Commentary: The long and short of why taller people are healthier and live longer

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Research on life course epidemiology and the fetal and childhood origins of chronic disease has grown steadily during the past two decades. It has long been observed that height, a stable marker of early childhood conditions, has a strong negative association with all-cause mortality but heterogeneous association with cause-specific mortality. In their meta-analysis, the Emerging Risk Factors Collaboration1 provides one of the most systematic and comprehensive pieces of evidence of these associations to date, overall and for a large number of causes. These robust findings confirm what has been previously reported in single studies and systematic reviews (e.g. the negative association between height and coronary heart disease) and add clarity where results were previously discrepant (e.g. pancreatic cancer) or lacking (e.g. bladder cancer).

The range of sensitivity analyses provided gives further confidence in the reported associations. A particularly noteworthy finding is that of similar associations for men and women, whereas previous studies found effect modification by gender.2 Although income was not measured, and residual bias due to this and other omitted or poorly measured covariates are a concern, these findings are consistent with what we know: the association between height and morbidity/mortality is robust to adjustment, including adjustment for socio-economic position.3 The same has been found for the association between height and intergenerational outcomes,4 although the association between height and non-health components of well-being may be largely explained by income and education.5 In contrast, there is evidence that the association between traditional measures of socio-economic position and health outcomes is overestimated when height is not included in analytic models.6

The association between height and health outcomes is rarely discussed in a systematic way. Furthermore, from a policy standpoint, separating the importance of investments (e.g. in nutrition, disease reduction or poverty alleviation) from heritability is of particular substantive interest.

Before discussing potential mechanisms, it is important to understand how height is determined. For example, this study states that height ‘…reflects the interplay of genetic endowment and various early life experiences and exposures (such as fetal, dietary, social and psychological circumstances)’. Although correct, this statement flattens what is a hierarchy of determinants. Height is determined by two factors: genetics and net nutrition—net nutrition being the combination of nutritional input and the demands on it from disease, stress, physical activity and so on. The only known mechanism by which socio-economic factors impact on height is through net nutrition.7 Furthermore, growth occurs principally in two periods: in utero up to age 2 years and adolescence before puberty. As they are differentially related to the two periods, analysis of components of adult stature—leg length and trunk length—holds promise in understanding the importance of different childhood conditions as they affect adult disease.

Briefly outlined are six mechanisms that account for the association between height and adult outcomes—including mortality, morbidity, well-being, socio-economic and intergenerational outcomes. The mechanisms are comprehensive; however, they are not distinctly bound; there is overlap between some categories (e.g. between biological and biomechanical: the distinction between organ size and function is not always clear). The purpose is to present a common language that allows for systematic thinking about the association between height and outcomes. There is a complex interrelationship between these mechanisms, and all are likely to be functioning to some extent. An important area of research is both in understanding these relationships and the relative weight of each mechanism in determining outcomes.
Genetic

Genetic factors influencing growth may be tied to mortality or risk of specific diseases through pleiotropic effects (genes related to both height and disease) or due to linkage disequilibrium (variants controlling both height and disease being transmitted together). Although, as the authors state, investigating whether height-related genetic loci are associated with height-related diseases may bear fruit, as height is a polygenic inherited trait—controlled by many genes each with small effects—endowment is likely to account for only a small portion of the variance in the association between height and health outcomes.

Biological

Height is an indicator of health capital. Growth, as well as rate of growth at different periods, has metabolic impacts that translate into lifelong and intergenerational health consequences. In this sense, it is not height per se but height as intrinsically a part of, and inseparable from, good growth and good health that accounts for the observed association. For example, childhood malnourishment impacts on both stature and health, including cognitive development, leading to fewer years of schooling completed and reduced capacity to work. Poor nutrition or disease in early life may induce adaptations in organ function and metabolism which initially raise survival but increase morbidity and mortality in adulthood.

Biomechanical

Height confers advantages and disadvantages related to body and organ size, which have health, productive and reproductive consequences. This mechanism is the most discussed in the present article (e.g. larger coronary vessel diameter for coronary disease or larger organs and greater number of cells for risk of malignancies).

Epigenetic

Changes in gene expression (without changes in DNA sequence) related to environmental influences, through DNA methylation and histone modifications, have been shown to underlie a number of common conditions. Although this is a promising area of research, perhaps in explaining the concurrent high heritability and sensitivity of height to environment, there is little evidence to date of this mechanism underlying any associations between height and outcomes.

Confounding or endogeneity

Other factors associated with both stature and outcomes may account for the associations observed. Income and education are positively correlated with both height and health. There are complex interrelationships and bidirectional causalities between height, health, income and education. Confounders may also be medical conditions causing both height loss and observed disease. Stature is also associated with risk factors, possibly confounding its association with health outcomes. For example, taller people generally smoke less and have lower blood pressure and better diets. However, across studies as in this study, the height association remains robust when controlling for potential confounders.

Psychosocial

Society places a premium on height, and those who are taller are conferred greater social status and exhibit greater confidence. This mechanism may relate to health outcomes principally through socio-economic position, but high self-esteem and social skills may themselves bring about better self-care and preventive behaviours.

The role of prenatal and early childhood influences on adult and intergenerational health has important implications for understanding disease aetiology and designing interventions. Distinguishing biological, biomechanical and epigenetic effects from purely additive genetic and confounding factors remains an important challenge. Birth cohorts with long-term follow-up, including multiple measurements of key covariates throughout the life course, are increasingly available from low- and middle-income countries (see http://www.cohortsgroup.org/) and are an important resource in this regard. Where genome-wide assays are available, new techniques, such as Mendelian randomization, hold promise in unravelling the complex pathways from childhood conditions to adult disease.

Conflict of Interest: None declared.

References

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