Commentary: The association between height growth and cholesterol levels during puberty: implications for adult health

Rebecca Hardy and Claudia Langenberg

High cholesterol is an important risk factor for coronary heart disease (CHD). It is involved in the development of atherosclerotic changes of the arterial wall, which have been shown to begin at an early age, even in apparently healthy children and adolescents. Levels of cholesterol and other cardiovascular risk factors in childhood are also known to track into adult life and influence subsequent subclinical and clinical cardiovascular disease. Studies of predictors of childhood cholesterol are therefore of importance for primary prevention and may help to identify those particularly susceptible to later hyperlipidaemia, atherosclerosis, and cardiovascular disease at an early age. A paper in this issue of the International Journal of Epidemiology reports negative associations between height and total cholesterol in Japanese boys and girls around the time of puberty. As well as cross-sectional associations the authors find that changes in height were negatively associated with changes in total cholesterol. An important question is what are the implications of these findings for later health?

Short stature in adulthood has been consistently linked with increased CHD morbidity and mortality of CHD and also with unfavourable levels of CHD risk factors, including cholesterol. Adult height is determined by genetic factors as well as early environmental influences on growth. Which particular period of growth may be crucial for the initiation and progression of cardiovascular disease remains to be determined.

Few studies have considered the association between childhood growth and later cholesterol levels. A previous study in...
Japan found that a greater percentage change in height between 3 and 20 years was associated with lower total cholesterol at age 20 years. A study of an elderly Finnish cohort which considered lipoprotein subfractions found that rapid height growth between 7 and 15 years of age was associated with lower levels of high density lipoprotein (HDL) cholesterol, but not with low density lipoprotein (LDL). Total cholesterol was not studied. The Japanese study thus implies that rapid childhood and adolescent growth is beneficial in that it lowers total cholesterol. The Finnish study, however, suggests that such rapid growth may reduce the HDL- rather than the LDL-subfraction.

Indirect evidence regarding the important stage of growth is provided by the observation that leg length in adulthood is the component of height most strongly associated with CHD risk and its risk factors, including cholesterol, possibly through it’s link with insulin resistance and associated metabolic disturbances. Prepubertal height growth is influenced to a greater extent by leg rather than trunk growth, while both components of height grow equally in the postpubertal period. These findings therefore suggest that it is prepubertal growth, possibly acting through childhood exposures such as infection, diet, and stress, which influence later CHD risk and cholesterol levels.

It has been argued that the association between height and CHD might be due to fetal growth, possibly through lower risk of adult disease and also leading to shorter adult stature. Findings relating birthweight, a commonly used proxy measure for fetal growth, and CHD events and blood pressure have been somewhat stronger and more consistent than findings for cholesterol. It has been suggested, however, that other measures of birth size such as short length are associated with the programming of lipids. There are several pieces of evidence suggesting that fetal growth and height growth are independently associated with CHD risk. Specific to cholesterol, the study in Japanese 20 year olds indicated that the effect of change in height between 3 and 20 years was independent of the effect of birthweight. Similarly, a study in Jamaican children suggested that being both short at birth and short in childhood were independently related to cholesterol levels. Further, studies have found similar correlations between birthweight and both leg and trunk length. If birthweight explained the association between height and CHD risk, then birthweight should be more strongly predictive of leg length than trunk length.

How can the new evidence be understood in the light of previous research? Two important issues must be considered in the interpretation of the findings presented by Kouda et al. First, changes in height are generally accompanied by changes in body composition and weight is correlated with height. Body mass index, designed to be a measure of adiposity that is independent of height, may be more appropriate in this context. In addition, previous studies have suggested that central adiposity, rather than total obesity, is more closely associated with insulin resistance and dyslipidaemia compared with weight or relative weight. Hence, the role of changes in adiposity may have been underestimated in this study and may be of greater importance than height growth.

Secondly, puberty is highly influenced by early growth, possibly even prenatal growth, and late puberty has been shown to lead to taller adult height. Height changes dramatically during pubertal growth spurs and the precise stage of puberty is an important factor to consider in any study of height during that period of life. Many physiological changes occur during puberty, and decreases in cholesterol are just one example. To distinguish transitional from long-term changes which may reflect a risk that tracks into adulthood is therefore crucial. Although the Japanese study indicates that changes in height were negatively related to changes in total cholesterol over the same time period, it is impossible to distinguish whether postpubertal levels of cholesterol are dependent on the extent and/or timing of pubertal growth or whether once all children have gone through puberty cholesterol level is simply dependent on final attained height.

Given the potential influence of early life factors on adult cholesterol levels, we might wish to screen those children who will be at risk of CHD in later life so that preventive measures can be implemented early. The long-term implications of the findings from studies in childhood must therefore be understood. The importance of further information from birth cohorts followed through to adulthood where repeated measures of height and weight throughout childhood and pubertal development are available is thus highlighted.

References