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REVIEW
Sports cardiology

Does too much exercise damage the heart? A narrative review of the long-term cardiovascular effects of intense training

Alessandro Zorzi [1,2]*, Mark Abela [3,4,5], Vincent L. Aengevaeren [6], Dimitra Antonakaki[5,7], Silvia Castelletti [8], Luna Cavigli [9], Guido Claessen [10,11,12], Clea Colombo[13], Thijs M.H. Eijsvogels [14], Gherardo Finocchiaro [15,16], Sabiha Gati[17,18], Francesca Graziano [1], Giuseppe Limongelli[19], Viviana Maestrini [20,21], Aneil Malhotra [22], Harald T. Jorstad[23,24], David Niederseer [25], Marina Ostojic[26], and Flavio D'Ascenzi [9]

[1]Department of Cardiac, Thoracic and Vascular Sciences and Public Health, University of Padua, Via Giustiniani 2, Padova 35128, Italy; [2]Ospedale Riabilitativo di Alta Specializzazione (ORAS), Via P. Lo Bello 3/C, Motta di Livenza, TV 31045, Italy; [3]Department of Cardiology, Mater Dei Hospital, Msida, MSD2090, Malta; [4]Faculty of Medicine, University of Malta, Msida, MSD2080, Malta; [5]City St George's University, London, UK; [6]Department of Cardiology, Radboud University Medical Center, Nijmegen, The Netherlands; [7]Department of Cardiology, Onasseio Hospital, Athens, Greece; [8]Cardiothoracic Department, University Hospital Santa Maria della Misericordia, Piazzale Santa Maria della Misericordia 15, Udine 33100, Italy; [9]Department of Medical Biotechnologies, Sports Cardiology and Rehab Unit, University of Siena, Siena, Italy; [10]Faculty of Medicine and Life Sciences, Biomedical Research Institute, LCRC, UHasselt, Hasselt, Belgium; [11]Hartcentrum Hasselt, Jessa Ziekenhuis, Hasselt, Belgium; [12]Department of Cardiovascular Diseases, KU Leuven, Leuven, Belgium; [13]Sao Leopoldo Mandic Medical School, Campinas, Sao Paulo, Brazil; [14]Department of Medical BioSciences, Radboud University Medical Center, Nijmegen, The Netherlands; [15]Cardiovascular Clinical Academic Group and Cardiology Research Section, St George's, University of London, London, UK; [16]St George's Hospital NHS Foundation Trust, London, UK; [17]National Heart and Lung Institute, Imperial College of London, London, UK; [18]Royal Brompton Hospital and Guys and St. Thomas' Hospital NHS Trust, London, UK; [19]Monaldi Hospital, Ao Colli, University of Campania 'Luigi Vanvitelli', Napoli, Italy; [20]Department of Medical and Cardiovascular Science, Sapienza University of Rome, Rome, Italy; [21]Institute of Sports Medicine and Science, National Italian Olympic Committee, Rome, Italy; [22]Institute of Sport, Manchester Metropolitan University, Manchester, UK; [23]Department of Cardiology, Amsterdam UMC, University of Amsterdam, Amsterdam, The Netherlands; [24]Movement Sciences, Amsterdam Cardiovascular Sciences, Meibergdreef 9, Amsterdam, The Netherlands; [25]Hochgebirgsklinik, Medicine Campus Davos, Davos, Switzerland; and [26]Cardiology Clinic, University Clinical Center of Serbia, Belgrade, Serbia

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Abstract

Long-term exposure to high-intensity endurance exercise induces significant physiological cardiovascular remodelling. However, recent evidence suggests that prolonged exercise in athletes, particularly those exposed to decades of intensive training, may be associated with features of cardiovascular 'wear and tear'. These include electrical changes such as an increased risk of sinus node dysfunction and atrial fibrillation as well as structural abnormalities such as focal non-ischaemic fibrosis and coronary artery calcification, more frequently observed in middle-aged and older endurance athletes. These observations suggest that long-term high-intensity exercise may have deleterious cardiovascular effects in susceptible individuals, although the available data are primarily derived from male Caucasian athletes. While the clinical significance of these potentially maladaptive changes at the individual level remains uncertain, at a population level, high intensities and volumes of physical activity are consistently associated with reduced cardiovascular and all-cause mortality compared with sedentary behaviour, although the incremental survival benefit may plateau at very high exercise volumes. This review critically appraises the available evidence on chronic adverse cardiovascular adaptations associated with intense endurance exercise, highlighting unresolved controversies, sex- and ethnicity-related knowledge gaps, and the need to balance potential risks against the well-established survival benefits of lifelong physical activity.

* Corresponding author. Tel: +390498218666, Email: alessandro.zorzi@unipd.it

Graphical Abstract

Keywords Arrhythmias • Athletes • Atrial fibrillation • Coronary atherosclerosis • Exercise-induced cardiac remodelling • Myocardial fibrosis • Sports cardiology

Introduction

Regular physical exercise is widely recognized as one of the most effective strategies to promote health and prevent diseases.[1] Its benefits extend well beyond the cardiovascular system, positively influencing metabolic profile, mental health, musculoskeletal function, and overall longevity.[2–6] Accordingly, international guidelines consistently recommend regular physical activity as a cornerstone of primary and secondary prevention.[7–10]

However, some people engage in high-intensity and high-volume recreational exercise, significantly exceeding guideline recommendations. In recent decades, participation in competitive and non-competitive endurance sports has also markedly increased, leading to a growing population of athletes who train for several hours per week.[11] Of particular interest is the group of so-called 'master athletes' (≥35 years old according to current guidelines[12]): middle-aged or older individuals who often practice individual endurance disciplines such as running or cycling, with training intensities and volumes that may approach those of professional athletes.[13] As already suggested by Paracelsus more than 500 years ago, 'the dose makes the poison'. Therefore, as these extreme levels of sport exposure emerge in society, a question has emerged: can excessive exercise regimes lead to deleterious effects on the cardiovascular system?[8,14–16]

The aim of this review is to examine the available evidence on the possible association between intense and prolonged exercise training and cardiovascular abnormalities, focusing on both electrical and structural findings, to help distinguish between commonly held beliefs and evidence-based conclusions and to discuss the clinical implications.

Writing methodology

This narrative review was independently developed by members of the Sports Cardiology Nucleus of the European Association of Preventive Cardiology with complementary expertise in electrophysiology, cardiac imaging, inherited heart diseases, and exercise physiology. An initial outline was shared among all authors, after which the two coordinators assigned individual sections to pairs of authors according to their expertise. For each section, the responsible authors selected the most relevant literature, prioritizing large cohort studies published within the last 10 years and systematic reviews. The complete draft and reference list were subsequently reviewed by all co-authors in two revision rounds, allowing additional relevant studies to be suggested and incorporated. This approach is consistent with the methodology of a narrative review.

Review of evidence: electrical changes

Sinus bradycardia/atrioventricular block

Sinus bradycardia (SB) is a well-established physiological finding among athletes, occurring in up to 85% of all athletes and exceeding 90% of endurance athletes. It is often accompanied by sinus pauses, ectopic atrial rhythm, junctional rhythm, and atrioventricular (AV) conduction anomalies. Profound SB (<40 b.p.m.) occurs in ~4% of athletes, with extreme bradycardia (<30 b.p.m.) reported in a few asymptomatic endurance athletes.[17–21] First-degree AV block and Mobitz I second-degree AV block are also often registered during rest or sleep and tend to normalize with exercise. In contrast, marked PR prolongation (>400 ms) or high-grade AV block is considered pathological and may be associated with metabolic, autoimmune, infectious, or genetic cardiac diseases.[17–21]

Traditionally, SB and AV block in athletes were attributed to increased vagal tone and thought to be reversible after detraining.[22] However, recent experimental data obtained from animal models suggest that exercise may cause intrinsic modification of the sinus node and AV node function, through downregulation

of ion channels (such as the HCN4) involved in impulse generation and conduction.[23] This hypothesis is supported by the observation that dual pharmacological blockade (beta-blockers plus atropine) does not completely reverse SB in athletes.[24–26] At variance with extrinsic (vagal) modulation, these intrinsic (epigenetic) modifications may not be completely reversible after cessation of the athletic career and predispose former athletes to clinically relevant bradycardia later in life.[27]

From a clinical point of view, some studies suggest that most former elite athletes continue to experience bradycardia, even after detraining, and that individuals with a history of endurance sport are also more likely to receive a pacemaker due to idiopathic bradyarrhythmias (i.e. in the absence of structural heart disease) at a younger age than non-athletes.[19,28–30] A large Swedish study observed that male cross-country skiers, especially those with the highest training levels, had higher rates of bradycardia and pacemaker implantation than non-skiers. Interestingly, no associations were found among female skiers, and mortality rates did not differ between male skiers with and without a pacemaker.[31]

These findings suggest that high-intensity and high-volume endurance exercise may accelerate sinus or AV node dysfunction over time, through intrinsic changes in nodal cells (ion-channel downregulation) possibly in combination with genetic predisposition and/or stress-induced fibrosis of the SA or AV node.[23] Although these arrhythmias appear to suggest a more complex picture, beyond physiologically driven adaptation, they affect only a small subset of athletes and do not seem to have any negative impact on long-term survival.

Atrial fibrillation

Moderate physical activity has been shown to reduce the risk of developing atrial fibrillation (AF) in the general population.[32] Endurance sports, however, appear to be a risk factor for AF, with a 10-fold increased risk quoted in some studies when compared with sedentary individuals.[33] This association is well-established in male master endurance athletes (>40 years)[12,34–40] with a dose-response U-shaped pattern,[10,27,41] independent of traditional cardiovascular risk factors. Data on female athletes are scarce and contradictory, suggesting possible sex-specific remodelling patterns.[42–45] These observations should, however, be interpreted with caution, as females are generally underrepresented in athletic cohorts. Interestingly, longitudinal studies showed that, despite the increased prevalence of AF, former athletes showed a lower incidence of stroke than non-athletes.[38,43]

Several mechanisms have been implicated in the development of AF in athletes. Originally, AF in athletes was linked to left atrial (LA) dilation, which is a typical feature of the athlete's heart. Left atrial dilatation is a risk factor for AF in the general population as it is associated with increased wedge pressure and atrial fibrosis.[46] However, LA dilation in athletes without AF is a physiological response to increased volume load and is usually not associated with impaired indexes of atrial function suggesting atrial myopathy.[46,47] In master athletes with AF, LA end-systolic volume is similar to athletes without AF; on the other hand, LA function quantified by emptying fraction, expansion index, reservoir, and contractile strain is worse.[47] These findings indicate that LA dilation does not *per se* increase the risk of AF as long as functional indexes are preserved. Atrial dysfunction suggests an underlying atrial myopathy, potentially caused by the stress of prolonged exercise and characterized by chronic inflammation and fibrosis acting as key substrates for arrhythmogenesis.[48–51] High vagal tone, typical of athletes, may also lead to an increased propensity to AF as it decreases the atrial refractory period.[52] Premature atrial beats originating from the pulmonary veins often trigger AF, but there is no evidence that endurance sport increases the atrial ectopic burden.[53,54] Finally, changes in calcium currents, ion channels, and gap junctions can lead to modifications of electrophysiological characteristics of atrial myocytes independently of LA structural remodelling.[55,56]

Detraining may partially reverse some of the structural and functional adaptations that are thought to trigger AF in athletes, which is why endurance athletes with paroxysmal AF may be advised to reduce their training intensity and volume.[32] A blanket, one-size-fits-all approach is, however, not justified. A shared decision-making model is warranted, incorporating symptom burden, sporting discipline, treatment options (rhythm vs. rate control strategies), and the presence of underlying structural heart disease. Athletes with AF requiring anticoagulation should avoid collision or body contact sports.[57] Definitive long-term human studies on reversibility remain lacking;[58] therefore, guidelines on exercise prescription in AF continue to rely heavily on expert consensus.

Premature ventricular beats

The relationship between physical activity and premature ventricular beats (PVBs) remains a matter of debate. The few studies that have investigated the acute effects of exercise on ventricular arrhythmic burden have reported inconsistent results. Two studies conducted in athletes completing a marathon did not demonstrate any significant effect on ventricular arrhythmias,[59,60] whereas a study performed during an ultramarathon observed a slight increase in the number of individuals presenting at least one PVB on a 1-min electrocardiogram (ECG) recorded before and after the race.[61] However, the more clinically relevant question is whether long-term training leads to a chronic increase in ventricular arrhythmic burden. This issue has important clinical implications since demonstrating a higher PVB burden with exercise could potentially downplay the diagnostic significance of such a finding in athletes.

Two recent studies have shown that, on average, athletes exhibit a relatively low burden of PVBs. The first study enrolled non-elite volunteer athletes of different ages and compared them with sedentary controls. It reported that 24-h Holter monitoring identified a low frequency of PVBs (6% had >100 PVBs/24 h) and non-sustained ventricular tachycardia (NSVT) (2%), similar to sedentary controls. Notably, 80% of athletes with frequent PVBs showed infundibular (left bundle branch block/inferior axis moprhology) or fascicular morphologies[62]: commonly observed PVB morphologies that are rarely associated with underlying structural heart disease.[63] The likelihood of frequent (>100 PVBs/24 h) or repetitive PVBs increased with age, but was not related to gender or training intensity/volume.[62] The second study recruited 296 highly trained young elite athletes and 138 endurance master athletes (mean age 56 years) in the pro@heart and master@heart registries. Most athletes had a low PVB burden over a 24-h period (median 1 PVB/day in young elite athletes and 6 PVB/day in middle-aged athletes). Only a minority of athletes exhibited >100 PVBs/24 h or NSVT, with a significantly higher prevalence among middle-aged athletes (13% and 8%,

respectively) compared with younger athletes (5% and 2%, respectively) and middle-aged non-athletes (6% and 2%, respectively).[64] Another study from the same group suggested that male athletes exhibited a higher prevalence of NSVT compared with female athletes (11% vs. 2%), although the median number of PVBs was similar between sexes (3.2/day vs. 1.4/day).[65] In summary, these studies suggest that frequent and repetitive PVBs are observed only in a minority of athletes and to what extent exercise directly contributes to the development of ventricular arrhythmias remains unclear. A recent study suggested that PVBs in athletes with structurally normal hearts may also be attributed to underlying metabolic (e.g. iron deficiency) or inflammatory states.[66]

Another knowledge gap is the impact of detraining on ventricular arrhythmic burden. More than 20 years ago, Biffi *et al.* observed that detraining significantly reduced the number of PVBs in athletes with frequent PVBs and structurally normal hearts.[67] However, the interpretation of these findings is limited by the selection of athletes with frequent ectopy at baseline and the possibility that spontaneous variability or regression to the mean may have contributed to the observed reduction. Of note, the same group reported that resuming physical activity was not associated with recurrence of PVBs.[68] Another study conducted in non-elite athletes demonstrated a spontaneous variability of PVB burden over time, regardless of whether athletes continued training or not.[69] More recently, Di Gioia *et al.* evaluated a cohort of 109 athletes with frequent PVBs and normal cardiac magnetic resonance imaging (MRI) over a mean follow-up of 3 years. In 40% of these athletes, ventricular arrhythmias resolved despite ongoing training. Notably, in 2 of 67 athletes with persistent PVBs, repeat MRI revealed the development of structural heart disease.[70] In conclusion, although detraining may be associated with a reduction in ventricular arrhythmic burden in some athletes, a clear cause–effect relationship has not yet been established.[71]

Exercise-induced long-QT syndrome

The QT interval on the surface electrocardiogram reflects the duration of ventricular depolarization and repolarization and results from the complex interplay between inward depolarizing currents, mainly mediated by sodium and calcium channels, and outward repolarizing potassium currents. QT prolongation may occur as a consequence of genetic conditions, such as congenital long QT syndrome, or as an acquired phenomenon, commonly driven by electrolyte disturbances, drugs, or systemic conditions.[72] In athletes, as in the general population, heart rate-corrected QT (QTc) is on average longer in females.[73,74] When average QTc was compared between athletes and non-athletes, studies showed mixed results with some reporting slightly higher values in athletes and others showing no differences.[75–78] In addition, the use of the Bazett method of correction may become less accurate in athletes with bradycardia, hampering the comparison with sedentary individuals usually showing higher heart rates.[79] However, the extent to which QTc prolongation reported in athletic cohorts reflects the correction formula rather than true repolarization changes remains uncertain.

There is some evidence that a small minority of athletes may develop marked QTc prolongation because of intense training: the so-called 'exercise-induced long-QT syndrome'. In a cohort of 2000 asymptomatic elite mixed and endurance athletes who underwent a 12-lead ECG and an echocardiogram, the prevalence of prolonged QT was only 0.4%. However, none of the athletes with a QTc between 460 and 500 ms exhibited a disease-causing genetic variant or had a first-degree relative with long QT syndrome, suggesting a possible effect of exercise on this mild QT interval prolongation.[80] One of the key studies supporting the hypothesis of a possible 'exercise-induced long QT' involved 310 competitive athletes referred for suspected long QT, in whom QT intervals normalized after a period of detraining in a subset of asymptomatic individuals with negative genetic testing and no family history.[81] Other investigations confirmed that long QT in athletes with negative genetic testing/family history usually resolves with detraining.[82]

Several mechanisms have been proposed to explain this presumed exercise-induced QT prolongation phenomenon. These include delayed ventricular repolarization related to increased left ventricular mass and chronic mechanical stretch. Specifically, chronic exposure to increased haemodynamic load during intensive training has been suggested to influence ventricular repolarization via mechano-sensitive pathways, including the upregulation of stretch-activated sarcolemmal Ca^{2+} channels.[80,81,83,84]

In conclusion, a significant QT prolongation that normalizes after detraining may occur in a small minority of athletes. It is not known if the arrhythmic risk of the 'exercise-induced long QT' is lower than that of congenital long QT caused by gene mutations, and therefore, the management of these athletes remains equivocal.

Table 1 and *Figure 1* summarize the available evidence on the relationship between prolonged intense training and electrical abnormalities, as well as response to detraining.

Review of evidence: structural abnormalities and troponin release

Exercise-induced arrhythmogenic cardiomyopathy phenotype

An expanding body of clinical, imaging, molecular, and electrophysiological evidence supports the concept that chronic, high-intensity endurance exercise can promote disease penetrance in desmosomal-gene mutation carriers and worsen the phenotype in patients with arrhythmogenic right ventricular (RV) cardiomyopathy.[85–88] However, a minority of athletes may exhibit RV abnormalities extending beyond the expected spectrum of physiological exercise adaptation, including disproportionate RV dysfunction and complex ventricular arrhythmias, even in the absence of pathological variants or familial disease.[89] Based on these observations, the term 'exercise-induced arrhythmogenic right ventricular cardiomyopathy (ExI-ACM)' has been proposed to describe a potentially maladaptive and arrhythmogenic phenotype at the extreme end of the athletic RV remodelling spectrum,[90,91] although its precise prevalence and even its definition as a distinct disease entity remain debated.[92]

The hypothetical pathophysiological basis for ExI-ACM rests on the uniquely high wall stress imposed on the thin-walled RV during sustained endurance exercise.[93] Because pulmonary vascular resistance decreases far less than systemic resistance during exertion, the RV experiences disproportionately elevated afterload and mechanical strain. Over time, this may exceed the

Table 1 Summary of evidence of an association between high cumulative sport dose and electrical abnormalities, reversibility with detraining and implications for athletes

	Sinus or AV node dysfunction
Evidence of an association	Emerging evidence of an association between persistent sinus node dysfunction and advanced/complete atrioventricular block and lifelong intense endurance sports activity in former male athletes. No evidence in female athletes
Type of studies	Cross-sectional observational, retrospective observational, longitudinal cohort (long-term follow-up and population-based)
Frequency and comparison with controls	Endurance athletes may exhibit a modestly increased long-term risk of sinus node dysfunction and pacemaker implantation compared with controls (relative risk increase ~15–20% in large cohort studies), particularly in male athletes with high cumulative training exposure
Reversibility with detraining	Although sinus bradycardia and first-degree/second-degree Mobitz type 1 atrioventricular block usually improve or disappear with detraining, there is evidence that sinus node and atrioventricular node function may not completely normalize, possibly reflecting intrinsic sinus node remodelling and only partial reversibility
Implications for athletes	A minority of master or former athletes may require pacemaker implantation at a younger age than non-athletes, with no impact on mortality
	Atrial fibrillation
Evidence of an association	There is strong evidence of an association between atrial fibrillation and male master endurance athletes. Limited evidence in female athletes
Type of studies	Observational studies (large population-based cohorts, matched cohort studies, case-control studies, cross-sectional imaging/mechanistic studies, retrospective/registry-based studies, prospective and longitudinal cohort studies), reviews, and scoping/umbrella reviews
Frequency and comparison with controls	In comparisons with sedentary controls, matched general population controls, or lower-exposure athletes, endurance athletes tend to show a higher prevalence and/or incidence of AF (2–5-fold). However, former athletes did not show an increased risk of stroke
Reversibility with detraining	Detraining may reduce the overall burden of atrial fibrillation, though studies to address the effect of such an intervention are still lacking
Implications for athletes	Management should be individualized through shared decision-making, considering symptoms, AF pattern, sport type, treatment strategy, anticoagulation, and structural heart disease. Reduction of training intensity or volume may be considered in selected endurance athletes, but the optimal degree of reduction remains undefined. Significant physiological remodelling of the sinus node and atrioventricular node often leads to intolerance to drug therapy, which is why athletes are often referred for ablative procedures instead
	Premature ventricular beats
Evidence of an association	Overall weak and inconsistent evidence: most athletes show a low premature ventricular beats (PVBs) burden, with a possible increase only in a minority of master endurance athletes
Type of studies	Cross-sectional Holter studies; limited longitudinal/detraining data
Frequency and comparison with controls	Low burden in most athletes; >100 PVBs/day in a minority (5–15%), with higher prevalence in older than younger athletes and inconsistent differences vs. controls
Reversibility with detraining	Detraining may reduce PVB burden in some athletes, but spontaneous variability and lack of a clear cause–effect relationship limit definitive conclusions
Implications for athletes	Although PVBs are usually a benign finding in athletes, they should be investigated to rule out the presence of an underlying structural heart disease or metabolic disturbance
	Exercise-induced long QT
Evidence of an association	Although average QTc interval in athletes is similar or just slightly prolonged compared with sedentary individuals, a small minority of athletes show significant QT prolongation in the absence of genetic disease
Type of studies	Cross-sectional ECG studies; small detraining cohorts; limited longitudinal data
Frequency and comparison with controls	Rare finding; clearly prolonged QTc reported in <1% of athletes in large cohorts, with uncertain excess vs. controls
Reversibility with detraining	QT prolongation may normalize after detraining in athletes with long-QT but negative genetic testing and no family history, with recurrence after resumption of intensive training only in some of them
Implications for athletes	There is no evidence about the arrhythmic risk attributable to an exercise-induced long QT phenotype in athletes without underlying genetic or structural heart disease. Therefore, management of these athletes remains equivocal

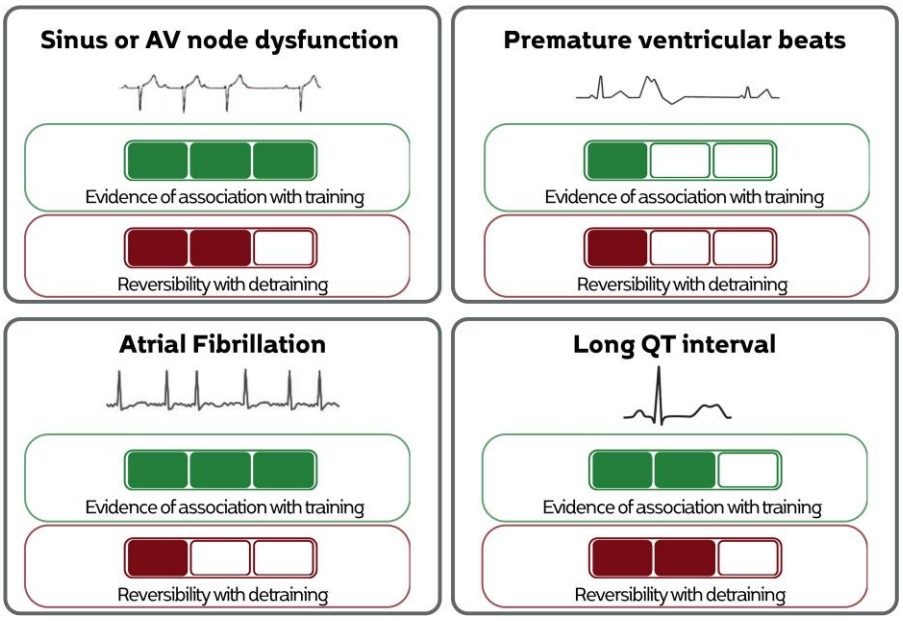

Figure 1 Electrical abnormalities associated with long-term training. Schematic summary of the main electrical adaptations and arrhythmic findings reported in athletes. The strength of evidence for an association with training and the degree of reversibility with detraining are qualitatively graded based on the available literature (0/3: no evidence; 1/3: low or preliminary evidence; 2/3: moderate evidence; 3/3: strong evidence).

myocardium's 'desmosomal reserve,' leading to progressive microstructural disruption of cardiomyocyte junctions.[87,94] In addition to mechanical overload, prolonged high-intensity exercise may activate myocardial inflammatory pathways and promote autoimmune responses directed against desmosomal proteins, thereby weakening intercellular coupling and increasing susceptibility to electrical instability.[91] This mechanistic framework provides a plausible explanation for why arrhythmogenic phenotypes may arise even in athletes without desmosomal gene variants and why the condition appears to cluster in individuals with long-standing, high-intensity training histories.[95,96] However, a strong individual predisposition possibly related to a still unknown genetic background is needed to develop ExI-ACM, as in the vast majority of athletes with a long history of intense sports activity, no signs of RV cardiomyopathy can be found[97,98] (*Figure 2*). Importantly, physiological RV and right atrial enlargement are common findings in endurance athletes and, in isolation, should not be considered evidence of cardiomyopathy.

Clinically, the proposed ExI-ACM phenotype is primarily characterized by complex ventricular arrhythmias, typically occurring in the setting of subtle, disproportionate, or functionally abnormal RV remodelling rather than isolated chamber enlargement alone and in the absence of desmosomal gene mutations.[89] These arrhythmias frequently exhibit a RV outflow tract (RVOT) morphology, which is traditionally regarded as a benign entity in otherwise healthy young individuals.[18,99,100] In athletes, however, distinguishing truly benign RVOT ectopy from an early arrhythmogenic cardiomyopathy requires careful contextual evaluation. Resting structural cardiac evaluations may reveal subtle findings that can overlap with physiological

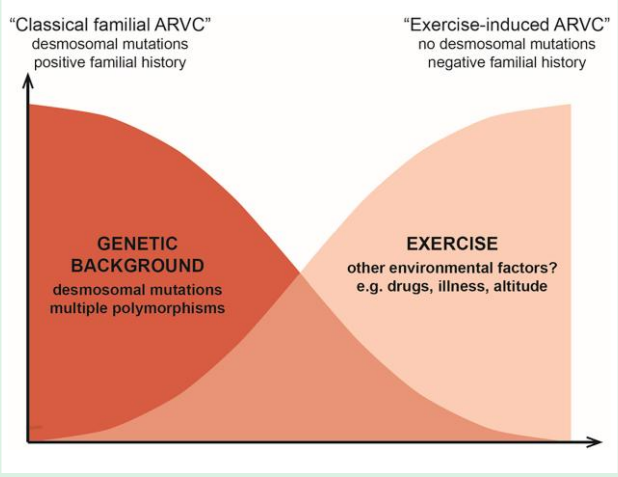

Figure 2 Spectrum of arrhythmogenic cardiomyopathy expression and its relation to cumulative exercise exposure.

athletic remodelling, including mildly asymmetric RV enlargement or borderline reductions in RV strain, and no detectable fibrosis on MRI.[101,102] Regional wall-motion abnormalities, characteristic of established familial ARVC, are uncommon in early exercise-associated remodelling, further blurring diagnostic boundaries.[101] However, exercise echocardiography and exercise MRI can uncover impaired RV contractile reserve, stress-induced dilation, or functional deterioration not evident at rest,[101] underscoring the potential of stress-integrated imaging to identify early pathological remodelling before overt

structural changes become apparent. Finally, an electrophysiological study with endocardial voltage mapping may be indicated in athletes with persistent symptomatic and complex RVOT-type ventricular arrhythmias combined with no or subtle imaging abnormalities, as patchy regions of abnormal myocardial voltage on electro-anatomical mapping, indicating early arrhythmogenic substrate below the resolution of conventional imaging, may be revealed.[103–105]

In athletes with clinically significant ventricular arrhythmias and suspected arrhythmogenic RV remodelling, exercise restriction remains a central management strategy as detraining reduces arrhythmic risk in both genetic and gene-elusive arrhythmogenic cardiomyopathies; however, complete resolution of structural abnormalities is not typically observed.[88,95]

Isolated non-ischaemic left ventricular scar

Cardiac magnetic resonance imaging is the reference modality for myocardial tissue characterization, through late gadolinium enhancement (LGE) imaging and parametric mapping techniques. In the chronic setting, the presence of LGE is traditionally considered a hallmark of replacement fibrosis, although other mechanisms, such as expansion of the myocardial extracellular space, may also be involved.[105]

In athletic populations, an increased prevalence of non-ischaemic (subepicardial/midmyocardial) LV LGE has been reported, particularly among master endurance athletes, whereas ischaemic-pattern LGE is uncommon.[27,64,106–110] Earlier cohorts of long-distance runners, triathletes, and cyclists reported highly variable prevalence estimates, partly because some studies included RV insertion-point LGE within the overall LGE burden, whereas others analysed non-hinge-point patterns separately.[107–109,111] In several cohorts, a substantial proportion of the observed LGE involved the RV insertion points, located at the junctions between the RV free wall and the interventricular septum.[107,111,112] Hinge-point LGE is a frequent finding in athletes, is thought to be related to increased mechanical stress, and has not been associated with ventricular arrhythmias or adverse outcomes.[64,106,111,112] More recent studies of older athletes with long-term endurance training reported non-hinge-point LGE in up to 48% of participants, particularly involving the basal inferolateral wall.[64,106,110] Across studies including control groups, non-ischaemic LGE was generally more prevalent among athletes than among sedentary controls, although findings were not uniform, underscoring the influence of age, training regimen and cumulative exposure, and methodological variability, including CMR acquisition and interpretation, on prevalence estimates.[113–115] Notably, no increased diffuse fibrosis has been reported in master athletes compared with controls using T1 mapping techniques.[116–119] In some studies, the presence of LGE has also been associated with a higher prevalence of ventricular arrhythmias, supporting the concept that fibrotic areas may serve as arrhythmogenic substrates in susceptible individuals.[64,106,111,120,121] Overall, MRI studies indicate that non-ischaemic LV fibrosis is not rare in well-trained athletes, but a direct causal association between sport and fibrosis has not been demonstrated.

Two leading aetiological mechanisms have been proposed to explain these MRI findings. One hypothesis suggests that a proportion of fibrosis represents residual scar from prior clinically overt or subclinical myocarditis.[120–122] This is supported by the observation that many LGE patterns in athletes, particularly subepicardial or mid-wall enhancement, mirror those typically seen following viral myocarditis. Given the exposure of athletes to viral illness and the possibility of continuing training during minor illness, silent or incompletely recognized myocarditis remains a biologically plausible contributor. In this context, persistence of scar tissue despite clinical recovery may explain focal non-ischaemic fibrosis detected in otherwise asymptomatic athletes.[115] The second hypothesis relates to repetitive haemodynamic loading and mechanical myocardial stress associated with chronic high-intensity endurance training.[115,123] Sustained exposure to increased preload and afterload, repetitive ventricular wall stress, and pressure-volume fluctuations may trigger repeated microinjury, focal myocyte necrosis, and subsequent replacement fibrosis. This mechanism is particularly relevant to hinge-point fibrosis, occurring where mechanical stress is greatest.[111,112,115] Furthermore, prolonged endurance exposure has been associated in some studies with transient biomarker elevation, temporary ventricular dysfunction, atrial and ventricular remodelling, and, in certain cases, ventricular arrhythmias and reduced systolic function, as highlighted in more recent endurance athlete cohorts.[64,106,110] It is likely that both mechanisms coexist, influenced by age, genetic predisposition, inflammatory susceptibility, training intensity, and cumulative lifetime exercise exposure.[115] Finally, in a minority of cases, non-ischaemic LGE can be the hallmark of an underlying genetic cardiomyopathy with predominant LV involvement. According to a recent study evaluating 40 athletes with non-ischaemic LGE and ventricular arrhythmias who underwent systematic genetic testing and family screening, seven carried pathological gene mutations, while familial recurrence was identified in two additional cases despite negative genetic testing.[121]

Continued prospective evaluation is required to clarify causality, prognostic implications, and stratification strategies to differentiate benign adaptive findings from early pathological change in the athletic heart.

Coronary artery disease

Coronary atherosclerosis, measured by coronary artery calcification scores (CACS), is strongly linked to adverse cardiovascular outcomes.[124] Unexpectedly, multiple independent international cohorts reported increased CACS and atherosclerotic plaque prevalence in middle-aged and older male athletes in comparison with their less active peers.[125–129] In 2008, Mohlenkamp *et al.* first reported higher CACS in male marathon runners compared with age- and risk factor-matched individuals from the general population (the Heinz Nixdorf Recall study).[127] This was initially presumed to be (partially) explained by a large proportion of (ex-)smokers among the marathon runners. However, subsequent studies confirmed the higher prevalence of CACS and atherosclerotic plaques in male athletes compared with non-athletes[126] and an association between the prevalence of CACS, atherosclerotic plaque, and lifelong exercise volume.[125] Larger general population studies have also shown increased CACS in individuals engaging in greater exercise volumes.[128] The impact of exercise intensity vs. exercise duration is not yet clear, since the MARC-2 study reported an association between a higher proportion of very vigorous intensity exercise and more progression of CACS,[130] whereas a cross-sectional analysis from the Cooper Clinic Longitudinal Study showed more CACS with a longer duration of exercise,

but less CACS with higher exercise intensity.[131] A subsequent longitudinal analysis of the Cooper Clinic found no association between physical activity volume and progression of CAC,[132] in line with findings from the MARC-2. Exercise volume thus appears to be associated with CAC prevalence but not with its progression.

Interestingly, exercise characteristics were not related to coronary atherosclerosis in female athletes.[133] The explanation for this difference between men and women is not clear, but may relate to the protection of oestrogen and the relatively low burden of atherosclerosis in women, or to sex-specific haemodynamic or inflammatory factors.

Early studies involving male athletes reported a higher prevalence of calcified plaques and a lower prevalence of mixed morphology plaques in the most active individuals.[125,126] These observations, coupled with lower mortality in athletes, led to the hypothesis that increased CACS in athletes might indicate accelerated calcification and plaque stabilization, similar to the effect of statins.[134] Unfortunately, the Master@Heart study found no differences in plaque morphology between lifelong male endurance athletes and controls. It is worth noting that the controls were allowed to engage in up to 3 h of exercise per week and were physically fit (122% of estimated VO2max).[129] Additionally, both groups had predominantly calcified plaques and only a few of the athletes exhibited plaques with high vulnerability characteristics (defined as at least 2 high-risk features: low attenuation, spotty calcification, positive remodelling, or napkin-ring sign, which are associated with a higher coronary event risk).[135] This suggests a less detrimental plaque morphology in all groups. The lack of difference may therefore be explained by the high fitness status and associated coronary plaque remodelling of the control group.[129] Future studies should further focus on the association between exercise characteristics and plaque characteristics, including high-risk plaque features and coronary inflammation, as this may importantly impact the risk associated with coronary plaques.[135]

The mechanisms underlying increased coronary atherosclerosis in male athletes are not yet elucidated. Traditional cardiovascular risk factors such as smoking, hypertension, and a genetic component as indicated by a family history of coronary heart disease, but also lipoprotein(a) values, are important predictors of coronary atherosclerosis in athletes,[136,137] but do not explain the degree of atherosclerosis observed. Exercise-related factors such as sustained increases in blood pressure and heart rate, mechanical stress, disrupted laminar flow, and exercise-induced inflammation are potential contributors but a recent study did not find differences in these factors after a single endurance cycling test between athletes with and without atherosclerosis.[138] Exercise-induced changes in lipid metabolism and calcium homeostasis, along with an atherogenic diet and genetic factors, may also contribute.[139] Future studies are needed to investigate the role of these proposed mechanisms. It is recommended that risk factors are adequately managed in athletes, according to general population guidelines.[39]

Outcome data in athletes with coronary atherosclerosis are limited. A recent follow-up study from the American Cooper Center Longitudinal Study showed that individuals with the highest exercise volume [>3000 metabolic equivalent of task (MET)-min/week] had the lowest risk for all-cause mortality, but interestingly found that these individuals with the highest exercise volume had a similar risk for myocardial infarction and revascularization as the individuals with low exercise volume (<500 MET-min/week).[140] The individuals with a moderate exercise volume (500–3000 MET-min/week) had the lowest risk of myocardial infarction and revascularization. Importantly, the association between CACS and increased CV risk was independent of exercise volume, suggesting that the most active individuals are not protected from the health risks of increased CACS. Similarly, data from the same cohort previously showed an increased risk of adverse outcomes among the fittest individuals with high CACS vs. without CACS, although higher cardiovascular fitness was associated with a lower risk of cardiovascular events independently of CAC.[141]

Based on current studies, there is no evidence that reducing exercise intensity would slow the progression of CAC or lower the incidence of CV events. It is, however, important for athletes to consider that exceedingly high exercise volumes will not yield a further reduction in CV risk, as optimal risk reduction occurs below the typical training volume of athletes.[3]

Troponin release

Due to the presence of cardiac-specific isoforms, assessment of circulating cardiac troponins [cTn, either subunit I (cTnI) or T (cTnT)] is currently the gold standard to assess myocardial injury in clinical practice,[142] defined as a cTn value above the 99th percentile upper reference limit (URL). Acute or chronically elevated cTn concentrations are associated with a higher event rate in patients[143,144] and the general population.[145]

Exercise is known to produce elevated cTn concentrations. A recent meta-analysis among 7289 athletes from 129 studies has shown that 36% of athletes demonstrated cTn concentrations above the URL following exercise, with extreme between-study heterogeneity (range 2–98%).[146] Post-exercise cTn elevations >URL were more common with high-sensitivity and cTnT assays, in middle-aged athletes, and after 3–6 h of endurance exercise. Importantly, sex and training status did not impact cTn elevations. The onset of exercise-induced cTn elevations already occurs during the exercise bout,[147] while complete normalization to resting concentrations typically occurs within 24–72 h post-exercise.[148] Hence, the kinetics of exercise-induced cTn release are different from clinical scenarios such as a patient with an acute myocardial infarction.[149]

The magnitude of exercise-induced cTn release is highly variable among athletes.[14] At least 10 factors have been identified as predictors of the magnitude of post-exercise cTn concentrations, with exercise intensity and exercise duration being listed as the greatest contributors.[150,151] Nevertheless, the predictive capacity of these combined predictors remains low, indicating that other unknown factors play an important role.[152]

The high prevalence and large inter-individual variability of exercise-induced cTn elevations have raised questions on the clinical implications of these observations. A cross-sectional study linked the presence of occult coronary artery disease to abnormal cTn release patterns, predominantly a delayed recovery to normal kinetics.[153] However, a recent large study did not find a difference in the prevalence and magnitude of coronary atherosclerosis between recreational athletes with very high vs. low post-exercise cTn concentrations.[154] Also, a well-controlled laboratory study did not find differences in exercise-induced elevations in cTn concentrations between athletes with vs. without coronary artery disease.[155] These findings indicate that the inter-

individual variation in post-exercise cTn concentrations is unlikely attributable to occult coronary artery disease.

Prospective studies linking post-exercise cTn concentrations to adverse health outcomes are scarce. A study among older long-distance walkers found that post-exercise cTnI concentrations above the URL were associated with an increased risk for all-cause mortality and major adverse cardiovascular events.[156] In contrast, no associations between elevated post-exercise cTnI concentrations and the incidence of adverse events[157] or cardiac dysfunction[158] were observed in German marathon runners during long-term follow-up. These contradictory findings indicate that elevated post-exercise cTn concentrations may hold prognostic value, but confirmation in larger, younger, and more athletic cohorts is warranted to establish whether these outcomes can be extrapolated to the larger athletic community. The 10-year clinical outcomes of preregistered prospective cohort studies are, therefore, eagerly anticipated.[159]

The underlying mechanisms of exercise-induced cTn elevations remain unknown. Previous studies showed that it may have a pathologic (i.e. necrosis or apoptosis) or physiologic origin (i.e. increased cardiomyocyte permeability or turnover), or a combination of these mechanisms.[147] For example, marathon running compromised cardiomyocyte integrity[160] which may allow cytosolic cTn fragments to leak from the cell into the circulation.[161] On the other hand, a recent MRI study showed no evidence to support the notion that first-time marathon running in healthy middle-aged men has a detrimental impact on the heart.[162] Further *in vitro* and *in vivo* studies are needed to establish the underpinnings of the physiological vs. pathological nature of exercise-induced cTn elevations in athletes.

As a final note, it is noteworthy that exercise produces elevations in other biomarkers, including N-terminal pro-B-type natriuretic peptide (NTproBNP). Nevertheless, exercise-induced elevations in NTproBNP are generally mild and transient, and often do not cause clinical confusion.[14]

Aortic dilatation

Aortic dilatation in young athletes is rare, but appears more frequently in master athletes and retired athletes, potentially linked to specific sport disciplines. In young (elite) athletes, aortic diameters are modestly larger than those of controls (~2–3 mm sinus of Valsalva, ~1–2 mm aortic valve annulus),[163,164] yet rarely exceed sex-specific or absolute cut-offs (prevalence 0.2–4.2%).[163–168] These differences diminish after indexing for height, body surface area,[164] and ventricular size,[65,169,170] supporting the concept that mild aortic dilatation in this group reflects physiological remodelling rather than aneurysmal pathology. Longitudinal studies reinforce this interpretation, reporting slow annual growth (typically 0.2–0.5 mm/year), well below clinically relevant thresholds—and an absence of major aortic events.[166,168] In contrast, up to 30% of master or retired athletes show aortic dilatation.[171] This appears associated with hypertension, exercise-induced hypertension,[172,173] and specific sports (e.g. rowing, rugby, American-style football).[171,174,175] Although robust data on longitudinal cohorts are not available, reported growth rates appear modest, with a very low incidence of major aortic events.[176]

Endurance disciplines, characterized by isotonic load, are associated with modest but consistent aortic root dilation compared with strength athletes and non-athletic controls.[177] Strength athletes, exposed predominantly to isometric load, may experience localized aortic wall stress, but without consistent evidence of dilation.[163,168,177,178] Sports with predominant isotonic components have been associated with greater aortic dimensions than predominantly isometric activities,[178] although these differences often diminish after indexation and rarely exceed physiological limits.[164,177] Large meta-analyses involving over 5000 athletes further demonstrated that those engaged in predominantly isotonic training exhibited larger aortic roots than their isometric-trained counterparts; however, the magnitude of this increase was minor and clinically insignificant.[163,164] In contrast, in master athletes, a clinically relevant prevalence of aortic dilatation has been reported in up to 20–25%. This prevalence was the highest in athletes with both isotonic and isometric loading (e.g. rowers).[171] Overall, predominantly endurance sports may confer a slightly higher long-term risk of aortic dilatation, while more research is needed on aortic dilatation in combined endurance and strength disciplines in master athletes.

Table 2 and *Figure 3* summarize the available evidence on the relationship between prolonged intense training and adverse structural remodelling as well as response to detraining.

Sex and ethnic differences: what are the main knowledge gaps?

Although exercise-induced cardiac remodelling has been studied for decades, our understanding of how these changes differ by sex and ethnicity remains limited.[179–181] Most research has focused on male Caucasian endurance athletes, which makes it difficult to apply the findings to women or athletes from diverse backgrounds. This leaves potentially important variations underexplored.

Sex differences

As previously discussed, men consistently show higher rates of SB, conduction abnormalities, pacemaker implantation, AF, non-sustained ventricular arrhythmias, myocardial fibrosis, and coronary atherosclerosis compared with women. Atrial fibrillation is particularly sex-specific: master male endurance athletes have a higher risk, while an increased incidence has not been demonstrated in female athletes.[43] In addition, one study found that highly trained men had more bradycardia and were more likely to require pacemaker implantation, whereas female skiers showed no such associations.[31]

Similar to men, training in women induces significant electrical and structural adaptation.[179] However, compared with men, female athletes have smaller atrial and ventricular dimensions for their age, less cardiomyocyte hypertrophy, and milder exercise-induced bradycardia, all of which reduce the arrhythmogenic substrate. Female athletes also have lower resting and peak exercise blood pressures, contributing to fewer conduction abnormalities and atrial remodelling. Interestingly, the arrhythmic triggers such as premature atrial or ventricular beats appear similar between sexes, suggesting that it is the structural and electrophysiological environment, rather than trigger frequency, that drives the difference in arrhythmic risk.[65,182,183]

Sex differences are probably shaped by hormonal, autonomic, and ion-channel factors. Women generally have a higher

Table 2 Summary of evidence of an association between high cumulative sport dose and structural cardiovascular abnormalities/troponin release, reversibility with detraining and implications for athletes

	Exercise-induced arrhythmogenic cardiomyopathy phenotype
Evidence of an association	Moderate evidence supports an association between long-term high-intensity endurance exercise and the development of an exercise-associated arrhythmogenic RV phenotype in a small subset of susceptible athletes, even in the absence of identifiable desmosomal gene mutations
Type of studies	Observational cohort and case-control studies in endurance athletes with ventricular arrhythmias, electrophysiological substrate mapping studies, advanced imaging studies demonstrating localized RV scar, and genotype-positive/genotype-negative arrhythmogenic cardiomyopathy cohorts showing exercise-related disease acceleration
Frequency and comparison with controls	Compared with non-athletic controls, endurance athletes presenting with complex ventricular arrhythmias demonstrate a higher prevalence of RV structural, functional, and electrophysiological abnormalities, including localized RV scar and impaired RV function. However, the true prevalence (possibly <1%) in the general athletic population remains uncertain because most studies are referral-based and include highly selected athlete cohorts. In contrast, isolated RV enlargement is common in endurance athletes and frequently represents physiological remodelling
Reversibility with detraining	Detraining is associated with a reduction in arrhythmic burden and disease progression, although complete reversal of structural or functional abnormalities is uncommon
Implications for athletes	The exercise-induced arrhythmogenic cardiomyopathy phenotype may present with ventricular arrhythmias that mimic benign right ventricular outflow tract arrhythmias, requiring careful differential diagnostic evaluation. Importantly, isolated right ventricular enlargement in athletes should not be considered diagnostic of cardiomyopathy in the absence of accompanying arrhythmic, functional, or structural abnormalities suggestive of arrhythmogenic disease. Exercise restriction represents a cornerstone of management, while catheter ablation or ICD implantation may be considered in selected high-risk individuals
	Non-ischaemic left ventricular fibrosis
Evidence of an association	No definitive evidence demonstrating a causal association between sport participation and myocardial fibrosis detected by late gadolinium enhancement (LGE). Some cohorts show a higher prevalence in master endurance athletes compared with controls
Type of studies	CMR studies mainly in middle-aged endurance athletes; limited longitudinal/outcome data
Frequency and comparison with controls	Highly variable prevalence, from <5% to ~50%, depending on LGE definition, inclusion of hinge-point LGE, and CMR methods; generally reported as more frequent than in controls
Reversibility with detraining	Myocardial fibrosis is not reversible. However, other causes of LGE (such as myocardial oedema) are potentially reversible. There are no longitudinal studies assessing reversibility of LGE with detraining
Implications for athletes	Management depends on the clinical context, including the presence of ventricular arrhythmias, extent and pattern of LGE, and symptoms. Right ventricular hinge-point LGE is considered a benign finding and is not associated with adverse prognosis
	Coronary atherosclerosis
Evidence of an association	Positive association between exercise intensity/volume and coronary atherosclerosis in middle-aged and older male athletes. Exercise characteristics were not related to coronary atherosclerosis in female athletes
Type of studies	Cross-sectional and longitudinal CT studies, mainly in middle-aged male endurance athletes
Frequency and comparison with controls	Coronary artery calcification/plaque burden is often higher than in controls; prevalence of coronary plaques ranges widely in master athletes (from approximately 30 to 70%) depending on age, risk profile, and exercise exposure. High-risk plaque features appear uncommon
Reversibility with detraining	Coronary artery calcification is non-reversible. No evidence is available on the effect of detraining on non-calcified plaques
Implications for athletes	Athletes do not show an increased rate of coronary events compared to less active individuals. However, a higher CAC burden remains associated with increased cardiovascular risk also in athletes. At present, there is no evidence to support changing exercise training patterns in asymptomatic athletes with non-obstructive coronary atherosclerosis. Cardiovascular risk factors in athletes should be managed according to general population guidelines
	Cardiac troponin elevations
Evidence of an association	Positive associations between exercise-induced workload (i.e. exercise duration and exercise intensity) and post-exercise cardiac troponin concentrations

Continued

Table 2 *Continued*

	Cardiac troponin elevations
Type of studies	Mostly acute exercise studies; meta-analyses; limited long-term outcome data
Frequency and comparison with controls	Common after endurance exercise; recent meta-analysis reports cTn > upper reference limit in ~one-third of athletes post-exercise, with wide variability by assay, exercise duration/intensity, and sampling time
Reversibility with detraining	Troponin concentrations show peak values at 3–6 h post-exercise, with complete normalization within 24–72 h following exercise cessation
Implications for athletes	Exercise-induced elevations of cardiac troponin concentrations are transient and common in athletes. Abnormal troponin concentrations are prognostic for adverse outcomes in older long-distance walkers, but it remains unclear whether these findings can be extrapolated to younger and low-risk athletes
	Aortic dilatation
Evidence of an association	Aortic dilatation is uncommon in young athletes and generally reflects physiological remodelling, characterized by slow progression and no excess risk of adverse events. Prevalence is higher in master and retired athletes, particularly those engaged in endurance or mixed-load sports.
Type of studies	Echocardiographic and CMR/CT studies; mostly cross-sectional, with limited longitudinal data
Frequency and comparison with controls	Rare in young athletes (<5%); more frequent in master/retired athletes (~20–30%), especially in endurance or mixed-load sports; usually mild, with low event rates
Reversibility with detraining	Data on reversibility are limited, but available evidence suggests that aortic dimensions in athletes tend to show slow progression rather than regression, and there is no evidence that detraining leads to a reduction in aortic size once dilatation is present
Implications for athletes	In young athletes, mild aortic enlargement is usually benign and rarely exceeds clinical thresholds. In contrast, in master athletes, periodic surveillance may be warranted due to a higher prevalence of clinically relevant dilatation despite low event rates

sinoatrial node automaticity, lower sympathetic activity, and oestrogen-related cardioprotection, whereas testosterone and lower oestrogen states in men may promote hypertrophy and arrhythmogenesis.[65,180] Despite these insights, female athletes remain underrepresented in longitudinal studies, leaving important gaps in understanding how arrhythmias develop and progress over time and how cumulative exercise exposure affects these risks.[184]

Ethnic differences

Evidence regarding ethnic variations in exercise-induced cardiac adaptations is even more limited. Most studies involve White athletes, with minimal representation from Black, Asian, or other underrepresented ethnic groups. Ethnic differences in ECG patterns, repolarization, QT interval, autonomic function, and sudden cardiac death risk are well-established in the general population.[17,181] However, whether these translate into differential arrhythmic susceptibility in athletes is largely unknown. Interactions between ethnicity, sex, and genetic predisposition for conduction disease, atrial or ventricular arrhythmias, and cardiomyopathies remain largely unexamined.

Clinical impact of sport-related chronic adverse cardiovascular effects

As discussed, observational studies have reported structural and electrical findings such as focal myocardial fibrosis, increased coronary artery calcification, and a higher prevalence of AF among endurance athletes compared with sedentary individuals. These observations have prompted debate over whether long-term, high-intensity, and high-volume exercise might predispose some athletes to adverse cardiovascular outcomes later in life.[57] However, the clinical significance, causality, reversibility, and long-term prognostic implications of these observations remain incompletely understood. In particular, the extent to which exercise itself contributes to these changes, as opposed to unmasking or interacting with individual susceptibility, remains uncertain. The generalizability of these observations also remains uncertain, as the available evidence is derived predominantly from cohorts of Caucasian male endurance athletes.

A central theme in this discussion is the so-called 'mortality paradox'. Despite imaging or arrhythmic findings that could be interpreted as maladaptive, large-scale cohort studies generally show that former elite athletes enjoy equal or greater longevity than the general population and that extreme doses of exercise do not increase the risk of adverse outcomes.[185–192] A striking example is the recent analysis of the first 200 men to run a sub-4-minute mile, who lived an average of 4.7 years longer than expected for age, sex, country, and birth cohort, supporting the concept that even extreme levels of aerobic training are compatible with exceptional longevity.[193] These data suggest that any potential structural or arrhythmic risks associated with lifelong athletic training do not outweigh the benefits of physical activity, even if intense, at a population level.

For example, the association between AF and endurance exercise is one of the most consistently reported. However, some observational studies suggest that athletes with AF may have a lower stroke risk profile than non-athletic AF populations, although AF in athletes remains associated with an increased stroke risk compared with athletes without AF.[38,43] CAC is also more

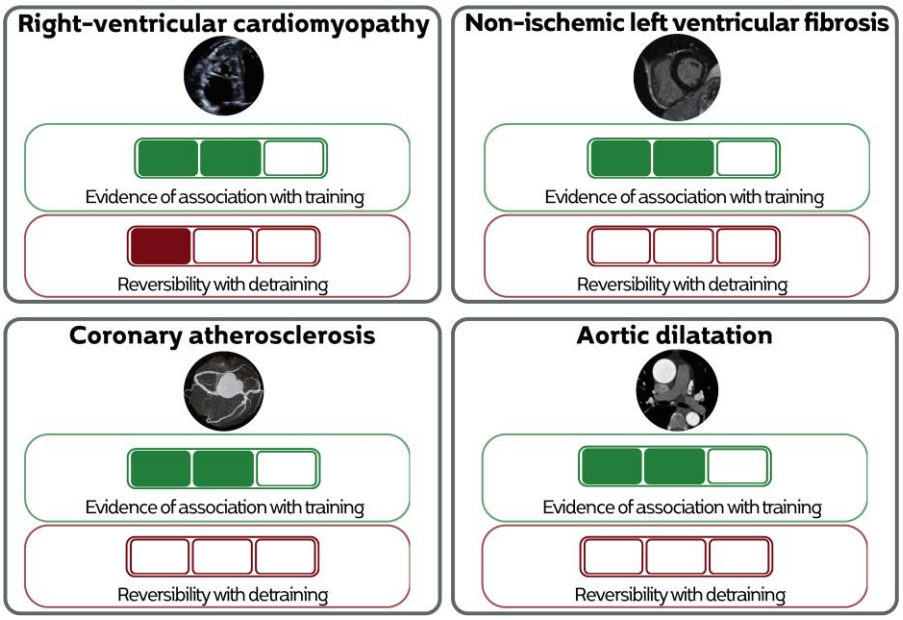

Figure 3 Structural cardiovascular abnormalities associated with long-term training. Schematic summary of the main myocardial and vascular abnormalities reported in athletes. The strength of evidence for an association with training and the degree of reversibility with detraining are qualitatively graded based on the available literature (0/3: no evidence; 1/3: low or preliminary evidence; 2/3: moderate evidence; 3/3: strong evidence).

frequently identified in veteran endurance athletes, but this does not seem to translate into an increased risk of clinical coronary events.[140,141,194] A possible explanation is that in most studies, coronary plaques tend to be stable and heavily calcified, implying a lower propensity for rupture compared with the mixed or lipid-rich plaques seen in sedentary individuals.[194] Finally, small areas of non-ischaemic myocardial fibrosis are common in master athletes and associated to PVBs, but their clinical meaning remains to be established.[195]

However, these observations must be interpreted with appropriate caution. Lifelong athletes likely represent a highly selected population enriched for favourable genetic, behavioural, and socioeconomic characteristics that may independently confer protection against cardiovascular disease. Healthy behaviours and lifestyle after retirement likely contribute importantly to these outcomes. In addition, athletes undergo repeated medical evaluations throughout their careers, which may increase the likelihood of identifying and excluding individuals with occult cardiovascular disease. Thus, the observed mortality benefit may reflect, at least in part, genetic and biological resilience, healthier lifestyles, and repeated medical screening, all of which may both enable sustained athletic participation and contribute to improved long-term outcomes. This selection effect, often referred to collectively as the 'healthy athlete effect', complicates direct causal inference regarding the long-term clinical impact of sport-related chronic adverse cardiovascular effects.

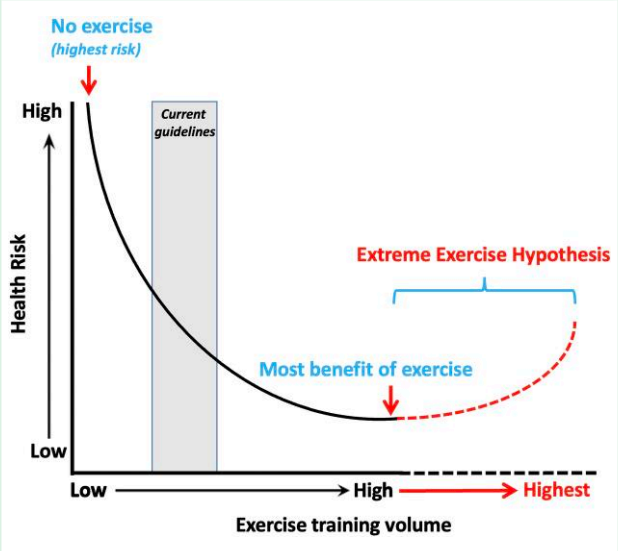

Figure 4 Conceptual visualization of the Extreme Exercise Hypothesis. Adapted from Eijsvogels *et al.*[196] and reprinted from Franklin *et al.*[10] under Creative Commons Attribution 4.0 International License (http://creativecommons.org/licenses/by/4.0/).

Conclusions

In conclusion, the available evidence suggests that while long-term participation in intensive sport is safe for most athletes and generally associated with favourable health and survival outcomes, in some athletes, it may be associated with features of cumulative cardiovascular stress. These observations support the concept that chronic, high-volume exercise loads may, in susceptible subjects,

contribute to structural, electrical, and vascular remodelling beyond the traditionally recognized physiological spectrum (*Figure 4*).[196]

Current evidence remains largely observational, and important uncertainties persist regarding causality, dose-response relationships, reversibility, and the contribution of genetic or acquired susceptibility factors. In particular, no uniform threshold of 'excessive' exercise above which cardiovascular risk consistently increases can be identified. A major limitation is that most data predominantly derive from studies conducted in male Caucasian endurance athletes. Evidence regarding female athletes and individuals from other ethnic backgrounds is scarce or lacking, despite well-recognized sex- and ethnicity-related differences in cardiovascular structure, adaptation, and disease expression. Addressing these gaps represents a key priority for future research.

The observations discussed in this review should not be dismissed as clinically irrelevant, because in selected athletes they may identify early disease expression, increased susceptibility, or a subgroup requiring closer clinical evaluation and longitudinal follow-up. However, any discussion of potential adverse cardiovascular adaptations should be interpreted within the broader context of the well-established cardiovascular and overall health benefits associated with regular physical activity compared with sedentary behaviour. The goal should therefore not be to discourage exercise, but rather to promote a more individualized approach to training, cardiovascular evaluation, and longitudinal follow-up, particularly in ageing athletes and in those with markers of increased susceptibility. In this context, sports cardiology plays a central role in guiding athletes across their life course.

Author contributions

Alessandro Zorzi (Conceptualization, Supervision, Writing—original draft [lead]), Mark Abela (Conceptualization, Writing—original draft, Writing—review & editing [supporting]), Vincent L. Aengevaeren (Conceptualization, Writing—original draft, Writing—review & editing [supporting]), Dimitra Antonakaki (Conceptualization, Writing—original draft, Writing—review & editing [supporting]), Silvia Castelletti (Conceptualization, Writing—original draft, Writing—review & editing [supporting]), Luna Cavigli (Writing—original draft, Writing—review & editing [supporting]), Guido Claessen (Conceptualization, Writing—original draft, Writing—review & editing [supporting]), Clea Colombo (Conceptualization, Writing—original draft, Writing—review & editing [supporting]), Thijs M.H. Eijsvogels (Conceptualization, Writing—original draft, Writing—review & editing [supporting]), Gherardo Finocchiaro (Conceptualization, Writing—original draft, Writing—review & editing [supporting]), Sabiha Gati (Conceptualization, Writing—original draft, Writing—review & editing [supporting]), Francesca Graziano (Writing—original draft, Writing—review & editing [supporting]), Giuseppe Limongelli (Conceptualization, Writing—original draft, Writing—review & editing [supporting]), Viviana Maestrini (Conceptualization, Writing—original draft, Writing—review & editing [supporting]), Aneil Malhotra (Conceptualization, Writing—original draft, Writing—review & editing [supporting]), Harald T. Jorstad (Conceptualization, Writing—original draft, Writing—review & editing [supporting]), David Niederseer (Coneptualization, Writing—original draft, Writing—review & editing [supporting]), Marina Ostojic (Conceptualization, Writing—original draft, Writing—review & editing [supporting]), and Flavio D'Ascenzi (Conceptualization, Supervision, Writing—review & editing [equal], Writing—original draft [supporting])

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