Contents lists available at SciVerse ScienceDirect

European Economic Review

Child height, health and human capital: Evidence using genetic markers

Stephanie von Hinke Kessler Scholder [a,b,*], George Davey Smith [c], Debbie A. Lawlor [c], Carol Propper [d,e], Frank Windmeijer [d,f]

[a] Department of Economics and Related Studies, University of York, Heslington, York YO10 5DD, UK
[b] CMPO, University of Bristol, 2 Priory Road, Bristol BS8 1TX, UK
[c] MRC Centre for Causal Analyses in Translational Epidemiology (CAiTE), School of Social and Community Medicine, University of Bristol, Oakfield House, Oakfield Grove, Bristol BS8 2BN, UK
[d] CMPO and Department of Economics, University of Bristol, 2 Priory Road, Bristol BS8 1TX, UK
[e] Imperial College London, South Kensington Campus, London SW7 2AZ, UK
[f] Centre for Microdata, Methods and Practice, UK

ARTICLE INFO

ABSTRACT

Height has long been recognized as being associated with better outcomes: the question is whether this association is causal. We use children's genetic variants as instrumental variables to deal with possible unobserved confounders and examine the effect of child/adolescent height on a wide range of outcomes: academic performance, IQ, self-esteem, depression symptoms and behavioral problems. OLS findings show that taller children have higher IQ, perform better in school, and are less likely to have behavioral problems. The IV results differ: taller girls (but not boys) have better cognitive performance and, in contrast to the OLS, greater height appears to increase behavioral problems.

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1. Introduction

The association between height and wealth has been noted in the academic literature for many decades. As early as the 17th Century, Guarinoni – one of the founders of preventive medicine – pointed to the difference in growth rates between the rich in towns and the poor in the countryside (Tanner, 1982). More recent studies find height to be positively related to education (Magnusson et al., 2006) and income (Persico et al., 2004). The advantages associated with greater height have also been reported for children. For example, Case and Paxson (2008) find that taller children perform better in school tests compared to shorter children and suggest that the relationship between childhood height and income and education in adulthood is due to height being associated with greater intelligence.

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* Corresponding author at: Department of Economics and Related Studies, University of York, Heslington, York YO10 5DD, UK. Tel.: +44 1904 32 3084.
E-mail addresses: Stephanie.Scholder@york.ac.uk (S. von Hinke Kessler Scholder), KZ.Davey-Smith@bristol.ac.uk (G. Davey Smith), D.A.Lawlor@bristol.ac.uk (D.A. Lawlor), Carol.Propper@bristol.ac.uk (C. Propper), F.Windmeijer@bristol.ac.uk (F. Windmeijer).

One problem in estimating the relationship between height and outcomes is that the relationship may not be causal. Height is influenced by a wide range of environmental factors experienced in childhood which may be the determinants of the outcomes, rather than height per se, for example, unobserved family wealth or differences in children's nutrition. To the extent that some of these unobserved differences are family specific, one approach is to identify the causal impact from twin or sibling differences in height and outcomes. Case and Paxson (2010) use this approach, exploiting differences between siblings. They conclude that taller children perform better in school, progress faster through school and consider themselves more scholastically competent than their shorter siblings.

However, accounting for fixed unobserved family effects using twin (or sibling) differences does not necessarily eliminate the inconsistency of the conventional cross-sectional estimator and can even aggravate it (Griliches, 1979; Bound and Solon, 1999). The intuition is that taking twin or sibling differences filters out some, but not all, endogenous variation but also filters out exogenous variation. If the endogenous variation comprises as large a proportion of the remaining within-sibling variation as it does of the between-sibling variation, the parameters using within-sibling estimation are as vulnerable to endogeneity bias as that found in between-sibling estimation. For example, some potentially endogenous variation that may remain in a within-sibling estimation are unobserved differences between siblings in nutrition and physical activity, both of which affect growth and final attained height. To argue that the within-sibling estimator is more consistent than the between-sibling estimator, this endogenous variation *as a share of the total variation* should be less in the within than the between-sibling estimation. There is no reason to be confident that this is the case, as the within-sibling analysis also removes exogenous variation, which – together with the endogenous variation – determines the inconsistency (Bound and Solon, 1999).

This paper therefore takes a different approach to estimate the causal effect of child height on children's cognitive and non-cognitive outcomes. Our approach is also called Mendelian randomization, which refers to the use of genetic variants as instrumental variables (IV) to examine the causal effect of an exposure (here height) on outcomes. It exploits the random assignment of an individual's genotype at conception (Davey Smith and Ebrahim, 2003) to enable genetic variants to instrument for a particular phenotype (the trait that the genetic variants is related to, e.g. height).[1] At conception, genes are randomly allocated from parents to offspring. Whilst this random allocation is at a family trio level, at a population level it has been demonstrated that genetic variants are largely unrelated to the many socioeconomic and behavioral characteristics that are closely linked with each other and that confound conventional observational studies (Bhatti et al., 2005; Davey Smith et al., 2008; Kivimäki et al., 2008; Lawlor et al., 2008). Furthermore, since genetic variation is determined at conception, it cannot be affected by later outcomes. Hence, in addition to dealing with fixed characteristics that affect both height and the outcome, Mendelian randomization can also deal with *time-varying* characteristics that affect height and outcomes. Therefore, under certain assumptions that we discuss below, genetic variants will allow us to isolate the causal effect of child height on the outcome of interest.

This paper is the first to exploit genetic variants for height in an attempt to estimate the causal effect of height on cognitive and non-cognitive outcomes for children. We begin therefore by outlining the conditions needed to use genetic variants as instruments. To examine and indirectly test the validity of the IV approach in our context, we show first that the genetic variants are uncorrelated with a large set of family background variables which may confound the relationship between height and outcomes. We then discuss biological pathways of our genetic variants, and run two 'falsification checks'. First, we examine the effect of height on an outcome for which we have clear theoretical reasoning that there should *not* be an effect (maternal education). And second, we investigate the effect of height on an outcome for which we have strong beliefs that there *should* be an effect (body weight). Finding no evidence against the validity of the instruments, we then use the genetic variants as instruments to examine the relationship between height and an extensive set of cognitive, mental health and behavioral outcomes. In so doing, we add to the range of outcomes examined in the previous literature. In addition to children's academic attainment, scholastic competence and self-worth studied by Case and Paxson (2010), we investigate the effects of height on IQ, symptoms of depression and behavioral problems, including hyperactivity, emotional, conduct and peer problems. Note here, that our IQ measure is an index of general intellectual functioning, which is shaped by both inherited and acquired attributes, including any family and environmental influences. In other words, it does not simply measure 'innate' ability.

We use data from a cohort of UK children currently in their late teens (the ALSPAC survey, described below). The OLS results show that taller children perform better in school tests, have higher IQ, and are less likely to have emotional and peer problems, though these relationships differ slightly by gender. Tall girls have higher depression scores, but we find no evidence of differences in self-esteem for children of different heights. The IV results suggest there is a causal relationship between height and cognitive functioning, though only robustly for girls. In contrast to Case and Paxson (2010), we find no evidence that height explains variation in scholastic self-esteem, global self-worth or depression. Further, we find evidence that height confers disadvantage rather than advantage as it *increases* hyperactive behavior (girls), emotional and peer problems (boys). These findings are robust to a set of instrument specification and robustness checks. We discuss the results, relating back to the assumptions made in Mendelian randomization, and speculate about possible reasons for these findings.

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[1] For a brief overview of some of the genetics terms used here, see the glossary in Table 1 and the Appendix.