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Commentary

Beyond moving more: Physical activity intensity, physiological reserve, and cardiovascular aging

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Received: 20 June 2026 | Approved: 06 July 2026 | Online: 06 July 2026

Physical activity has long been recognized as a cornerstone of chronic disease prevention and healthy aging. Current guidelines are largely structured around weekly volume, recommending specified durations of moderate physical activity (MPA), vigorous physical activity (VPA), or an equivalent combination of the two[1]. This volume-based framework has clear public health utility, yet it may obscure a critical question for cardiovascular aging: does the intensity distribution of physical activity provide information beyond that captured by total accumulated volume? In the aging cardiovascular system, the preservation of physiological reserve may depend not only on how much individuals move, but also on whether the movement is sufficiently intense to elicit adaptive vascular, metabolic, and cardiorespiratory responses[2]. The study by Wei *et al.*, *Volume vs. intensity of physical activity and risk of cardiovascular*

and non-cardiovascular chronic diseases, offers a timely opportunity to revisit this question[3].

The central contribution of this study lies in its treatment of physical activity volume and intensity as related yet distinct exposure dimensions, rather than regarding VPA merely as a time-efficient substitute for MPA. Using data from the UK Biobank, the authors analyzed 96,408 participants with wrist-worn accelerometer measurements and 375,730 participants with self-reported physical activity[3]. In the device-measured cohort, the proportion of VPA (%VPA) was defined as VPA MET-min/week divided by total physical activity MET-min/week. Importantly, the highest accelerometer-derived category was > 4% VPA, a threshold reflecting the population distribution of measured activity rather than an athletic or high-volume exercise exposure[3]. This distinction carries clinical relevance: the study does not suggest that only structured vigorous exercise matters, but rather that even a modest vigorous fraction within total activity may convey prognostic information. A long walk, a brisk climb up stairs, a short burst of cycling, and prolonged light occupational movement may all contribute to energy expenditure, yet they may differ substantially in hemodynamic load, autonomic activation, skeletal muscle recruitment, and metabolic demand[2,4]. By examining both cardiovascular and non-cardiovascular outcomes, the authors position intensity at the center of a broader prevention framework[3].

Wei *et al.* found that a higher %VPA was associated with lower risks of multiple chronic disease outcomes, even after accounting for total physical activity volume[3]. These findings challenge the assumption that activity intensity can be fully reduced to minutes or metabolic-equivalent totals. Prior investigations have similarly suggested that both total physical activity and the fraction performed at higher intensity are associated with cardiovascular risk, mortality, and other major outcomes[4-6]. The present study extends this literature by systematically comparing the relative contributions of volume and intensity across a broad spectrum of cardiovascular and non-cardiovascular diseases.

For the clinician and researcher, the most important implication lies in the biology of cardiovascular aging. Aging is accompanied by declining endothelial function, arterial stiffening, impaired mitochondrial capacity, reduced cardiorespiratory fitness,

autonomic dysregulation, chronic low-grade inflammation, and diminished metabolic flexibility[2,7]. VPA may provide a more potent physiological challenge than an equivalent volume of low-intensity activity, with potential relevance to endothelial adaptation, nitric oxide–dependent vascular function, oxidative stress regulation, inflammatory signaling, mitochondrial fitness, and cardiorespiratory reserve[2,7]. In this sense, intensity may not simply increase the "dose" of physical activity; it may also shape the biological quality of the stimulus imposed on an aging cardiovascular system. Nevertheless, this interpretation remains mechanistic and hypothesis-generating, and requires direct testing in interventional studies, particularly among older adults with reduced functional capacity.

A further strength of the study is its disease-sensitive perspective. The authors reported that the relative contribution of %VPA and total physical activity volume differed across disease outcomes[3]. Immune-mediated inflammatory diseases showed a stronger intensity-associated pattern, whereas cardiometabolic outcomes, including type 2 diabetes, metabolic dysfunction-associated steatotic liver disease, and chronic kidney disease, appeared to benefit from both volume and intensity[3]. This heterogeneity argues against a universal dose-equivalence model. Instead, different disease pathways may respond to distinct features of physical activity: some may be more sensitive to cumulative energy expenditure, whereas others may depend more strongly on peak physiological challenge, inflammatory modulation, or vascular adaptation[3,4].

These findings have implications for exercise prescription, though clinical translation should remain cautious and individualized[3]. For sedentary adults with limited time, small increments of vigorous lifestyle activity, such as stair climbing, short uphill walking, fast walking intervals, or brief cycling bursts, may represent a feasible means of introducing higher-intensity stimuli, consistent with emerging evidence on vigorous intermittent lifestyle physical activity[5,8]. However, the message should not be simplified to "vigorous activity for everyone." For older adults and individuals with atrial fibrillation, established cardiovascular disease, frailty, multimorbidity, or musculoskeletal limitations, intensity should be prescribed as a tolerable progression rather than a categorical mandate. Screening, gradual dose escalation, symptom monitoring, and shared decision-making remain essential[1,9]. For some individuals, reducing sedentary time, increasing total activity volume, improving strength, or

enhancing balance and functional capacity may represent a safer and more clinically appropriate first step[1,9].

Several methodological considerations warrant caution in interpretation. First, %VPA is a compositional measure embedded within total physical activity volume. Its interpretation is therefore closer to a substitution question: what happens when a larger fraction of activity is performed vigorously at a given total volume, than to an independent exposure contrast. Second, the absence of vigorous activity may reflect more than behavioral preference; it may indicate subclinical disease, reduced physiological reserve, chronic symptoms, functional limitation, socioeconomic disadvantage, or early frailty. Although Wei *et al.* performed multivariable adjustment and sensitivity analyses, including analyses addressing frailty, residual confounding and reverse causation remain difficult to exclude in observational data[3]. Third, accelerometer measurements were obtained over a limited observation window and may not capture long-term trajectories of physical activity intensity, which are likely more relevant to cardiovascular aging than a single baseline measurement. Finally, relative risk estimates and population-attributable modeling should be interpreted alongside absolute risk, feasibility, safety, adherence, and equity considerations.

In summary, Wei *et al.* advance the field by shifting attention from how much people move to how movement is distributed across intensity levels. The study does not imply that volume no longer matters, nor that VPA is universally appropriate[3]. Rather, it supports a multidimensional view of physical activity as a behavioral exposure with distinct volume, intensity, and patterning components. Future research should clarify how volume and intensity interact across different diseases, populations, and levels of functional capacity, and whether intensity-specific associations can be replicated using repeated objective measurements, vascular and biological aging biomarkers, compositional modeling, and pragmatic randomized trials.

DECLARATIONS

Authors' contributions

Conceived the commentary: B.X; J.W;

Drafted the manuscript: J.W;

Critically revised the manuscript: Y.W, Z.G, M.Z, and B.X.

All authors read and approved the final version of the manuscript.

Availability of data and materials

Not applicable.

AI and AI-assisted tools Statement

Not applicable.

Financial support and sponsorship

This work was supported by the National Key Research and Development Plan: Real-Time Intelligent Active Intervention on Integration of Ten Important Chronic Diseases (2020YFC2003504-2).

Conflicts of interest

All authors declared that there are no conflicts of interest.

Ethical approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

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