THE EDUCATION-HEALTH GRADIENT

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Much of the policy discussion about reducing health disparities across socio-economic groups has focused on improving health insurance coverage and access to health care. However, increasing attention is being paid to the social determinants of health (Commission on Social Determinants of Health 2008). Several scholars have noted that better health early in life is associated with higher educational attainment. More educated individuals, in turn, have better health later in life and better labor market prospects (Janet Currie 2009, David Cutler and Adriana Lleras-Muney 2009).

This paper summarizes our current research examining the early origins of health disparities by education (Gabriella Conti, James J. Heckman & Sergio Urzua 2009, henceforth CHU). It contributes to a growing literature that establishes a strong relationship between health and education and more generally between early childhood conditions and adult outcomes. Gaps in both the cognitive and noncognitive abilities of children of different socioeconomic groups emerge at early ages (Flavio Cunha, Heckman, Lance J. Lochner and Dimitriy V. Masterov 2006). So do gaps in health (Anne Case, Darren Lubotsky & Christina

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Paxson 2002). Various studies suggest that it is possible to enrich adverse early environments and promote child development.

CHU identify the causal effect of education on health and health-related behaviors. We determine the role played by cognitive, noncognitive, and health endowments measured in the early years in explaining numerous adult outcomes. Family background characteristics, and cognitive, noncognitive and health endowments are all important determinants of health disparities at age 30. Our methodology allows us to determine the fraction of health gaps by education that can be explained by selection into education on early life endowments and the fraction that can be attributed to the causal effect of education. Not accounting for personality traits overestimates the importance of cognitive ability in determining adult health and participation in healthy behaviors. Selection into education on early life factors explains more than half of the observed difference by educational level in poor health, depression and obesity. Education has an important causal effect in explaining differences in smoking rates, and participation in many other health behaviors, as well as on a number of other outcomes. We find significant gender differences in the effect of education on health. We go beyond the current literature that typically estimates mean causal effects to compute distributions of treatment effects. We show how the returns to education vary among individuals who are similar in their observed characteristics. Mean effects hide gains and losses to treatment in the population. Our analysis highlights the important role played by factors determined in the early years in promoting health.

A positive correlation between health and schooling is one of the most well-established findings in the social sciences. Figure 1 shows mean educational differentials for a variety of outcomes in the British Cohort Study (BCS70) data we analyze. The full length of the bar for each outcome is the raw educational differential. The extent to which this association reflects causality is still subject to much debate. Three explanations for this correlation are offered in the literature: that causality runs from schooling to health (Grossman 2006), that it runs from health to schooling (Currie 2009), and that both are determined by a third factor, such as time or risk preferences. Understanding the relative importance of each of these mechanisms in generating observed differences in health by education is helpful in designing policies to promote health.

Our research joins the literatures in economics, epidemiology and psychology.

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1 Obesity is measured by BMI ≥ 30 for males and BMI ≥ 25 for females.
2 We discuss the decomposition into causal and selection components later.
Figure 1: Disparities in outcomes by education by gender in the British Cohort Study 1970 (outcomes measured at age 30)

Note: The bar heights show the difference in outcomes by educational level (post-compulsory schooling level vs. compulsory schooling). The darker region within each bar shows the fraction of the raw gap arising from the causal contribution of education. The rest is due to selection.
We explore the relationship between health and cognitive ability. We also explore the relationship between personality traits and health. This research contributes to understanding the nonmarket returns to education.

1 Models, Methods, and Data

CHU estimate a semiparametric structural model of the choice of schooling and the causal effect of schooling on a variety of health outcomes and healthy behaviors. Agents are assumed to select into schooling based on expected market and nonmarket returns. We have precise measures of a number of early childhood environmental factors, $X$. In addition, we have proxies for a vector of latent capabilities for early life cognition, personality, and health endowments, $\theta$, that, in addition to the $X$, affect both the choice of education and the adult outcomes studied. (See Pedro Carneiro, Karsten Hansen and Heckman 2003 and Heckman, Jora Stixrud and Urzua 2006 for descriptions of the methodology.) If we could condition on observed characteristics $X$ and unobserved characteristics $\theta$, any remaining association between education and adult outcomes would be causal.

While we cannot directly measure $\theta$, we have a large number of proxies for the low-dimensional $\theta$ in our data. These measures are taken early in life (at age 10). Using proxies for $\theta$ accounting for the measurement error in the proxies and controlling for observables, $X$, we estimate causal effects of education on adult health and healthy behaviors. Our method is a form of matching on both observables and unobservables where the unobservables are proxied, and we account for the errors in the proxies for the unobservables. It can also be interpreted as a latent variable structural model with the key unobservables measured up to error that is accounted for in the estimation procedure. We find substantial evidence of measurement error.\(^4\)

We estimate the model two ways: using matching and using structural methods. Both methods produce results that are in agreement for all parameters identified by both methods. For the sake of brevity, we only report the results from the structural method.\(^5\)

The BCS70 data that we analyze is a survey of all babies born (alive or dead) in one week in April 1970. We have panel records on schooling, family background, a variety of health and healthy behaviors, and labor market outcomes. Schooling choices depend on expected market and nonmarket returns. In this paper, school-

\(^4\)For further details, see CHU or our Web Appendix at http://jenni.uchicago.edu/gradient/

\(^5\)The matching results are available in our Web Appendix.
ing is a binary decision and refers to attendance beyond the compulsory school-leaving age. We have multiple measures of cognitive and noncognitive skills and early health, taken at age 10. See the Web Appendix for further details.

To avoid dependence of estimates on distributional assumptions, we use mixtures of multivariate normals models to characterize the distributions of the latent capabilities. We can generate all treatment effects.\footnote{Our identification strategy does \textit{not} rely on “identification at infinity” or conventional exclusion restrictions. Like matching, it relies on conditional independence assumptions. Unlike conventional matching, we allow the conditioning variables to be measured with error. See the discussion in Carneiro, Hansen & Heckman (2003), Jaap H. Abbring & Heckman (2007) or Heckman, Stixrud & Urzua (2006). The support of the estimated probability of schooling is essentially the full unit interval so that identification of the model is over the full support of the unobservables in the choice equation.}

We can also estimate the distribution of treatment effects following Carneiro, Hansen & Heckman (2003) and Abbring & Heckman (2007). The estimated model passes tests of goodness of fit.\footnote{See CHU and the Web Appendix for details.}

\section{Findings}

We relate early measures of endowments to the adult outcomes measured at age 30 that are shown in Figure\textsuperscript{1}. We find sorting of individuals by schooling in terms of cognitive, noncognitive, and early health endowments. Early health endowments are weakly associated with schooling for women but not at all for men. Thus, in our data, evidence on the link between early health and education emphasized by Currie (2009) is at best weak. However, consistent with Currie (2009), we find that early health has a statistically significant direct effect on adult outcomes. Noncognitive factors play a powerful role in predicting participation in healthy behaviors. Introducing noncognitive factors substantially weakens the predictive power of cognitive factors in promoting adult outcomes, but cognitive ability remains an important determinant of education and labor market outcomes.\footnote{See the Web Appendix for the full results.}

Education is estimated to have a strong causal effect on most outcomes examined. Figure\textsuperscript{1} displays for each outcome the fraction of the observed educational differential that can be attributed to the causal effect of education — the part of each bar labeled “causal component.”\footnote{These are Average Treatment Effects.} Note the gender differential. Education plays a much more important causal role for males than females in accounting for
gaps in obesity rates, exercise, and employment.

Our analysis moves beyond the traditional literature which only considers mean treatment effects and estimates distributions of treatment effects. Knowledge of these distributions is fundamental in uncovering what lies behind a zero estimated average treatment effect, and the proportion of individuals who actually benefit from the treatment (education). We find substantial heterogeneity in treatment responses. Consider the case of smoking for females. The proportion of people who stop smoking is much bigger than the proportion of people who start smoking, so the average treatment effect turns out to be negative (see Figure 2).

Compare these results to the results for obesity for females. Underlying a statistically insignificant average treatment effect of education on obesity there are gains and losses which balance each other out: the same proportion of women (roughly 20 percent) lose and gain from the treatment. For males there is a net negative effect.

### Treatment Effect Heterogeneity: the Role of Early Endowments

CHU study how the average treatment effect of education varies with the level of endowment of cognitive and noncognitive skills, and early health. While there is a significant amount of heterogeneity in the effect of education across outcomes by levels of endowments, some patterns emerge. Education compensates for low endowments of noncognitive skills for poor health. See Figure 3 which plots the effect of education on poor health for different quantiles of the cognitive, noncognitive and health endowments. A similar pattern is found for depression. For males, on most outcomes, the beneficial effect of education is much bigger at the bottom of the noncognitive ability distribution. The effect is greater at the top of the cognitive ability distribution in the case of smoking. This finding is consistent with the interpretation that the information content on the dangers of smoking provided by post-compulsory education needs to be combined with the capacity to process that information in order for it to be effective.

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10 Results are the same for males.
Figure 2: Population distribution of the average treatment effect for females - health behaviors at age 30
Note: The figures display the distribution of the average treatment effect. The outcomes are simulated from the estimates of the model.
Figure 3: Treatment effect heterogeneity - poor health (males) at age 30
The Role of Cognitive and Noncognitive Ability

We find strong evidence that noncognitive traits promote health outcomes and healthy behaviors. If noncognitive traits measured at age 10 are not included in the estimated model, early cognitive ability has a strong and statistically significant effect for many outcomes. There is a smaller estimated effect of cognitive traits in models where we analyze health endowments, cognition, and noncognitive traits jointly. This comes as no surprise since the correlation between cognitive and noncognitive endowments is 0.54.11 Our analysis sounds a warning for research in the area of cognitive epidemiology that has not given adequate attention to personality traits and focuses exclusively on the role played by intelligence (see, e.g., Linda S. Gottfredson & Ian J. Deary 2004). If anything, noncognitive factors are relatively more important determinants of health and healthy behaviors.

Summary

The research reported in CHU examines the early origins of health disparities across education groups. We determine the role played by early cognitive, noncognitive, and health endowments. We identify the causal effect of education on health and health-related behaviors. We develop an empirical model of schooling choice and post-schooling outcomes, where both schooling and the outcomes determined in part by schooling are influenced by measured early family environments and latent capabilities (cognitive, noncognitive and health). We show that family background characteristics, and cognitive, noncognitive, and health endowments developed by age 10, are important determinants of labor market and health disparities at age 30. Not properly accounting for personality traits overestimates the importance of cognitive ability in determining adult health. Selection on factors determined early in life explains more than half of the observed difference by education in poor health, depression, and obesity. Education has an important causal effect in explaining differences in many adult outcomes and healthy behaviors. We uncover significant gender differences. We go beyond the current literature which typically estimates mean effects to compute distributions of treatment effects. We show how the health returns to education can vary among individuals who are similar with respect to their observed characteristics, and how a mean effect can hide gains and losses for different individuals. Our research highlights the important role played by the early years in producing health.

11We display the joint distributions of the endowments in the Web Appendix.
References


