Commentary: Physical functioning and coronary heart disease development: is physical activity the missing link?

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SF-36 is a measure of non-condition-specific health status that was originally designed to assess the patient’s subjective efficacy of interventions. Although SF-36 has been in use for nearly 20 years, its utility as a predictor for cardiovascular disease (CVD) risk has received little attention. In this issue, Myint et al. used SF-36 to assess the relationship between ‘physical functional health’ and incident coronary heart disease (CHD) over an average follow up of 9 years in a British cohort of 40–79-year olds at baseline. The relative risk reduction for every standard deviation of the SF-36 physical component summary (PCS) score was 24%. Results were robust to comprehensive adjustments for common confounders (including body mass index, physical activity, blood pressure, diabetes and smoking) and generous steps to address reverse causality, such as exclusions of events occurring during the first 6 years of follow-up.

SF-36 includes eight subscales: physical functioning, social functioning, role limitation due to emotional problems, mental health, energy/vitality, pain and general health perception. The three dominant subscales that contributed the most to the PCS score in the Myint et al. study was physical functioning (limitations in physical activity of various intensities due to poor health), role-physical (role limitation due to physical problems) and bodily pain. The study findings suggest a temporal sequence of CHD development that has face plausibility: chronic exposure to unhealthy behaviours and adverse psychosocial states may lead to the development of non-specific symptoms of poor health (such as compromised daily activities, bodily pain, etc.) that have not reached levels of clinical severity and specificity to warrant medical attention. This compromised health state that is captured by SF-36 may accelerate further declines in physical activity and deterioration of emotional states and eventually lead to the clinical expression of CHD (or stroke, as noted previously by the same authors).

In particular, the role of physical inactivity in interpreting these results merits further discussion. Physical inactivity was the most prevalent risk factor in this cohort (like in most population cohorts) ranging from >65% (bottom tertile of PCS score) to approximately 48% (top tertile). There is good cross-sectional and some randomised controlled trial (RCT) evidence, suggesting a relationship between physical activity and physical functioning. Physical inactivity is considered by many clinical researchers and practitioners only as a ‘predisposing’ CVD risk factor due to the unfeasibility and non-ethicality of long-term physical activity RCT studies with mortality as an end point and due to lack of complete understanding of its mechanisms of action. Nevertheless, it is generally accepted that physical activity is causally linked to surrogate CVD risk factors such as obesity, dyslipidemia, and hypertension that have causal status in medical literature. Cardiorespiratory fitness, an attribute that is largely determined by physical activity, has been shown to be more important than weight status in determining the relationship between PCS score and diabetes. A meta-analysis of RCTs among adults aged ≥50 years found that physical activity leads to measurable improvements in physical functioning. The benefits of physical activity in physical functioning seem to be independent of weight status. Although Myint et al. did adjust for physical activity, it may be that this is not an adequate measure to eliminate the possibility of dependence of the observed relationships from physical activity behaviour: first, self-reported physical activity contains unquestionably larger measurement error than other confounders like smoking that can be measured relatively easily or are objectively assessed, such as body mass index (BMI) and blood pressure. Secondly, due to the inherent limitations of longitudinal designs with exposure assessments only at baseline, age-related decreases of physical activity during follow-up may dilute considerably its effect on mortality. This latter
point is particularly pertinent here as the compromised physical functioning that SF-36 captured would accelerate the age-related physical activity reductions between baseline and follow-up. For these reasons, and despite the good design of the study, the independence of SF-36 from current and previous and baseline physical activity may be questionable.

As the authors suggest, further research is needed to understand the plausible mechanisms of how functional health affects the risk of CHD, or this could be another example of well-conducted black-box epidemiology. Disentangling these mechanisms will not be an easy task, considering how broad the concept of functional health is. Working towards understanding better how physical activity at each life stage influences current and future functional health might be a good place to start from.

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**References**