuracy of the genetic test is very high – more than ninety-nine percent. Everyone who carries the gene will eventually develop the disease and treatment options are extremely limited. Furthermore, there is no way to delay onset or alter the disease's progression. Therefore, there is not the need to adjust the analysis for the possibility that people will change their behavior to affect the probability, severity, or date of onset of the disease.[5] Finally, Huntington's disease is sufficiently common compared to many other genetic diseases that it is practical to obtain a large enough sample for empirical analysis.

Theoretical basis for effects of morbidity and mortality risk on educational attainment

Background and prior research

Mortality and morbidity risks have been long recognized as a factor in human capital investments. In *Human Capital* (Becker 1993), Becker discusses the idea that decreases in mortality and morbidity rates raise the rate of return to human capital investments[6] (because there is a longer time period to collect the returns) and thereby encourage human capital accumulation. Since investment in education is considered an important factor in economic growth,[7] it is important to measure how sensitive the investment in education is to mortality and morbidity risks. Currently, however, there is no consensus in the literature on the sensitivity of human capital investments to such risks. For example, Preston (1980) provides

[3]People could select into the "tested negative" or "tested positive" groups, but only since 1993. In addition, less then 10% of people at risk choose to take the genetic test. Finally, the test is almost always taken years after the studied human capital investments were made.

[4]For example, a common complication in estimating the returns to college education is that people who select to go to college may earn more because they are more talented and not only because of their college degree.

[5]If this were not true, one would have to worry about possibilities like people at risk getting more education because they think that "training the brain" may delay onset.

[6]This idea depends on the assumption that decreased productivity which results from the increase in the labor force is not too large.

[7]See for example the theoretical work of Becker, Murphy, and Tamura (1990) or the empirical work of Barro (1989).

some calculations regarding the effect of mortality decline in Mexico from 1921 to 1966 on the internal rate of return from completing high school (conditional on reaching age fifteen), and finds that the change in the internal rate of return is about one percent. Preston concludes that although mortality decline has a role in encouraging investment in education, it is likely to be "relatively small." Paroush and Silber (1980) use cross-country data to test the hypothesis that lower mortality risk encourages investment in education, and they discover that people in countries with lower mortality risk make larger investments in schooling. Ram and Schultz (1979) attempt to link improvements in health to economic growth, and find that districts in India that experienced the greatest decline in mortality due to malaria eradication experienced the greatest increase in productivity following the eradication.

Meltzer (1992) argued that mortality decline may have a larger than expected effect on human capital accumulation as a result of overestimated rates of return due to the following factors: ability bias,[8] failure to account for parental inputs such as nutrition, health expenditures, or training outside school,[9] failure to control for hours worked,[10] ignoring non-market earnings,[11] or limiting data collection to wage earners rather than including the self-employed.[12]

In addition, the estimation of how human capital accumulation responds to changes in mortality and morbidity risks is constrained by the quality of the available data. For example, when comparing human capital investment in different countries, it is difficult to control the numerous variables that may be different among them. When comparing different time periods in the same country, similar problems arise. Moreover, regressions based on such data can be sensitive to specification or causation problems. For example, a negative relationship between mortality rates and educational enrollment could also reflect a positive effect of education on health.[13]

In an influential recent paper, Jayachandran and Lleras-Muney (2009) used the substantial maternal mortality decline in Sri Lanka between 1946 and 1953 to identify the effect of decreased mortality on female human capital investment, using males as a natural control group. Jayachandran and Lleras-Muney find that for every extra year of life expectancy, years of education increase by 0.11. This result could be used as a benchmark for our results. However, we cannot expect our results to be directly comparable because we use a dataset from a different time period and country, but if our results are at least close it could lend support to the findings of both papers.

In order to further motivate our empirical approach, consider a simple model where p(a) is the probability that the investor will survive a years from the time of investment and B(a) and C(a) are the benefits are costs of investment in year a. Then, the internal rate of return is defined as that value of ρ for which the net present value of the stream of net benefits to investing equals zero:

$$P.V.=\int_0^\infty e^{-\rho a}\, p(a)[\,B(a)-C(a)]\partial a=0$$

[8]The classic reference on ability bias is Rosen and Willis (1986).
[9]See Behrman (1987).
[10]See Lindsay (1971).
[11]See Rosen and Willis (1986).
[12]See Chiswick (1976).
[13]See Grossman (1972a) and Grossman (1972b).

Under the reasonable assumption that investments in education have negative "yearly" present value during educational investment (because foregone earnings and tuition costs are realized immediately while increased earnings due to higher wages will only be realized after school is finished), an increase in mortality risk will make investment in education less beneficial and decrease the rate of return. This is because after the investment is already done, the individual expects positive present value from it, simply because the first year after investment has a negative "yearly" present value, so the other years must have positive aggregate present value, or the investment would not have been made. [14]

Changes in morbidity risk are also an important influence on human capital investment. Morbidity is more common than mortality in young individuals, and therefore morbidity risks may affect the human capital decision even more than mortality risks. The theoretical analysis is very similar to the analysis of mortality risk. [15]

Empirical strategy

The papers mentioned above and the human capital models on which they are based provide some predictions about the relationship between mortality and morbidity risks and educational attainment. By studying the economic choices of people who are at risk for Huntington's disease, we were able to identify the effects of mortality and morbidity risks on education. Many people in the sample did not know about their risk for Huntington's disease until their thirties or forties, and a comparison of their educational choices to the educational choices of people who learned about their risk status in their teens was the mechanism used to identify the effect of increased mortality and morbidity risks.

Data were collected by interviewing people who are (or were) at risk for Huntington's disease. Table 1 reports summary statistics for the primary variables of interest for our sample. Table 2 reports summary statistics for the primary variables of interest for people who knew before age 18 that they are at risk for Huntington's disease, while Table 3 reports summary statistics for the primary variables of interest for people who did not know before age 18 that they are at risk for Huntington's disease

To identify the effects of mortality and morbidity risk on education, we estimated the following model:

$$\begin{aligned} AdjustedEducation_i = {} & \beta_1 + \beta_2 Male_i + \beta_3 FatherEducation_i + \beta_4 MotherEducation_i \\ & + \beta_5 KnewRiskat18_i + \beta_6 Caucasian_i + \beta_7 Caregiver_i + \beta_8 HighParentIncome_i \\ & + \beta_9 Earlyonset_i + \beta_{10} Earlyonset_i * KnewRiskat18_i + \varepsilon_i \end{aligned}$$

The dependant variable, or "adjusted education", is the total years of schooling the subject has minus the average years of schooling for his/her age cohort.[16] We do not follow the usual approach of using education as the dependent variable and controlling for cohort effect by using an age variable as one of the independent variables. Doing so would not result in precise estimation of the variable of interest, because there is a strong correlation between subject age and whether the subject learned about his risk before or after age eighteen, our main variable of interest. This correlation is the result of the fact that our subjects could not be recruited and interviewed before learning that they are at risk for HD, so young subjects

[14]It is possible for a mortality increase to not change the rate of return, for example if the increase in mortality risk is after the age of retirement, but this is not the kind of change that we study in this paper.
[15]For further details, please see Meltzer (1992) p. 29, section 4.
[16]Cohort education was calculated using the census data available at http://www.census.gov/population/www/socdemo/education/ppl-169.html.

must have learned about their risk at a very young age, creating a strong positive correlation between current age and the age at which the subject learned about his or her HD risk.

The strong correlation between the age the subject learned about his HD risk and his current age combined with the modest sample size results in substantial multi-collinearity problem which makes it difficult to accurately estimate the variable of interest.[17] We cannot use education as the dependent variable without using age as an independent variable, because this means we are not controlling for cohort effects. Collecting more data is not practical. Therefore, our solution is to import external data that tells us what the expected education is for a person of the same cohort as the subject and from the same state. This involves making the assumption that the relationship between schooling and age in our sample is the same as the relationship in the general population from which average cohort schooling was calculated. Considering that expected cohort education is calculated using people from the same state and same time period, we think this mild assumption is justified as it allows us to prevent the more serious problem of multi-collinearity.

Other independent variables include the following standard variables that are known to affect schooling: the “Male” variable is equal to 1 if the subject is male, 0 otherwise; The “Father Education” variable measures the number of years of schooling the subject’s father has; The “Mother Education” variable measures the number of years of schooling the subject’s mother has; The “Caucasian” variable equals 1 if the subject is Caucasian, 0 otherwise, and the “High Parent Income” variable equals 1 if the subject reported that the income of his parents during his childhood was at least $50,000 a year in 2004 dollars, 0 otherwise. Such variables are known to be relevant for human capital decisions and therefore are included to prevent omitted variable bias and in order to facilitate comparisons of the results with the literature.

In addition to the common variables affecting the human capital investment decision, we need to add several variables specific to our study. The first such variable is the “Caregiver” variable. We add it because patients with Huntington’s disease will generally have an affected parent, so we must control for the possibility that having to provide care to an ill parent might make it more difficult for the subject to complete high school or college. The “Caregiver” variable is equal to one if the subject was a caregiver on a daily basis and for at least two hours a day during any period between the ages of 15 and 25. Otherwise, it is equal to zero.

The next variable we add is the “Early onset” variable. It is equal to 1 if the affected parent age on onset of HD was below 45. Otherwise, it is equal to zero. The early onset variable is important for several reasons. First, there is some correlation between the age of onset in the parent and the age of onset in his affected children. Second, even if subjects do not know about this correlation, the naive, natural assumption for a person to make is that, if he is affected, his age of onset will be the same as that of his parent. This may result in estimates that are an average of heterogeneous outcomes for different subjects. Because the age of onset in the parent can affect the educational attainment of the subject only when the subject knows he has an affected parent (i.e., is aware of his HD risk), we interact the “Early onset” variable with the “Knew risk at 18” variable.

Finally, we add to the regression the “Early onset” variable without interacting it with any other variables. This addition is important because it helps protect our estimation against

[17]We have tried that approach and results are exactly as expected in the case of multicollinearity. The “age” variable is estimated to have a positive coefficient, exactly the opposite of what we know in practice, and the” knew risk at 18” variable is now only statistically significant at the 10% level and about 30% smaller in magnitude. The “age” variable and the “knew risk at 18” variable are jointly significant at the 1% level.

identification problems. For example, people who learn before age 18 that they are at risk for HD almost always have an affected parent. People who learn at a later age that they are at risk for HD as a result of late onset in the parent are different in two important ways: first, they did not grow up with a disabled parent; and second, they could not adjust their education decision to their risk status. If growing up with a disabled parent affects schooling decisions, we might have a problem separating this effect from the effect of HD risk on schooling. The "Early onset" variable controls for differences between subjects who learned about their risk late as a result of late parental onset and subjects who learned about their risk early, because it is strongly correlated with growing up with a disabled parent and other effects of having a parent with HD while growing up. Subjects who learned about their risk late because of misdiagnosis are assumed to be similar to subjects who learned about their risk early. For example, in both cases the subjects would grow up with a disabled parent. If there are systematic differences between those two groups that are not captured by the variables we use, or if the "Early onset" variable is not strongly correlated with growing up with a disabled parent, our identification strategy would be threatened.

Results

Table 4 reports regression results for this model. Regression results are consistent with our expectations: The main variable of interest, "Knew Risk at 18", has a negative, large, and statistically significant coefficient, suggesting that subjects who learned about their risk for Huntington's disease at age 18 or earlier attained 2.45 less years of education compared to subjects who learned about their risk for Huntington's disease after age 18.

The results are also generally consistent with the existing human capital literature: The coefficient for father's education is positive and statistically significant, as expected. The coefficient for mother education is weak and not statistically significant. This can be explained by the fact that almost all mothers in the sample only had a high school diploma.

The coefficients for the "Male", "Mother education" and "Caucasian" variables are positive as expected, but not statistically significant, likely due to the modest sample size. The coefficient for care giving is not significant[18], suggesting that this variable is not important in the regression, a suggestion confirmed by discussions with subjects.[19] The coefficient for high parental income is not statistically significant and in the wrong direction. This may be both due to sample size and measurement error, because it is hard to expect people to accurately report, or being aware of, parental income. The "Early onset" coefficient and the "Early onset" interacted with "Knew risk at 18" coefficient are not statistically significant, suggesting that either our concerns about identification these variables are supposed to resolve are not important, or that the various concerns approximately offset each other.

Alternative explanations and robustness of assumptions

Self-selection—First, we considered the possibility of bias due to selection into the study sample. This is a particular concern when participation rates are low, since a sample may not be representative of the population. However, the participation rate was approximately 75%, and most of the people who refused to participate stated that they did so because they had other obligations.[20] There is no basis to believe that refusal to participate for such a reason

[18]A potential concern is that the caregiver variable may be endogenous. This can happen if, for example, less talented siblings are selected (or self-selected) into giving care. It is not clear whether this is a serious empirical problem, because Huntington's disease is special in that it appears at a relatively young age, so siblings, spouses, and a parent are usually able to care for the affected individual. To address this concern we repeated our regression analysis using only subjects who did not provide care (therefore omitting from the regression the observations representing the nine individuals who did provide care). The results were almost identical.

[19]Only 9 subjects provided care as defined in this study, and several of them clearly said that it was not a factor preventing them from furthering their education.

is correlated with any of the choices studied in this paper. These facts suggest that self-selection into the study is unlikely to be a major source of bias. There is, however, the potential for some selection based on the fact that recruitment was done in Rush Hospital which is a relatively prestigious hospital, but the nature of any resulting bias is not clear.[21]

Parental intervention—Another interesting possibility that may result in bias is parental intervention. In many cases, parents can control the age at which their children are informed of their risk status. If parents want their children to attend college, then they may delay sharing this information until after their children have completed college.[22] However, examination of the data suggests that this is not a likely source of bias. If it were, we would expect many subjects to have learned of their risk status soon after completing college. We would also expect that subjects who have learned of their risk status at ages 22–26 would have accumulated more years of education than other subjects. The data do not support either expectation.

Exogeneity—An implicit assumption in our regressions is that the age at which a person learns of his risk status is exogenous. If it were not exogenous, estimates of its effects from our model could be biased. To address this, it is useful to consider which variables determine the age at which a person learns of his Huntington's disease risk. The main variables affecting the age in which a person learns of his or her risk status are the variables that determine his or her age when his or her parent was first diagnosed with Huntington's disease. This is so because in most cases, people who are at risk do not know they are at risk until a parent is diagnosed with the disease.[23] If this is the case, such people cannot learn that they are at risk before the affected parent is diagnosed with Huntington's disease.[24]

Important factors affecting the age of when the affected parent was first diagnosed with Huntington's disease include the following: accuracy and speed of the diagnosis of the affected parent,[25] the age of Huntington's disease onset of the affected parent,[26] the age of the affected parent when the subject was born,[27] and the age of death of the affected parent.[28] These variables are unlikely to be correlated with educational choice.[29]

Depression—another possibility we wanted to examine is whether the news about being at risk for Huntington's disease induce depression which causes a reduction in educational attainment. Even if that were so, it would still be an effect of mortality and morbidity risk on

[20]Typical answers were "I have to leave before traffic" and "I have another doctor's appointment right after this appointment."
[21]This is probably why subjects have, on average, more education than their healthy peers (as shown by table 1), although being at risk for Huntington's disease reduces educational attainment.
[22]Withholding the information may be inconsistent with maximizing the child's utility. However, some parents may be paternalistic.
[23]As a matter of fact, most people in the sample never knew they are at risk until they were actually diagnosed.
[24]This is strongly supported by the data. Only three out of sixteen subjects who had a parent with onset before age 45 knew about their risk before age eighteen. On the other hand, twenty out of thirty five subjects who had a parent with onset after age 45 knew about their risk before age eighteen. Surprising as it seems (because Huntington's disease is the result of a mutation in a single, dominant gene), it is uncommon for people to learn that they are at risk because of the illness of a grandparent.
[25]Incorrect diagnosis, which is common with Huntington's disease, would have delayed the age at which the affected parent was diagnosed and as a result, the age at which the person at risk would have learned about his or her risk status. Since Huntington's disease is rare, incorrect diagnosis is common and depends on random factors such as whether the examining doctor have seen other Huntington's disease patients during his or her residency.
[26]The later the age of onset in the affected parent, the later he or she is likely to be diagnosed with Huntington's disease.
[27]This is because the age at which a person at risk can first learn about his or her risk status (if there is no knowledge of Huntington's disease in the family before the affected parent is diagnosed) is his current age when the affected parent is diagnosed, which is the age of diagnosis in the affected parent minus the age of the affected parent when the person at risk was born.
[28]Early death of the affected parent from reasons unrelated to Huntington's disease can prevent diagnosis, preventing the subject from learning about his risk status.
[29]The age of onset of Huntington's disease in the affected parent might be an exception. We checked this by repeating our regression with the addition of a variable controlling for the age of onset in the affected parent. The results were practically the same, and the age of onset variable was not statistically significant.

educational attainment; but it would not be due to changes in the rate of return to educational attainment, which is the effect we are more interested in. However, this seems implausible because every subject in the regression dataset has at least a high school diploma. What this means is that all subjects who learned about being at risk for Huntington's disease before age 18 actually completed high school, which seems contradictory to the depression explanation. It may not be a property of the general at risk population, but all that means is that this population, inasmuch as it suffers from depression in addition to a reduced rate of return to education, will have reduced educational attainment.

Early onset of HD (in subject)—Although uncommon, early onset of Huntington's disease is possible. This introduces a problem, because early onset may result in a person obtaining less education because he is simply unable to do so (for example, he cannot pass the entrance examinations). While this is a consequence of Huntington's disease, it is not a response to a change in the rate of return to education and not the effect we would like to estimate. To check whether this is a problem, we examined the age of diagnosis of every subject who has Huntington's disease. Two subjects were diagnosed before age 25, and were dropped from the analysis. Two subjects who were diagnosed at ages 28 and 29 were included in the regression. [30]

Parent disability—While we control for the effects of parent disability to some extent (using the "care giver" variable, the "high parent income" variable, and the "early onset" variable), it is still very likely that learning one's own HD risk status very frequently occurs at the same time as a parent's disability. If the latter affects subjects in ways not controlled for by the variables mentioned above, it could present a threat to identification. To provide support for our interpretation of the results, we looked for studies that relate parent disability status to the schooling of their children. We were able to find a World Bank policy research paper[31] which studies the relation between parent disability status and children's enrollment rate and achievement in primary and secondary schools. The authors find that children to disabled parents are eight percentage points less likely to enroll in school, while there is no statistically significant relation between parent disability and achievement in school. We expect the relevant drop in enrollment rate in our study to be substantially lower, because the eight percentage points drop in the schooling rate reported by Cuong and Mont includes the possible effects of care giving as well as parent financial difficulty. We try to control for both these factors in our regression. Moreover, even if we do not control for these factors fully, the United States has policies and programs supporting disabled parents and children of disabled parents[32] that should result in a smaller effect in the population we study compared to Vietnam. If this is indeed the case, then parent disability is not likely to be the major factor explaining our results, because the effect of Huntington Disease risk status on schooling is much larger.[33]

Other approaches to address this problem in future research could be collecting data on whether the affected parent was misdiagnosed. If this is the case, then the child of that parent may know about the parent disability (depending on the exact diagnosis) while still not learning about his Huntington's disease risk status. As long as the incorrect diagnosis

[30]An alternative regression not including the data for these subjects was tried, but results were practically the same.
[31]See Cuong and Mont (2011).
[32]See for example http://ssa.gov/pubs/10085.html for a discussion of support of children of disabled parents in the United States.
[33]Indeed, if one considers the meaning of a reduction of more than 2 years in schooling that we find and the fact that all of our subjects except one have a high school degree, it would translate into more than 50% decline in the likelihood to attend college (assuming that a college degree takes 4 years to complete and that people do not drop out). This is a much larger effect than the effect reported by Cuong and Mont, and although they focus on primary and secondary school attendance and not on college, we feel the difference is so big even such an inaccurate comparison has some value.

has similar effects to the diagnosis of Huntington's disease, a comparison of subjects with misdiagnosed parents to subjects who learned about their Huntington's disease before age 18 could help identify the effect of risk status on schooling. Another compelling identification strategy would be a study of adoptees. An adopted person could learn of his or her own HD risk status, but their adoptive parents' health would be unaffected. Conversely, an adoptee could learn of an adoptive parent's HD diagnosis, without it having any implications for their own at-risk status. However, because Huntington's disease is so rare, we do not expect this approach to be easily feasible in future studies.

Conclusions, generalizations, and future research

In this paper we examined the effects of heightened risks of early mortality and morbidity on human capital decisions. A strong negative relation was found between heightened risks of early mortality and morbidity and educational attainment. Other things equal, people who knew they were at risk for Huntington's disease before making their education decisions accumulated 2.45 less years of education compared to people who learned about their risk only later in life.

This supports the broader conclusion that high mortality risk among young adults may reduce investment in education. While young adult mortality is low among most modern populations, sub populations of persons, such as low-income urban youth and young adults in developed countries and youth and young adults in low income settings continue to have elevated risk of mortality. Among those groups, improved incentives to invest in education might be an important dividend of efforts to reduce mortality risk.

Our results are generally consistent with the results reported by Jayachandran and Lleras-Muney. They find that for every extra year of life expectancy, years of schooling increase by 0.11. Unlike Jayachandran and Lleras-Muney, We do not use life expectancy because with HD, morbidity prevents the individual from realizing the return on his human capital investment long before death.[34] However, considering that a person with HD typically becomes disabled around age 40, we can calculate the years of income lost because of HD. Worklife expectancy (i.e., expected number of years in the labor force) for 40 year old active men is 21.25 years (Skoog and Ciecka 2010). Dividing the effect we find by 21.25, we find that a gain of a year of income will cause years of schooling for men to increase by 11.5%, an effect that is almost identical to that found by Jayachandran and Lleras-Muney.[35]

We have considered how the methodology of this project could be generalized. The first way to generalize it is to undertake similar studies using other diseases. We believe that the prospects for generalizing the methodology to other diseases are low for the following reasons: first, a disease that appears too early in life will affect the production function of human capital and not just the later returns. Second, a disease that appears too late in life will not affect the rate of return to human capital investments much because of the cumulative effects of discounting and because its effects may happen after the working age. For example, a person who knew today he has a high risk to get Alzheimer's disease would not change his human capital accumulation as a result, at least not because of change in the returns. Third, to be able to isolate the effects of mortality and morbidity risks on human capital investment, the disease to be studied must involve a substantial risk. For example, suppose a certain individual knew that because of her genetic makeup, she is five percent

[34]Jayachandran and Lleras-Muney do not need to worry about the effect of morbidity because death during child birth is not associated with a period of morbidity.

[35]We caution against giving too much weight to the similar coefficients, because there are many reasons why they could differ (different time, different country, different rates of return to schooling, the fact we use expected number of years in the labor force for men, etc); but nevertheless we think that the similar magnitude is encouraging.

more likely to get breast cancer at a young age. While this should have an effect on human capital accumulation, it might be hard to identify because it would be relatively small. Fourth, the disease to be studied must be common enough to allow for reasonable sample sizes. Fifth, the disease to be studied must be predicted in advance, before it affects the production function of human capital. We do not know any other disease except Huntington's that meets all of these conditions. On a positive note, we believe that generalizing the methodology in the sense of using variations in genetic information to study economic questions can be gainfully used to study a variety of other economic questions.

Another question is the extent to which our results generalize to how our other diseases might affect schooling. In addressing this question, it is useful to consider the theoretical model, which suggests that the reason people who are at risk for a disabling disease reduce their schooling is the lower returns to schooling. The expected returns are affected by the probability of illness, the severity of illness[36], and the likely age at which illness will appear. Given that Huntington's disease is associated with very severe disability, appears relatively early in life, and is associated with very high risk for at-risk populations, we believe that for most other diseases the effect of disease risk on schooling will be much smaller, although there are exceptions. Meltzer (1992) sites several possibly relevant examples. These include the risk of HIV, especially in countries such as many in Africa where prevalence is quite high. Malaria may have similar effects if adequate treatment is not available. The risk of Alzheimer's disease should not affect schooling decisions because Alzheimer's disease almost always appears after retirement, so it does not affect earnings, but it might alter children's expectation about their parents need for care in old age, and thus their expectation about their own level of labor force participation in late middle age when their parents may need their care. Thus, while the example of Huntington's disease may have a unique combination of factors, the concept

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Bibliography

Barro; Robert, J. Economic Growth in a Cross Section of Countries. Prepared for the conference "Human Capital and Economic Growth."; SUNY Buffalo. 1989.

Becker, Gary S. Demographic and Economic Change in Developed Countries. Princeton University Press; 1960. An economic analysis of fertility.

Becker, Gary S. Human Capital. 3. University of Chicago Press; 1993. p. 85-86.

Becker, Gary S.; Murphy, Kevin M.; Tamura, Robert. Human Capital, Fertility, and Economic Growth. Journal of Political Economy. 1990; 98(5):S12–S37.

Behrman, Jere R. Schooling and Other Human Capital Investments: Can the Effects be identified? Economics of Education Review. 1987; 6(3):301–305.

Ciecka, James; Skoog, Gary. Measuring Years of Inactivity, Years in Retirement, Time to Retirement, and Age at Retirement within the Markov Model. Demography. 2010; 47(3):609–628. [PubMed: 20879680]

Chiswick, Carmela. On Estimating Earning Functions for LDC's. Journal of Development Economics. 1976:67–78.

Cuong, Nguyen; Mont, Daniel. Does Parental Disability Matter to Child Education? World Bank Policy Research Working Paper. 2011 Aug.5743

[36]The severity of illness is not included in the model we present, but such an extension is straightforward. that knowledge of genetic risk may provide information that can affect investment in education seems to have broader potential relevance.

Grossman, Michael. The Demand for Health: A Theoretical and Empirical Investigation. New York: Columbia University Press; 1972a.

Grossman, Michael. On the Concept of Health Capital and the Demand for Health. Journal of Political Economy. 1972b; 80(2):223–255.

Hotz, Joseph; Klerman, Alex; Willis, Robert. Handbook of Population and Family Economics. Vol. 1A. Elsevier Science; 1997. The Economics of Fertility in Developed Countries; p. 275-347.

Jayachandran, Seema; Lleras-Muney, Adriaana. Life Expectancy and Human Capital Investments: Evidence from Declines in Maternal Mortality. Quarterly Journal of Economics. 2009; 124(1): 349–397.

Lindsay CM. Measuring Human Capital Returns. Journal of Political Economy. 1971; 79(6):1195–1215.

Mascolell, A.; Whinston, M.; Green, J. Microeconomic Theory. Oxford University Press; 1995.

Meltzer, David O. PhD dissertation. University of Chicago; 1992. Mortality Decline, the Demographic Transition and Economic Growth.

Paroush, Jacob; Silber, Jacques. The Stochastic Dominance Criteria, Mortality Risk and Investment in Education: A Cross-country Comparison. Research in Finance. 1980; 2:111–120.

Preston, Samuel H. Population and Economic Change in Developing Countries. University of Chicago Press; 1980. Mortality Declines in Less Developed Countries.

Ram R, Schultz TW. Life Span, Health, Savings and Productivity. Economic Development and Cultural Change. 1979; 27(3):399–421.

Rosen, Sherwin; Willis, Robert J. Education and Self-Selection. Journal of Political Economy. 1979; 87(5):S7–S36.

Von Neumann, J.; Morgenstern, O. Theory of Games and Economic Behavior. Princeton University Press; 1953.

Table 1

Summary Statistics

	N	Mean	SD	Min	Max	p25	p75
Aware of risk at 18?	56	0.32	0.47	0.00	1.00	0.00	1.00
Adjusted education	56	1.55	2.54	−1.46	6.84	−1.32	2.70
Father education	56	12.68	2.65	8.00	18.00	12.00	15.00
Mother education	56	12.29	2.17	8.00	20.00	12.00	12.00
Male	56	0.41	0.50	0.00	1.00	0.00	1.00
Caucasian	56	0.89	0.31	0.00	1.00	1.00	1.00
Caregiver	56	0.16	0.37	0.00	1.00	0.00	0.00
High parent income	56	0.45	0.50	0.00	1.00	0.00	1.00
Age	56	45.96	9.99	27.00	72.00	38.00	53.00
Education	56	14.82	2.46	12.00	20.00	12.00	16.00
Symptomatic	56	0.48	0.50	0.00	1.00	0.00	1.00
Age learned of risk	56	29.59	15.73	6.00	59.00	16.50	45.00

Note: the table shows summary statistics of the sample used in the regression analysis.

Table 2

Conditional Summary Statistics

	N	Mean	SD	Min	Max	p25	p75
Aware of risk at 18?	18	1.00	0.00	1.00	1.00	1.00	1.00
Adjusted education	18	0.22	2.01	−1.46	4.61	−1.37	2.61
Father education	18	13.56	2.53	8.00	18.00	12.00	16.00
Mother education	18	12.56	1.79	8.00	16.00	12.00	14.00
Male	18	0.22	0.43	0.00	1.00	0.00	0.00
Caucasian	18	0.94	0.24	0.00	1.00	1.00	1.00
Caregiver	18	0.22	0.43	0.00	1.00	0.00	0.00
High parent income	18	0.50	0.51	0.00	1.00	0.00	1.00
Age	18	38.44	6.57	29.00	52.00	33.00	43.00
Education	18	13.56	2.01	12.00	18.00	12.00	16.00
Symptomatic	18	0.61	0.50	0.00	1.00	0.00	1.00
Age learned of risk	18	13.50	3.31	6.00	18.00	10.00	16.00

Note: the table shows summary statistics of the sample used in the regression analysis, Conditional on the “Aware of risk at 18?” variable being equal to one.

Table 3

Conditional Summary Statistics

	N	Mean	SD	Min	Max	p25	p75
Aware of risk at 18?	38	0.00	0.00	0.00	0.00	0.00	0.00
Adjusted education	38	2.19	2.55	−1.46	6.84	−0.75	4.70
Father education	38	12.26	2.64	8.00	18.00	12.00	14.00
Mother education	38	12.16	2.34	8.00	20.00	12.00	12.00
Male	38	0.50	0.51	0.00	1.00	0.00	1.00
Caucasian	38	0.87	0.34	0.00	1.00	1.00	1.00
Caregiver	38	0.14	0.38	0.00	1.00	0.00	0.00
High parent income	38	0.42	0.50	0.00	1.00	0.00	1.00
Age	38	49.53	9.38	27.00	72.00	41.00	57.00
Education	38	15.42	2.46	12.00	20.00	12.00	18.00
Symptomatic	38	0.42	0.50	0.00	1.00	0.00	1.00
Age learned of risk	38	37.21	13.31	19.00	59.00	25.00	50.00

Note: the table shows summary statistics of the sample used in the regression analysis, Conditional on the “Aware of risk at 18?” variable being equal to zero.

Table 4

Regression Results

Variable	Coef.	Std. Err.	t	P>|t|	95% Conf. Interval	
Aware of risk at 18	−2.45**	0.95	−2.57	0.013	−4.36	−0.53
Father education	0.42***	0.14	3.08	0.003	0.15	0.70
Mother education	0.07	0.15	0.49	0.625	−0.23	0.37
Male	0.91	0.64	1.43	0.160	−0.37	2.19
Caucasian	0.62	1.17	0.53	0.597	−1.74	2.99
Caregiver	−0.07	0.99	−0.07	0.942	−2.07	1.92
High parent income	−0.76	0.68	−1.12	0.269	−2.14	0.61
Early onset	−0.23	0.88	−0.26	0.798	−2.00	1.55
Aware risk 18 * Early onset	0.48	1.54	0.31	0.757	−2.62	3.57
Constant	−4.49**	2.13	−2.10	0.041	−8.78	−0.19
Other statistics	N=56	P>F=0.008		R^2=0.36	Adj R^2=0.24	