Sports Medicine
https://doi.org/10.1007/s40279-026-02429-0

SYSTEMATIC REVIEW

# Coronary Artery Disease in Masters Athletes: A Systematic Review

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Accepted: 18 March 2026

## Abstract

**Background** Despite undertaking large volumes of structured exercise training, evidence suggests that masters athletes (MAs) remain susceptible to coronary artery disease (CAD). This review evaluated the prevalence and nature of CAD in MAs.

**Methods** PubMed, Medline, EMBASE, Web of Science and Cochrane Library were searched on 23 November 2024, from 1 January 2000. MAs were defined as apparently healthy individuals aged $\geq$ 35 years with an exercise training history indicative of regular engagement in sporting activity. The Appraisal tool for Cross-Sectional Studies (AXIS) was used to assess study quality.

**Results** A total of 17 studies reporting various measures of CAD in 2749 MAs (2127 male, 622 female) were included. Coronary artery calcium (CAC) was present in a significant proportion, with 565 MAs scoring $>$ 0, and 179 and 43 MAs exceeding thresholds of $>$ 100 and $>$ 400, respectively. Evidence of plaque was detected in at least 458 MAs. This was predominantly calcified in the majority of cases. Obstructive disease ($>$ 50% stenosis) was also identified in at least 51 MAs. Conventional cardiovascular (CV) risk factors such as smoking, dyslipidaemia, hypertension and diabetes were prevalent in this population.

**Discussion** Clinical and methodological heterogeneity made it challenging to perform accurate quantitative analyses. However, our data reiterate that MAs are not invulnerable to CAD. Long-term and high-intensity endurance training may be associated with changes that predispose to atherosclerotic disease. Female MAs, and those from low- and middle-income countries, are markedly underrepresented in literature.

**Conclusion** A more individualised approach to CV risk assessment may be warranted in the future to guide early CAD detection and subsequent management.

**Registration** PROSPERO 2024 CRD42024607539.

**Key Points**

Lifelong high-volume exercise training may be associated with a distinct CAD phenotype in MAs, despite the generally protective effects of exercise.

Our review identifies subgroups of MAs that may be at a higher risk of CAD and explores plausible mechanisms underlying this risk.

Most studies report exercise history and traditional CV risk factors, but few consider risk stratification or follow-up.

Tabulating reported and non-reported data highlights the need for longitudinal research to clarify causality and risk pathways.

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## 1 Introduction

Masters athletes (MAs) are individuals aged 35 years or above who engage in structured exercise training and regularly take part in athletic events [1]. They include former elite competitors who continue training after the peak of their careers, those who return to competition after periods of inactivity and individuals who were not active in their youth or young adulthood but became active in later life [2]. Whilst some maintain strict training regimens, others train intermittently and participate for personal enjoyment and leisure [3].

The benefits of exercise on cardiovascular (CV) health are firmly established [4]. However, there is increasing evidence recognising the association between long-term endurance exercise and diminishing CV returns [5]. This has contributed to the concept of a ‘U-shaped’ relationship between exercise dose and CV health, whereby both very low and very high levels of endurance training may attenuate—or even reverse—the protective effects of exercise [6–8]. Within this context, coronary artery disease (CAD) is of particular relevance in MAs, as it represents the leading cause of sudden cardiac death (SCD) in this group [9]. Acute coronary syndrome in MAs results from disruption of atherosclerotic plaques with subsequent coronary thrombosis [10]. Myocardial ischaemia can also arise owing to a mismatch between oxygen supply and demand, due to stable calcified plaque and fixed coronary stenosis [10]. Importantly, this risk may be present in asymptomatic MAs with no prior diagnosis of CAD, including those in whom traditional CV risk factors appear to be controlled [10, 11].

Although invasive coronary angiography remains the gold standard for diagnosing CAD, the degree of obstruction can also be evaluated non-invasively using coronary computed tomography (CT) imaging [12, 13]. Non-contrast CT scanning enables the measurement of coronary artery calcium (CAC) by determining a CAC score (CACS) [14]. The Agatston CACS is most commonly used to predict atherosclerotic burden and works by assigning each calcified lesion within the coronary vasculature a score on the basis of its peak attenuation and area [15]. The score of each lesion is its weighting factor multiplied by its area, and the Agatston CACS is the sum of all individual lesion scores across the coronary arteries [15].

Alternatively, contrast-enhanced CT angiography (CCTA) has been used to assess luminal stenosis and plaque morphology in MAs [16]. Obstructive (luminal stenosis > 50%) disease and the number of segments affected are both strong predictors of CV events [17, 18]. The morphology of plaques can be divided into calcified, non-calcified and mixed [13]. Mixed plaques convey the highest CV risk, whereas calcified plaques are associated with more favourable prognoses [19]. Other indicators of CAD in MAs include carotid intima-media thickness (IMT), and pulse wave velocity (PWV) and augmentation index (AIx), which are measured using carotid ultrasound and applanation tonometry respectively [20–23]. Maximal oxygen uptake ($VO_2$max) is a useful marker of cardiorespiratory fitness and is inversely proportional to CAD risk: higher levels are associated with improved CV health, reduced arterial stiffness and greater endothelial function [24]. Tools such as the Framingham Risk Score [25] (FRS) and Systematic Coronary Risk Evaluation 2 [26] (SCORE2) can be used to further stratify disease risk.

The relationship between significant volumes of endurance exercise and CAD has been recognised [4–6, 13]. However, the underlying mechanisms remain incompletely understood [11]. We aimed to systematically review and evaluate the existing evidence, concentrating on the interplay between training history, traditional risk factors, risk stratification and CT findings. We attempted to extend prior syntheses by mapping risk factors and mechanistic variables which have been investigated against those that may remain underreported. We hypothesised that the prevalence and phenotype of CAD in MAs varied according to training characteristics, with higher CV burden amongst those exposed to greater exercise volumes, and that this risk is further modified by the presence of traditional CV risk factors.

## 2 Methods

The search strategy and reporting of this review were conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. The review protocol was registered with the International Prospective Register of Systematic Reviews (PROSPERO 2024 CRD42024607539).

### 2.1 Eligibility Criteria

Studies were eligible if they had been published in English and assessed CAD in MAs. MAs were defined as apparently healthy males or females aged 35 years or older with no known CV disease, who were participating in athletic events and had a training history suggestive of sustained engagement in competitive or organised recreational sporting activity [3]. No minimum duration of training was set. The distinction between MAs and highly active individuals was based on the presence of structured exercise training undertaken in preparation for athletic competition. Although high levels of activity may involve substantial amounts of structured exercise, studies which did not explicitly clarify this were excluded [27]. We acknowledge the lack of objective criteria that define MAs; therefore, this conceptual and intent-based distinction was used to determine eligibility. Where participants had been characterised according to exercise volume, only those in the highest group were included. Studies in which relevant data were not reported or could not be extracted were excluded.

Measures of CAD included CACS [14], plaque morphology [19] and luminal stenosis [17, 18], carotid IMT [20], PWV [21], AIx [22, 23], $VO_2$max [24], or an otherwise new diagnosis of CAD. Assessment using invasive coronary angiography was not required, as its routine use for investigating CAD is impractical and ethically challenging. Studies examining acute CV responses to exercise were excluded, as this offers little insight into long-term prevalence or progression of CAD. Other systematic reviews were excluded; however, their reference lists were screened for potentially relevant primary studies. Abstracts, letters, journal articles and case reports or series were not considered.

### 2.2 Search Strategy and Data Sources

A comprehensive electronic literature search was performed independently by two researchers across PubMed, Medline, EMBASE, Web of Science and Cochrane Library on 23 November 2024, with all results shown from 1 January 2000. The search strategy used was as follows: (master athlete* OR endurance athlete* OR endurance activit* OR middle-aged athlete* OR veteran athlete* OR endurance exercise* OR vigorous exercise* OR intense exercise* OR lifelong exercise*) AND (‘coronary artery disease’ OR coronary athero* OR cardiovascular adaptation* OR physiological remodel* OR calcification).

The search results were imported into the Covidence systematic review software (Veritas Health Innovation, Melbourne, Australia) with duplicate records identified and removed. Subsequent title and abstract screenings were performed independently by the same two reviewers. The full texts of relevant articles were then retrieved for further assessment, with studies meeting the eligibility criteria being included. Any disagreements regarding the eligibility of a study involved the opinion of a third researcher and were resolved by consensus.

### 2.3 Data Extraction and Analysis

Data extracted from the eligible studies included: study characteristics (author, year, country, study design, criteria for MAs, sample size, age, sex, training history and type of exercise training, weekly training volume, maximal oxygen update), CV risk factors [smoking history, low-density lipoprotein [LDL] cholesterol, high-density lipoprotein [HDL] cholesterol, total cholesterol [TC], hypertension, diabetes, family history of CAD), CV risk stratification and follow-up (risk stratification tool, risk scoring, follow-up duration, CV events) and assessment of CAD (diagnosis of CAD, CACS, location of CAC, presence of atherosclerotic plaque, coronary luminal stenosis, atherosclerotic plaque type and distribution, carotid IMT, PWV, AIx, $VO_2$max). CACS can be interpreted as follows: 0 (no detectable coronary calcium, very low risk), 1–99 (mild atherosclerosis, low risk), 100–399 (moderate atherosclerosis, intermediate risk), $\geq 400$ (severe atherosclerosis; high risk) [19].

Since it was not anticipated that all included studies would report data across every domain, relevant information was collected where available [28]. Meta-analysis was not performed owing to the clinical and methodological heterogeneity of our data: specifically, studies differed markedly in their definition of MAs, methods used to quantify exercise exposure and outcome reporting. In addition, many outcomes were reported incompletely or without consistent denominators. Therefore, a narrative synthesis was considered the most appropriate approach and was conducted with an emphasis on training history, CV risk factors, risk stratification and CT findings.

We also prospectively recorded whether variables of interest were explicitly reported in each study. Variables were classified as ‘reported’ if quantitative or clearly defined qualitative data were provided in the manuscript or supplementary material. Variables that were not mentioned, incompletely described or not extractable were classified as ‘non-reported’.

### 2.4 Quality Assessment

Two reviewers independently assessed the methodological quality of each study using the Appraisal Tool for Cross-Sectional Studies (AXIS) [29]. The AXIS tool consists of 20 items and serves as a means of critical appraisal by addressing study designs, reporting quality and risk of bias. Four items (3, 7, 13 and 14) were removed from scoring, as it was not appropriate to assess non-response. Scores were reported as percentages to account for the changes made to the scoring system. Studies were given one (1) point for items met and none (0) for items not met or where this was unclear. Interpretation of overall study quality was left to the discretion of the research team. Any disagreements regarding the quality of a study involved the opinion of a third researcher and were resolved by consensus.

## 3 Results

### 3.1 Study Selection

The initial literature search identified 9301 studies, with 6342 remaining after duplicate removal. Title and abstract screening excluded 6232 studies, which left 110 for full-text screening. From these, 17 studies met the inclusion criteria. Reasons for exclusion were: participants not considered MAs ($n = 42$); abstracts, letters, journal articles, case reports or case series ($n = 29$); unable to accurately extrapolate data ($n = 19$); other systematic reviews ($n = 2$); and same population of MAs ($n = 1$). No further eligible studies were identified from screening the reference lists of relevant reviews during the literature search.

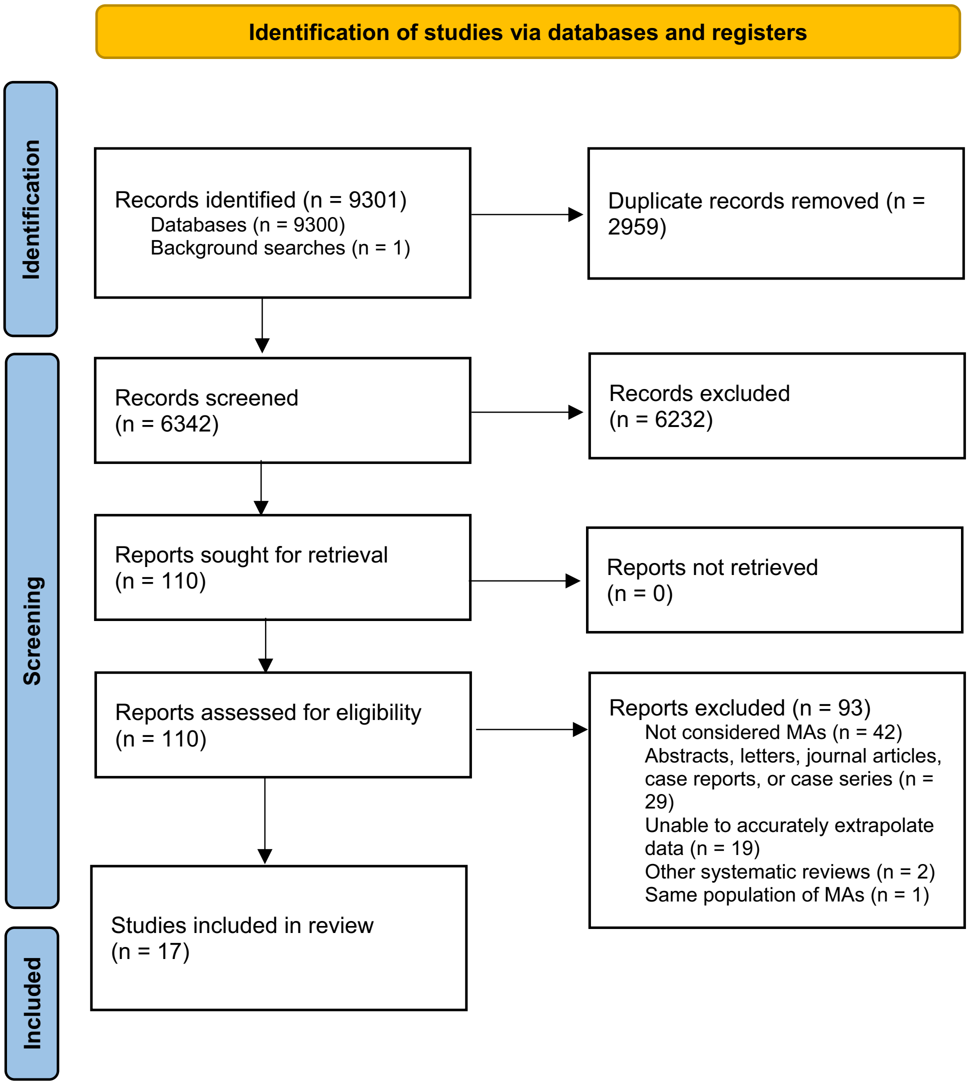
Although 18 studies initially met the inclusion criteria, four studies [30–33] comprised two pairs that reported different analyses from the same athlete cohorts. To avoid duplication, data were extracted from the study within each pair that the authors felt provided the most comprehensive and relevant outcome information. One of these studies [31] did not contribute any extractable data to our measures of interest and was excluded from quality assessment, as it did not inform the evidence synthesis. After accounting for cohort overlap and data availability, a final total of 17 studies were included. No study was excluded on the basis of quality assessment. A full breakdown of the selection process is shown in Fig. 1.

### 3.2 Study and Participant Characteristics

A total of 17 studies were published over a 17-year period between 2008 and 2024. Collectively, 6 studies were from the USA [34–39]; two each were from Italy [40, 41], Germany [42, 43] and Canada [32, 33]; and one each was from the Netherlands [44], Belgium [45], Portugal [30], Korea [46] and the UK [47]. Nine studies were cross-sectional [33, 34, 36, 37, 39, 41, 42, 45, 47] and six were observational [30, 32, 35, 38, 44, 46]. The other studies were retrospective [40] and prospective [43].

A total of 2749 apparently healthy MAs were included, of whom 2127 were male and 622 were female. Participant age, training history and type of exercise training, and/or weekly training volume were reported in all studies.

**Fig. 1** PRISMA flow chart detailing study selection process



$VO_2$max was reported in five studies [34, 39, 45–47]. Study characteristics are presented in Table 1.

### 3.3 CV Risk Factors

All but three studies [35, 41, 46] presented CV risk factor data in MAs (Table 2). At least 549 (20%) MAs had a positive smoking history. At least 160 and 42 MAs had a history of hypertension and diabetes, respectively. A positive family history of CAD was reported in at least 304 MAs.

In total, seven studies [30, 32, 34, 40, 42, 43, 47] calculated CV risk of MAs (Table 3); four studies [32, 42, 43, 47] used FRS, two studies used SCORE/SCORE2 and one study [34] used the Multi-Ethnic Study of Atherosclerosis (MESA). Two studies [32, 43] followed up MAs and reported a total of 14 CV events.

### 3.4 Evaluation of CAD

CAD in MAs was evaluated differently across all studies (Table 4). Collectively, 11 studies [30, 34–36, 39, 42–47] quantified CAD in terms of CAC. At least 565 MAs were found to have CACS > 0. Of these, at least 179 MAs had CACS > 100 and at least 57 and 43 had CACS > 300 and CACS > 400, respectively.

Seven studies [30, 37, 40, 44–47] discussed the presence of plaque. Plaque was identified in at least 458 MAs and was obstructive in at least 51 MAs. At least 246 plaques were calcified, which was the most common type. A further 135 and 94 plaques were identified as mixed and non-calcified, respectively.

Two studies [34, 41] measured carotid IMT; however, objective numerical data could not be extracted in one study [34]. This study [34] also measured AIx in MAs whilst separately reporting PWV data which could not be extracted.

Two other studies [33, 38] highlighted the number of MAs who had been diagnosed with CAD ($n = 79$), although the means of investigation was not specified.

### 3.5 Reported Data Compared with Non-reported Data

Across most studies, several factors and characteristics were inconsistently reported (Table 5). Nearly all studies included information on the exercise histories and training volumes of MAs and most addressed traditional CV risk factors. However, CT findings such as CAC and atherosclerotic plaque location were not always present. Risk stratification and follow-up assessments were absent in the majority of studies.

### 3.6 Quality Assessment

Studies were scored out of 16. All but seven studies [34, 35, 39, 41–43, 45] scored 16/16 (100%). The remainder scored 15/16 (94%). Studies were rated as high quality. A full scoring breakdown is provided in the Supplementary Material.

## 4 Discussion

This review summarises the prevalence of CAD in MAs, in terms of their training history and CV risk profiles. Our data indicate that atherosclerotic disease and plaque formation is present in over 20% and 16% of Mas, respectively. These findings support existing concerns that MAs may still be vulnerable to the development of CAD despite their high levels of exercise.

### 4.1 Assessment of CAD

The reporting of CAC and plaque burden continues to challenge the notion that high-volume endurance exercise is uniformly protective against CAD [48]. However, these results must be interpreted with caution, as the included studies differed substantially in their definitions of MAs, approaches to quantifying training load, imaging modalities and reporting of CV risk factors. This limits direct comparability between studies and precludes definitive conclusions regarding causality or risk.

Seven studies [30, 35, 36, 43, 45–47] detected CACS > 100 in a significant number of MAs, which is suggestive of moderate atherosclerotic plaque and possibly obstructive CAD [49]. In addition, 43 MAs across three studies [36, 43, 45] were found to have CACS > 400, which is thought to carry a high likelihood of at least one coronary artery stenosis [49]. However, across the ten studies [30, 34–36, 42–47] that reported CAC distributions, just over 46% of MAs had a CACS of zero, indicating a very low likelihood of obstructive disease [50].

Aengevaeren et al. detected coronary plaques in more than 77% of MAs, which was the highest amongst reviewed cohorts [44]. Follow-up analyses revealed exercise intensity—rather than volume—to be more strongly associated with elevated CAC and subsequent plaque formation [51]. Although exercise intensity could not be directly quantified, chronic endurance training may paradoxically promote changes conducive to atherosclerotic development through altered haemodynamic forces and endothelial injury [52]. This may be especially true in areas of turbulent blood flow, such as the proximal left anterior descending artery (LAD) [52].

Most plaques in MAs were reported as calcified, which are considered to be stable and less prone to rupture [13].

**Table 1** Characteristics of included studies and study participants

| Study | Year | Country | Study design | Criteria for MAs | Sample size | Sex (M/F) | Age (years) | Training history (years) | Weekly training volume | $VO_2$max (mL/kg/min) |
|---|---|---|---|---|---|---|---|---|---|---|
| Aengevaeren et al. [44] | 2017 | The Netherlands | Observational | Healthy male athletes aged ≥ 45 years who engaged in competitive or recreational leisure sports (> 2000 MET-minutes per week) | 75 | M | 55.9 ± 6.9 | 40.0 [35.0–47.0] | 5.7 [4.6–7.3] h | N/A |
| Bachman et al. [34] | 2021 | USA | Cross-sectional | Healthy athletes aged 40–65 years. Most competed annually in ultra-endurance events such as long-distance cycling races, ultramarathons and Ironman triathlons | 25 | 14 M, 11 F | 50.0 ± 1.0 | 19.0 ± 2.0 | 11.5 ± 0.6 h | 53.0 ± 1.6 |
| [a]Certo Pereira et al. [30] | 2024 | Portugal | Observational | Veteran male asymptomatic athletes aged ≥ 40 years old, participating in regular exercise for ≥ 4 h (and ≥ 66 MET-hours) per week, for ≥ 5 consecutive years | 56 | M | 47.5 ± 5.4 | 16.8 ± 9.2 | 10.2 ± 5.1 h | N/A |

**Table 1** (continued)

| Study | Year | Country | Study design | Criteria for MAs | Sample size | Sex (M/F) | Age (years) | Training history (years) | Weekly training volume | $VO_2$max (mL/kg/min) |
|---|---|---|---|---|---|---|---|---|---|---|
| De Bosscher et al. [45] | 2023 | Belgium | Cross-sectional | Male athletes aged 45–70 years. MAs reported engagement in: cycling ≥ 8 h or running ≥ 6 h per week, or triathlon (combination of swimming, cycling and running) ≥ 8 h per week for at least 6 months prior to baseline. Lifelong and late-onset athletes started regular endurance exercise training at < 30 years and > 30 years of age, respectively | 382 (MAs: 191; LOAs: 191) | M | MAs: 56.0 [51.0–61.0]; LOAs: 55.0 [51.0–60.0] | MAs: 36.0 [29.0–44.0]; LOAs: 14.0 [9.0–20.0] | MAs: 5070.0 [4500.0–6240.0]; LOAs: 4884.0 [3720.0–5790.0] MET-minutes | MAs: 48.0 [43.0–53.0]; LOAs: 46.0 [41.0–51.0] |
| Gervasi et al. [40] | 2019 | Italy | Retrospective | Athletes aged > 35 years, practicing competitive sports and having participated in competitions for at least 1 year | 167 | 143 M, 24 F | M: 53.8 ± 10.1; F: 53.8 ± 8.0 | ≥ 1.0 | Not specified | N/A |
| Gori et al. [41] | 2015 | Italy | Cross-sectional | Athletes aged 40–60 years, participating in ≥ 4.4 ± 2.6 h of weekly physical activity | 100 | 82 M, 18 F | 50.0 ± 6.7 | Not specified | 7.0 ± 2.6 h | N/A |

**Table 1** (continued)

| Study | Year | Country | Study design | Criteria for MAs | Sample size | Sex (M/F) | Age (years) | Training history (years) | Weekly training volume | $VO_2$max (mL/kg/min) |
|---|---|---|---|---|---|---|---|---|---|---|
| Jafar et al. [35] | 2019 | USA | Observational | Group A comprised runners aged ≥ 45 years who had competed in at least ten ultra-marathons (races covering > 50 km) and/or Ironman competitions (consisting of a 3.2-km swim, 161-km bicycle ride and 42-km run) in 10 years. Group B included runners aged ≥ 45 years who had participated in more than nine marathons over 10 years. Group C comprised runners aged ≥ 45 years who had competed in more than nine shorter races, defined as races covering less than 13.1 miles over 10 years | 56 | 37 M, 19 F | A: 60.9 ± 7.6; B: 53.0 ± 7.7; C: 59.3 ± 7.3 | A: 35.4 ± 11.7; B: 19.1 ± 7.7; C: 27.3 ± 15.0 | A: 42.9 ± 12.3; B: 40.0 ± 10.9; C: 33.5 ± 15.6 miles | N/A |

**Table 1** (continued)

| Study | Year | Country | Study design | Criteria for MAs | Sample size | Sex (M/F) | Age (years) | Training history (years) | Weekly training volume | $VO_2$max (mL/kg/min) |
|---|---|---|---|---|---|---|---|---|---|---|
| Kim et al. [46] | 2020 | Korea | Observational | Athletes aged ≥ 40 years and < 60 years, exercise experience ≥ 3 years, exercise frequency ≥ 2 times per week and number of marathons completed ≥ 5. Subjects who showed resting SBP/DBP < 140/90 mm Hg and maximal exercise SBP < 210 mm Hg were classified in the normal blood pressure group, while subjects with resting SBP/DBP < 140/90 mm Hg and maximal exercise SBP ≥ 210 mm Hg were placed in the exercise-induced hypertension group | 50 | M | NBPG: 51.7 ± 4.9; EIHG: 54.5 ± 5.0 | NBPG: 14.1 ± 4.9; EIHG: 14.3 ± 5.5 | Not specified | NBPG: 53.3 ± 6.8; EIHG: 51.9 ± 6.5 |
| Kröger et al. [42] | 2010 | Germany | Cross-sectional | Male runners > 50 years and had completed ≥ 5 full-distance marathon races (42.195 km) during the preceding 3 years | 100 | M | 57.0 ± 6.0 | Not specified | 57.0 ± 16.0 km | N/A |

**Table 1** (continued)

| Study | Year | Country | Study design | Criteria for MAs | Sample size | Sex (M/F) | Age (years) | Training history (years) | Weekly training volume | $VO_2$max (mL/kg/min) |
|---|---|---|---|---|---|---|---|---|---|---|
| Merghani et al. [47] | 2017 | UK | Cross-sectional | MAs were > 40 years of age, ran ≥ 10 miles or cycled ≥ 30 miles per week, and have continued to do so for ≥ 10 years, and competed in ≥ 10 endurance events, including marathons (26.2 miles, 42.2 km), half marathons (13.1 miles, 21.1 km), 10-km races or endurance cycling races ranging from 41.1 to 161.5 miles, 66 to 260 km) over a 10-year period | 152 | 106 M, 46 F | 54.4 ± 8.5 (M: 55.1 ± 9.1; F: 53.1 ± 7.1) | M: 33.4 ± 12.9; F: 26.1 ± 10.9 | M: 7.5 ± 3.8; F: 7.7 ± 2.9 h | M: 44.4 ± 7.0; F: 40.4 ± 7.3 |
| Mohlenkamp et al. [43] | 2008 | Germany | Prospective | Male marathon runners aged ≥ 50 years and had completed ≥ 5 full-distance marathons (42.195 km) during the preceding 3 years | 108 | M | 57.2 ± 5.7 | Not specified | 4686.0 ± 2285.0 MET-minutes | N/A |
| [b]Morrison et al. [33] | 2018 | Canada | Cross-sectional | Male and female recreational and competitive athletes aged ≥ 35 years, who engaged in moderate-to-vigorous-intensity physical activity, ≥ 3 days per week over the preceding 3 months | 798 | 500 M, 298 F | 54.6 ± 9.5 | 35.1 ± 14.8 | 10.9 ± 6.4 h | N/A |

**Table 1** (continued)

| Study | Year | Country | Study design | Criteria for MAs | Sample size | Sex (M/F) | Age (years) | Training history (years) | Weekly training volume | $VO_2$max (mL/kg/min) |
|---|---|---|---|---|---|---|---|---|---|---|
| Roberts et al. [36] | 2017 | USA | Cross-sectional | Male runners who had participated in 25 consecutive Twin Cities Marathon races | 50 | M | 59.4 ± 0.9 | Age started: 28.1 ± 1.2 (7.0–48.0) | Not specified | N/A |
| Roberts et al. [37] | 2016 | USA | Cross-sectional | Female runners who had participated in the Twin Cities Marathon for ≥ 10 consecutive years | 26 | F | 56.0 ± 10.0 | Age started: 36.0 ± 13.0 (with CAC); 25.0 ± 9.0 (without CAC) | 56.0 ± 24.0 (with CAC); 59.0 ± 36.0 (without CAC) miles | N/A |
| Shapero et al. [38] | 2016 | USA | Observational | Men and women over the age of 35 years who participate in organised competitive sport | 591 | 391 M, 200 F | 50.0 ± 9.0 | 21.3 ± 12.9 | 10.3 ± 5.5 h | N/A |
| Wilund et al. [39] | 2008 | USA | Cross-sectional | Subjects aged 60–80 years who were actively training and had competed in middle- or long-distance races at the local or national level for ≥ 2 years | 13 | 7 M, 6 F | M: 66.3 ± 4.4; F: 66.8 ± 6.5 | Not specified | Not specified | M: 36.5 ± 6.5; F: 29.4 ± 5.9 |

Data reported as either: *n*, mean ± SD, or median [interquartile range]

*MET* metabolic equivalent, *LOAs* late-onset athletes, *NBPG* normal blood pressure group, *EIHG* exercise-induced hypertension group, *SBP* systolic blood pressure, *DBP* diastolic blood pressure

[a] Dores et al. [31] utilised the same population of MAs as Certo-Pereira et al. [30]

[b] Morrison et al. [32] utilised the same population of MAs as Morrison et al. [33]

**Table 2** Cardiovascular risk factors reported in masters athletes

| Study | Smoking history | Low-density lipoprotein (LDL) cholesterol | High-density lipoprotein (HDL) cholesterol | Total cholesterol | Hypertension | Diabetes | Family history of CAD |
|---|---|---|---|---|---|---|---|
| Aengevaeren et al. [44] | 35 (46.7%) | – | – | 5.4±1.0 mmol/L | 6 (8%) | 2 (3%) | 25 (33%) |
| Bachman et al. [34] | | 2.8±0.1 mmol/L | 1.9±0.1 mmol/L | 5.1±0.2 mmol/L | – | – | 5 (20%) |
| Certo Pereira et al. [30] | 7 (12.5%) | 121.2±32.6 mg/dL | 66.7±15.9 mg/dL | 193.4±34.7 mg/dL | 3 (5.4%) | | 1 (1.8%) |
| De Bosscher et al. [45] | – | MAs: 121.0 [105.0–139.0]; LOAs: 124.0 [105.0–143.0] | MAs: 64.0 [55.0–74.0]; LOAs: 64.0 [56.0–76.0] mg/dL | MAs: 193.0 [172.0–212.0]; LOAs: 196.0 [177.0–215.0] mg/dL | – | – | MAs: 12 (6.3%); LOAs: 13 (6.8%) |
| Gervasi et al. [40] | 16 (9.6%) | – | – | – | 34 (20.4%) | 6 (3.6%) | 25 (15%) |
| Kröger et al. [42] | 57 (57%) | 121.0±30.0 mg/dL | 74.0±17.0 mg/dL | 227.0±43.0 mg/dL | 12.1% | – | – |
| Merghani et al. [47] | None | M: 2.9±0.4; F: 2.8±0.3 mmol/L | – | M: 4.6±0.4; F: 4.5±0.4 mmol/L | – | 26 (17.1%) | – |
| Mohlenkamp et al. [43] | 61 (56.5%) | 121.0±29.0 mg/dL | 73.8±17.3 mg/dL | 227.0±42.0 mg/dL | – | None | – |
| Morrison et al. [33] | 207 (34.6%) | – | – | – | 61 (7.7%) | 8 (1.0%) | – |
| Roberts et al. [36] | 24 (48%) | 111.9±3.7 [54.0–174.0] mg/dL | 58.0±1.6 [35.0–83.0] mg/dL | 186.6±4.1 [135.0–257.0] mg/dL | 12 (24%) | None | 21 (42%) |
| Roberts et al. [37] | 5 (20%) | 103.0±23.0 mg/dL | 73.0±15.0 mg/dL | 189.4±31.9 mg/dL | 3 (12%) | None | 13 (50%) |
| Shapero et al. [38] | 137 (23.2%) | – | – | – | 36 (6.1%) | – | 189 (32%) |
| Wilund et al. [39] | | 123.0±9.5 mg/dL | 56.9±3.5 mg/dL | 193.0±12.0 mg/dL | – | – | – |

Data reported as either *n* (%), mean ± standard deviation (SD) or median [interquartile range]

*LOAs* late-onset athletes

However, mixed plaques are vulnerable and rupture prone and are more commonly seen in non-athletic populations [47]. Their relative paucity in MAs could be explained by favourable metabolic adaptations associated with consistent exercise training, which support a shift towards plaque stabilisation [53]. Yet, the prevalence of CAC amongst otherwise asymptomatic MAs raises questions about its prognostic value, implying that plaque presence may represent a benign and adaptive response to mechanical stress [54, 55]. However, five studies [30, 40, 45–47] detected obstructive plaque in a subset of MAs. Obstructive disease is clinically relevant, as fixed coronary stenosis can limit myocardial perfusion during periods of increased demand, and even predominantly calcified plaques may be implicated in exercise-induced ischaemia under extreme physical stress [56].

This could help to explain why CAD remains the leading cause of SCD in MAs [11]. SCD represents the most catastrophic CV event and continues to impose a substantial global burden [57]. Unfortunately, successful resuscitation following sudden cardiac arrest (SCA) in athletes remains poor [58]. Sports-related SCA and SCD accounts for a small proportion of all events; however, it disproportionately affects middle-aged male athletes during endurance exercise [59, 60]. Female athletes have demonstrated higher rates of successful resuscitation following SCA, as well as a lower prevalence of underlying structural heart disease in these cases [61]. Owing to study heterogeneity and limited follow-up, we could not establish an association between CAD burden and subsequent CV events. However, CV risk in MAs likely arises from a combination of factors that collectively heighten arrhythmic susceptibility in MAs, which highlights the need to further elucidate the determinants of SCD in this population.

**Table 3** Cardiovascular risk stratification in masters athletes

| Study | Risk stratification tool | Risk scoring | Follow-up duration | CV events |
|---|---|---|---|---|
| Bachman et al. [34] | MESA | 2.3 ± 0.5 | – | – |
| Certo Pereira et al. [30] | SCORE2 | Low-to-moderate: 45 (80.4%); high 10 (17.9%) | – | – |
| Gervasi et al. [40] | SCORE | Low: 56 (36.8%); moderate: 101 (66.4%); high 7 (4.6%); very high 2 (1.3%) | – | – |
| Kröger et al. [42] | FRS | 7.0 ± 3.7 | – | – |
| Merghani et al. [47] | FRS | M: 4.3 ± 3.3; F: 1.3 ± 0.8 | – | – |
| Mohlenkamp et al. [43] | FRS | 7.0 [4.0–9.0] | 21.0 [18.6–24.0] months | Sudden (hard) coronary event (VT during exercise, stent/CABG): 2; revascularisation (stent/CABG): 2 |
| Morrison et al. [32] | FRS | Low: 535 (67%); intermediate: 197 (25%); high: 67 (8%) | 5 years | Myocardial infarction: 5; stroke: 3; other cardiac mortality (non-exertional and exertional SCD): 2 |

Data reported as either *n* (%), mean ± SD or median [interquartile range]

*MESA* Multi-Ethnic Study of Atherosclerosis, *SCORE* Systematic Coronary Risk Evaluation, *SCORE2* Systematic Coronary Risk Evaluation 2, *FRS* Framingham Risk Score, *VT* ventricular tachycardia, *CABG* coronary artery bypass graft, *SCD* sudden cardiac death

### 4.2 Demographics and Characteristics of MAs

Only one study [37] exclusively investigated CAD in female MAs, who were otherwise markedly underrepresented in our review [62]. This contributes to our incomplete understanding of sex-specific CV adaptations to prolonged exercise training. Another letter [63] assessed CAD in a cohort of 196 apparently healthy female MAs (aged ≥ 40 years, with ≥ 10 years of systematic endurance training and participation in ten or more endurance events) and mostly detected CAD in MAs older than 65 years ($n = 18$), with 78% and 22% demonstrating evidence of coronary plaque and CACS > 100, respectively. Although excluded from our review, these data recognise age as a dominant risk factor in the development of atherosclerotic disease; however, the relatively lower prevalence of mixed plaques is also suggestive of a more benign plaque phenotype in female MAs.

Different findings have been observed in male cohorts. Potential protective mechanisms in female MAs can be explained by sex-based differences in anatomy and physiology and through hormones such as testosterone and oestrogen [64]. Testosterone influences skeletal muscle, bone and haemoglobin concentration, which enables male MAs to sustain higher absolute exercise intensities and training volumes [65]. This could also contribute to differences in $VO_2$max between male and female athletes [66]. In contrast, oestrogen exerts several cardioprotective effects, including enhanced endothelial function via nitric oxide-mediated vasodilation and favourable modulation of lipid profiles [67]. These mechanisms may help to clarify the lower prevalence of adverse coronary phenotypes in female MAs [63]. However, the modifying effect of menopausal status warrants additional consideration in future research, as the decline in circulating oestrogen following menopause is associated with an increase in CV disease risk [67].

Meaningful comparison between athlete cohorts was limited most by study heterogeneity in quantification of exercise exposure. Whilst some studies reported exercise volumes using metabolic equivalent (MET)-based estimates, others used distance covered or hours of training per week. These inconsistencies are particularly harmful, as accurate characterisation of exercise exposure is central to understanding dose–response relationships between exercise and CAD. Interpretation of METs presents additional challenges, as they reflect the energy cost of physical activity (PA) relative to resting metabolic rate rather than structured exercise training. Thus, reliance on PA-based thresholds may misrepresent true training load in MAs [68]. Tools such as the Compendium of Physical Activities provide standardised estimates of energy exposure; however, their retrospective application to athletic training histories is imprecise [69]. During our study screening, several articles were excluded because training histories were insufficiently detailed, and exercise exposure was reported only as PA volume. This highlights the absence of a consensus definition for MAs within literature and suggests that standardised reporting of training characteristics is necessary to facilitate robust comparison between studies.

Most data were derived from economically developed countries, which are often characterised by increasingly

**Table 4** Coronary artery disease evaluation and findings in masters athletes

| Study | CAC (AU) | CAC > 0 | CAC > 100 | CAC > 300 | CAC > 400 | Location of CAC | Presence of plaque | Luminal stenosis > 50% | Plaque type | Plaque location | Carotid IMT | AIx |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Aengevaeren et al. [44] | 9.4 [0.0–60.9] | 51 (68%) | – | – | – | LAD: 43 (84%); RC: 20 (39%); RCA: 21 (41%); proximal segments: 39 (77%) | 58 (77.3%) | – | Calcified: 41 (71%); non-calcified: 16 (28%); mixed: 28 (48%) | LAD: 49 (85%); RC: 24 (41%); RCA: 27 (47%); proximal segments: 45 (78%) | – | – |
| Bachman et al. [34] | – | 8 (32%) | – | – | – | – | – | – | – | – | – | 17.0 ± 2.0 |
| Certo Pereira et al. [30] | – | – | 2 (3.6%) | – | – | – | 22 (39.3) | 2 (3.6%) | Calcified (only): 17 (30.4%) | LMS: 3 (5.4%); 3- or 2-vessels including proximal LAD: 10 (17.9%) | – | – |
| De Bosscher et al. [45] | MAs: 8.5 [0.0–90.9]; LOAs: 1.3 [0.0–54.0] | 209 (54.7%) | 75 (19.6%) | – | 19 (5%) | – | 230 (60.2%) | 32 (8.3%) | Calcified: 183 (47.9%); non-calcified: 78 (20.4%); mixed: 107 (28%) | Proximal: 193 (50.5%) | – | – |
| Gervasi et al. [40] | – | – | – | – | – | – | 76 (50%) | 8 (5.3%) | – | – | – | – |
| Gori et al. [41] | – | – | – | – | – | – | – | – | – | – | M: 629.0 ± 92.5; F: 601.0 ± 87.9 | – |
| Jafar et al. [35] | – | 28 (50%) | 13 (23.2%) | – | – | – | – | – | – | – | – | – |
| Kim et al. [46] | NBPG: 2.8 ± 6.0; EIHG: 42.6 ± 67.8 | 21 (42%) | 5 (10%) | – | – | – | 12 (24%) | 1 (8.3%) | – | – | – | — |
| Kröger et al. [42] | 41.0 [0.0–228.5] | 74 (74%) | – | – | – | – | – | – | – | – | – | – |
| Merghani et al. [47] | | 61 (40.1%) | 23 (15.1%) | 14 (9.2%) | – | – | 55 (36.2%) | 8 (5.3%) | – | – | – | – |

**Table 4** (continued)

| Study | CAC (AU) | CAC > 0 | CAC > 100 | CAC > 300 | CAC > 400 | Location of CAC | Presence of plaque | Luminal stenosis > 50% | Plaque type | Plaque location | Carotid IMT | AIx |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Mohlenkamp et al. [43] | 36.0 [0.0–217.0] | 77 (71.3%) | 39 (36.1%) | – | 14 (13%) | – | – | – | – | – | – | – |
| Roberts et al. [36] | 273.8 ± 77.4 (0.0–3153.0) | 34 (68%) | 22 (44%) | – | 10 (20%) | – | – | – | – | – | – | – |
| Roberts et al. [37] | – | – | – | – | – | – | 5 (19.2%) | – | Calcified: 5 (19.2%) | LAD: 5 (71.4%); RCA 2 (28.6%) | – | – |
| Wilund et al. [39] | 70.0 ± 26.0 | – | – | – | – | – | – | – | – | – | – | – |

Data reported as either *n* (%), mean ± SD or median [interquartile range]

*AU* Agatston units, *LAD* left anterior descending artery, *RC* ramus circumflexus (circumflex artery), *RCA* right coronary artery, *LMS* left main stem artery, *LOAs* late-onset athletes, *NBPG* normal blood pressure group, *EIHG* exercise-induced hypertension group

obesogenic environments, including sedentary occupations and availability of processed foods [70, 71]. These factors may elevate the baseline prevalence of traditional CV risk factors amongst active populations [72]. For example, the USA derives a higher proportion of dietary energy from processed foods compared with Italy and South Korea [73]. Thus, the risk factor profiles of MAs included may not fully represent those from less economically developed areas. Countries such as Kenya and Ethiopia produce a substantial number of long-distance runners, with many continuing to train and compete into middle age and beyond, qualifying them as MAs [74, 75]. Thus, more geographically and socioeconomically diverse research is required to accurately characterise the risk profile of CAD in MAs.

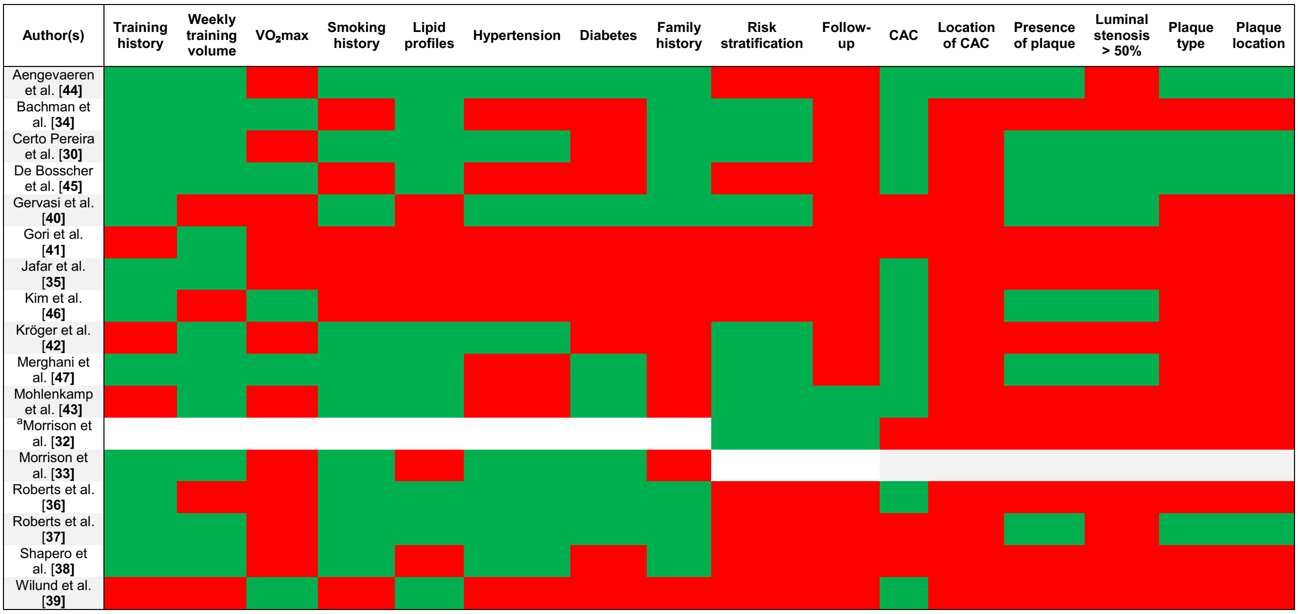
## 4.3 CV Risk Factors

Our findings reinforce that conventional CV risk factors persist in MAs in spite of their high exercise volumes [76]. Although MAs generally demonstrate more favourable health profiles than their sedentary counterparts, they are not immune to the development of CAD. Several studies reported a positive smoking history amongst MAs, challenging assumptions regarding optimal health behaviours in this population. Whilst these data may include former smokers, smoking remains strongly associated with the initiation and progression of CAD through mechanisms such as endothelial dysfunction, increased oxidative stress and reductions in HDL cholesterol [77]. Given the long-term impact of smoking, risk assessment in MAs should not only consider traditional CV risk factors but also exercise characteristics and broader behavioural patterns [78, 79].

Consistently elevated HDL cholesterol and reduced LDL cholesterol are regarded as hallmarks of lifelong endurance training [80]. However, mean TC values often exceeded recommended thresholds, with two studies [42, 43] reporting average TC concentrations ≥ 200 mg/dL. This suggests that certain high-volume training modalities may influence circulating lipids in ways that are not currently understood. Lipid metabolism is strongly influenced by genetic predisposition—often independently of lifestyle behaviours—which may explain the variability in blood profiles observed amongst MAs [81]. Exercise modality itself may also play a role, with combined aerobic and resistance training shown to be more effective than endurance training alone in optimising lipid parameters [82].

Our data suggest that the prevalence of hypertension in MAs is lower than that of the general population [83]. Nevertheless, hypertension remains strongly implicated in the development of CAD [84]. Long-term endurance training is associated with reductions in resting blood pressure (BP); however, hypertension is still the most commonly diagnosed CV disorder in MAs [85, 86]. This illustrates that age-related

**Table 5** Reported data compared with non-reported data across included studies



| Author(s) | Training history | Weekly training volume | $VO_2$max | Smoking history | Lipid profiles | Hypertension | Diabetes | Family history | Risk stratification | Follow-up | CAC | Location of CAC | Presence of plaque | Luminal stenosis > 50% | Plaque type | Plaque location |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Aengevaeren et al. **[44]** | 🟩 | 🟩 | 🟥 | 🟩 | 🟩 | 🟩 | 🟩 | 🟩 | 🟥 | 🟥 | 🟩 | 🟩 | 🟩 | 🟥 | 🟩 | 🟩 |
| Bachman et al. **[34]** | 🟩 | 🟩 | 🟩 | 🟥 | 🟩 | 🟥 | 🟥 | 🟩 | 🟩 | 🟥 | 🟩 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 |
| Certo Pereira et al. **[30]** | 🟩 | 🟩 | 🟥 | 🟩 | 🟩 | 🟩 | 🟥 | 🟩 | 🟩 | 🟥 | 🟩 | 🟥 | 🟩 | 🟩 | 🟩 | 🟩 |
| De Bosscher et al. **[45]** | 🟩 | 🟩 | 🟩 | 🟥 | 🟩 | 🟥 | 🟥 | 🟩 | 🟥 | 🟥 | 🟩 | 🟥 | 🟩 | 🟩 | 🟩 | 🟩 |
| Gervasi et al. **[40]** | 🟩 | 🟥 | 🟥 | 🟩 | 🟥 | 🟩 | 🟩 | 🟩 | 🟩 | 🟥 | 🟥 | 🟥 | 🟩 | 🟩 | 🟥 | 🟥 |
| Gori et al. **[41]** | 🟥 | 🟩 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 |
| Jafar et al. **[35]** | 🟩 | 🟩 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟩 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 |
| Kim et al. **[46]** | 🟩 | 🟥 | 🟩 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟩 | 🟥 | 🟩 | 🟩 | 🟥 | 🟥 |
| Kröger et al. **[42]** | 🟥 | 🟩 | 🟥 | 🟩 | 🟩 | 🟩 | 🟥 | 🟥 | 🟩 | 🟥 | 🟩 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 |
| Merghani et al. **[47]** | 🟩 | 🟩 | 🟩 | 🟩 | 🟩 | 🟥 | 🟩 | 🟥 | 🟩 | 🟥 | 🟩 | 🟥 | 🟩 | 🟩 | 🟥 | 🟥 |
| Mohlenkamp et al. **[43]** | 🟥 | 🟩 | 🟥 | 🟩 | 🟩 | 🟥 | 🟩 | 🟥 | 🟩 | 🟩 | 🟩 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 |
| [a]Morrison et al. **[32]** | | | | | | | | | 🟩 | 🟩 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 |
| Morrison et al. **[33]** | 🟩 | 🟩 | 🟥 | 🟩 | 🟥 | 🟩 | 🟩 | 🟥 | | | | | | | | |
| Roberts et al. **[36]** | 🟩 | 🟥 | 🟥 | 🟩 | 🟩 | 🟩 | 🟩 | 🟩 | 🟥 | 🟥 | 🟩 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 |
| Roberts et al. **[37]** | 🟩 | 🟩 | 🟥 | 🟩 | 🟩 | 🟩 | 🟩 | 🟩 | 🟥 | 🟥 | 🟥 | 🟥 | 🟩 | 🟥 | 🟩 | 🟩 |
| Shapero et al. **[38]** | 🟩 | 🟩 | 🟥 | 🟩 | 🟥 | 🟩 | 🟥 | 🟩 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 |
| Wilund et al. **[39]** | 🟥 | 🟥 | 🟩 | 🟥 | 🟩 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 | 🟩 | 🟥 | 🟥 | 🟥 | 🟥 | 🟥 |

Does not include carotid IMT or AIx.

[a]Morrison et al. [32] utilised the same population of MAs as Morrison et al. [33]

🟩 Reported

🟥 Non-reported

increases in BP can still occur in spite of exercise training [87]. MAs also appear to exhibit higher systolic and diastolic BP responses during exercise compared with sedentary individuals, reflecting adaptations such as increased myocardial mass and augmented cardiac output [88].

Exercise modality appears to be another important modifying factor. Resistance-trained MAs have demonstrated higher resting and exertional BP, perhaps owing to vascular remodelling or the cumulative effects of chronic hypertension over time [89]. In contrast, endurance-trained MAs tend to exhibit lower overall BP readings and a reduced prevalence of hypertension, which may suggest healthier ageing processes [90]. Additional athlete-specific factors including medical conditions, medication use and ergogenic supplement intake can also influence BP profiles [89]. Repeated exposure to elevated BP during training imposes significant mechanical stress on the endothelium, which can promote atherosclerotic progression or plaque instability [91].

Dietary habits should also be considered as part of CAD risk. Aerobic training paired with a heart-healthy diet has been shown to improve lipid profiles and reduce TC levels [92]. However, MAs represent a distinct group whose dietary patterns may differ from sedentary controls. MAs are thought to consume greater total energy, as well as higher intakes of macro- and micronutrients—including fruits, vegetables and dietary fibre—which are associated with improved glycaemic control [93]. This could explain the relatively low prevalence of diabetes observed in our data. In addition, the growing popularity of low-carbohydrate and high-fat dietary approaches amongst MAs further complicates the interpretation and stratification of CAD risk factors [94]. Although these diets may enhance performance through increased fat oxidation, their long-term effects on lipid profiles and overall CAD risk remain unclear.

## 4.4 Risk Stratification and Clinical Implications

Having not been developed with athlete-specific physiology in mind, conventional CV risk calculators may underestimate the true burden of CAD in MAs [95]. Several studies in this review found that athletes categorised as low- or moderate-risk on the basis of traditional scoring tools had elevated CACS or detectable plaques on imaging. We know that MAs who present with favourable metabolic profiles may still harbour subclinical CAD; therefore, this apparent discordance likely reflects the inability of standard risk calculators to account for physiological adaptations associated with long-term and high-volume training.

The majority of studies in our review used non-contrast CT imaging to assess CAC. Albeit practical and widely available, this modality does not detect non-calcified plaque and provides limited information regarding plaque morphology [96]. Nevertheless, CACS remains a valuable metric, and scores > 100 have been independently associated with increased CAD. Although higher exercise levels are generally associated with lower CV event rates for a given CACS, a rise from < 100 to > 100 seems to confer a disproportionately greater risk in MAs [27]. This suggests that closer surveillance of MAs with CACs in this intermediate-to-high range may be warranted, particularly where targeted risk factor modification could yield meaningful clinical benefit.

Individualised assessments incorporating imaging findings, biomarkers, performance metrics and detailed training histories could offer insight into CAD prevalence and potential mechanisms. However, routine screening of all MAs is somewhat unrealistic, and a one-size-fits-all approach is unlikely to be appropriate. Rather, such evaluation may be most relevant for athletes with prolonged exposure to high-intensity endurance training and additional CV risk factors. Only two studies [32, 43] included longitudinal follow-up, indicating that further prospective and outcome-based research is required to guide the development of athlete screening protocols and whether assessment for subclinical CAD should occur earlier in MAs [97]. For now, clinical practice should remain cognisant of the fact that, although high levels of training confer substantial cardiometabolic benefits, they do not negate the influence of other risk factors in CAD development.

### 4.5 Who is at Increased Risk—and Why?

Our data indicate that CAD risk is not uniform across all MAs, but our understanding of the mechanisms underpinning its development remain incomplete [11].

Aengevaeren et al. reported that higher exercise intensity, rather than total exercise volume, was more strongly associated with progression of CAC and plaque formation [44]. This may be explained by haemodynamic stresses, as higher-intensity exercise can disrupt laminar blood flow and promote endothelial dysfunction and plaque development to a greater extent [98]. Strenuous exercise can also trigger acute inflammatory responses at higher intensities, and it has been hypothesised that significant volumes of high-intensity training may create a proinflammatory environment that accelerates CAD progression [97]. Conversely, other studies have reported associations between the higher exercise intensities and lower CAC levels compared with exercise volumes, emphasising the complexity and non-linearity of these relationships [99].

Ultimately, athlete risk is unlikely to be attributable to a single exposure or mechanism. Beyond the factors discussed, other contributors include parathyroid hormone levels, with elevated circulating levels associated with increased arterial calcification [100]. Our comparison of reported versus non-reported data provides further insight into current evidence gaps, and the currently limited scope constrains our understanding of how and why CAD develops in MAs. Without more integrated and longitudinal approaches to research moving forward, we risk drawing oversimplified or potentially misleading conclusions.

### 4.6 Limitations

Certain limitations should be considered when interpreting the findings of this review. The included studies displayed heterogeneity in their definitions of MAs and study designs. The latter ranged from cross-sectional to observational and retrospective approaches, which altogether limited comparison. Furthermore, the lack of validated criteria to objectively differentiate MAs from highly physically active individuals meant that eligibility decisions were based on reported training intent and competitive participation, which may have influenced study inclusion. Finally, many studies enrolled relatively small athlete cohorts, and methodological inconsistencies in data collection and reporting hindered our ability to quantitatively synthesise results.

## 5 Conclusions

Despite the significant volumes of exercise undertaken by this unique population, MAs are not immune to the development of CAD. Subclinical CAD is also prevalent in a noteworthy proportion of MAs, and although atherosclerotic plaques are considered to be more stable, they remain clinically significant. Lifelong training generally promotes healthy cardiometabolic profiles; however, the interplay between traditional CV risk factors, exercise type and individual behavioural characteristics reinforces that exceeding a certain exercise threshold does not eliminate CAD risk. Moreover, evidence suggests that exercise intensity, rather than total volume alone, may play a more influential role in driving vascular changes in MAs. Future research should prioritise larger diverse cohorts alongside standardised definitions and methodologies, and there is a need to investigate underrepresented populations, such as female MAs and athletes from low- and middle-income countries. Eventually, a more individualised approach to risk assessment may better identify high-risk MAs and guide early interventions aimed at optimising long-term CV health.

**Supplementary Information** The online version contains supplementary material available at https://doi.org/10.1007/s40279-026-02429-0.

**Funding** This paper received no specific grant from any funding agency in the public, commercial or not-for-profit sectors. The authors affirm that this manuscript is an honest, accurate and transparent account of the study being reported, that no important aspects of the study have been omitted and that any discrepancies from the study as planned (and, if relevant, registered) have been explained.

## Declarations

**Conflict of interest** None declared.

**Ethical approval** Not applicable.

**Data availability statement** Not applicable.

**Author contributions** All authors contributed to the study design. SG and KS drafted the manuscript and all authors contributed to the writing of the manuscript. All authors revised the draft manuscript and approved the final version.

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