The Education-Health Gradient

By Gabriella Conti, James Heckman, and Sergio Urzua*

Much of the policy discussion about reducing health disparities across socioeconomic 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 2010).

This paper summarizes our current research examining the early origins of health disparities by education (Conti, Heckman, and Urzua forthcoming, 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 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, and Christina 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.[1] 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

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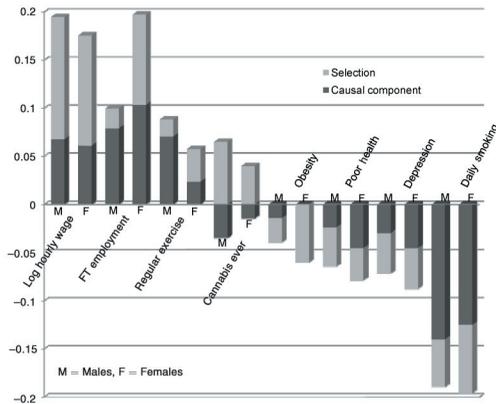[1] Obesity is measured by BMI $\geq$ 30 for males and BMI $\geq$ 25 for females.

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.

differential.[2] The extent to which this association reflects causality is still subject to much debate.[3] 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. 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.

I. 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 may 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, θ, 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 θ, any remaining association between education and adult outcomes would be causal.

While we cannot directly measure θ, we have a large number of proxies for the low-dimensional θ in our data. These measures are taken early in life (at age ten). Using proxies for θ 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 (Heckman, Susanne Schennach, and Benjamin Williams 2010). 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, schooling

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[2] We discuss the decomposition into causal and selection components later.

[3] See Michael Grossman (2008).

[4] For further details, see CHU or our online Appendix at http://www.aeaweb.org/articles.php?doi=10.1257/aer.100.2.234.

[5] The matching results are available in our online Appendix.

is a binary decision and refers to attendance beyond the compulsory school-leaving age. We have multiple measures of cognitive and non-cognitive skills and early health, taken at age 10. See the online 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.[6] We can also estimate the distribution of treatment effects following Carneiro, Hansen, and Heckman (2003) and Abbring and Heckman (2007). The estimated model passes tests of goodness of fit.[7]

II. Findings

We relate early measures of endowments to the adult outcomes measured at age 30 that are shown in Figure 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 other 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.[8]

Education is estimated to have a strong causal effect on most outcomes examined. For each outcome, Figure 1 displays 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."[9] 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.[10] 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.

III. 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 with early health. While there is a significant amount of heterogeneity in the effect of education across outcomes by levels of endowments, some patterns emerge. On most outcomes for males, the beneficial effect of education is much bigger at the bottom of the noncognitive ability distribution and at the top of the cognitive ability distribution. See Figure 3, which plots the average treatment effect of education on smoking of different quantiles of the cognitive, noncognitive, and health endowments. The evidence on differential effects of education by level of cognitive skill is consistent with the interpretation that the

[6] Our identification strategy does *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, and Heckman (2003), Jaap H. Abbring and Heckman (2007), Heckman, Stixrud, and Urzua (2006), or Heckman, Schennach, and Williams (2010). In fact, 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 so that an identification at infinity identification strategy would be valid in our data.

[7] See CHU and the online Appendix for details.

[8] See the online Appendix for the full results.

[9] These are average treatment effects.

[10] Results are the same for males.
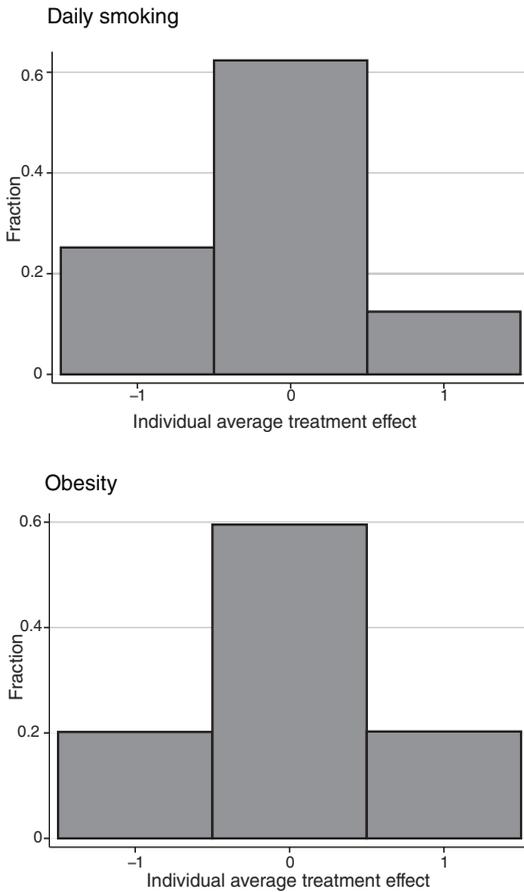
Daily smoking

Obesity

Figure 2. Population Distribution of the Average Treatment Effect for Females–Health Behaviors at Age 30

Notes: The figures display the distribution of the average treatment effect. The outcomes are simulated from the estimates of the model.

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.

IV. 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 ten are not included in the estimated model, early cognitive ability has a strong and statistically significant effect for many outcomes. There is
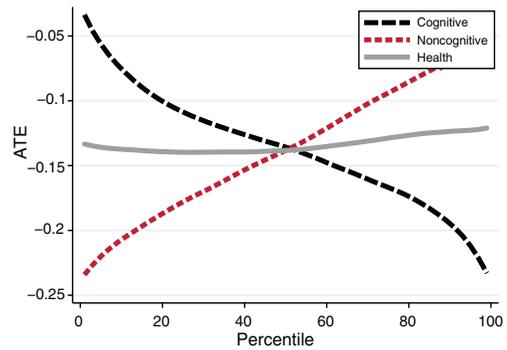

Figure 3. Treatment Effect Heterogeneity— Smoking (males) at age 30

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 and Ian J. Deary 2004). If anything, noncognitive factors are relatively more important determinants of health and healthy behaviors.

V. 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 ten, are important

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[11] We display the joint distributions of the endowments in the online Appendix.

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.

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