Diet, height, and health

Joseph Yeboah

Heart and Vascular Center of Excellence, Wake Forest University School of Medicine, Winston-Salem, NC

A careful review of secular trends from the 20th century, especially in the Western world, shows a general increase in average adult height, a reduction in cardiovascular and infectious diseases, and an increase in cancer diagnosis and life expectancy, among others (1, 2). However, whether these secular trends are parallel and unrelated events driven by our progress in science and development or intricately related to a common trigger remains a mystery today. Nonetheless, these observations continue to intrigue investigators and have led to important publications in the literature.

Adult height results from a combination of genetic and environmental factors, especially during childhood (3–5). The heritability of height is reported as being between 0.60 and 0.95 (3), and height is less heritable in women than in men (4, 5). Thus, 60–95% of the variance in height within a population can be accounted for by the variance of genes within that population. It is plausible that the increasing average adult height is a preferred trait being propagated by natural selection. However, unlike genetically specified traits, environmental factors that begin during gestation also play a significant role in determining adult height. In this issue of the Journal, Morency et al. (6) provide an example of an association between diet (environment) and childhood height in the TARGet Kids Collaboration study. With the use of a cross-sectional approach, the authors show a dose-dependent association between higher noncow-milk consumption and lower height in 5034 children. It should be noted that several environmental factors also influence the transition from childhood height to adult height.

Numerous but sometimes conflicting findings in the literature have associated adult height with morbidity and mortality. These conflicting results are often attributed to confounders in these studies, such as differences in genetic and socioeconomic factors, education, health care delivery, and lifestyle. For example, although greater adult height has been associated with lower rates of cardiovascular disease and all-cause mortality in Western societies, scores of studies in some developed and developing populations have shown contrary findings (7). In addition, despite the increasing prevalence of cardiovascular diseases in the developing world, Western populations who are much taller still have a significantly higher cardiovascular disease prevalence and burden than do shorter populations in developing countries. Also in this issue of the Journal, Ma et al. (8) used data from the Nurses’ Health Study to assess the association between adult height and healthy aging, defined as the absence of 11 major chronic illnesses and no reported impairment of subjective memory, physical impairment, or mental health limitations in women. The authors also assessed the effects of various lifestyle-including dietary and physical activities on the association between adult height and healthy aging. The authors found that greater adult height was associated with a modest decrease in the likelihood of healthy aging, and among the lifestyles assessed, a prudent diet rich in fruit and vegetables might modify the relation. Thus, as the average adult height in women increases according to current secular trends, the prevalence of healthy aging will decrease.

Morency et al. (6) and Ma et al. (8) should be commended for their contributions to this subject. So, should we encourage more noncow-milk consumption in childhood in order to produce relatively shorter individuals and ultimately promote healthy aging in our societies? To address this question, one must consider the quality of data so far produced on this subject and the difficulty of proving causality in general. Almost all of the data on the association between diet and height, and height and diseases, are retrospective or observational. Observational and retrospective studies tend to be heavily confounded, and hence often produce conflicting results in different cohorts and populations as evidenced by the literature on the present topic (9). Observational and retrospective studies are, at best, hypothesis-generating and their findings should be interpreted or adopted with caution. Undoubtedly, childhood diet, such as with the consumption of milk other than cow milk, may have an effect on childhood height as shown by Morency et al. (6); however, even though more and more Canadians are feeding noncow milk to their children according to the authors, secular trends show that the average height, including of Canadians, is increasing. Thus, despite the plausible “lack of IGF-I [insulin-like growth factor I] in noncow milk” mechanism or hypothesis provided by some authors, the association between noncow-milk consumption and childhood height may be more complicated and the results provided may not tell the whole story.

As shown by Ma et al. (8), adult height may be associated with or may influence healthy aging. However, it is also plausible that the increasing adult height and the upward and downward trends of various diseases in society are parallel and unrelated events brought about by advances in science, modernization, and...
changes in environmental factors as a whole. If the latter were true, then targeting and changing the current trend of increasing adult height, even from childhood by feeding children noncow milk, would have no effects on the prevalence of these diseases in adulthood, because they are parallel and unrelated events. Until better statistical methods and study designs that eliminate confounding in observational and retrospective studies are developed, we should all fasten our seat belts and enjoy the seesaw ride.

The author did not report any conflicts of interest.

REFERENCES

1. Fogel RW, Grothe M. Major findings from the changing body, health, nutrition and human development in the Western world since 1700. J Econ Asymmetries 2011;8:1–9.