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A Short Note on Link between Weight Gain, Aging, and Coronary Atherosclerosis

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Introduction

The escalating global prevalence of obesity has become a recurring topic of discussion in both medical and scientific literature, as well as in the media. Data provided by the NCD Risk Factor Collaboration network present disheartening statistics. For instance, projections indicate that by 2025, the worldwide prevalence of obesity will reach 18% among men and exceed 21% among women [1]. Various population-based cohorts have consistently demonstrated a progressive escalation in the risk of morbidity and mortality corresponding to higher body mass index (BMI) values. BMI is widely utilized as an anthropometric index to broadly classify individuals into normal weight, overweight, and obesity categories [2]. Numerous studies have also established a correlation between obesity and cardiovascular disease (CVD) on a population level [2,3]. However, despite overwhelming evidence that increased body weight, adjusted for height, heightens the risk of cardiovascular outcomes, obesity has not been identified as an independent risk factor for CVD when intermediate risk factors such as blood pressure, lipids, and diabetes are considered [4]. Consequently, although excess adiposity, as assessed by elevated BMI, contributes to intermediate risk factors, there is currently insufficient evidence that weight loss through lifestyle modification programs reduces CVD outcomes. It is crucial to note that the surge in obesity is not limited to adults alone. The prevalence of overweight and obese children and adolescents is alarmingly high and escalating in various regions worldwide [5]. This trend elicits significant concern, not only due to the associated short-term cardiometabolic complications like dyslipidemia, hypertension, and type 2 diabetes [6], but also because it facilitates the early development of atherosclerosis long before adulthood [7]. Indeed, the seminal work of Henry McGill has documented that atherosclerosis initiation occurs at a young age, with a growing number of young individuals developing atherosclerosis, particularly when risk factors are present [8]. For example, the Pathobiological Determinants of Atherosclerosis in Youth (PDAY) study investigated the natural history of atherosclerosis in approximately 3,000 young subjects aged 15 to 34 years who died accidentally [8,9]. Post-mortem arterial examinations were conducted along with lipoprotein-lipid profiling and assessment of other cardiometabolic risk factors, including panniculus adiposus thickness and BMI as indicators of adiposity. Young men with obesity displayed a higher prevalence of pronounced fatty streaks and raised lesions specifically in the right coronary artery [8,10]. Additional analyses from the PDAY study revealed significant links between obesity and accelerated coronary atherosclerosis, particularly in young men [10]. The relationship between this phenomenon and an increased risk of developing coronary heart disease was also explored, yielding mixed results. Some studies found that increased body weight during adolescence or early adulthood correlated with an elevated risk of coronary heart disease [11,12], while others did not find the same association [13].

To further investigate the impact of excessive body weight during young adulthood and the long-term effects of weight changes on coronary atherosclerosis, researchers from the Swedish Cardiopulmonary bio Image Study (SCAPIS) employed noninvasive imaging techniques. Their aim was to quantify the extent of atherosclerosis in the coronary and carotid arteries and gather data from proteomics, metabolomics, and genomics technologies to enhance the prediction of cardiovascular disease (CVD) risk. In the present study published in Atherosclerosis, Bergström et al. [14] explore the risk of midlife coronary atherosclerosis based on body weight factors such as weight at 20 years of age (as self-reported by participants), weight measured during midlife, and changes in weight over that period. The SCAPIS sample consisted of 25,181 participants, with 51% being females, and no history of myocardial infarction, percutaneous coronary intervention, or coronary artery bypass graft. The age of the participants was 57 years. Coronary atherosclerosis was assessed using coronary computed tomography angiography, and the total burden of coronary atherosclerosis was calculated using the segment involvement score (SIS), which measures the overall number of coronary segments affected by atherosclerosis regardless of the degree of stenosis.

After accounting for various factors such as site, age, height, LDL-cholesterol, smoking, alcohol intake, education status, and level of physical activity, it was observed that a higher body weight at age 20 correlated with an increased likelihood of having coronary atherosclerosis in both men and women. Similarly, body weight during midlife (50-64 years) was also associated with a greater probability of having a segment involvement score (SIS) greater than zero. Even after adjusting for additional factors such as systolic blood pressure, type 2 diabetes, and treatment for dyslipidemia and hypertension, these associations remained largely unchanged. When alternative measures such as Duke scores or coronary artery calcification scores were utilized instead of SIS, similar conclusions were drawn. However, when analyzing weight changes from age 20, the associations were less pronounced and only significant in men with higher body weight during early adulthood. No significant relationship was found between weight changes and coronary atherosclerosis in women. The SCAPIS investigators deserve praise for conducting a large-scale cardiometabolic imaging study that included coronary artery images from over 25,000 men and women. Firstly, the results confirm that higher body weight during early adulthood serves as a predictive factor for increased midlife coronary atherosclerosis, regardless of the scoring method employed. This finding highlights the significance of primordial prevention [15].

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Regrettably, the alarming rise in obesity among children, attributed to sedentary behaviors and insufficient physical activity, paints a grim picture for the future [16]. It is crucial that promoting healthy lifestyles becomes a top priority, starting with educational institutions and the creation of environments that facilitate healthy eating habits and an active lifestyle for everyone. Additionally, the study revealed that midlife body weight exhibited an association with coronary atherosclerosis, which aligns with the existing literature on the subject [17]. However, the lack of a substantial correlation between weight changes since early adulthood and midlife coronary atherosclerosis may initially seem counterintuitive. Nevertheless, several factors could account for these findings. For instance, using body weight as a sole anthropometric measure to assess overweight and obesity may have led to misclassification of individuals in terms of changes in body composition and distribution of body fat [18]. While some weight gain is a natural consequence of healthy aging, it is important to consider the composition and distribution of this weight gain. For example, an individual who engages in regular physical activity may experience an increase in muscle mass with limited changes in body fat, whereas a sedentary individual may experience a loss of muscle mass and an increase in adipose tissue, particularly in the abdominal visceral region [19]. These individual variations in body composition and changes in adipose tissue distribution are likely to significantly influence the rate of coronary atherosclerosis progression. Therefore, the researchers have an opportunity to further investigate these aspects within their extensive database. What are the outcomes for male and female participants who exhibit similar weight gain but differ in terms of their level of physical activity or visceral adiposity? In addition, since the authors possess imaging data, it would be intriguing to investigate the impact of ectopic fat depots on coronary atherosclerosis, beyond just weight changes. Variations in visceral adiposity, epicardial adiposity, liver fat, and skeletal muscle fat are all factors that could potentially elucidate why individuals with the same weight gain experience varying degrees of coronary atherosclerosis in midlife. Additionally, menopause has been linked to an accelerated accumulation of visceral adipose tissue [20]. Considering that approximately 75% of the female participants in the SCAPIS study were above 54 years of age, it is highly probable that a significant subset of them were post-menopausal during the examination. This aspect has not been accounted for in the analyses and warrants further investigation. Furthermore, exploring the use of hormonal replacement therapy, known to affect body fat distribution, could provide additional insights. Once again, the SCAPIS investigators deserve commendation for their exceptional cardiometabolic imaging study. The study's findings, specifically the association between current coronary atherosclerosis assessed in midlife and self-reported body weight at age 20, underscore the importance of early intervention, both in clinical settings and through population-based initiatives. These findings align with the notion that tracking weight changes over several decades is an overly simplistic approach to identifying individuals at high risk of coronary atherosclerosis. Monitoring weight changes throughout adulthood is akin to merely observing the tip of a potentially life-threatening iceberg. However, given the extensive data collected by SCAPIS, we can anticipate numerous additional discoveries in the coming months/years that will emphasize the significance of focusing on key behaviors (such as measuring moderate to vigorous physical activity using accelerometry) as well as indices of body fat distribution (such as visceral and ectopic adiposity) and simple clinical tools like waist circumference.

Conclusion

In the meantime, the findings of this study highlight the crucial need to take early action and bridge the divide between clinical and population-based approaches. This is essential if we aim to prevent future generations from entering adulthood with premature coronary atherosclerosis. The ongoing epidemic of obesity can be better characterized as a lifestyle epidemic, stemming from a socioeconomic model that frequently hinders the adoption of healthy behaviors. Addressing the issue of premature atherosclerosis caused by high-risk forms of overweight and obesity will necessitate more than just medical interventions. It calls for comprehensive solutions that address the broader societal and environmental factors contributing to unhealthy lifestyles.

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Not applicable.

Conflict of Interest

Author declares no conflict of interest.

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