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ORIGINAL RESEARCH ARTICLE

Wearable-Derived Training Load and Coronary Atherosclerosis in Middle-Aged and Older Athletes and Physically Active Controls: A New Perspective From the Master@Heart Study

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BACKGROUND: Middle-aged and older endurance athletes have increased prevalence of coronary artery disease (CAD) on coronary computed tomography angiography compared with healthy controls, despite similarly low cardiovascular risk. Previous studies relied on self-reported data to quantify training load (TL), which poorly correlates with objective wearable-derived TL and may bias outcomes. The effect of objective TL on CAD risk remains unknown.

METHODS: In this observational, cross-sectional analysis of the Master@Heart study, 222 men (median age, 54 [49–59] years) were included: 77 lifelong athletes, 98 late-onset athletes, and 47 controls. TL was assessed using objective wearable-derived training duration and intensity (12 consecutive months), as well as self-reported training measures. Coronary computed tomography angiography–derived CAD prevalence was compared across TL quartiles (Q) using a global unadjusted chi-square test and logistic regression, adjusted for cardiovascular risk factors and years of endurance exercise, to estimate odds ratios between Q4 and Q1. In addition, adjusted logistic regression models were fitted with continuous TL, using smoothing splines to capture potential nonlinear associations.

RESULTS: Across quartiles of objective Edwards training impulse (training duration × heart rate–weighted intensity), unadjusted global differences were observed for ≥1 plaque ($P<0.001$), coronary artery calcification (CAC)>0 ($P=0.002$), and CAC>100 ($P=0.012$). Q4 participants had significantly higher adjusted odds of ≥1 plaque (odds ratio, 5.85; 95% CI, 2.33–14.71), CAC>0 (odds ratio, 5.03; 95% CI, 2.04–12.35), and CAC>100 (odds ratio, 3.50; 95% CI, 1.22–10.00) versus Q1. Similar associations were found for objective training duration, whereas no clear associations were observed for relative time spent in high-intensity zones. In continuous analyses, Edwards training impulse and objective training duration showed significant positive associations with ≥1 plaque and CAC>100 ($P<0.05$), whereas self-reported training duration was only significantly associated with CAC>100 ($P<0.05$). Metabolic equivalent of task-minutes per week based on self-reported TL was not associated with CAD ($P>0.05$).

CONCLUSIONS: High training duration (hours/week), particularly when combined with cumulative high-intensity TL (Edwards training impulse), was independently associated with increased prevalence of subclinical CAD in middle-aged and older athletes and physically active controls. Exercise intensity alone, in the absence of high duration, was not clearly linked to CAD. These findings underscore the potential of objectively measured TL for understanding associations with subclinical CAD in endurance athletes.

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Key Words: coronary artery disease ■ athletes ■ endurance training

Editorial, see p 1023

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Clinical Perspective

What Is New?

- Objectively captured high-volume endurance exercise (training duration), particularly when combined with cumulative high-intensity training load (Edwards training impulse), is associated with increased prevalence of subclinical coronary artery disease in middle-aged and older male athletes.
- Exercise intensity alone, in the absence of high-duration training, is not clearly linked to increased subclinical coronary artery disease prevalence, challenging previous assumptions about intensity as an independent risk factor.
- Wearable-derived training load data can be processed automatically at scale, with 12 months of training data showing strong associations with coronary artery disease characteristics in highly active men, independent of traditional cardiovascular risk factors and years of endurance exercise training.

What Are the Clinical Implications?

- The strong association between recent, objectively measured training load and subclinical coronary artery disease highlights the value of wearable-based monitoring for improving insight into the relationship between endurance exercise exposure and subclinical coronary atherosclerosis.
- Although wearable-derived objective training load metrics are not yet readily implemented in routine clinical practice, future efforts may focus on incorporating objective training load to refine cardiovascular risk stratification in endurance athletes.

Nonstandard Abbreviations and Acronyms

AU	arbitrary units
CAC	coronary artery calcification
CAD	coronary artery disease
CCTA	coronary computed tomography angiography
eTRIMP	Edwards training impulse
HDL	high-density lipoprotein
HR	heart rate
HRmax	maximal heart rate
LDL	low-density lipoprotein
MET-min/week	metabolic equivalent of task-minutes per week
OR	odds ratio
Q	quartile
TL	training load

Regular physical activity is well established to reduce cardiovascular risk and all-cause mortality.[1–3] Moderate-intensity leisure-time exercise is linked to a lower risk of coronary artery disease (CAD) compared with sedentary behavior,[4] and current guidelines recommend 150 to 300 minutes of moderate or 75 to 150 minutes of vigorous physical activity per week to promote cardiovascular health.[5]

Paradoxically, emerging evidence suggests that middle-aged and older male endurance athletes, despite favorable cardiovascular risk profiles, may have a higher prevalence of coronary artery calcification (CAC) and plaques on coronary computed tomography angiography (CCTA) compared with less active peers.[6–8] The Master@Heart study confirmed this observation and, in contrast with earlier reports, also found a higher burden of less favorable plaque types, such as partially calcified and noncalcified plaques, in lifelong athletes.[9] This is clinically relevant because these plaque types are more vulnerable to plaque rupture than calcified plaques.[10,11] These findings raise the possibility of a reversed J-shaped relationship between lifelong endurance exercise and CAD, in which very high levels of endurance exercise may confer increased atherosclerotic risk.[12]

The mechanisms linking high-volume endurance training to CAD remain incompletely understood.[12] Previous studies have yielded conflicting results on the role of training load (TL), with some implicating high-duration, low-intensity exercise,[13] and others suggesting high-intensity training as the key driver.[6,14] However, this evidence is exclusively based on self-reported TL, which is prone to recall bias and showed weak correlation with objectively measured data.[15] As a result, the reliability of these findings is questionable, and any conclusions drawn from self-reported TL may not accurately reflect objective TL.

A major barrier to progress in this area has been the technical challenge of processing large-scale wearable-derived training data. Assessing TL objectively requires integration of duration and intensity from many exercise files per participant, something that has not previously been feasible at scale. In the present study, we overcome this limitation by applying a novel, semiautomated software platform capable of processing and aggregating wearable data over a 12-month time period.[15] This enables a comprehensive and unbiased assessment of exercise exposure, incorporating training duration and intensity. We included the Edwards training impulse (eTRIMP), a heart rate (HR)–based composite measure of internal TL that combines training duration and intensity.[15]

Leveraging this approach, we aimed to evaluate the association between both objectively measured and self-reported TL and CCTA-derived subclinical CAD characteristics in a cohort of middle-aged and older athletes and physically active controls. We hypothesized that the combination of high-duration and high-intensity exercise

would be associated with an increased prevalence of subclinical CAD in athletes.

METHODS

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Study Design and Study Population

The rationale, design, and methodology of the Master@Heart study have been described previously.[9,16] Briefly, the Master@Heart study is a Belgium-based multicenter prospective cohort study conducted at University Hospitals Leuven, Jessa Hospital Hasselt, and Antwerp University Hospital, with participants enrolled between 2018 and 2021. Eligible participants were men age 45 to 70 years, stratified into 3 groups based on lifelong physical activity patterns. Athletes were defined as individuals engaged in >8 hours/week of cycling or triathlon, or >6 hours/week of running, for at least 6 months before baseline evaluation. Lifelong athletes had initiated regular endurance training before the age of 30 years, whereas late-onset athletes began after age 30 years. Nonathletes engaged in <3 hours of physical activity per week and had no history of regular endurance training. Participants were excluded if they had a history of cardiovascular disease, known traditional CAD risk factors (including diabetes, hypercholesterolemia, arterial hypertension, body mass index >27.2 kg/m^2, or history of smoking), or an allergy to iodine-based contrast agents.

The current study is a predefined secondary observational, cross-sectional analysis of the Master@Heart study, starting from the initial cohort of 558 participants. From this cohort, only participants with wearable-derived objective TL data available for 12 consecutive months (before or after the CCTA) were included. This resulted in a subset of 222 participants: 77 lifelong athletes, 98 late-onset athletes, and 47 healthy active controls. Participants without complete wearable TL data were excluded (Figure 1).[16] The research protocol was approved by the ethics committee of Katholieke Universiteit Leuven. All participants gave written informed consent.

Training Load

Participants completed an online questionnaire assessing lifelong TL, including sports type, self-reported training duration (hours/week), and perceived intensity (Supplemental Questionnaire).[16] As a composite measure of training volume and intensity, metabolic equivalent of task (MET)-minutes per week (MET-min/week) was calculated by multiplying the MET score of each self-reported activity by the self-reported weekly training duration in minutes.[17]

TL was objectively measured using wearable device data over a continuous 12-month period to ensure a representative training snapshot. When wearable devices (eg, sports watches) were connected to the TrainingPeaks platform (Peaksware, Boulder, CO), all training sessions were automatically synchronized without requiring manual uploads by participants. These centrally collected data were extracted by our research team, and training parameters were semiautomatically processed using our internally developed, custom-made, and validated software tool. The methodology of this tool has been described in detail.[15]

To ensure adequate capture of training intensity, participants were included in the objective TL analysis if ≥70% of total objectively measured training time was recorded with a HR monitor. Maximal HR (HRmax) was defined as the highest HR value recorded during any training session, following a validated approach, as HRmax achieved during training sessions often exceeds values obtained during cardiopulmonary exercise testing.[15] A 5-zone HR model was constructed using percentages of HRmax: zone 1 (50%–59% HRmax), zone 2 (60%–69% HRmax), zone 3 (70%–79% HRmax), zone 4 (80%–89% HRmax), and zone 5 (90%–100% HRmax).[15,18]

The software tool extracted key TL metrics over the 12-month period, including average objective training duration (hours/week), total time spent in each HR zone, and the percentage of time spent in each zone to estimate training intensity. In addition, the average weekly eTRIMP was calculated, a validated composite metric incorporating both training duration and intensity. eTRIMP was computed by multiplying the time spent in each HR zone (in minutes) by zone-specific weighting factors (HR zone 1–5: weighting factors 1–5, respectively), summing the values across the training period, and dividing by the number of weeks. Results were expressed in arbitrary units (AU) per week.[15,18,19]

For 136 participants, TL data were collected prospectively during the 12 months preceding CCTA. In an additional 86 participants, only post-CCTA data (12 months after the scan) were available and were included to enhance statistical power. In 102 of the 136 participants with pre-CCTA TL, additional data were also available for the 12 months after CCTA, resulting in 24 months of observation. This allowed us to examine potential changes in training behavior related to the CCTA and its results.

Coronary Computed Tomography Angiography

CCTA was performed using a 2x192-slice dual-source computed tomography scanner (Siemens Somatom Force, Siemens Healthineers, Forchheim, Germany), a 256-slice computed tomography scanner (GE Revolution, GE Healthcare, Milwaukee, WI), or a 320-slice computed tomography scanner (Aquilion ONE ViSION, Canon Medical Systems, Otawara, Japan). Esmolol was administered intravenously when needed to achieve target HR. All subjects received 0.4 mg nitroglycerin before scanning. The Agatston method was used to quantify coronary calcium, resulting in the CAC score. Age-adjusted CAC percentile was calculated to estimate CAC burden relative to age.[20]

Subsequently, an ECG-triggered CCTA was performed to quantify coronary plaques after intravenous injection of iodinated contrast into a peripheral vein, followed by a saline chaser. Coronary plaques were assessed using Syngo.Via (Siemens) or GE Advanced Workstation (GE Healthcare). CCTA readers were blinded to participants' exercise history. Plaque phenotypes were defined based on plaque composition and coronary location. Plaque composition was classified as calcified (attenuation >130 Hounsfield units), noncalcified (absence of calcification), or partially calcified (presence of both calcified and noncalcified components). In addition, plaques were categorized by location, with proximal plaques defined as those located in coronary segments 1 (right coronary artery), 5 (left main coronary artery), 6 (left anterior descending artery), and 11 (left circumflex artery).[21–23]

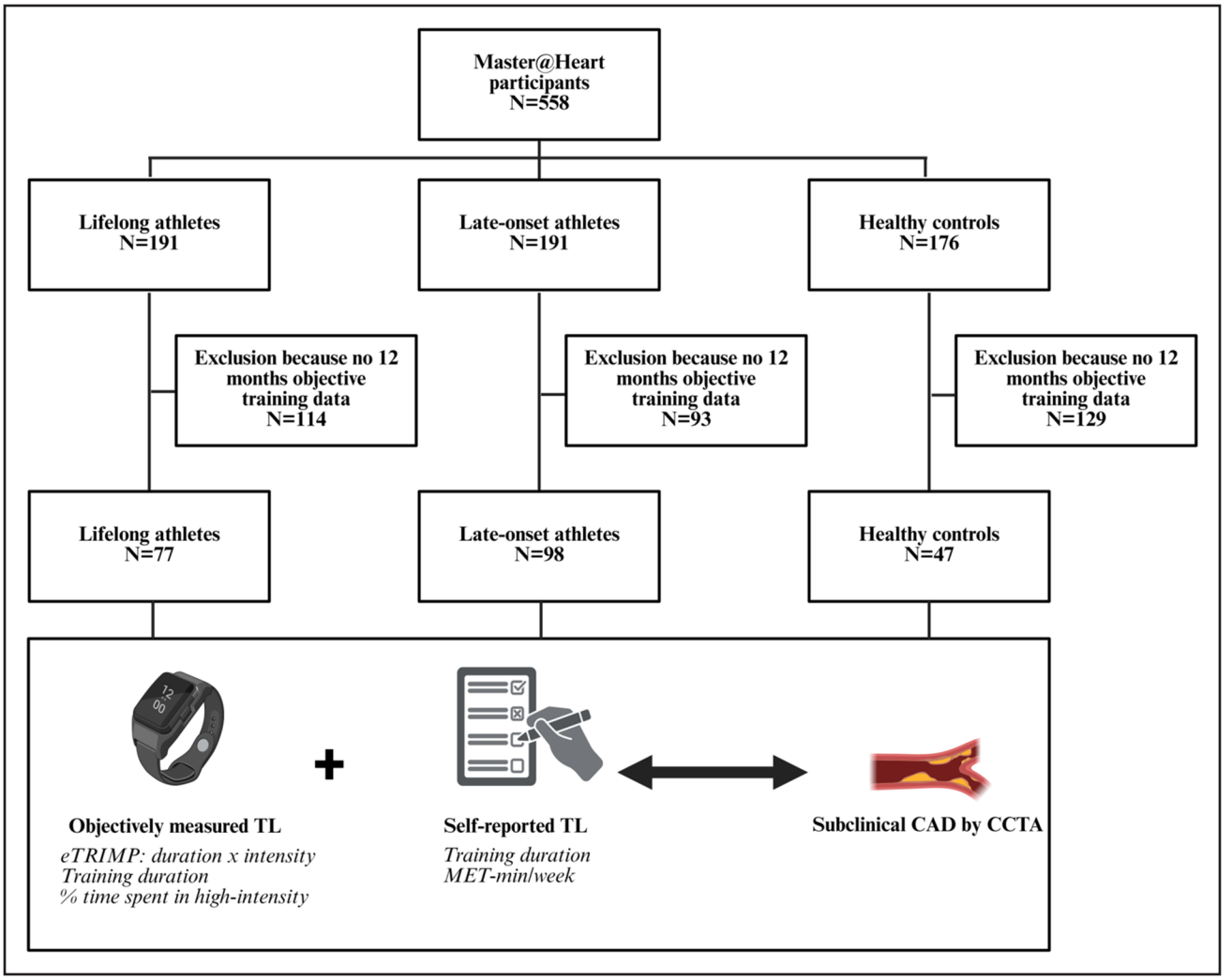

Figure 1. Flow chart of the study design.
CAD indicates coronary artery disease; CCTA, coronary computed tomography angiography; eTRIMP, Edwards training impulse; MET-min/week, metabolic equivalents of task multiplied by minutes of training per week; and TL, training load.

Cardiopulmonary Exercise Testing

Cardiorespiratory fitness was assessed as peak oxygen uptake, defined as the highest 30 seconds average of oxygen uptake, and measured during an incremental cycling stress test with respiratory exchange monitoring.[16]

Cardiovascular Risk Calculation With SCORE2 (Systematic Coronary Risk Evaluation 2)

Although individuals with known cardiovascular risk factors were excluded in accordance with the study design,[16] the presence of unrecognized risk factors could not be ruled out. Therefore, the SCORE2 (Systematic Coronary Risk Evaluation 2) algorithm was to estimate 10-year risk of fatal and nonfatal cardiovascular disease, following the 2021 European Society of Cardiology guidelines.[5]

Based on age-specific thresholds defined in these guidelines, participants were stratified into low-to-moderate, high, or very high cardiovascular risk categories. For individuals age <50 years, risk categories were defined as follows: low-to-moderate (<2.5%), high (2.5%–7.5%), and very high (≥7.5%). For individuals age 50 to 69 years, the respective cutoffs were low-to-moderate (<5%), high (5%–10%), and very high (≥10%).

Statistical Analysis

An explanation of the rationale for the initial sample size can be found elsewhere.[9,16] For the current study, we included all participants with availability of high-quality, objective TL.

Statistical analysis and creation of figures were performed with SPSS statistics (version 29.0.2.0), SAS (version 9.4), and GraphPad Prism (version 10.4.1). Continuous variables are presented as medians with interquartile range or means with SD, depending on normality. Categorical variables are presented as absolute numbers with percentages. Between-group differences were evaluated using chi-square test, Kruskal-Wallis test, or 1-way ANOVA where appropriate.

TL parameters were divided into quartiles based on the 25th, 50th, and 75th percentiles. The associations between TL quartiles and binary CAD characteristics were first assessed using unadjusted chi-square test and subsequently using adjusted multiple logistic regression. Odds ratios (ORs) with 95% confidence intervals (CIs) are reported for comparisons

between quartiles (Q), with the highest quartile Q4 and the lowest quartile Q1. Models were adjusted for traditional cardiovascular risk factors, including age, body mass index, systolic and diastolic blood pressure, HbA1c, measured LDL (low-density lipoprotein) cholesterol, triglycerides, lipoprotein(a), family history of CAD (defined as a first-degree relative with myocardial infarction before age 50 years in men or 60 years in women), and years of endurance exercise. Smoking was not included in the model because ex-smokers and current smokers were excluded by study design. The models assessing quartiles of objective training duration were in addition adjusted for the percentage of time spent in high-intensity exercise (intensity zones 4 and 5). Similarly, models assessing quartiles of high-intensity training were adjusted for objective training duration. This approach was used to isolate the independent associations of training duration and intensity with CAD.

In addition, a logistic regression model was fitted to obtain ORs with 95% CIs, with continuous TL parameters in relation to CAD characteristics (≥1 plaque, CAC>0, CAC>100). eTRIMP was divided by 100 and MET-min/week by 60, such that the ORs represent the change in odds per 100 AU increase in eTRIMP or per 60 MET-min/week increase, respectively, for easier interpretation. The model was adjusted for traditional cardiovascular risk factors and years of endurance exercise, as previously highlighted. The model assessing objective training duration was in addition adjusted for the percentage of time spent in high-intensity exercise (intensity zones 4 and 5). Penalized smoothing splines were used for the training parameter, allowing for a flexible, potentially nonlinear relationship between the log-odds and the TL parameter.[24] To minimize the risk of overfitting, the smoothing parameter was selected by generalized cross-validation. If no nonlinear relationship was observed, a linear relationship on the logit scale was plotted. This approach nonetheless allowed us to assess potential nonlinear patterns. There were no missing data for baseline characteristics or CCTA data in the cohort of 222 participants. A 2-sided *P* value <0.05 was considered statistically significant.

RESULTS

Study Population and TL Quantification

Median weekly average eTRIMP across the entire cohort was 914 AU (478–1281; Table S1). Quartiles of objectively measured weekly average eTRIMP were defined as follows: Q1, 80 to 477; Q2, 478 to 908; Q3, 919 to 1276; and Q4, 1293 to 4574 AU. Cardiorespiratory fitness, expressed as peak oxygen uptake and peak oxygen uptake predicted, was superior for higher eTRIMP quartiles (*P*<0.001). Median age did not differ across groups (*P*=0.342; Table 1). Median values of most cardiovascular risk factors were within normal reference ranges in the entire cohort, except for a slightly elevated LDL and total cholesterol (Table S1). Across eTRIMP quartiles, body mass index, systolic blood pressure, and HDL (high-density lipoprotein) differed significantly (*P*=0.005, *P*=0.030, and *P*<0.001, respectively). SCORE2 risk classification showed that the majority of participants fell into low to intermediate risk, with a small proportion categorized as high-risk, and none meeting criteria for very high risk (Table 1). Those in the high-risk group were more likely to have subclinical CAD (Table S2). Baseline characteristics of participants with available 12-month wearable TL data (n=222), included in this analysis, were comparable with those of the remaining Master@Heart participants without wearable data (n=336), suggesting minimal risk of selection bias (Table S3).

Objective and self-reported training duration increased significantly across eTRIMP quartiles (*P*<0.001). MET-min/week also differed significantly between quartiles (*P*<0.001), although values did not increase progressively across the groups. Years of endurance exercise training were similar (*P*=0.209) across quartiles (Table 1).

Baseline characteristics for quartiles of objective training duration, MET-min/week based on self-reported TL data, and self-reported training duration are available in Tables S4 through S6.

A total of 42 079 real-world training sessions, corresponding to 74 636 objectively measured training hours, were semiautomatically analyzed using our software tool (Table S1). To account for potential bias caused by timing of TL collection (ie, data collected 12 months before or after CCTA), a timing covariate was included in multivariable logistic regression models. This covariate was not significantly associated with CAD characteristics (Table S7). In addition, among 102 participants with 12 months of objective training data both before and after CCTA, no significant differences in TL parameters between pre- and post-CCTA were observed (Table S8).

Association Between Objectively Measured TL and CAD

Across eTRIMP quartiles, there was a significant, unadjusted global difference in the prevalence of ≥1 plaque (*P*<0.001), proximal plaques (*P*=0.016), calcified plaques (*P*=0.008), noncalcified plaques (*P*=0.047), partially calcified plaques (*P*=0.013), CAC>0 (*P*=0.002), and CAC>100 (*P*=0.012), whereas differences for CAC percentile ≥75 (*P*=0.178) did not reach statistical significance. In adjusted logistic regression controlling for traditional cardiovascular risk factors and years of endurance exercise, participants in the highest eTRIMP quartile (Q4, 1293–4574 AU) had significantly higher prevalence of ≥1 plaque (74.5% versus 36.4%; OR, 5.85; 95% CI, 2.33–14.71; *P*<0.001) compared with those in the lowest quartile (Q1, 80–477 AU). The presence of proximal plaques was also more common in Q4 versus Q1 (61.8% versus 32.7%; OR, 3.60; 95% CI, 1.48–8.77; *P*=0.005), as were calcified plaques (58.2% versus 27.3%; OR, 3.69; 95% CI, 1.54–8.85; *P*=0.003), and partially calcified plaques (40.0% versus 14.5%; OR, 5.18; 95% CI,

ORIGINAL RESEARCH ARTICLE

Table 1. Baseline and Exercise Characteristics of eTRIMP Quartiles (Weekly Average)

Quartiles of eTRIMP	Q1	Q2	Q3	Q4	*P* value
Quartile cutoffs, arbitrary units	80–477	478–908	919–1276	1293–4574	
Baseline characteristics					
Total number, No.	55	56	56	55	
Lifelong athletes, No.	10	21	24	22	
Late-onset athletes, No.	11	24	31	32	
Controls, No.	34	11	1	1	
VO_2 peak, ml/kg per min	41±8	44±7	48±7	49±8	<0.001*
VO_2 peak predicted, %	136±26	148±25	159±19	165±24	<0.001*
Age, years	52 (48–58)	53 (50–60)	54 (50–59)	55 (49–61)	0.342
BMI, kg/m^2	23 (22–25)	24 (23–26)	24 (22–25)	23 (22–24)	0.005*
HR in rest, bpm	55±8	54±8	53±8	52±10	0.286
Systolic BP, mm Hg	121 (113–130)	129 (118–139)	120 (115–130)	125 (115–130)	0.030*
Diastolic BP, mm Hg	75 (70–80)	78 (71–86)	78 (70–80)	75 (70–80)	0.317
HbA1c, %	5.4 (5.2–5.6)	5.5 (5.2–5.6)	5.5 (5.3–5.6)	5.5 (5.3–5.7)	0.157
Triglycerides, mg/dl	84 (61–101)	80 (68–108)	74 (61–96)	81 (59–109)	0.478
Total cholesterol, mg/dl	185 (161–212)	193 (171–214)	199 (171–212)	199 (172–217)	0.363
LDL cholesterol, mg/dl	118 (100–143)	125 (106–140)	132 (108–144)	119 (102–135)	0.603
HDL cholesterol, mg/dl	60 (48–68)	61 (53–69)	63 (54–71)	70 (60–80)	<0.001*
Lipoprotein(a), nmol/l	13 (7–66)	15 (7–51)	20 (7–38)	13 (7–49)	0.987
Risk according to SCORE2					
Low-intermediate risk, No. (%)	46 (83.6)	47 (83.9)	51 (91.1)	48 (87.3)	0.630
High risk, No. (%)	9 (16.4)	9 (16.1)	5 (8.9)	7 (12.7)	0.630
Very high risk, No. (%)	0	0	0	0	
Exercise characteristics based on wearables					
Objective training duration, h/week	1.9 (1.1–2.9)	4.7 (3.7–5.7)	7.3 (6.2–8.2)	10.4 (9.5–11.8)	<0.001*
Maximal HR (bpm) derived from training files	181 (172–187)	181 (172–185)	177 (170–183)	174 (165–181)	0.008*
Training time with HR monitoring, %	100 (95–100)	97 (90–100)	97 (91–100)	96 (91–99)	0.007*
TL 12 months before CCTA, No. (%)	24 (43.6)	31 (55.4)	42 (75.0)	39 (70.9)	0.002*
TL 12 months after CCTA, No. (%)	31 (56.4)	25 (44.6)	14 (25.0)	16 (29.1)	0.002*
Self-reported training duration, h/week	3 (2–8)	10 (6–12)	11 (10–13)	13 (11–15)	<0.001*
MET-min/week based on self-reported TL	1350 (990–4500)	4950 (2895–5400)	4905 (4102–6300)	4500 (3600–6030)	<0.001*
Years of endurance exercise	15 (5–32)	19 (14–34)	25 (9–34)	22 (13–37)	0.209

Results are presented as mean±SD, median with interquartile range, or total number with percentage. Differences were compared using ANOVA, Kruskal-Wallis, or chi-square test as appropriate. BMI indicates body mass index; BP, blood pressure; bpm, beats per minute; CCTA, coronary computed tomography angiography; eTRIMP, Edwards training impulse; HDL, high-density lipoprotein; HR, heart rate; LDL, low-density lipoprotein; MET-min/week, metabolic equivalent of task multiplied by minutes of training per week; Q, quartile; TL, training load; and VO_2, oxygen uptake.

*Statistically significant.

1.79–14.93; *P*=0.002). Similarly, a significantly higher proportion of participants in Q4 had CAC>0 (69.1% versus 32.7%; OR, 5.03; 95% CI, 2.04–12.35; *P*<0.001), and CAC>100 (38.2% versus 14.5%; OR, 3.50; 95% CI, 1.22–10.00; *P*=0.012). Trends for noncalcified plaques and CAC percentile ≥75 were directionally similar but did not reach statistical significance (Table 2; Figure 2).

When stratified by quartiles of objective training duration, global unadjusted *P* values indicated significant differences for ≥1 plaque (*P*=0.008), calcified plaques (*P*=0.034), partially calcified plaques (*P*=0.005), CAC>0 (*P*=0.011), and CAC>100 (*P*=0.001), and nonsignificant trends for other characteristics. In adjusted logistic regression models correcting for traditional risk factors, high-intensity exercise, and years of endurance exercise, participants in Q4 (9.2–26.5 hours/week) had a significantly higher prevalence of ≥1 plaque (70.9% versus 40.0%; OR, 4.42; 95% CI, 1.63–11.90; *P*=0.003) compared with Q1 (0.5–3.3 hours/week). The prevalence of ≥1 proximal plaque was higher in Q4 versus Q1 (60.0% versus 36.4%; OR, 3.45; 95% CI, 1.27–9.35; *P*=0.015), as were calcified plaques (54.4% versus 30.9%; OR, 3.21; 95% CI, 1.21–8.47; *P*=0.019), partially calcified plaques (43.6% versus 16.4%; OR, 8.70; 95% CI, 2.54–29.41; *P*<0.001), CAC>0 (65.5% versus 36.4%;

Table 2. Relationship Between Objectively Measured Training Load (eTRIMP, Objective Training Duration, and Training Intensity) and Coronary Artery Disease

	Q1	Q2	Q3	Q4	Unadjusted *P* value measured using chi-square test	Adjusted OR Q4 vs Q1	Adjusted *P* value Q4 vs Q1
eTRIMP, AU	80–477	478–908	919–1276	1293–4574			
≥1 plaque, No. (%)	20 (36.4)	29 (51.8)	36 (64.3)	41 (74.5)	<0.001*	5.85 (2.33–14.71)*	<0.001*
≥1 proximal plaque, No. (%)	18 (32.7)	27 (48.2)	31 (55.4)	34 (61.8)	0.016*	3.60 (1.48–8.77)*	0.005*
≥1 calcified plaque, No. (%)	15 (27.3)	27 (48.2)	29 (51.8)	32 (58.2)	0.008*	3.69 (1.54–8.85)*	0.003*
≥1 noncalcified plaque, No. (%)	7 (12.7)	5 (8.9)	12 (21.4)	15 (27.3)	0.047*	2.90 (0.99–8.55)	0.053
≥1 partially calcified plaque, No. (%)	8 (14.5)	11 (19.6)	16 (28.6)	22 (40.0)	0.013*	5.18 (1.79–14.93)*	0.002*
CAC >0, No. (%)	18 (32.7)	28 (50.0)	31 (55.4)	38 (69.1)	0.002*	5.03 (2.04–12.35)*	<0.001*
CAC percentile ≥75, No. (%)	9 (16.4)	8 (14.3)	14 (25.0)	16 (29.1)	0.178	2.28 (0.85–6.13)	0.103
CAC >100, No. (%)	8 (14.5)	9 (16.1)	13 (23.2)	21 (38.2)	0.012*	3.50 (1.22–10.00)*	0.020*
Objective duration, h/week	0.5–3.3	3.3–6.1	6.1–9.2	9.2–26.5			
≥1 plaque, No. (%)	22 (40.0)	30 (53.6)	35 (62.5)	39 (70.9)	0.008*	4.42 (1.63–11.90)*	0.003*
≥1 proximal plaque, No. (%)	20 (36.4)	27 (48.2)	30 (53.6)	33 (60.0)	0.085	3.45 (1.27–9.35)*	0.015*
≥1 calcified plaque, No. (%)	17 (30.9)	25 (44.6)	31 (55.4)	30 (54.4)	0.034*	3.21 (1.21–8.47)*	0.019*
≥1 noncalcified plaque, No. (%)	6 (10.9)	9 (16.1)	8 (14.3)	16 (29.1)	0.065	3.29 (0.95–11.36)	0.059
≥1 partially calcified plaque, No. (%)	9 (16.4)	11 (19.6)	13 (23.2)	24 (43.6)	0.005*	8.70 (2.54–29.41)*	<0.001*
CAC >0, No. (%)	20 (36.4)	26 (46.4)	33 (58.9)	36 (65.5)	0.011*	4.13 (1.52–11.11)*	0.005*
CAC percentile ≥75, No. (%)	10 (18.2)	8 (14.3)	13 (23.2)	16 (29.1)	0.252	2.96 (0.94–9.26)	0.064
CAC >100, No. (%)	9 (16.4)	7 (12.5)	12 (21.4)	23 (41.8)	0.001*	4.46 (1.36–14.71)*	0.014*
High-intensity zones 4+5, % of time	0–12	12–20	20–31	32–89			
≥1 plaque, No. (%)	29 (52.7)	37 (64.9)	33 (60.0)	27 (49.1)	0.326	2.18 (0.82–5.80)	0.119
≥1 proximal plaque, No. (%)	25 (45.5)	33 (57.9)	26 (47.3)	26 (47.3)	0.535	2.89 (1.07–7.80)*	0.036*
≥1 calcified plaque, No. (%)	22 (40.0)	32 (56.1)	27 (49.1)	22 (40.0)	0.246	2.22 (0.85–5.81)	0.104
≥1 noncalcified plaque, No. (%)	9 (16.4)	12 (21.1)	10 (18.2)	8 (14.5)	0.827	2.09 (0.58–7.50)	0.257
≥1 partially calcified plaque, No. (%)	13 (23.6)	17 (29.8)	14 (25.5)	13 (23.6)	0.860	3.13 (0.98–10.06)	0.055
CAC >0, No. (%)	27 (49.1)	34 (59.6)	30 (54.5)	24 (43.6)	0.361	1.93 (0.73–5.14)	0.185
CAC percentile ≥75, No. (%)	8 (14.5)	13 (22.8)	14 (25.5)	12 (21.8)	0.541	3.96 (1.18–13.28)*	0.026*
CAC >100, No. (%)	13 (23.6)	14 (24.6)	13 (23.6)	11 (20.0)	0.944	2.54 (0.77–8.34)	0.125

The differences across quartiles of eTRIMP (combination duration and intensity), objective training duration (h/week), and training intensity (percentage time training in high-intensity zones 4+5) in relation to coronary artery disease (CAD) characteristics are presented with, first, a global *P* value derived from the chi-square test across all quartiles. This is followed by the odds ratio (OR) for Q4 vs Q1 along with the corresponding 95% CI and adjusted *P* value, derived from multivariable logistic regression adjusting for age, body mass index, systolic and diastolic blood pressure, HbA1c, low-density lipoprotein cholesterol, triglycerides, lipoprotein(a), family history for CAD, and years of endurance exercise. The model assessing quartiles of objective training duration was in addition adjusted for exercise intensity (the continuous percentage of time spent in high-intensity exercise zones 4 and 5), whereas the model assessing quartiles of intensity was in addition corrected for continuously measured objective duration. To measure training intensity, a 5-zone heart rate model was used based on percentages of maximal heart rate (HRmax): zone 1 (50%–59% of HRmax), zone 2 (60%–69%), zone 3 (70%–79%), zone 4 (80%–89%), and zone 5 (90%–100%). High-intensity exercise was defined as time spent in zones 4 and 5. Coronary artery calcification (CAC) scores are expressed in Agatston units. AU indicates arbitrary units; eTRIMP, Edwards training impulse; and Q, quartile.

*Statistically significant.

OR, 4.13; 95% CI, 1.52–11.11; *P*=0.005), and CAC>100 (41.8% versus 16.4%; OR, 4.46; 95% CI, 1.36–14.71; *P*=0.014). Although trends comparing Q4 versus Q1 for noncalcified plaques and CAC percentile ≥75 were directionally similar, these associations did not reach statistical significance (Table 2; Figure S1).

For quartiles of relative time spent in high-intensity zones (4 and 5), no significant unadjusted overall differences were observed (all *P*>0.05). However, adjusted logistic regression, correcting for traditional risk factors, objective training duration, and years of endurance exercise, showed that participants in Q4 (32%–89% of training time at high-intensity) had higher odds of proximal plaques (47.3% versus 45.5%; OR, 2.89; 95% CI, 1.07–7.80; *P*=0.036) and CAC percentile ≥75 (21.8% versus 14.5%; OR, 3.96; 95% CI, 1.18–13.28; *P*=0.026) compared with Q1 (0%–12%) (Table 2; Figure 3).

Additional analyses of eTRIMP and objective training duration showed that Q4 was generally associated with higher odds of subclinical CAD characteristics compared with Q2. Comparisons of Q4 versus Q3 were directionally similar but mostly not significant, except for objective

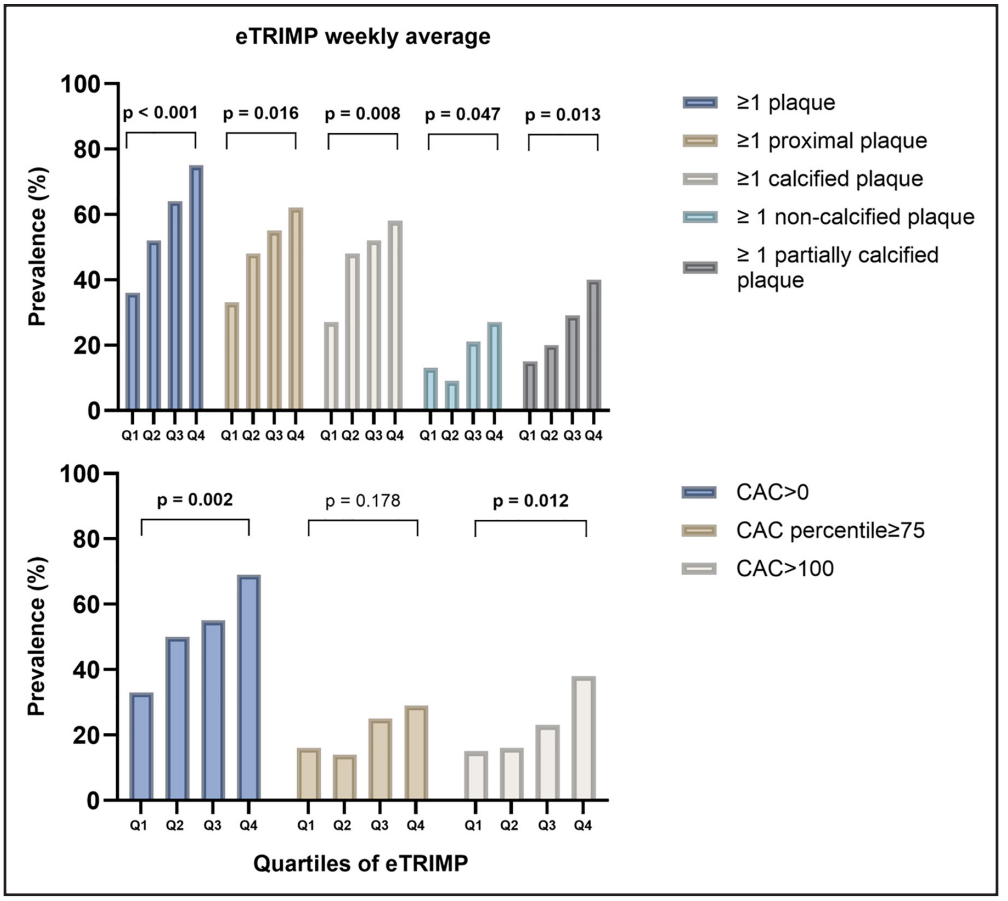

Figure 2. The relationship between objectively measured training load by eTRIMP and coronary artery disease.
Unadjusted global *P* values were derived from chi-square tests and are shown in the figure. Adjusted odds ratios comparing Q4 versus Q1 (adjusted for age, body mass index, systolic and diastolic blood pressure, HbA1c, low-density lipoprotein cholesterol, triglycerides, lipoprotein(a), family history of coronary artery disease, and years of endurance exercise) and corresponding *P* values are presented in Table 2. Quartiles were defined as follows: Q1, 80–477 AU; Q2, 478–908 AU; Q3, 919–1276 AU; and Q4, 1293–4574 AU. AU indicates arbitrary units; CAC, coronary artery calcification; eTRIMP, Edwards training impulse; and Q, quartile.

training duration, where CAC>100 and partially calcified plaque remained significant. In contrast, no clear dose-response or significant differences were observed for relative time in high-intensity exercise comparing Q4 with Q2 to Q3 (Table S9).

Association Between Self-Reported TL and CAD

For quartiles of MET-min/week based on self-reported TL and self-reported training duration in relation to CAD, unadjusted global *P* values did not reach statistical significance (all *P*>0.05). For MET-min/week, the adjusted OR was only significant for CAC>100 in Q4 versus Q1 (OR, 3.36; 95% CI, 1.05–10.76; *P*=0.041), whereas the adjusted OR for self-reported training duration was only significant for noncalcified plaques when comparing Q4 versus Q1 (OR, 5.13; 95% CI, 1.56–16.80; *P*=0.007) (Table 3; Figures S2 and S3). Additional analyses comparing Q4 with Q2 to Q3 of MET-min/week and self-reported training duration were not significant (Table S10).

Continuous Associations Between TL and CAD

All associations between continuously measured TL and CAD were linear on the logit scale using penalized smoothing splines, adjusted for traditional risk factors and years of endurance exercise. Higher eTRIMP was associated with higher odds of ≥1 plaque (OR, 1.14 [95% CI, 1.06–1.21]; *P*<0.001), CAC>0 (OR, 1.12 [95% CI, 1.05–1.19]; *P*<0.001), and CAC>100 (OR, 1.11 [95% CI, 1.03–1.20]; *P*<0.001). Similarly, higher objective training duration was associated with higher odds of ≥1 plaque (OR, 1.16 [95% CI, 1.05–1.27]; *P*=0.004), CAC>0 (OR, 1.15 [95% CI, 1.05–1.27]; *P*=0.004), and CAC>100 (OR, 1.16 [95% CI, 1.07–1.30]; *P*=0.011). Higher self-reported training duration was associated with higher odds of CAC>100 (OR, 1.07 [95% CI, 1.00–1.15]; *P*=0.047), whereas associations with ≥1 plaque and CAC>0 were borderline but did not reach statistical significance (*P*>0.05). MET-min/week based on self-reported TL was not significantly associated with those CAD markers (*P*>0.05) (Table 4; Figure 4).

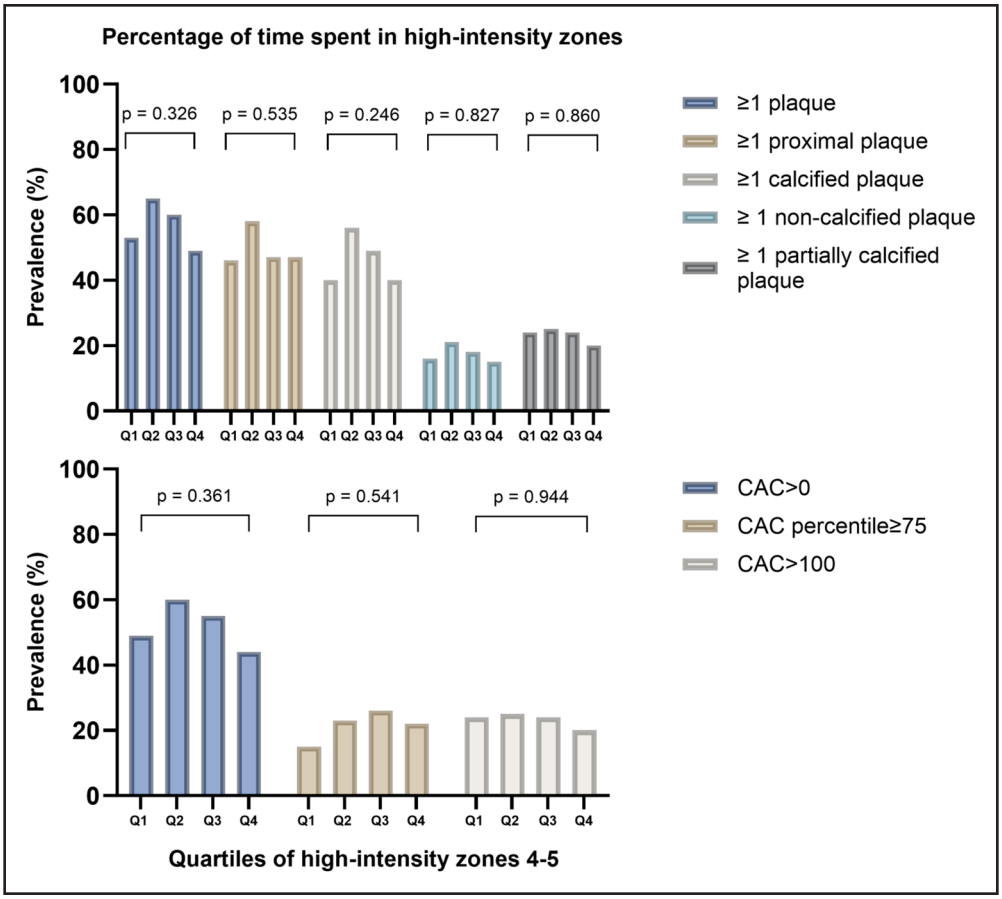

Figure 3. Association between relative time spent in high-intensity zones and coronary artery disease.
Unadjusted global *P* values were derived from chi-square tests and are shown in the figure. Adjusted odds ratios comparing Q4 versus Q1 (adjusted for age, body mass index, systolic and diastolic blood pressure, HbA1c, low-density lipoprotein cholesterol, triglycerides, lipoprotein(a), family history of coronary artery disease, and years of endurance exercise) and corresponding *P* values are presented in Table 2. Models were in addition adjusted for objectively measured training duration (continuous). Although the highest-intensity quartile showed elevated odds for proximal plaque and CAC percentile ≥75 compared with the lowest, no consistent dose-response pattern was observed across quartiles. Quartiles were defined as follows: Q1, 0%–12%; Q2, 12%–20%; Q3, 20%–31%; and Q4, 32%–89% of total exercise time spent in zones 4 and 5. High-intensity zones were based on a 5-zone heart rate model derived from percentages of maximum heart rate (HRmax): zone 1 (50%–59%), zone 2 (60%–69%), zone 3 (70%–79%), zone 4 (80%–89%), and zone 5 (90%–100%). CAC indicates coronary artery calcification; and Q, quartile.

DISCUSSION

The current analysis from the Master@Heart study provides a detailed evaluation of the dose-response relationship between endurance exercise training characteristics and subclinical CAD in athletes rigorously screened for traditional cardiovascular risk factors.[9,16] By combining self-reported and objectively measured TL in 222 middle-aged and older athletes and physically active controls, we demonstrate that high-volume endurance exercise, particularly when combined with sustained high-intensity effort (eTRIMP), is associated with a significantly higher prevalence of subclinical CAD. In contrast, high-intensity exercise alone was not clearly associated with CAD after accounting for objective training duration. Although the highest intensity quartile showed an elevated odds for selected CAD features compared with the lowest quartile, no consistent dose-response pattern across intensity categories was observed.

These findings extend the literature by showing that objectively measured TL, captured over 12 consecutive months using an automated algorithm, is strongly associated with CCTA-derived CAD characteristics, even after adjustment for traditional cardiovascular risk factors and years of endurance exercise training. Objective TL metrics such as training duration and eTRIMP, integrating training duration and intensity in a standardized and reproducible way, provide a robust approach for assessing recent training exposure in highly active populations. This is particularly relevant, as lifelong exercise exposure in sports cardiology research is often estimated using self-reported TL expressed as MET-min/week, a complex and potentially biased approach. The strong association between recent, objectively measured TL and subclinical CAD highlights the value of wearable-based monitoring for studying relationships between exercise exposure and subclinical coronary atherosclerosis.

Table 3. Relationship Between Self-Reported Training Load (MET-min/week Calculated Based on Self-Reported Training Load and Self-Reported Training Duration) and Coronary Artery Disease

	Q1	Q2	Q3	Q4	Unadjusted *P* value – chi-square	Adjusted OR Q4 vs Q1	Adjusted *P* value Q4 vs Q1
Self-reported MET-min/week	0–2880	2940–4500	4590–5430	5580–14 700			
≥1 plaque, No. (%)	27 (49.1)	41 (60.3)	24 (54.5)	34 (61.8)	0.508	1.73 (0.73–4.06)	0.212
≥1 proximal plaque, No. (%)	25 (45.5)	34 (50.0)	21 (47.7)	30 (54.5)	0.806	1.23 (0.52–2.89)	0.636
≥1 calcified plaque, No. (%)	20 (36.4)	34 (50.0)	21 (47.7)	28 (50.9)	0.382	1.83 (0.78–4.27)	0.165
≥1 noncalcified plaque, No. (%)	7 (12.7)	14 (20.6)	8 (18.2)	10 (18.2)	0.719	1.66 (0.54–5.13)	0.381
≥1 partially calcified plaque, No. (%)	11 (20.0)	24 (35.3)	9 (20.5)	13 (23.6)	0.174	1.31 (0.47–3.66)	0.609
CAC >0, No. (%)	24 (43.6)	39 (57.4)	21 (47.7)	31 (56.4)	0.383	1.70 (0.72–4.02)	0.226
CAC percentile ≥75, No. (%)	8 (14.5)	18 (26.5)	8 (18.2)	13 (23.6)	0.387	1.67 (0.58–4.84)	0.345
CAC >100, No. (%)	6 (10.9)	19 (27.9)	10 (22.7)	16 (29.1)	0.084	3.36 (1.05–10.76)*	0.041*
Self-reported duration, h/week	0–6	7–10	11–12	13–31			
≥1 plaque, No. (%)	26 (43.3)	38 (63.3)	32 (65.3)	30 (56.6)	0.074	1.78 (0.77–4.13)	0.177
≥1 proximal plaque, No. (%)	25 (41.7)	31 (51.7)	27 (55.1)	27 (50.9)	0.523	1.34 (0.58–3.13)	0.492
≥1 calcified plaque, No. (%)	20 (33.3)	33 (55.0)	25 (51.0)	25 (47.2)	0.096	1.71 (0.74–3.96)	0.207
≥1 noncalcified plaque, No. (%)	5 (8.3)	12 (20.0)	8 (16.3)	14 (26.4)	0.082	5.13 (1.56–16.80)*	0.007*
≥1 partially calcified plaque, No. (%)	12 (20.0)	13 (21.7)	14 (28.6)	18 (34.0)	0.303	2.45 (0.92–6.51)	0.073
CAC >0, No. (%)	24 (40.0)	34 (56.7)	28 (57.1)	29 (54.7)	0.199	1.86 (0.80–4.32)	0.150
CAC percentile ≥75, No. (%)	11 (18.3)	12 (20.0)	8 (16.3)	16 (30.2)	0.307	1.78 (0.69–4.59)	0.232
CAC >100, No. (%)	10 (16.7)	11 (18.3)	12 (24.5)	18 (34.0)	0.124	2.28 (0.84–6.19)	0.107

The differences across quartiles of MET-min/week based on self-reported training load and self-reported training duration (h/week) in relation to coronary artery disease (CAD) characteristics are presented with, first, a global *P* value derived from the chi-square test across all quartiles. This is followed by the odds ratio (OR) for Q4 vs Q1 along with the corresponding 95% CI and adjusted *P* value, derived from multivariable logistic regression adjusting for age, body mass index, systolic and diastolic blood pressure, HbA1c, low-density lipoprotein cholesterol, triglycerides, lipoprotein(a), family history for CAD, and years of endurance exercise. MET-min/week indicates metabolic equivalents of task multiplied by minutes of training per week; and Q, quartile.

*Statistically significant.

TL and Subclinical CAD in Middle-Age and Older Athletes

Our analysis supports the hypothesis of a reversed J-shaped association between exercise dose and CAD as moderate physical activity offers cardiovascular protection, whereas excessive endurance training may increase atherosclerotic burden.[6,7,9,12] In the present study, this pattern was most evident among participants with the highest TL, defined by training duration and eTRIMP, within a cohort composed exclusively of endurance athletes and physically active, low-risk controls. Sedentary individuals and those with traditional cardiovascular risk factors, who typically populate the higher-risk, low-activity end of the curve, were not represented. Consequently, our analyses represent a selected part of the physical activity spectrum, likely starting at the nadir with coverage up to extreme TL exposures. These participant characteristics

Table 4. Relationship Between Continuous Training Load (TL) Parameters and Coronary Artery Disease

	≥1 plaque		CAC >0		CAC >100	
TL parameter	OR (95% CI)	*P* value	OR (95% CI)	*P* value	OR (95% CI)	*P* value
eTRIMP, AU	1.14 (1.06–1.21)*	<0.001*	1.12 (1.05–1.19)*	<0.001*	1.11 (1.03–1.20)*	<0.001*
Objective duration, h/week	1.16 (1.05–1.27)*	0.004*	1.15 (1.05–1.27)*	0.004*	1.16 (1.04–1.30)*	0.011*
MET-min/week based on self-reported TL	1.01 (0.99–1.13)	0.234	1.05 (0.99–1.12)	0.282	1.08 (0.99–1.17)	0.114
Self-reported duration, h/week	1.11 (1.00–1.12)	0.062	1.06 (1.00–1.12)	0.073	1.07 (1.00–1.15)*	0.047*

Logistic regression using penalized smoothing splines to allow potential nonlinear associations was used, adjusted for age, body mass index, systolic and diastolic blood pressure, HbA1c, low-density lipoprotein cholesterol, triglycerides, lipoprotein(a), family history for coronary artery disease, and years of endurance exercise. The model of objective training duration was in addition adjusted for exercise intensity (the continuous percentage of time spent in high-intensity exercise zones 4 and 5). All relationships were linear on the logit scale. eTRIMP was divided by 100 and MET-min/week by 60, such that the ORs represent the change in odds per 100 AU increase in eTRIMP or per 60 MET-min/week increase, respectively, for easier interpretation. Data from 1 study participant with an eTRIMP of 4574 AU and 26.5 objective training hours were excluded from the objective training load analysis because it was an extreme outlier. AU indicates arbitrary units; CAC, coronary artery calcification; eTRIMP, Edwards training impulse; MET-min/week, metabolic equivalent of task multiplied by minutes of training per week; and OR, odds ratio.

*Statistically significant.

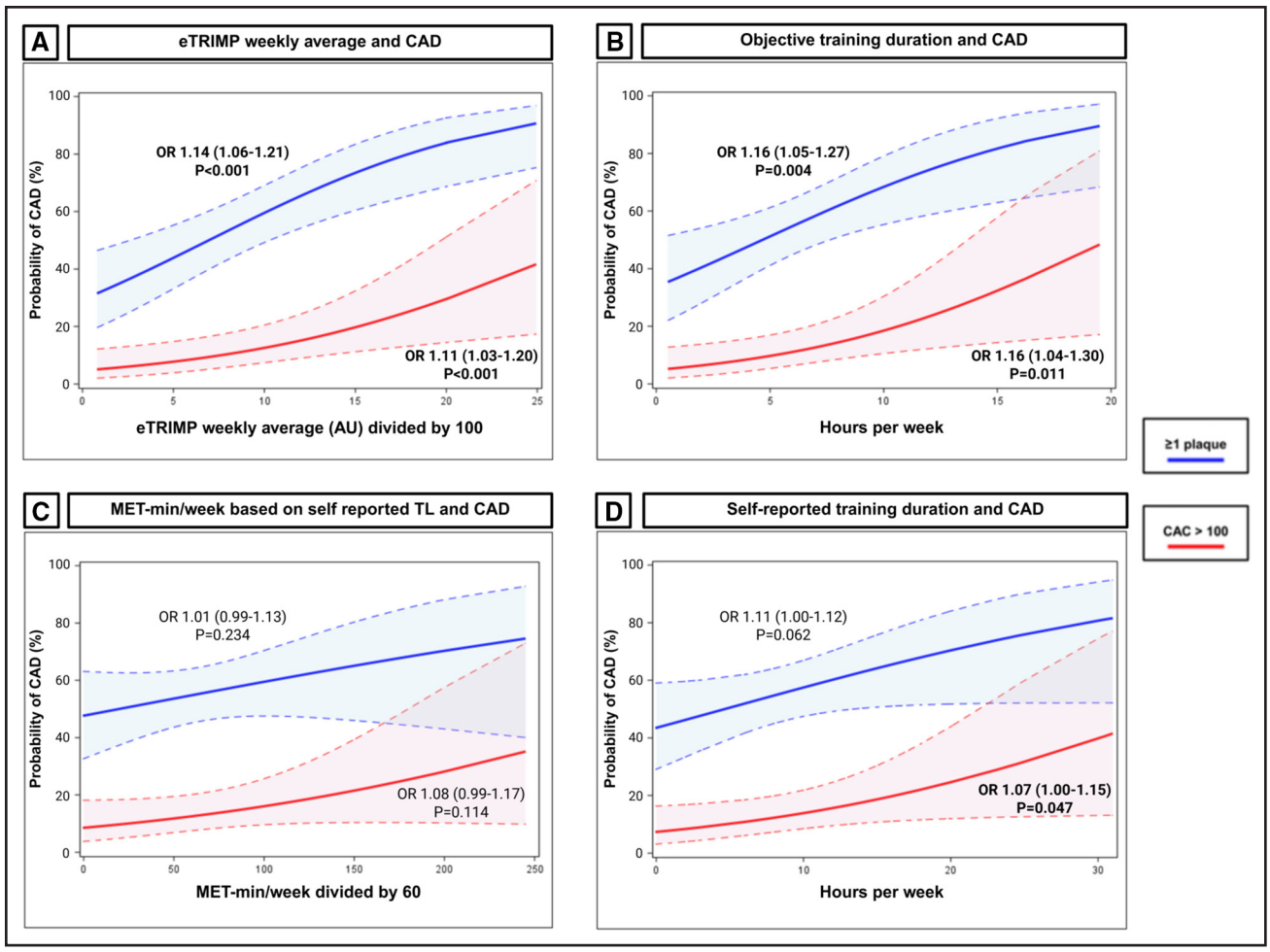

Figure 4. Relationship between continuous training load (TL) parameters and coronary artery disease (CAD).
Logistic regression with penalized smoothing splines was used to model potential nonlinear associations, adjusted for age, body mass index, systolic and diastolic blood pressure, HbA1c, low-density lipoprotein cholesterol, triglycerides, lipoprotein(a), family history of coronary artery disease, and years of endurance exercise. The model of objective training duration was in addition adjusted for exercise intensity (the continuous percentage of time spent in high-intensity exercise zones 4 and 5). All relationships were linear on the logit scale. Odds ratios (ORs) with 95% CIs and *P* values are described for each relationship. Graphs are presented with 95% CIs. eTRIMP was divided by 100 and MET-min/week by 60, such that the ORs represent the change in odds per 100 AU increase in eTRIMP or per 60 MET-min/week increase, respectively, for easier interpretation. Data from 1 study participant with an eTRIMP of 4574 AU and 26.5 objective training hours was excluded from the objective training load analysis because it was an extreme outlier. **A**, A significant association was observed between Edwards training impulse (eTRIMP) and ≥1 plaque as well as with coronary artery calcification (CAC) >100. **B**, A significant relationship was detected for objectively measured training duration with ≥1 plaque and CAC >100. **C**, A significant association for self-reported training duration with CAC >100 was observed, whereas ≥1 plaque was not significant. **D**, The associations between metabolic equivalents of task-minutes per week (MET-min/week) and CAD characteristics were not significant. AU indicates arbitrary units.

likely explain the linear associations between TL and CAD characteristics in our study population, as association across the full activity spectrum is hypothesized to be J-shaped.[12]

Previous studies examining the link between training characteristics and CAD have yielded conflicting results, probably resulting from differences in TL definitions and measurement approaches.[12–14] Aengevaeren et al showed that high-volume exercise (>2000 MET-min/week) was associated with higher CAC and plaque prevalence,[6] and later reported a relationship between high-intensity exercise, rather than exercise volume, with CAD progression.[14] However, they defined "very vigorous" exercise intensity as >9 METs, a threshold commonly exceeded by endurance athletes and insufficient to distinguish training extremes in this population.[14,17] Similarly, Pavlovic et al found an association between high-duration, low-intensity training and CAC, but used an even lower threshold for "high intensity" (>6 METs). These limitations underscore the need for more nuanced TL metrics in highly trained cohorts.[13] Our findings, using wearable-derived objective TL, suggest that the likelihood of subclinical CAD increases mainly when a high training volume is combined with sustained high-intensity effort (eTRIMP).

CAC and Clinical Events in Athletes

Emerging evidence supports the clinical relevance of CAC in athletes.[25,26] Berry et al reported that higher CAC was associated with increased risk of acute coronary events irrespective of physical activity level, even among individuals reporting very high activity volumes (>3000 MET-min/week).[26] In that study, CAC>100 was associated with a fourfold higher risk of acute myocardial infarction compared with CAC 0, although high-volume physical activity remained associated with lower all-cause mortality.[26]

In our study, participants in the highest eTRIMP category showed a higher prevalence of partially calcified, noncalcified, and proximal plaques. Plaque features such as noncalcified or partially calcified composition have been linked to greater plaque vulnerability, and proximal lesion location may be associated with larger myocardial territories at risk in the event of an acute coronary syndrome.[27] These findings raise concern that a subset of middle-aged and older endurance athletes may unknowingly harbor high-risk plaque characteristics, despite a healthy lifestyle and low cardiovascular risk profile as demonstrated by SCORE2 (Table 1).

Potential Mechanisms Explaining CAD in Athletes

The 12-month objective TL characteristics captured in this study likely reflect participants' habitual exercise patterns maintained over many years. This long-term consistency in exercise behavior may explain the strong associations observed between this wearable-based assessment and subclinical CAD, a pathophysiological process that evolves slowly over time.[5] Several pathophysiological mechanisms may underlie this association.[12,28]

First, repeated high-intensity exercise may alter hemodynamics by increasing shear stress, particularly in proximal coronary segments, potentially promoting focal atherosclerosis.[12,29,30] The observed higher prevalence of proximal plaques in high-TL athletes supports this hypothesis. Second, intense exercise is known to acutely elevate inflammatory markers,[31] and repeated exposure may contribute to vascular inflammation and plaque vulnerability, consistent with our finding of more partially calcified and noncalcified plaques in the highest TL quartile. High-volume training is associated with transient increases in parathyroid hormone,[32] which may promote vascular calcification, aligning with higher CAC observed in athletes. In addition, athletes often consume higher total caloric and fat intake to meet training demands.[12] Although such dietary patterns could influence lipid metabolism and atherosclerotic risk, diet was not assessed in our study. Berge et al found an inverse association between higher fat intake and coronary plaque burden in athletes, likely reflecting the mitigating effects of adherence to a Mediterranean diet,[33] but this needs to be validated. Last, genetic predisposition could interact with intensive training to influence CAD risk, and future analyses incorporating polygenic risk scores may help clarify this interaction.[12,34]

Objective Wearable-Derived TL: A New Frontier

This study is the first to leverage large-scale wearable-derived data to objectively assess TL over a defined period in a well-characterized cohort. Our semiautomated software platform enabled extraction of training duration, intensity, and eTRIMP with high fidelity. Previous work in this cohort has shown that self-reported training duration correlates poorly with objective training duration, whereas wearable-derived eTRIMP closely matches gold standard physiological indices such as the Lucia TRIMP (Training Impulse).[15]

Although self-reported MET-based questionnaires remain acceptable in general populations, their application in endurance athletes with high-volume, variable-intensity training is challenging. In our study, self-reported MET-min/week showed limited associations with coronary atherosclerosis, whereas objectively measured TL demonstrated more consistent and stronger associations across multiple CAD markers. Continuous analyses suggested an approximately linear relationship between objective TL and subclinical CAD, precluding identification of a clear threshold for "high" TL. Self-reported training data remain essential for estimating lifetime exposure, whereas objective measures may improve sensitivity for detecting dose-response associations in highly active populations.

Interpretation of these findings must account for the challenges inherent in assessing long-term exercise exposure using wearable-derived data. Although such measures provide detailed and objective information, their systematic collection, harmonization across devices, long-term storage, and analysis require substantial infrastructure and are not readily implemented in routine clinical practice. In the present study, assembling these data demanded considerable effort, including retrospective data retrieval, extensive data processing, and careful management of data governance and privacy in accordance with applicable regulations, including General Data Protection Regulation. These challenges illustrate why objective TL remains difficult to incorporate in large-scale and lifespan-spanning studies. In parallel, ongoing efforts are focused on developing standardized and privacy-compliant approaches to facilitate scalable analysis of wearable-derived training data already routinely collected by physically active individuals. Although such initiatives may help address current methodological barriers in future research, the clinical implications of objectively measured TL should, at present, be interpreted cautiously.

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Limitations

The cohort consisted exclusively of White men age 45 to 70 years, limiting the generalizability of findings to women and other ethnicities. Men were selected to optimize statistical power given the higher prevalence of CAD in men, particularly in the age group studied.[35] Furthermore, previous studies have found no association between lifelong exercise and CAD in female athletes, supporting our focus on males in this analysis.[36] Although the majority of objective TL was collected prospectively, TL for 86 participants was captured during the 12 months after CCTA. However, inclusion of a timing covariate in multivariable models showed no significant association with CAD characteristics, supporting the assumption that timing of TL data collection did not bias the results. Although 12 months of training data offer a robust estimate of recent exercise behavior,[15] they may not fully reflect lifelong exposure. However, analysis of 102 participants with training data 12 months before and after CCTA showed no change in TL, indicating that CCTA did not affect training behavior and that the data likely reflect consistent long-term TL. Next, it is possible that occasional training sessions were performed without a wearable, potentially leading to minor underestimation of TL; however, this bias is expected to be nondifferential.

CONCLUSIONS

In this well-characterized cohort of middle-aged and older male athletes and active controls, high training volume (training duration, hours/week), particularly when combined with cumulative high-intensity TL (eTRIMP), was independently associated with increased prevalence of subclinical CAD. Exercise intensity alone, in the absence of high duration, was not clearly linked to CAD burden. These findings underscore the potential of objectively measured TL for understanding associations with subclinical CAD in endurance athletes.

ARTICLE INFORMATION

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Disclosures

None.

Supplemental Material

Supplemental Methods
Tables S1–S10
Figure S1–S3
STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) Checklist

APPENDIX

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