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Review

Physical activity, sedentary behavior, and long-term cardiovascular risk in young people: A review and discussion of methodology in prospective studies

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Abstract

The long-term effects of physical activity (PA) or sedentary behavior on cardiovascular health in young people are not well understood. In this study, we use a narrative format to review the evidence for a prospective association with adiposity and other well-established biological cardiovascular risk factors in healthy young people, considering only studies with at least 2 years of follow-up. PA appears to elicit a long-term beneficial effect on adiposity and particularly markers of cardiovascular health. With adiposity, however, a few studies also reported that higher levels of PA were associated with higher levels of adiposity. Time spent sedentary does not appear to be related to adiposity or markers of cardiovascular health independent of PA. We then discuss the uncertainties in the underlying causal chain and consider a number of alternative modeling strategies, which could improve our understanding of the relationship in future studies. Finally, we consider the current methodology for assessing PA and sedentary time.

Keywords: Adiposity; Adolescents; Cardiovascular health; Children; Long-term; Metabolic syndrome score

1. Introduction

Cardiovascular disease (CVD) continues to be the number one cause of death and disability in the world.[1,2] While CVDs such as coronary heart disease and stroke manifest clinically in middle age or older adulthood, their origins begin much earlier.[3] Exposures acting during growth and maturation may exert long-term effects on cardiovascular physiology and have a major impact on the development of CVD. For example, evidence shows that alterations in fetal environment such as exposure to maternal smoking and nutritional status, childhood socioeconomic position and overweight independently predispose to development of CVD in adulthood.[4–6] Similarly, lack of physical activity (PA) during childhood and youth may lead to cardiovascular disturbances and progression of atherosclerosis that could contribute to development of CVD in adulthood. Indeed, organizations and governments have identified a population-wide promotion of healthy lifestyle including PA in young people as a key priority for primordial prevention of CVD.[7] A major advance in population-based studies of the influence of childhood or youth PA on long-term cardiovascular health outcomes has been the application of objective methods to assess PA that prevents bias related to recall and social desirability.[8] This has also facilitated the possibility to obtain detailed information on frequency, duration, and intensity of PA.

Two earlier reviews, published in 2010 and 2011, respectively, systematically addressed studies on the observational associations between objectively assessed PA and adiposity outcomes.[9,10] These reviews highlighted the importance of distinguishing between cross-sectional and prospective studies as Jimenez-Pavon and colleagues[9] found a negative association

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in 32 of 41 cross-sectional studies (78%), while Wilks and colleagues,[10] who only considered prospective studies, found a negative association in 4 of 10 studies (40%). Because cross-sectional studies have a number of limitations, including inability to infer causality, this inconsistency is important to address by considering recently published experimental and prospective studies. In 2014, Tanaka and colleagues[11] published a review on the prospective association between changes in objectively measured sedentary behavior (SED) and measures of adiposity. However, by imposing this restriction the authors were only able to include 3 studies. The review found evidence for a positive association in 1 of the 3 studies. In summary, the previous evidence synthesis did not clearly support a prospective association between PA/SED and adiposity outcomes. In contrast to these reviews on adiposity outcomes, we are aware of no published reviews on the prospective association between PA and biological risk factors in young people.

This narrative review aims to give an overview of the evidence from population-based studies relating objectively assessed PA and SED in childhood or youth with long-term (≥2 years) cardiovascular risk factor progression. In addition, we will discuss the current methodological challenges and future direction in the objective assessment of PA in large-scale studies following young people over time.

2. Scope of the review

In our discussion we will consider PA as a behavior separately from SED. This is because a body of literature has identified behaviors such as TV-viewing[12,13] and total sitting time[13] to predict all-cause and cardiovascular mortality in adults, independent of other domains of PA. Our discussion will consider observational studies which

- Used an objective measure to quantify whole-day PA (i.e., total activity, light PA (LPA), moderate PA (MPA), moderate-to-vigorous PA (MVPA), or vigorous PA (VPA)) or SED at baseline.
- Included healthy, population-based samples of children and adolescents aged ≤18 years at baseline and followed the same individuals for a period of ≥2 years.
- Related an exposure to any form of the conventional biological risk factors (recommended for use in CVD risk stratification among asymptomatic adults[14]) with the addition of indices of insulin-resistance. We considered all forms of adiposity outcomes.
- Were published after October 29, 2009, as this was the final search date in the review by Wilks and colleagues[10] (applies for PA–adiposity investigations only).

We identified relevant studies from our records by conducting a search on PubMed using combinations of the relevant exposures and outcomes and by going through the reference lists of the identified studies. As there are a greater number of studies on adiposity outcomes than studies on biological risk factors, we will address these in separate paragraphs. Furthermore, we will briefly discuss findings from controlled or randomized controlled intervention studies conducted in a general (young) population.

3. How do PA and SED associate with adiposity and biological risk factors?

3.1. PA and adiposity

We identified a total of 13 published studies which fulfilled our criteria.[15–27] The duration of follow-up ranged from 2 to 7 years with 9 studies covering ≤3 years.[15–18,22–25,27] The studies included samples aged 4–18 years at baseline. The median (25th–75th quartiles) study size was 554 (315–984). Twelve studies used accelerometry as their exposure variable, while 1 used a combination of accelerometry and heart rate.[16] The outcome variables were more heterogeneous. Nine studies (69%)[17–20,22–24,26,28] reported a significant association between PA and a measure of adiposity such that a higher level of PA was associated with a lower level of adiposity (negative association). Surprisingly, 3 studies reported significant associations in the opposite direction.[16,20,23] When examining the relative importance of specific PA intensities, significant negative associations were apparent with LPA in 0 of 3 studies (0%), with MPA in 1 of 4 studies (25%)[17] and with VPA in 2 of 4 studies (50%).[17,24] Finally, significant negative associations with adiposity were found for MVPA in 7 of 8 studies (87%).[18–20,23–26] Four studies included SED as a covariate, which generally had a small effect on the estimates and only affected the conclusion in 1 study.[25] The choice of outcome measure in adiposity studies appeared of importance as all studies (4 in total) that considered MVPA and used a reference method for assessing adiposity such as DXA or densitometry[29] reported a negative association with adiposity.[18,20,23,26] In contrast, 6 of the MVPA studies used a body mass index (BMI)-based measure with significant negative associations to adiposity found in 3 studies, while 2 studies found significant positive associations. The latter appears to be explained by an increase in muscle or bone tissue as both of these studies also reported significant associations between PA and fat free mass. If PA associates with fat free mass, which is suggested in the literature,[30,31] future studies should use methods which are accurate enough to separate fat mass from lean mass.

3.2. SED and adiposity

We identified a total of 9 published studies which fulfilled our criteria.[15,20,21,24–27,32,33] The duration of follow-up ranged from 2 to 7 years with 5 studies having ≤2.5 years of follow-up.[15,24,25,27,32] The studies included youth 4–18 years at baseline. The median (25th–75th quartiles) study size was 554 (403–984). Eight studies used accelerometry, while 1 used a combination of accelerometry and heart rate.[32] The measures used for outcomes were more heterogeneous. Two studies (22%)[25,33] reported a significant association between SED and adiposity; more time spent on SED was associated with a higher level of adiposity. One study found an association in the opposite direction, i.e., higher SED associated with lower adiposity.[20] Of the 2 studies reporting a positive association, 1 used a relatively high cut-point to define SED (<1100 counts/min (CPM)),[25] which is very likely also to include time spent on LPA.[34] This finding is surprising as none of the 3 studies examining LPA separately from SED found an association and 1 of these even found a negative association.[16,17,27] Six studies used a