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# Personal Activity Intelligence (PAI): A new standard in activity tracking for obtaining a healthy cardiorespiratory fitness level and low cardiovascular risk☆

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ABSTRACT

Despite all the evidence of health benefits related to physical activity (PA) and cardiorespiratory fitness (CRF), low levels of PA have reached pandemic proportions, and inactivity is the fourth leading cause of death worldwide. Lack of time, and inability to self-manage are often cited as main barriers to getting adequate PA. Recently, a new personalized metric for PA tracking named Personal Activity Intelligence (PAI) was developed with the aim to make it easier to quantify how much PA per week is needed to reduce the risk of premature mortality from non-communicable diseases. PAI can be integrated in self-assessment heart rate devices and defines a weekly beneficial heart rate pattern during PA by considering the individual's sex, age, and resting and maximal heart rates. Among individuals ranging from the general population to subgroups of patients with cardiovascular disease (CVD), a PAI score ≥100 per week at baseline, an increase in PAI score, and a sustained high PAI score over time were found to delay premature death from CVD and all causes, regardless of whether or not the current PA recommendations were met. Importantly, a PAI score ≥100 at baseline, maintaining ≥100 PAIs and an increasing PAI score over time was associated with multiple years of life gained. Moreover, obtaining a weekly PAI ≥100 attenuated the deleterious association between CVD risk factor clustering and prolonged sitting time. PAI and objectively measured CRF (as indicated by $VO_{2peak}$) were positively associated in a graded fashion, and individuals with a PAI score between 100 and 150 had expected age and sex specific average $VO_{2peak}$ values. A PAI score ≥100 was associated with higher $VO_{2peak}$ in both men (4.1 $mL \cdot kg^{-1} \cdot min^{-1}$; 95% CI, 3.5 to 4.6) and women (2.9 $mL \cdot kg^{-1} \cdot min^{-1}$; 95% CI, 2.4 to 3.3), compared to the reference group of <100 PAI. The combined analysis of PAI, PA and $VO_{2peak}$ demonstrated that a PAI score ≥100 was associated with high $VO_{2peak}$ values regardless of meeting or not meeting the current PA recommendations. Collectively, these findings suggest that PAI has the potential to be a useful tool to motivate people to become and stay physically active by quantifying the amount of PA needed to produce significant health benefits.



**Contents**



*Abbreviations and acronyms:* CI, Confidence Interval; CRF, Cardiorespiratory Fitness; CVD, Cardiovascular Disease; HTN, Hypertension; HUNT, the Nord-Trøndelag Health Study; NCDs, Non-communicable Diseases; PA, Physical activity; PAI, Personal Activity Intelligence.

☆ Statement of conflict of interest: see page XX.

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The National Institute on Aging and the World Health Organization (WHO) rank the dramatic increase in average life expectancy in the 20th century as one of the greatest societal achievements.[1] However, this crowning achievement does not come without other challenges. Changes in demographics have shifted the leading causes of disease and death from infectious to non-communicable diseases (NCDs), which more commonly affect the aging adult population.[1,2] This, in turn, has created an unparalleled demand for treatments with societies struggling to afford the mounting healthcare costs together with the availability of cost-effective and feasible interventions to prevent NCDs[3]. Some estimates have suggested that these costs may become unsustainable in the not too distant future.[4]

According to the WHO, insufficient physical activity (PA) is one of the leading risk factors for NCDs and death worldwide.[5] Inadequate PA level increases the risk of NCDs by 20–30%, shortens lifespan by 3–5 years, and accounts for >5 million deaths each year worldwide.[6–8] Indeed, PA can contribute to prevention of different co-morbid conditions including hypertension (HTN), overweight, and obesity and also is an effective and inexpensive non-pharmacological therapy for these conditions along with improving mental health, quality of life, and delaying onset of dementia.[9,10] Evolving evidence suggests that cardiorespiratory fitness (CRF) might be a stronger predictor of adverse health outcomes when compared to PA,[11–13] and research shows that CRF improves in a dose-response manner through PA and/or exercise training.[12]

Yet the major challenge when it comes to PA effectiveness is adherence. Inactivity has reached pandemic proportions worldwide. A quarter of the world's population, or 1.4 billion adults, are not getting enough PA, putting them at risk of developing or exacerbating inactivity related diseases.[6,14] A recent study reported that the goal of reducing physical inactivity by 10% by the year 2025, as set out by the WHO, is unlikely to materialize.[14] Lack of time, and inability to self-manage (i.e., setting personal goals, monitoring PA progress through personalized feedback tailored to individual needs and preferences) are often cited as main barriers to getting adequate PA.[15–18]

Unfortunately, most available PA metrics have failed to translate physiological measures into meaningful, personal and scientifically proven information for the mainstream user.[18–20] For instance, goals such as “10,000 steps per day” or “30 minutes of PA per day” are too vague and may even be misleading. Research shows that intensity of exercise matters when it comes to health benefits.[10,12,21] Walking 6000 steps rapidly uphill at a high relative intensity results in superior physiological or psychological adaptations compared to walking slowly downhill at low relative intensity. The advice to walk 10,000 steps per day does not account for intensity, and a person able to walk only 6000 steps per day, but at a faster rate or with inclines (stairs or uphill) may be discouraged by not meeting the daily step count goal, even though the physiological workload can be much higher than walking 10,000 steps on flat terrain. Because it parallels metabolic rate, the most personalized and accurate reflection of the body's response to PA, regardless of the PA type, is heart rate.[19,22] However, current PA advice and most widely marketed PA trackers do not convert PA- induced changes in heart rate into a metric for understanding how much PA an individual needs to perform, and at what intensity in order to prevent NCDs and improve survival.

In keeping with the suggestions for overcoming PA barriers,[15] the Cardiac Exercise Research Group (CERG) (ntnu.edu/cerg) developed a personalized PA metric, named Personal Activity Intelligence (PAI),[19] with the aim to make it easier to quantify how much PA per week is needed to reduce the risk of premature mortality from NCDs.[19,23] PAI can be integrated in self-assessment heart rate devices and defines a weekly beneficial heart rate pattern during PA. PAI considers the individual's sex, age, and resting and maximal heart rates, and reflects an individuals' response to PA.[19] The metric translates heart rate variations over the course of a week, into a simple and easily understandable score (0 = inactive, and 100 = active enough). For instance, a score of 100 PAI could be obtained by performing various PA volumes and intensities, using individually preferred activities as long as the heart rate is elevated above a certain threshold.[19,24] Currently, the relationship between PAI and CRF is not known. Such a relationship may expand our understanding of biological adaptations elicited by PAI to obtain substantial health benefits.

The purpose of this paper is to summarize the epidemiological evidence regarding the effects of PAI on health and investigate the association between PAI and CRF in a large unselected population of healthy individuals.

## PAI and cardiovascular disease (CVD) risk

Among individuals ranging from the general population to subgroups of patients with CVD, a PAI score ≥100 per week at baseline, an increase in PAI score, and a sustained high PAI score over time were found to delay premature death from CVD and all causes, regardless of whether or not the current PA recommendations were met.[19,24,25]

In a study on 19,269 men, and 20,029 women free from known CVD at baseline and followed up for 26.2 (SD 5.9) years,[19] a PAI score of ≥100/week was associated with a 13% (95% confidence interval [CI], 6%–20%) lower all-cause mortality, and 17% (95% CI, 7%–27%) lower CVD mortality in men, compared with inactive group. The corresponding risk reduction in women was 17% (95% CI, 6%–26%) for all-cause mortality, and 23% (95% CI, 4%–38%) for CVD mortality (Fig. 1). In subgroups of participants, i.e., pre-specified age groups, or those with known CVD risk factors, such as diabetes, HTN, smoking, or overweight/obesity, obtaining ≥100 weekly PAI was associated with a significantly lower risk of all-cause and CVD mortality. For example, HTN patients with ≥100 PAI had 30% (95% CI, 21%–38%) lower CVD mortality, and the corresponding risk reduction in diabetic patients was 54% (95% CI, 17%–75%).[19]

Among patients with established CVD (1123 women, 2010 men) followed for a mean of 12.5 years (39,157 person-years), those with a weekly baseline PAI score ≥100 had 24% (95% CI, 10%–35%) and 36% (95% CI, 21%–48%) lower risk of all-cause and CVD- mortality, respectively.[25] Furthermore, a PAI weekly score ≥100 was associated with similarly lower mortality risk among various subgroups of patients in both sexes and across age groups.[25]

The temporal changes in PAI were also predictive of a mortality risk. In a prospective cohort study of 11,870 men and 13,010 women without known CVD at baseline and followed up for 18.0 (SD 4.0) years, high PAI score that was sustained (≥100 PAI at both time points) and an increase in PAI score (<100 at first assessment and ≥100 at second assessment) over a 10-year period were associated with lower risk of mortality.[24] Participants obtaining a score ≥100 PAI at both time points had a 32% reduction in risk for CVD mortality (95% CI, 14%–46%), and a 20% reduction in risk for all-cause mortality (95% CI, 9%–29%), compared to participants with a PAI score <100 at both assessments. For those who increased their PAI score over time, the risk reduction was 14% (95% CI, 5%–21%) for all-cause mortality.[24]

Importantly, obtaining a PAI score ≥100 was associated with a lower risk of mortality regardless of meeting or not meeting the current PA recommendations.[19,25] However, among those with a PAI score <100 who adhered to the PA recommendations, the adjusted hazard ratios were 1.27 (95% CI, 1.09–1.48) for CVD mortality, and 1.13 (95% CI, 1.02–1.24) for all-cause mortality.[19]

An additional intriguing finding was that a PAI score ≥100 at baseline was associated with multiple years of life gained both in the general population free from CVD [4.7 (95% CI, 4.4–5.0) years],[19] as well as patients with known CVD [4.7 (95% CI, 3.2–6.3) years].[25] Furthermore, maintaining ≥100 PAIs and an increasing PAI score over time associated with 8.2 (95% CI, 7.4–9.0) and 6.6 (95% CI, 5.7–7.4) years of life gained, respectively.[24]

Emerging evidence indicates that sedentary behaviour, which is distinct from physical inactivity, is associated with a clustering of risk factors for CVD, several chronic conditions and mortality.[26–29] The results of a study on a large apparently healthy population (n = 29,950)

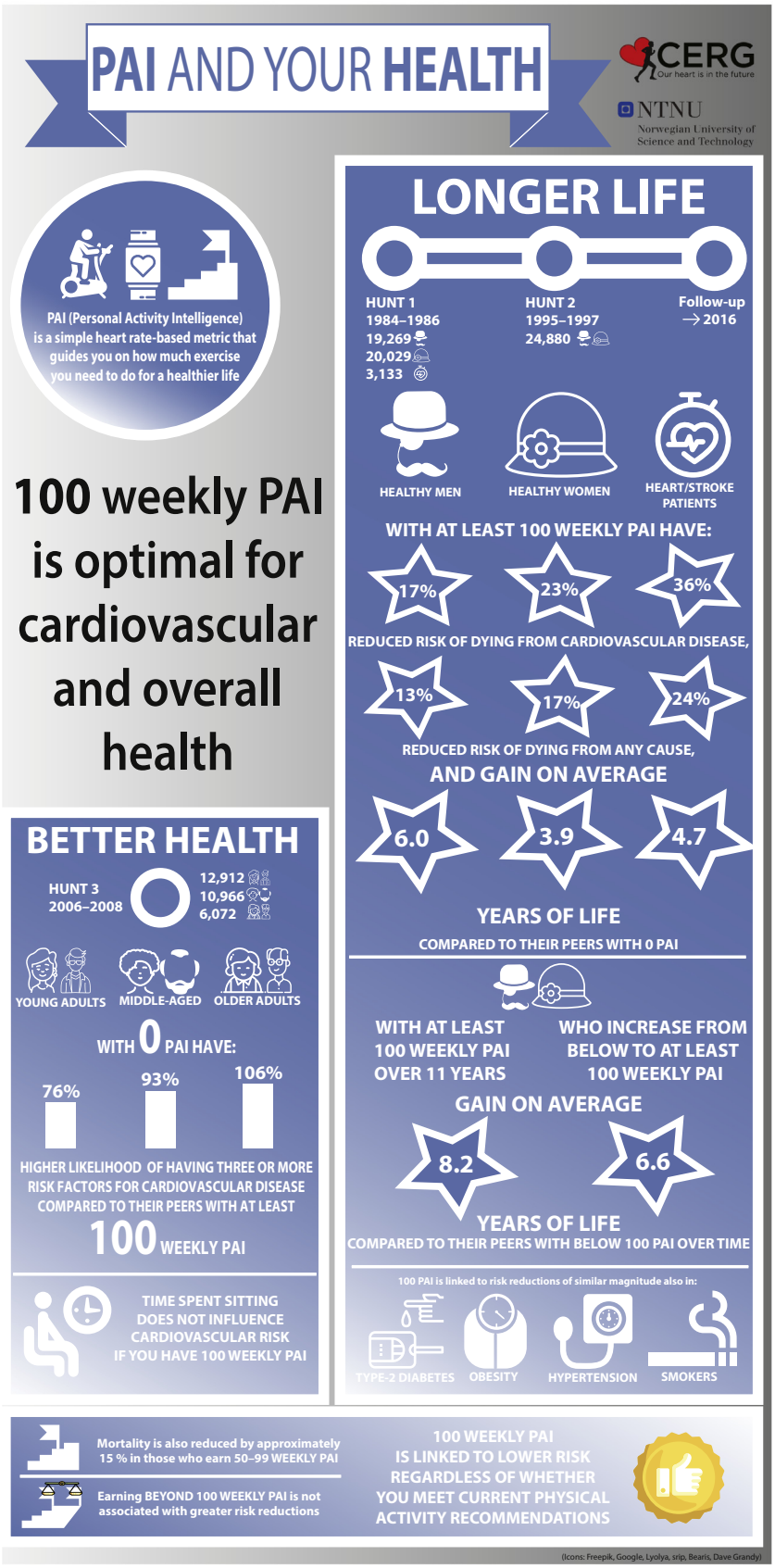



**Fig. 1.** Personal Activity Intelligence (PAI) and health.

show that participants with a weekly PAI score <100 had a higher likelihood of CVD risk factor clustering across age groups compared with those who had a weekly PAI ≥100.[30] Moreover, obtaining a weekly PAI ≥100 attenuated the deleterious association between CVD risk factor clustering and prolonged sitting time. For instance, for subjects between the ages of 45–59, the odds ratio for those sitting 5–<7 and ≥7 h per day

and having a weekly PAI score <100 were 2.20 (95% CI, 1.61–3.00) and 2.21 (95% CI, 1.61–3.01), respectively, compared with the reference group (≥100 PAI per week and sitting 4 or less hours per day).[30]

Collectively, these results suggest that PAI may be used as a very suitable prescription for PA, with a personalized dose required for significant health benefit.

## Linking PAI and CRF

The characteristics of the study participants used to establish the association between PAI and CRF are presented in Table 1 (for methods, see Online Supplement). The mean $VO_{2peak}$ was 36.0 ± 7.7 $mL \cdot kg^{-1} \cdot min^{-1}$ and 44.4 $mL \cdot kg^{-1} \cdot min^{-1}$ for women and men, respectively. In total, 55.3% achieved a ≥100 PAI score, and 7.6% of the participants (10.5% of men and 4.9% of women) were classified as inactive (0 PAI).

There was a graded positive association between PAI and $VO_{2peak}$ in both men and women as shown in Table 2. Compared with men in the reference group (0 PAI), a PAI score ≥100 was associated with a 5.4 $mL \cdot kg^{-1} \cdot min^{-1}$ higher $VO_{2peak}$ (95% CI, 4.5 to 6.3). In women, PAI ≥100 was associated with a 4.1 $mL \cdot kg^{-1} \cdot min^{-1}$ higher $VO_{2peak}$ (95% CI, 3.1 to 5.2), compared with inactive women. Similarly, a PAI score ≥100 was associated with higher $VO_{2peak}$ in both men (4.1 $mL \cdot kg^{-1} \cdot min^{-1}$; 95% CI, 3.5 to 4.6) and women (2.9 $mL \cdot kg^{-1} \cdot min^{-1}$; 95% CI, 2.4 to 3.3), compared to the reference group of <100 PAI (Table 2).

The combined analysis of PAI, PA and $VO_{2peak}$ is presented in Table 3. The results demonstrate that a PAI score ≥100 was associated with high $VO_{2peak}$ values regardless of meeting or not meeting the current PA recommendations. Compared with the reference group (<100 PAI and not meeting the recommendations), those with a PAI ≥100 and not meeting the recommendations had a 2.6 $mL \cdot kg^{-1} \cdot min^{-1}$ (95% CI, 2.2 to 3.0) higher $VO_{2peak}$. Among those with PAI ≥100 and meeting the recommendations, the adjusted difference in $VO_{2peak}$ was 4.4 $mL \cdot kg^{-1} \cdot min^{-1}$ (95% CI, 3.9 to 4.8), compared to the reference group.

Fig. 2 presents the $VO_{2peak}$ values according to weekly PAI scores, and show that individuals with a PAI score between 100 and 150 had expected age and sex specific mean $VO_{2peak}$ values. For instance, $VO_{2peak}$ for men with 100–150 weekly PAI and in the age groups of 40–59 and ≥60 years was 44.1 ± 8.1 $mL \cdot kg^{-1} \cdot min^{-1}$ and 38.5 ± 7.4 $mL \cdot kg^{-1} \cdot min^{-1}$, respectively. The analogous $VO_{2peak}$ values for women were 34.9 ± 6.0 $mL \cdot kg^{-1} \cdot min^{-1}$ and 30.0 ± 3.7 $mL \cdot kg^{-1} \cdot min^{-1}$, respectively. This is of great interest given that achieving average or above average age-specific CRF levels have been associated with substantial health and health economic benefits.[13,31,32] The figure also shows that there is a gradual increase in $VO_{2peak}$ with increasing levels of PAI categories in both men and women.

In terms of mortality risk, previous PAI studies[19,25] show no additional benefits in those having more than 100 weekly PAI (in our population exhibiting equal to or above age-specific CRF-levels, Fig. 2) compared to those having 100 PAI. This might appear to contrast studies using CRF-values to categorize risk indicating an inverse association between CRF and long-term mortality with no upper limit of benefit.[33] The lack of additional benefit in terms of risk reduction among those achieving higher than 100 weekly PAI might reflect a lack of statistical power in these studies given that there were relatively few events in each age category that achieved very high weekly PAI levels (>200 PAI). However, the seemingly contrasting observations may have another likely explanation. Currently, there is no consensus regarding classification of CRF categories, however, recent reports from the United States,[34–36]

**Table 1**
Descriptive characteristics of the participants.

| | Total | Men | Women | *P*-value[a] |
|---|---|---|---|---|
| No. of subjects | 4334 | 2112 | 2222 | |
| Age, mean (SD), years | 48.4 (13.5) | 49.0 (13.5) | 47.9 (13.6) | <0.01 |
| $VO_{2peak}$, mean (SD), $mL \cdot kg^{-1} \cdot min^{-1}$ | 40.1 (9.5) | 44.4 (9.2) | 36.0 (7.7) | <0.01 |
| Height, mean (SD), cm | 172.5 (9.1) | 179.4 (6.3) | 166.0 (5.8) | <0.01 |
| Weight, mean (SD), kg | 77.5 (13.7) | 85.6 (11.4) | 69.8 (11.2) | <0.01 |
| BMI[b], mean (SD), $kg/m^2$ | 25.9 (3.6) | 26.6 (3.2) | 25.4 (3.8) | <0.01 |
| RHR, mean (SD), bpm | 68.1 (10.8) | 66.6 (11.1) | 69.4 (10.2) | <0.01 |
| Cholesterol, mean (SD), mmol/L | 5.5 (1.1) | 5.5 (1.1) | 5.4 (1.1) | 0.15 |
| HDL, mean (SD), mmol/L | 1.4 (0.3) | 1.3 (0.3) | 1.5 (0.3) | <0.01 |
| Glucose, mean (SD), mmol/L | 5.4 (1.3) | 5.6 (1.5) | 5.2 (1.0) | <0.01 |
| Smoking status, No. (%) | | | | |
| Never | 2158 (49.8) | 1069 (50.6) | 1089 (49.0) | |
| Former | 1380 (31.8) | 673 (31.9) | 707 (31.8) | |
| Current | 471 (10.9) | 196 (9.3) | 275 (12.4) | |
| Occasional | 325 (7.5) | 174 (8.2) | 151 (6.8) | <0.01 |
| Occupational physical activity, No. (%)[c] | | | | |
| Mostly sedentary | 1478 (34.1) | 846 (40.1) | 632 (28.4) | |
| Frequent walking | 1535 (35.4) | 596 (28.2) | 939 (42.3) | |
| Frequent walking and lifting | 1043 (24.1) | 499 (23.6) | 544 (24.5) | |
| Heavy manual work | 92 (2.1) | 77 (3.7) | 15 (0.7) | <0.01 |
| Alcohol consumption, No. (%)[c] | | | | |
| 0 | 643 (14.8) | 220 (10.4) | 423 (19.0) | |
| 1–4 | 1750 (40.4) | 702 (33.2) | 1048 (47.2) | |
| ≥5 | 1796 (41.4) | 1138 (53.) | 658 (29.6) | <0.01 |
| Health status, No. (%)[c] | | | | |
| Poor | 13 (0.3) | 5 (0.2) | 8 (0.4) | |
| Not so good | 444 (10.2) | 187 (8.9) | 257 (911.6) | |
| Good | 2636 (60.8) | 1327 (62.8) | 1309 (58.9) | |
| Very good | 1170 (27.0) | 567 (26.9) | 603 (27.1) | <0.01 |
| Personal Activity Intelligence (PAI) | | | | |
| 0 | 329 (7.6) | 221 (10.5) | 108 (4.9) | |
| ≤50 | 1132 (26.1) | 545 (25.8) | 587 (26.4) | |
| 51–99 | 476 (11.0) | 208 (9.8) | 268 (12.1) | |
| ≥100 | 2397 (55.3) | 1138 (53.9) | 1259 (56.6) | <0.01 |

$VO_{2peak}$, peak oxygen uptake; BMI, body mass index; RHR, resting heart rate; bmp, beats per minute; HDL, High-density lipoprotein.
[a] Baseline group differences were examined by using t-test for continuous variables, and $\chi 2$ tests were used for proportions of categorical variables.
[b] Calculated as weight in kilograms divided by height in meters squared.
[c] Data is missing.

**Table 2**
Difference in $VO_{2peak}$ ($mL \cdot kg^{-1} \cdot min^{-1}$) according to Personal Activity Intelligence.

| PAI | Men | | | | Women | | | |
|---|---|---|---|---|---|---|---|---|
| | n | Crude mean $VO_{2peak}$ | Age-adjusted difference | Adjusted diff.[a] (95% CI) | n | Crude mean $VO_{2peak}$ | Age-adjusted difference | Adjusted diff.[a] (95% CI) |
| 0 | 221 | 40.6 | 0.0 (Ref.) | 0.0 (Ref.) | 108 | 32.2 | 0.0 (Ref.) | 0.0 (Ref.) |
| ≤50 | 545 | 40.0 | 1.4 | 1.2 (0.3–2.2) | 587 | 32.7 | 1.8 | 1.1 (0.0–2.2) |
| 51–99 | 208 | 43.7 | 3.6 | 2.8 (1.7–3.9) | 268 | 34.5 | 3.0 | 2.1 (0.9–3.2) |
| ≥100 | 1138 | 47.5 | 7.3 | 5.4 (4.5–6.3) | 1259 | 38.1 | 5.6 | 4.1 (3.1–5.2) |
| <100 | 974 | 40.9 | 0.0 (Ref.) | 0.0 (Ref.) | 963 | 33.2 | 0.0 (Ref.) | 0.0 (Ref.) |
| ≥100 | 1138 | 47.5 | 5.8 | 4.1 (3.5–4.6) | 1259 | 38.1 | 3.6 | 2.9 (2.4–3.3) |

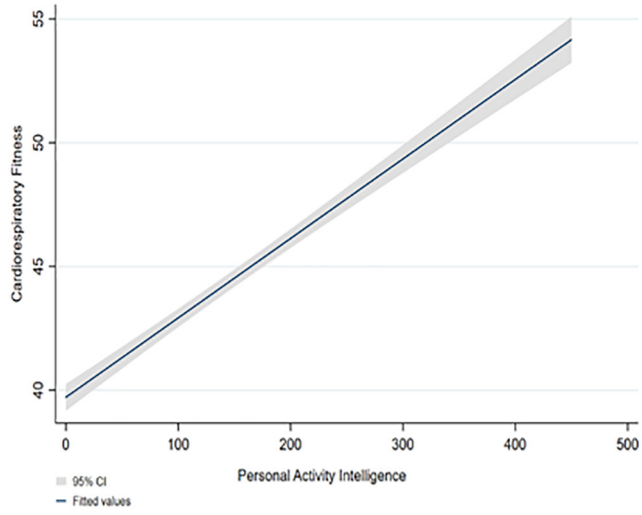
$VO_{2peak}$, objectively measured peak oxygen uptake; PAI, Personal Activity Intelligence.
[a] Adjusted for age, body mass index, smoking status, occupational physical activity, alcohol consumption, marital status, serum glucose, serum cholesterol, HDL and general health status.

**Table 3**
Adjusted difference in $VO_{2peak}$ ($mL \cdot kg^{-1} \cdot min^{-1}$) in combined categories of Personal Activity Intelligence and Physical Activity Recommendations.

| | n | Crude mean $VO_{2peak}$ | Age & sex adjusted difference | Adjusted diff.[a] (95% CI) |
|---|---|---|---|---|
| <100, Not meeting | 1937 | 37.1 | 0.0 (Ref.) | 0.0 (Ref.) |
| <100, Meeting | 0 | – | – | – |
| ≥100, Not meeting | 1221 | 40.8 | 3.6 | 2.6 (2.2–3.0) |
| ≥100, Meeting | 1176 | 44.4 | 5.8 | 4.4 (3.9–4.8) |

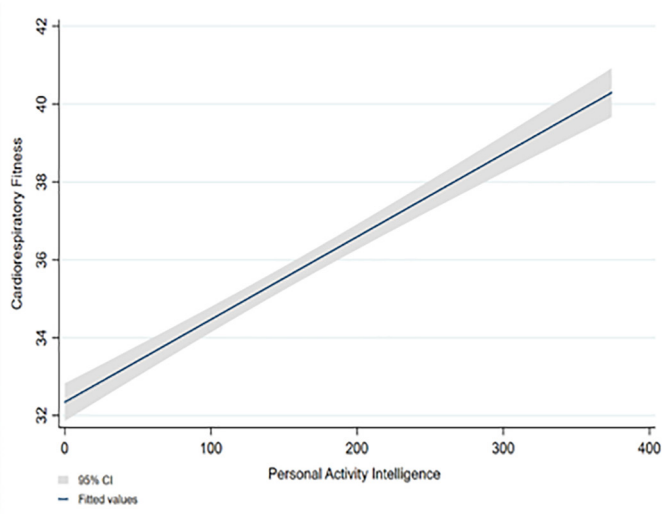
[a] Adjusted for age, sex, body mass index, smoking status, occupational physical activity, alcohol consumption, marital status, serum glucose, serum cholesterol, HDL, and general health status.

Norway,[37] Germany,[38] and Brazil[39] have made efforts to develop reference standards. It is well known that CRF varies by age, sex, and mode of exercise, and it seems clear that these reference classifications vary substantially (≈1–3 METs) in different regions around the world. A recent study from the Cleveland Clinic used a cohort-specific (n = 122,007) age and sex standardized classification of CRF estimated from treadmill speed and grade.[33] The study participants were categorized into low fitness (<25th percentile), below average (25th–49th percentile), above average (50th–74th percentile), high (75th–97.6th percentile) and elite (>97.7th percentile), and the results showed increasingly reduced risk across all CRF categories "with no upper limit of benefit".[33] Interestingly, the mean age-specific $VO_{2peak}$ value in our study was similar to that observed when combining the "above average" and "high" fitness categories of Mandsager et al.[33]. For instance, $VO_{2peak}$ for men aged 40–59 in the "above average" and "high" fitness groups together was 43 $mL \cdot kg^{-1} \cdot min^{-1}$ (≈12 METs), similar to what we




**Men**

| PAI | Crude mean $VO_{2peak}$: Age group (y) 20-39 | 40-59 | 60+ | Total |
|---|---|---|---|---|
| 0 | 44.9 | 40.8 | 32.7 | 40.6 |
| 1-50 | 45.7 | 40.8 | 34.7 | 40.0 |
| 51-99 | 46.6 | 42.8 | 36.7 | 43.7 |
| 100-150 | 47.9 | 44.1 | 38.5 | 44.1 |
| 151-200 | 52.9 | 46.5 | 40.3 | 46.5 |
| 201-250 | 52.3 | 46.4 | 39.4 | 45.6 |
| >250 | 56.7 | 50.6 | 40.6 | 50.9 |




**Women**

| PAI | Crude mean $VO_{2peak}$: Age group (y) 20-39 | 40-59 | 60+ | Total |
|---|---|---|---|---|
| 0 | 34.9 | 32.3 | 28.6 | 32.2 |
| 1-50 | 37.6 | 33.5 | 28.1 | 32.7 |
| 51-99 | 38.9 | 33.8 | 30.1 | 34.5 |
| 100-150 | 40.0 | 34.9 | 30.0 | 35.7 |
| 151-200† | - | - | - | 36.2 |
| 201-250 | 41.9 | 36.3 | 31.3 | 37.0 |
| >250 | 44.5 | 39.2 | 31.2 | 39.7 |

†Fewer observations

**Fig. 2.** Personal Activity Intelligence (PAI) and cardiorespiratory fitness. $VO_{2peak}$, peak oxygen uptake.

(44 mL·kg$^{-1}$·min$^{-1}$) observed as the age-specific average. Similarly, the $VO_{2peak}$ for women aged 40–59 years when combining "above average" and "high" CRF categories in the study of Mandsager and colleagues (37 mL·kg$^{-1}$·min$^{-1}$) was similar to ours (35 mL·kg$^{-1}$·min$^{-1}$). Regardless, the observation of Mandsager and colleagues showing that those with the highest CRF level categorized as "elite" had the greatest risk reduction is of high interest. Importantly, these fitness levels are not those observed in endurance athletes (for instance, "elite" corresponds to $VO_{2peak}$ approximately above 50 and 45 mL·kg$^{-1}$·min$^{-1}$ for men and women aged 40–59 years of age), but values that may be achievable for most healthy adults. In our view, future studies are needed to define region-specific and age-specific values for $VO_{2peak}$ needed to obtain optimal risk reduction. However, what our data clearly demonstrates is that PAI can be used to target a certain level of $VO_{2peak}$, and as such be a very practical way to secure a high enough level of PA in order to obtain a CRF level (even beyond average) that induces substantial, and perhaps, optimal risk reduction.

The new PA guidelines for Americans[9] suggest that adults should engage in at least 150–300 min a week of moderate intensity, or 75–150 min of vigorous intensity aerobic PA, or an equivalent combination without the stipulation to do PA in bouts of at least 10 min. In this regard, PAI seems to fit well with these recommendations as it offers individuals a variety of options and choices relating to quantity, quality, and intensity of PA. The key is to accumulate adequate active time above the relative heart rate threshold in order to earn PAI, and the higher the intensity, the shorter the time needed to obtain 100 PAI. For example, a PAI score of 100 equates to approximately 60 min per week of PA at an intensity of ≈75% heart rate reserve.

## Conclusions

PAI is an easily understandable and scientifically proven PA metric. If applied more broadly, it has the potential to be a useful tool to motivate people to become and stay physically active. The impact of higher PAI in reducing the risk of premature CVD and all-cause mortality, and attenuating the deleterious effects of prolonged sedentary time have been well documented in recent years. Furthermore, we observed a graded positive association between PAI and $VO_{2peak}$ in both men and women. These findings suggest that PAI may be a useful tool for quantifying the amount of PA needed to produce significant health benefits in individuals in the general population as well as subgroups of patients with CVD.

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## Statement of conflict of interest

Professor Wisløff is the inventor of PAI, and scientific advisor of a company (PAI Health Inc.) that holds the IP rights for PAI that develops applications that utilize data from diverse heart rate monitors to display PAI for users. Due to the potential conflict of interest, we are thankful to Professor Sigurd Steinshamn at Department of Circulation and Medical Imaging, Faculty of Medicine and Health Sciences, NTNU who monitored adherence to design, and statistical analysis in the current study.

We have read and understood the policy of the Progress in Cardiovascular Diseases and declare that design, statistical analyses, and reporting of results of the current study are in accordance with the directives of Regional Committee on Medical and Health Research Ethics of Norway (2017/2425/REK nord). There are no further disclosures or potential conflicts of interest to report.

## Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.pcad.2019.02.006.

## References

1. WHO. *Global health and ageing*. WHO: World Health Organization. 2015.
2. WHO. *The global strategy and action plan on ageing and health*. WHO: World Health Organization. 2018.
3. Beaglehole R, Bonita R, Alleyne G, et al. UN High-Level Meeting on Non-Communicable Diseases: addressing four questions. Lancet 2011;378:449-455.
4. Wise J, Moller A, Christie D, et al. The positive impacts of Real-World Data on the challenges facing the evolution of biopharma. Drug Discov Today 2018;23:788-801.
5. NCDs. *Physical activity*. WHO: World Health Organization. 2018.
6. Kohl 3rd HW, Craig CL, Lambert EV, et al. The pandemic of physical inactivity: global action for public health. Lancet 2012;380:294-305.
7. Kraus WE, Bittner V, Appel L, et al. The National Physical Activity Plan: a call to action from the American Heart Association: a science advisory from the American Heart Association. Circulation 2015;131:1932-1940.
8. Lee IM, Shiroma EJ, Lobelo F, Puska P, Blair SN, Katzmarzyk PT. Effect of physical inactivity on major non-communicable diseases worldwide: an analysis of burden of disease and life expectancy. Lancet 2012;380:219-229.
9. Piercy KL, Troiano RP, Ballard RM, et al. The physical activity guidelines for Americans. Jama 2018;320:2020-2028.
10. Wisloff U, Lavie CJ. Taking physical activity, exercise, and fitness to a higher level. Prog Cardiovasc Dis 2017;60:1-2.
11. Lee DC, Sui X, Ortega FB, et al. Comparisons of leisure-time physical activity and cardiorespiratory fitness as predictors of all-cause mortality in men and women. Br J Sports Med 2011;45:504-510.
12. Ross R, Blair SN, Arena R, et al. Importance of assessing cardiorespiratory fitness in clinical practice: a case for fitness as a clinical vital sign: a scientific statement from the American Heart Association. Circulation 2016;134:e653-e699.
13. Ozemek C, Laddu DR, Lavie CJ, et al. An update on the role of cardiorespiratory fitness, structured exercise and lifestyle physical activity in preventing cardiovascular disease and health risk. Prog Cardiovasc Dis 2018;61:484-490.
14. Guthold R, Stevens GA, Riley LM, Bull FC. Worldwide trends in insufficient physical activity from 2001 to 2016: a pooled analysis of 358 population-based surveys with 1.9 million participants. Lancet Glob Health 2018;6:e1077-e1086.
15. CDC. *Barriers to Physical Activity*. 2018.
16. Sallis JF, Hovell MF. Determinants of exercise behavior. Exerc Sport Sci Rev 1990;18:307-330.
17. Sallis R, Franklin B, Joy L, Ross R, Sabgir D, Stone J. Strategies for promoting physical activity in clinical practice. Prog Cardiovasc Dis 2015;57:375-386.
18. Thompson D, Peacock O, Western M, Batterham AM. Multidimensional physical activity: an opportunity, not a problem. Exerc Sport Sci Rev 2015;43:67-74.
19. Nes BM, Gutvik CR, Lavie CJ, Nauman J, Wisloff U. Personalized Activity Intelligence (PAI) for prevention of cardiovascular disease and promotion of physical activity. Am J Med 2017;130:328-336.
20. Peacock OJ, Western MJ, Batterham AM, et al. Multidimensional individualised Physical ACTivity (Mi-PACT) — a technology-enabled intervention to promote physical activity in primary care: study protocol for a randomised controlled trial. Trials 2015;16:381.
21. Wen CP, Wai JP, Tsai MK, et al. Minimum amount of physical activity for reduced mortality and extended life expectancy: a prospective cohort study. Lancet 2011;378:1244-1253.
22. Garber CE, Blissmer B, Deschenes MR, et al. American College of Sports Medicine position stand. Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: guidance for prescribing exercise. Med Sci Sports Exerc 2011;43:1334-1359.
23. Global, regional, and national age-sex specific all-cause and cause-specific mortality for 240 causes of death, 1990–2013: a systematic analysis for the Global Burden of Disease Study 2013Lancet 2015;385:117-171.
24. Kieffer SK, Croci I, Wisloff U, Nauman J. Temporal changes in a novel metric of physical activity tracking (personal activity intelligence) and mortality: the HUNT study, Norway. Prog Cardiovasc Dis 2018. https://doi.org/10.1016/j.pcad.2018.09.002.
25. Kieffer SK, Zisko N, Coombes JS, Nauman J, Wisloff U. Personal activity intelligence and mortality in patients with cardiovascular disease: the HUNT study. Mayo Clin Proc 2018;93:1191-1201.

26. Ekelund U, Steene-Johannessen J, Brown WJ, et al. Does physical activity attenuate, or even eliminate, the detrimental association of sitting time with mortality? A harmonised meta-analysis of data from more than 1 million men and women. Lancet 2016;388:1302-1310.
27. Biswas A, Oh PI, Faulkner GE, et al. Sedentary time and its association with risk for disease incidence, mortality, and hospitalization in adults: a systematic review and meta-analysis. Ann Intern Med 2015;162:123-132.
28. Grontved A, Hu FB. Television viewing and risk of type 2 diabetes, cardiovascular disease, and all-cause mortality: a meta-analysis. Jama 2011;305:2448-2455.
29. Nauman J, Stensvold D, Coombes JS, Wisloff U. Cardiorespiratory fitness, sedentary time, and cardiovascular risk factor clustering. Med Sci Sports Exerc 2016;48:625-632.
30. Zisko N, Skjerve KN, Tari AR, et al. Personal Activity Intelligence (PAI), sedentary behavior and cardiovascular risk factor clustering - the HUNT study. Prog Cardiovasc Dis 2017;60:89-95.
31. Myers J, Doom R, King R, et al. Association between cardiorespiratory fitness and health care costs: the veterans exercise testing study. Mayo Clin Proc 2018;93:48-55.
32. de Souza de Silva CG, Kokkinos P, Doom R, et al. Association between cardiorespiratory fitness, obesity, and health care costs: the Veterans Exercise Testing Study. Int J Obes (Lond) 2018. https://doi.org/10.1038/s41366-018-0257-0.
33. Mandsager K, Cleveland Clinic Foundation C, Ohio, Harb S, et al. Association of cardiorespiratory fitness with long-term mortality among adults undergoing exercise treadmill testing. JAMA Netw Open 2019;1.
34. Kaminsky LA, Arena R, Myers J. Reference standards for cardiorespiratory fitness measured with cardiopulmonary exercise testing: data from the Fitness Registry and the Importance of Exercise National Database. Mayo Clin Proc 2015;90:1515-1523.
35. Kaminsky LA, Myers J, Arena R. Determining cardiorespiratory fitness with precision: compendium of findings from the FRIEND registry. Prog Cardiovasc Dis January - February 2019;62(1):76-82.
36. Kaminsky LA, Imboden MT, Arena R, Myers J. Reference standards for cardiorespiratory fitness measured with cardiopulmonary exercise testing using cycle ergometry: data from the Fitness Registry and the Importance of Exercise National Database (FRIEND) registry. Mayo Clin Proc 2017;92:228-233.
37. Loe H, Rognmo O, Saltin B, Wisloff U. Aerobic capacity reference data in 3816 healthy men and women 20–90 years. PLoS One 2013;8, e64319.
38. Rapp D, Scharhag J, Wagenpfeil S, Scholl J. Reference values for peak oxygen uptake: cross-sectional analysis of cycle ergometry-based cardiopulmonary exercise tests of 10 090 adult German volunteers from the Prevention First Registry. BMJ Open 2018;8, e018697.
39. Rossi Neto JM, Tebexreni AS, Alves ANF, et al. Cardiorespiratory fitness data from 18,189 participants who underwent treadmill cardiopulmonary exercise testing in a Brazilian population. PLoS One 2019;14, e0209897.