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Does height modify the risk of angina associated with economic adversity?

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Abstract

Adult height partly reflects childhood exposures, and we hypothesise that some exposures impairing growth may also increase *susceptibility* to coronary heart disease—angina pectoris (angina)—risks, such that shorter adults may be more susceptible to some exposures in adulthood that are risks for heart disease. This hypothesis is tested among all adults who participated in the National Health Interview Survey (USA), 1997–2000 [The National Health Survey, 1997–2000. Data file documentation, National Health Interview Survey (machine-readable data file and documentation). National Center for Health Statistics, Hyattsville, Maryland, http://www.cdc.gov/nchs/nhis.htm]. In the entire study population, height was negatively associated with angina and after adjustment for potential confounding factors; the odds ratio (and 95% confidence interval) for angina risk associated with the tallest height fifth compared with the shortest fifth is 0.77 (0.97, 0.88). The association of low income (less than US$ 20,000) with angina was assessed separately in each of five height strata defined by fifths of the height distribution. The magnitude of this association is lower in the shortest than the tallest height fifth, with odds ratios of 1.18 and 1.60, respectively (effect modification). The unexpected results may be explained by the following: childhood adversity resulting in shorter stature may confer resilience against adult economic adversity; the relative disadvantage of low income may be perceived more keenly by those of taller stature thereby increasing stress and thus disease risk; or health-promoting characteristics associated with taller stature may be less effective in the face of adult economic adversity in the low-income group.

JEL classification: I1

Keywords: Height; Growth; Income; Angina; Resilience; Health; Morbidity; Physical stature; US; National Health Interview Survey

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1. Introduction

Taller stature is associated with reduced cardiovascular mortality and morbidity (Langenberg et al., 2005a). Shorter adults are at greater risk of death from cardiovascular causes and while this could be due to a direct effect on cardiovascular function (Smulyan et al., 1998) other explanations are likely. Adult height reflects genetic as well as childhood environmental factors and factors influencing growth may also influence later coronary heart disease (CHD) risk. This could be due to influences on physiological mechanisms relevant to blood pressure, as impaired growth in childhood has been associated with higher blood pressure in early old age, independent of adult height (Montgomery et al., 2000). This is plausible, as factors associated with CHD risk such as material circumstances and psychosocial exposures can also influence child growth (Skuse et al., 1996; Voss et al., 1998). If some childhood exposures – associated with impaired growth and shorter adult stature – influence physiological blood pressure control mechanisms as well as CHD risk, then shorter stature may be a marker of greater *susceptibility* to exposures that are risks for CHD. Such susceptibility has been demonstrated, as psychosocial stress in adult life was associated with an increase in systolic blood pressure only among those who experienced impaired childhood growth (Montgomery et al., 2000).

The majority of research into disease aetiology is concerned with risk or protective factors, where measures are associated with an increased or decreased risk of disease. However, this paper is concerned with effect modification: if height *modifies* the association between an exposure and CHD. Our a priori hypothesis is that among shorter adult subjects, adverse adult exposures (low income) will be more powerfully associated with CHD, denoting greater susceptibility to such exposures. Conversely, we hypothesise that taller stature will confer resilience against adult exposures associated with CHD such as low income.

Economic adversity in adult life is associated with an increased risk of CHD morbidity and mortality (Davey Smith et al., 1998; Hemingway et al., 2000). We use low income in adulthood as the exposure representing a CHD risk. CHD is defined by a diagnosis of angina pectoris. We test the hypothesis that the association of economic adversity (low income) in adulthood with angina risk is modified by height.

2. Subjects and methods

Each year the National Health Interview Survey (NHIS) selects a stratified random sample of households in the USA (The National Health Survey, 1997–2000). Information is collected on households and on one adult sampled from within these households (Botman et al., 2000; Pleis et al., 2003). The target for this analysis is all sampled subjects above 18 years of age in the years 1997–2000 ($n = 116{,}587$). A sub-analysis of more detailed income data was limited to 97,408 subjects (83.5%) with valid data for the relevant income questions. Survey participation is voluntary and the annual response rate of NHIS is greater than 90% of the eligible households in the sample.

3. Measures

This analysis uses information on adult height, weight, BMI, sex, smoking, and the answer to the question ‘‘Have you EVER been told by your doctor or other health professionals that you had angina, also called angina pectoris?’’ This information was collected every year throughout the study period. Data were collected through a personal household interview conducted by