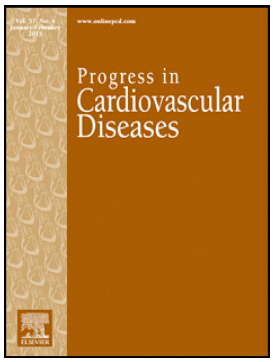

Impact of endurance exercise on the heart of cyclists: A systematic review and meta-analysis

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PII: S0033-0620(20)30141-9

DOI: https://doi.org/10.1016/j.pcad.2020.07.004

Reference: YPCAD 1117

To appear in: *Progress in Cardiovascular Diseases*

Please cite this article as: D.W.T. Wundersitz, B.A. Gordon, C.J. Lavie, et al., Impact of endurance exercise on the heart of cyclists: A systematic review and meta-analysis, *Progress in Cardiovascular Diseases* (2020), https://doi.org/10.1016/j.pcad.2020.07.004

Title page

Title: Impact of Endurance Exercise on the Heart of Cyclists: a Systematic Review and Meta-analysis

Running title: Endurance Cycling and the Heart

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Financial support and conflict of interest disclosure: there are no conflicts of interest or financial support related to this work.

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Keywords: athlete's heart, cardiac abnormality, cardiovascular, bicycle.

Abstract

Objective. To compare heart structure and function in endurance athletes relative to participants of other sports and non-athletic controls in units relative to body size. A secondary objective was to assess the association between endurance cycling and cardiac abnormalities.

Patients and Methods. Five electronic databases (CINAHL, Cochrane Library, Medline, Scopus, and SPORTdiscus) were searched from the earliest record to 14 December 2019 to identify studies investigating cardiovascular structure and function in cyclists. Of the 4865 unique articles identified, 70 met inclusion criteria and of these, 22 articles presented 10 cardiovascular parameters in units relative to body size for meta-analysis and five presented data relating to incidence of cardiac abnormalities. Qualitative analysis was performed on remaining data. The overall quality of evidence was assessed using GRADE. Odds ratios were calculated to compare the incidence of cardiac abnormality.

Results. Heart structure was significantly larger in cyclists compared to non-athletic controls for left ventricular: mass; end-diastolic volume, interventricular septal diameter and internal diameter; posterior wall thickness, and end-systolic internal diameter. Compared to high static and high dynamic sports (e.g., kayaking and canoeing), low-to-moderate static and moderate-to-high dynamic sports (e.g., running and swimming) and moderate-to-high static and low-to-moderate dynamic sports (e.g., bodybuilding and wrestling), endurance cyclists end-diastolic left ventricular internal diameter was consistently larger (mean difference 1.2-3.2 mm/m^2). Cardiac abnormalities were higher in cyclists compared to controls (odds ratio: 1.5, 95%CI 1.2-1.8), but the types of cardiac abnormalities in cyclists were not different to other athletes.

Conclusion. Endurance cycling is associated with a larger heart relative to body size and an increased incidence of cardiac abnormalities relative to controls.

Abbreviations

AC, Amateur cyclist; AV, Atrioventricular; CON, Control; CV , Cardiovascular ECG, electrocardiography; EET, Endurance exercise training; HH, High static & high dynamic sports; LA, Left atrium or atrial; LV, Left ventricular or ventricle; LMMH, Low-moderate static & moderate-high dynamic sports; MD, mean difference; MHLM, Moderate-high

static sports & low-moderate dynamic sports; ND, Not described; OR, odds ratio; PC, Professional cyclist, RV, Right ventricular or ventricle; SD, standard deviation.

Article Highlights

- Endurance cycling is a popular form of exercise training that requires non-weight-bearing isotonic and isometric muscle activity and elevated cardiovascular demand for long durations.
- Most systematic reviews and meta-analyses evaluating the heart of athletes, in particular cyclists, are reported in absolute units (i.e., mm, g, ml, etc.). This systematic review evaluated heart structure relative to body size to ensure that anthropometric differences are not responsible for any differences found.
- Evidence demonstrates that endurance cycling training impacts heart structure and affects heart function.
- Few studies have measured the structure of the right ventricle and atrium in relative units, which is likely due to complex geometry and a lack of specific reference landmarks when using two-dimensional echocardiography. More studies are needed using three-dimensional techniques to evaluate the effect of endurance exercise on the right side of the heart.
- This is the first study to systematically assess the incidence of cardiac abnormality in cyclists. Cyclists were 1.5 times more likely to develop cardiac abnormalities than non-athletic controls; however, the incidence of abnormality was no different to other sport athletes.

Introduction

Exercise, defined as skeletal muscle contraction during any bodily movement that elevates energy expenditure above basal levels [1,2], is associated with positive metabolic,

musculoskeletal and cardiovascular (CV) adaptations [3,4]. Endurance exercise training (EET), approximately 20 hours of intense exercise (15 metabolic equivalents) per week [5], provides additional health benefits [6]. As a result, more people are engaging in EET and competitive sport than ever before [7,8]. The chronic effects of EET are typically described under the umbrella term 'athlete's heart' [5], and include beneficial cardiac adaptations, such as bradyarrhythmia and cardiac enlargement in absence of CV disease [9].

Cycling is a popular competitive mode of EET and the number of recreational cyclists is also increasing around the world [10]. The CV demands of endurance cycling can be high [11,12]. For example, competitive endurance (and some recreational) cyclists train up to seven hours a day [13] with heart rate substantially elevated from rest [14] and at near maximal oxygen consumption [15]. Professional cyclists can cover ~35,000 km per year [16]. When competing, event durations range from ~1 to 5 hours [10] and professional cyclists compete for ~90 days of the year [16]. Competitions are also likely to include multiple back-to-back days of racing that may last as long as three weeks (i.e., the Tour de France, Giro d'Italia and Vuelta a España [10]).

When endurance cyclists are compared to non-athletic controls, heart mass (measured in grams) and diameter (measured in millimetres) have been reported to be significantly larger, and rate (measured in beats per minute) significantly lower [17]. Similar findings have been observed in meta-analyses that grouped cyclists with other endurance trained athletes [18,19], all athletes [20,21] or with rowers [14]. However, cardiac structural and functional outcomes have only been compared in absolute units rather than units relative to body size as suggested by echocardiographic associations/societies [22,23]. Comparison of cardiac parameters relative to body size is important because heart size has been shown to correlate with body size in athletes [24]. Consequently, previously reported comparisons in absolute heart structure and function might reflect anthropometric differences between groups rather than

the influence of a particular exercise regime (e.g. Kou et al. [25]). It is important to evaluate the effects of cycling on heart structure and function because, unlike most other modes of EET, endurance cyclists perform a large volume of non-weight-bearing isotonic and isometric exercise [11,14] that influences the CV demands.

Endurance cycling is shown to lower mortality by 41% [26] and increase longevity by 17% [27] when professional athletes are compared to the general population. However, endurance cyclists have higher cardiac abnormality than the general population [28-31] and an increased incidence of cardiac abnormality when compared to other exercise modes [32]. For example, Pelliccia et al. [32] assessed abnormal ECG patterns in 1005 athletes from 21 sporting modes and found that cyclists displayed the most distinctly abnormal ECG patterns of any sport (abnormalities were found in 35% of cyclists assessed). Further, logistic regression showed cycling was associated with a greater probability of an abnormal ECG pattern being present, such as R or S wave ≥ 35 mm and negative T wave (OR: 6.01, 95%CI: 1.22 to 29.7) [32]. This is of concern given the link between cardiac abnormalities and life-threatening events. For example, one study that followed 46 athletes (37 of whom were cyclists) with diagnosed cardiac abnormalities over 4.7 years and found 18 of these athletes had a major cardiac event and nine (all cyclists) died two years after the event [33]. Only one study to date has systematically compared cardiac abnormality incidence rates between pediatric athletes and non-athletic controls [20]. McClean et al. [20] reported the odds of a training-related cardiac abnormality in pediatric athletes was between 1.4 (1.2-1.7; early repolarization percentage) and 4.6 (3.5-5.6; first degree AV-block) times higher. However, the incidence of cardiac abnormalities in endurance cyclists has not been evaluated.

The primary aim of this systematic review and meta-analysis was to assess the chronic effect of endurance cycling performance on cardiac structure and function in units relative to body size. This was achieved through comparison of endurance cyclists (competitive and

recreational) with non-athletic controls. In addition, to assess potential differences between athletes as a result of the mode of exercise performed, cyclists were compared to athletes from other sports grouped by the mode of EET performed (as defined by Mitchell et al. [11]). A secondary aim was to systematically assess the incidence of cardiac abnormality in endurance cyclists.

Methods

Search strategy

Five electronic databases (CINAHL, Cochrane Library, Medline, Scopus, and SPORTdiscus) were systematically searched from the earliest record to 14 December 2019 using the preferred reporting items for systematic reviews and meta-analyses (PRISMA) statement [34]. Search terms were grouped into three constructs: 'cyclists', 'endurance training' and 'cardiac pathologies' (combined with the AND operator). Search terms for each construct and outcome of each database search are listed in supplementary Table 1. One investigator reviewed all studies by title and excluded studies according to the criteria below in Figure 1. Two investigators independently reviewed all studies by abstract, and subsequently by full text, with a unanimous result required to exclude a study. A third investigator reviewed split judgements and the majority ruled on whether that study was excluded.

Last, where it was suspected that two studies drew on the same pool of participants, data from the study with the larger participant numbers were included [35-37]. In addition, when multiple subgroups of cyclists were presented in a study they were combined into a single cyclist group [38].

*** Insert Figure 1 roughly here ***

Data extraction

The following relative structural and functional parameters were extracted from included studies: **structural;** aortic root diameter, end-diastolic interventricular septal diameter, end-diastolic left atrium (LA) diameter, left ventricular (LV) end diastolic volume, LV end systolic volume, end-diastolic LV internal diameter, end-systolic LV internal diameter, LV mass, end-diastolic posterior wall thickness, and end-diastolic right ventricular (RV) internal diameter, and **functional**; cardiac output and stroke volume.

To allow comparison among cyclists of different body sizes, structure and function, data were extracted from studies that report these indexed to units of body size (i.e., mm/m^2, ml/m^2, g/m^2, and $L/m^2/min$), as per the guidelines from the American Society of Echocardiography and the European Association of Cardiovascular Imaging [22,23]. Data presented only in figures were extracted using digitisation software (Visio professional, Version 10.0.525, Microsoft Corporation, USA). A previously validated formula [39] was used to convert data from median and range/interquartile range to mean and standard deviation (SD). In addition, Cochrane's formula for combining groups [40] was used to pool data for comparison where study data were presented only split for outcomes of interest (i.e., age, sex, etc.), but with no combined values.

Using the participant descriptive information provided in studies, cyclists were classified as: **amateur cyclists** (AC) cyclists who cycle recreationally alone or as members of a cycling club to highly trained cyclists competing at or below the national level, or **professional cyclists** (PC) cyclists who were paid to cycle, national team members or competing at the international level.

In studies with multiple sporting modes, three sporting categories were used to group sports athletes together for comparison with endurance cyclists as described previously [11]. These categories were: **high static and high dynamic sports** (HH; boxing, canoeing, kayaking and rowing); **low-to-moderate static and moderate-to-high dynamic sports** (LMMH;

basketball, swimming, team handball, cross-country skiing, field hockey, long distance running, soccer, tennis, American football, jumping field events and rugby), or **moderate-to-high static and low-to-moderate dynamic sports** (MHLM; bodybuilding, wrestling, diving, equestrian, bobsledding/luge, throwing field events, gymnastics, martial arts, sailing, weight lifting and sprinting) [11].

Assessment of Study Quality

Two researchers independently assessed the quality of evidence using GRADE. Studies were graded low, unclear or high quality of evidence based on five discrete categories: i) **risk of bias** (random sequence generation, blinding of outcome assessors, incomplete outcome data) [41]; ii) **indirectness** (differences in population, differences in outcome measures, and indirect comparisons) [42]; iii) **inconsistency** (variation in effect, confidence interval overlap, heterogeneity and I^2) [43]; iv) **imprecision** (sample size) [44]; and v) **publication bias** (selective publication) [45].

The quality of evidence was then accumulated for each outcome and a final rating given. Note that not all studies included were randomized control trials, hence initial quality was graded as low prior to assessment. Categories with an accumulated 'high risk' percentage above 20% were graded as low quality and marked down by one (if the percentage for the category was above 40% then they were marked down by two).

Data Analysis

Mean differences (MD) with 95% confidence intervals (95%CI) for structural and functional outcomes were calculated using Review Manager (Version 5.3 The Nordic Cochrane Centre; The Cochrane Collaboration, Copenhagen, Denmark). Due to the large number of meta-analyses performed and spread of MD between outcomes, standardised mean difference

(SMD) was reported instead of MD for ease of visual comparison in Figure 2. For dichotomous variables (cardiac abnormality outcomes), odds ratio (OR) with 95% CI were calculated [46].

Results

Study Selection

From the 4865 unique studies identified during the literature search, 70 studies met the inclusion criteria for review (Figure 1) and data were extracted from 25 studies [28,38,47-69] for analysis (Table 1). Of these, 18 studies [28,38,48,49,52-54,56-66] compared cyclists to control and 10 studies [28,47-55] compared cyclists to athletes participating in other sports, with cardiac outcomes reported in units relative to body size for meta-analysis. Seventeen studies were not included in the meta-analysis as they: potentially included the same participants as other included studies [36,37,70], presented control values from a clinical population [71,72] or another study from which the data were being used [73-75], evaluated cyclists who were retired [67,69,76] or presented no outcomes in units relative to body size [35,68,69,77-81].

Insert Table 1 roughly here

CV Structure

Compared to controls (Figure 2), endurance cyclists had larger end-diastolic interventricular septal diameter (MD: 0.9; 95%CI: 0.6 to 1.2 mm/m^2), LV end diastolic volume (13.4; 95%CI: 5.5 to 21.2 ml/m^2), end-diastolic LV internal diameter (3.4; 95%CI: 2.4 to 4.5 mm/m^2), end-systolic LV internal diameter (1.2; 95%CI: 0.5 to 1.9 mm/m^2), LV mass (53.6; 95%CI: 40.4 to 66.8 g/m^2) and end-diastolic posterior wall thickness (1.0; 95%CI: 0.6 to 1.4 mm/m^2).

*** Insert Figure 2 roughly here ***

Compared to all athletes combined, endurance cyclists had larger end-diastolic LV internal diameter (2.4; 95%CI: 1.3 to 3.5 mm/m^2), end-systolic LV internal diameter (1.8; 95%CI: 1.0 to 2.6 mm/m^2) and LV mass (17.6; 95%CI: 6.4 to 28.8 mm/m^2). End-diastolic LV internal diameter was larger in cyclists compared to HH (2.1; 95%CI: 0.1 to 4.1 mm/m^2), LMMH (2.2; 95%CI: 0.4 to 3.9 mm/m^2) and MHLM (32.5; 95%CI: 12.4 to 52.6 mm/m^2) athletes. LV mass was larger in cyclists compared to LMMH (17.6; 95%CI: 6.4 to 28.8 g/m^2) and MHLM (35.0; 95%CI: 9.7 to 60.4 g/m^2) athletes. Cyclists end-systolic LV internal diameter (1.9; 95%CI: 1.3 to 2.6 mm/m^2) and end-diastolic posterior wall thickness (0.39; 95%CI: 0.02 to 0.77 mm/m^2) was larger in cyclists compared to LMMH athletes (Figure 2). For all other CV structure outcomes there was no difference between endurance cyclists and LMMH or HH athletes (Table 2).

A number of studies presented values in relative units that could not be meta-analysed using consistent relative units (i.e., mm/$BSA^{-0.5}$ [38], mm/$m^{2/3}$ [64], g/$h^{2.7}$ [38], etc.). However, when cyclists were compared to CON the end-diastolic LV internal diameter [38,64], end-systolic LV internal diameter, LV mass and end-diastolic posterior wall thickness [38] presented in these studies support the outcomes of the meta-analysis.

Insert Table 2 roughly here

CV Function

Four studies [38,56,66,73] reported functional outcomes from cyclists and control in units relative to body size (Table 1-2). Endurance cyclists had higher stroke volume when compared to control (MD: 10.9; 95%CI 7.7 to 14.1 ml/m^2; Figure 2); however, when cardiac output was at rest there was no difference between cyclists and control (MD: 0.2; 95%CI -0.0 to 0.4 l/m^2/min). No data relative to body size were available for comparison with athletes of other sports.

Higher rates of cardiac abnormalities were observed in cyclists (current and retired) compared to control [54,58,67-69], but there were no differences between cyclists and LMMH athletes (Table 3 [50,51,54]). The most common cardiac abnormalities reported in cyclists were ventricular premature complexes, atrial premature complexes, ventricular ectopic beats, incomplete right bundle branch block, supraventricular tachycardia and atrioventricular block.

Insert Table 3 roughly here

Quality of the Studies and Strength of Evidence

We assessed the quality of the 10 CV outcomes and a total of 25 comparisons included in the meta-analyses (Table 2). Study quality was graded as very low or below for all comparisons. Twenty one of the 25 comparisons assessed were marked down by one for selection biases, 16 were marked down for inconsistency, six were marked down for imprecision and one was marked down for indirectness.

Discussion

This systematic review with meta-analyses is the first to explore cyclists' heart structure and function in units relative to body size. The majority of studies included employed case-control study designs. While methodological weaknesses create higher risk of bias, the results of this meta-analysis confirm that endurance cyclists have different heart structures than non-athletic controls and other athletes relative to body size. Cardiac abnormalities, such as atrial/ventricular premature complexes, are also 1.5 times higher in endurance cyclists than control, but no different to other LMMH athletes (the only athletic comparison possible).

Cardiac Structure and Function of Cyclists

All structural outcomes were significantly larger in endurance cyclists than control (Figure 2, Table 2). This finding expands on previous meta-analyses which found athlete's hearts are significantly larger than control populations when expressed in absolute units [14, 17-21]. The present study provides confidence that endurance cycling causes a disproportional increase in heart structure beyond that due to body size.

While cyclists' hearts were significantly larger than control, the impact of endurance cycling on the heart compared to other low-to-high static and dynamic modes of exercise is less clear (Figure 2, Table 2). The mean difference in CV structure was larger in cyclists for all outcomes assessed relative to body size. However, this trend was only significantly larger in approximately half the outcomes assessed. Endurance cyclists had significantly larger end-diastolic LV internal diameter than all athlete categories measured, and significantly larger end-systolic LV internal diameter than LMMH athletes (the only comparison possible for this outcome). Although not statistically compared, previous meta-analyses have shown that combined athlete categories, which included cyclists, had the highest end-diastolic LV internal diameter [14,18,19] and end-systolic LV internal diameter [18] values when measured in absolute units [14,18,19]. This finding is supported by Pelliccia et al. [55] and Spirito et al. [12] who calculated impact coefficients for different sports and found endurance cycling had the greatest effect on absolute end-diastolic LV internal diameter of the 29 [55] and 27 [12] sports they assessed. These findings suggest that cycling can enhance LV volume and hence increase capacity to perform endurance exercise. Although speculative, cycling might be particularly useful as a training modality to enhance aerobic capacity for athletes in aerobic-based sports.

In addition, endurance cycling had a mixed effect on the LV mass when compared to other modes of exercise training. Endurance cyclists LV mass was significantly larger than LMMH and MHLM athletes, but no different from HH athletes relative to body size. Although not

statistically compared, these findings are supported by previous meta-analyses [14,17,19,82] when cyclists alone [17] or athletes including cyclists [14,19,82] LV mass was reported in absolute units. Cyclists also have significantly larger end-diastolic posterior wall thickness to LMMH athletes (the only comparison possible). Previous meta-analyses have shown cyclists alone [17], or in combination with other HH athletes [14], have a larger end-diastolic posterior wall thickness compared to other LMMH athlete groupings measured in absolute units. Lastly, cyclists' end-diastolic RV internal diameter and end-diastolic left atrium diameter were no different to LMMH athletes (again the only athletic comparison possible). No previous meta-analyses have compared these outcomes, for two reasons: (1) the small number of studies that have measured heart outcomes in relative units and (2) the difficulty in measuring the RV using two-dimensional echocardiography owing to the complex geometry and a lack of specific reference landmarks [23]. This study recommends that further research on the influence of EET on the RV reports heart outcomes in units relative to body size and use three-dimensional imaging techniques.

The difference in endurance cyclists' hearts compared to other modes of exercise training might be due to the type of muscular requirement cycling places on the body and the volume of exercise endurance cyclists complete compared to other modes of exercise. When cycling the lower body musculature is isotonically contracting and at the same time the upper body musculature is isometrically active [11,14]. The metabolic demands of muscle contraction require matched muscle blood flow [83]. Hence, a relatively large mass of contracting muscle requires high and prolonged blood flow. Blood pressure regulation is important to ensure adequate perfusion pressure to all organs [83]. However, during isometric muscular action, as seen in cyclists' upper body musculature, intramuscular pressure increases due to swelling and stiffening of the muscle fibers. This activity results in compression of the intramuscular blood vessels, so blood is forced out of the veins and blood flow into the arteries is hindered

[84]. The addition of upper body isometric muscular strain during cycling is, therefore, suggested to lead to an increased pressure load on the heart [18,85], with elite cyclists systolic blood pressure ranging from 140 to 200 mmHg during exercise [30]. This can provide a greater stimulus for the supply of oxygenated blood to a larger relative muscle mass leading to greater subsequent cardiac adaptation than other modes of exercise. According to Mitchell et al. [11] apart from other HH sports like rowing and canoeing, the majority of sports assessed involve one form of muscular contraction. Therefore, the current findings support a conclusion that endurance cycling imposes additional adaptive stimuli onto the CV system which leads to greater heart adaptation in response. For LMMH and MHLM athlete categories, our findings support this conclusion.

If the cardiac adaptations seen in cyclists are due to this unique muscular contraction alone, then both cyclists and HH athletes should have similar LV mass and end-diastolic LV internal diameter outputs. However, although LV mass was no different (mean bias 0.47 g/m^2 in favour of cyclists), end-diastolic LV internal diameter was significantly larger in cyclists (mean bias 1.20 g/m^2). This suggests that another mechanism is in play. It is possible that the volume of exercise cyclists perform during training and competition, in combination with both dynamic and static muscular exercise, explains our findings. Cyclists train for considerable periods of time, with elite cyclists training for an average of six [14] to seven [13] hours per day. By comparison, elite rowers, another HH mode of exercise similar to cycling, train approximately 2.5 hours per day [86]. This difference in training volume could explain the different CV adaptations identified in cyclists.

Few studies have presented functional heart outcomes in units relative to body size. In the current study, stroke volume was significantly larger while cardiac output was no different in cyclists when compared to control (Figure 2, Table 2). This finding confirms one previous meta-analysis that found the stroke volume of all LMMH athletes (including cyclists) was

significantly larger than control when expressed in absolute units [19]. This is expected as EET is known to increase stroke volume, which in turn enhances an athlete's ability to perform exercise [87]. No previous meta-analysis has compared resting cardiac output between athletes and control in units relative to body size. Nevertheless, this finding is to be expected because it is well established that endurance trained cyclists' resting heart rates are significantly lower than control [58,60,88].

Cardiac Abnormalities in Cyclists

This was the first study to systematically assess cyclists' cardiac abnormality incidence, finding cyclists were 1.5 times more likely to develop cardiac abnormalities than controls; however, there was no difference in incidence when compared to LMMH sport athletes (Table 3). Previous research has confirmed an association between EET and cardiac abnormalities [20,67,89-96]. For example, Claessen et al. [93] found participants, which included cyclists, with a history of long-term endurance sports practice were more likely to develop atrial flutter (odds ratio 5.33, 95%CI 2.1 to 13.53), Heidbüchel et al. [96] found a history of endurance sport participation was a significant risk factor for atrial fibrillation (hazard ratio 1.96, 95% CI 1.19 to 3.22), and Pelliccia et al. [32] found a greater probability of an abnormal ECG pattern was associated with cycling EET. Similarly, Aizer et al. [92] found that men at three years follow-up who exercised 5-7 times per week had the greatest risk of developing atrial fibrillation compared to those who did not exercise at all. Interestingly, in a sub-analysis Aizer et al. [92] reported that men who cycled recreationally (n=2930) were at no greater risk of developing atrial fibrillation. Considering that the mean weekly total amount of time spent exercising in their study was approximately 108 minutes, this result does not apply to endurance trained athletes who complete much larger exercise doses. Hence, the present study likely included participants with a higher fitness level and

extended the dose-response curve to suggest a higher rate of cardiac abnormalities than in the general cycling population [92] as well as non-cyclists who are both inactive and recreationally active.

The increase in cardiac abnormalities found in cyclists compared to control could be due to multiple mechanisms. Firstly, exercise intensity might contribute to cardiac abnormality [95]. Cyclists train while working at elevated heart rates [14] and near maximal oxygen consumption [15], which is suggested to be increased relative to most exercise modes [11,12]. Further the volume of EET performed, including the number of training hours per day [97], days a week [92], and years [98] may also progressively increase the incidence of cardiac abnormalities. Professional cyclists train up to seven hours a day on most days of the week [13] and do so for the duration of their professional careers. Although training history data were sparse in studies included in this systematic review (e.g. Bekaert et al. [58], Palatini et al. [54], Van Ganse et al. [68]), one study reported that participants competed professionally for 11 ± 4 years and covered approximately 25,200 km/year [67] and another reported participants training for 24 years including 16 hours/week covering 522 km/week [69]. Combined, the high intensity and volume of cycling performed could be factors that contribute to cardiac abnormalities as shown in the current study. Further research is required to examine the link between exercise dose and cardiac abnormalities in cyclists. Further, to better understand the impact of the training history of participants on study outcomes, future studies should include in-depth surveys of participant training and competition data.

Widespread use of performance enhancing drugs in professional cycling has been reported previously [99] and the perceived benefits of using performance enhancing drugs are still seen to outweigh perceived risks [100], even in young elite cyclists [101]. Sex has also been suggested to potentially influence cardiac abnormality incidence, with a link between cardiac abnormality and physical activity reported in male athletes (e.g. Thelle et al. [102], Drca et al.

[103]), however the effects in women are less clear [97]. This study was not able to consider the influence of either sex or performance enhancing drugs on the incidence of cycling-induced cardiac abnormality. This was because the five studies only had male participants and only one reported on drug use [67], even though 70% of cyclists report using drugs such as anabolic steroids and amphetamines [67]. Further research is required to explore both these areas.

Abnormalities found in cyclists are likely the result of three mechanisms: the increase in diameter of the LA, myocardial fibrosis, or altered autonomic tone. The diameter of the LA is linked to an increased incidence of cardiac abnormalities [104]. Spataro et al. [49] reported on end-diastolic LA diameter, and found that cyclists' atrium diameter was significantly larger than control while Baldesberger et al. [67] reports similar findings for left atrial volume. This finding, along with no relative differences in LA diameter in the current study between cyclists and LMMH athletes, supports this suggestion.

In some athletes, life-long endurance exercise is associated with myocardial fibrosis [105-107], where collagen fibres become interspersed between cardiomyocytes and impede normal electrical conduction [97]. As a result, myocardial fibrosis is linked to increased incidence of cardiac abnormalities [97,108]. Fornes and Lecomte [109] assessed the pathology of sudden death in 31 recreational athletes, four of which were cyclists, and the cause of death for one was attributed to endomyocardial fibrosis. However, none of the studies included in this systematic review reported on fibrosis [54,58,67-69] and it is difficult to verify any link from the current study as a result. Further research is required to establish a link between cardiac abnormalities and myocardial fibrosis in cyclists.

Lastly, altered autonomic tone as a result of EET is associated with cardiac abnormality in both animals [110] and athletes [111]. Autonomic changes might have contributed to cardiac

abnormalities seen in the current study. However, this was not a focus of the study and further research is required to link cardiac autonomic activity and abnormalities.

The similarities in cardiac abnormalities between cyclists and LMMH athletes indicate that the dynamic component of exercise is more important to cardiac abnormality incidence than the static component. For example, the three included studies compared cyclists to runners [50,54] or soccer players [51] and these two sports are highly dynamic forms of exercise, similar to cycling, with large oxygen consumption requirements [11]. Although the intensity of exercise is similar between athletes, the volume of exercise performed is different (i.e., a soccer match lasts for 1.5 hours [112] and a marathon ends in 2.5 hours or less [113], but cyclists ride for five hours a day on average during the Tour de France [114]). This suggests that cardiac abnormality incidence may not increase once a certain (lower) volume of EET is exceeded or that the incidence could be related to exercise intensity.

This study has limitations that have potential to impact on the findings presented. First, data were largely limited to male cyclists, due to insufficient data available on female cyclists. Further research is required including both male and female cyclists. Second, there could be heart structure and function differences due to the large variance in study age (~34 years between Bekaert et al. [58] and Hong et al. [51]) and differences in measurement technology over this time. The more recent research would have used better imaging technology for measurements. Third, six out of the eight structural outcomes presented were limited to the LV due to insufficient data on the right atrium, LA, and RV reflecting the technical difficulty if measuring these chambers with two-dimensional echocardiography. It is recommended that further research is required to examine all chambers of the heart in units relative to body size. This is achievable through more advanced imaging technologies, such as magnetic resonance imaging. Fourth, the majority of included studies employed case-control study designs, and when pooled presented modest sample sizes. Fifth, selection of comparison athletic groups

matched for body size and EET performance may introduce errors as cycling is non-weight-bearing and is frequently associated with lower bone mass [115]. Last, even though body size parameters do not influence LV diastolic and systolic function [14], many functional outcomes were not included in this review because data were not available in relative units.

Conclusion

This systematic review and meta-analysis on endurance cyclists provides evidence that heart structure and function differ from non-athletic controls. The findings show that the heart of cyclists might differ from other athletes when grouped in relation to their static and dynamic exercise components. These differences in heart structure and function might increase the incidence of cardiac abnormalities in cyclists compared to non-athletic controls.

Declaration of interest

None of the authors have any conflicts of interests with regard to this publication.

Acknowledgements

We acknowledge the support of the Bendigo Tertiary Education Anniversary Foundation and Holsworth Research Initiative for Prof. Kingsley and Dr. Wundersitz's research, with special thanks to Penny Love and Luke Daly.

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Figure Legend

Figure 1. PRISMA flow diagram of the systematic searches, record exclusions and included studies for qualitative and quantitative syntheses (adapted from Moher et al. [34]).

Figure 2. Meta-analysis of relative structural and functional outcomes of cyclists compared to; control, all sport athletes combined, as well as HH, LMMH, and MHLM athletes. CO = Cardiac output; HH = High static & high dynamic sports; $IVST_d$ = End-diastolic interventricular septal diameter; LA_d = End-diastolic left atrium diameter; LVEDV = Left ventricular end diastolic volume; LMMH = Low-moderate static & moderate-high dynamic sports; $LVID_d$ = End-diastolic left ventricular internal diameter; $LVID_s$ = end-systolic left ventricular internal diameter; LVM = Left ventricular mass; MHLM = Moderate-high static sports & low-moderate dynamic sports; PWT_d = End-diastolic posterior wall thickness; SMD = Standardised mean difference; SV = Stroke volume.

Table 1. Descriptive characteristics, and primary CV structural and functional results.

Author, year	Number (male/ female)	Participant classification; description	Age (y)	Height (cm)	Weight (kg)	Vo2max (ml/kg/min)	Training history (y; description)	Primary Results
Case-Control Studies (Cyclists versus non-athletic CON; n=21)								
Abergel et al. 2004 [56]	286/0	PC; competing in the Tour de France	28±3	179±6	71±6	ND	ND	CO ↑; $LVID_d$ ↑; LVM ↑
	52/0	CON; male physicians	26±3	177±6	71±9	ND	ND	
Agati et al. 1985 [57]	12/0	PC; competitive national team members	ND	ND	ND	ND	ND	$LVID_d$ ↑
	8/0	CON; untrained	22±3	179±8	76±8	ND	ND	
Baldesberger et al. 2008 [67]	62/0	Former PC; participated in ≥1 Tour de Swiss	66±7	BMI = 25.6±2.8		ND	11±4y; 485km/wk (range 346-770), stopped competing 38±7y, achieved 342±181 bike years	Angina pectoris =; Atrial flutter/ fibrillation ↑; Atrial premature complexes =; AV block =; Bradycardia ↑; Dizziness ↑; Dyspnoea =; Left anterior/ posterior hemiblock =; Heart failure =; Hypertension =; Myocardial infarction =; Palpations =; RBBB complete/ incomplete =; Supraventricular tachycardia =; Syncope =; Ventricular premature complexes =; Ventricular tachycardia =
	62/0	CON; never performed professional endurance training	66±6	BMI = 25.9±2.4		ND	12±21 bike years (note one bicycle year = 1000km/y)	
Bekaert et al. 1981 [58]	14	PC; ≥3 months in current season's competitions	25±2	178±7	73±7	67±6	ND	$LVID_d$ =; LVM ↑; Left posterior hemiblock?; RBBB incomplete ?
	11	CON; no regular/ intense sporting activity in last 1y	26±2	174±6	64±8	43±7	ND	
Bonaduce et al. 1998 [59]	15/0	Trained AC; ≥3y competing in amateur national teams	21±4	175±8	69±9	67±4	≥3y; 21h/wk, 560-770km/wk	LVEDV ?; LVESV ? LVM ?
	15/0	CON; not involved in	21±3	169±5	71±6	43±4	ND	

		competitive sports						
Galanti et al. 1993 [60]	16/0	PC; evaluated in maximal training	20-31	ND	ND	ND	7y (range 5-17); >40h/wk	LVM ↑
	16/0	CON; no sports competition last 1y	Matched	ND	ND	ND	ND	
Giorgi et al. 2000 [61]	10/0	PC; elite	32±1	176±6	75±3	80±8	ND; 15-20h/wk, plus 1x/wk either 50km or 20km race	$LVID_d$ ↑; LVM ↑
	10/0	CON; sedentary	31±1	176±6	74±5	ND	ND	
Horowitz et al. 2014 [62]	49/7	AC; best-trained from 5 leading cycling clubs	38±10	175±7	72±10	ND	ND; 5x 13±6 h/wk, 5 ranked top10-Israel Cycling League	LVM ↑
	87/9	CON; sedentary	36±9	173±9	74±10	ND	ND	
Jordaens et al. 1994 [28]	15/0	PC; healthy PC	26±4	183±9	74±7	ND	ND	$LVID_d$ =; $LVID_s$ =; LVM ↑
	15/0	CON; no sports activity	25±3	183±6	76±13	ND	ND	
Larsen et al. 2000 [63]	13/4	PC; internationally competitive Danish national team members	20±1	ND	ND	70±8	≥4y; ND	LVM ↑
	10/5	CON; randomly selected from high schools	19±1	ND	ND	52±6	ND	
Lattanzi et al. 1992 [53]	14/0	PC; PC	22±3	ND	71±4	ND	ND	LVM ↑
	10/0	CON; sedentary	24±5	ND	75±5	ND	ND	
Miki et al. 1994 [64]	104/0	PC; PC	35±12	ND	ND	ND	8/29y; ND	$LVID_d$ ↑; LVM ↑
	40/0	CON; sedentary	36±12	ND	ND	ND	ND	
Milliken et al. 1988 [52]	9/0	AC; highly trained & competitive (US Cycling Federation cat 2/3)	25±1	182±3	77±2	72±2	6±1y; ND	LVM ↑
	9/0	CON; no organised training or sport ≤8y	27±1	177±3	80±4	44±2	ND	
Missault et al. 1993 [65]	26/0	PC; PC	26±3	183±7	74±6	ND	~5y; ~600-800km/wk	LVM ↑; RWT ↑
	21/0	CON; ≤recreationally active	26±4	179±9	ND	ND	ND; <1h/wk	
Nottin et al. 2004 [38]	22/0	AC; endurance-trained members of local clubs	17±6	165±17	55±17	ND	≥5y; 5-20h/wk	CO ?; SV ?
	24/0	CON; no regular training	18±6	166±16	61±6	ND	ND	
Palatini et	20/0	AC; highly trained,	21±7	BSA = 1.8±0.1		ND	≥3y; 317km/wk	$IVST_d$?; $LVID_d$?; LVM ?;

al. 1985 [54]		partake in competitions						PWT_d ?; $RVID_d$? Ventricular ectopic beats ?; 1st Degree AV block ?
	40/0	CON; no training in sport or other activities	19±5	BSA = 1.8±0.1		ND	ND	
Pellicca et al. 1996 [48]	0/20	PC; Italian National team members	22±6	BSA = 1.6±0.1		ND	ND	LVM ?
	0/65	CON; sedentary	23±5	BSA = 1.6±0.1		ND	ND	
Plium et al. 1998 [66]	21/0	PC; elite	42±8	181±6	77±8	ND	23±7y (13-35); ≥239km/wk	LVEDV ↑; LVM ↑; SV ↑
	12/0	CON; ≤recreationally active	47±8	182±7	78±10	ND	ND	
Spataro et al. 1985 [49]	44/0	PC; top-class international athletes	20±2	ND	ND	ND	ND	AR_d ?; $IVST_d$?; LA_d ?; $LVID_d$?; $LVID_s$?; LVM ?; PWT_d ?
	50/0	CON; untrained	23±4	ND	ND	ND	ND	
Van Ganse et al. 1970 [68]	30/0	AC; ≥semi-professional, ≥2 months competitive in current season	22±3	178±6	72±6	ND	>2y; ND	Multiple unifocal ventricular extrasystoles ?; RBBB incomplete =
	30/0	CON; untrained	22±3	177±5	72±7	ND	ND	
Vollmer-Larsen et al. 1989 [69]	16/0	AC [n=8]; veteran cyclists Former AC [n=8]; as good as the AC	45±3	180±5	81±6	46±12	Former AC: >11y (range 5-22); stopped competing 17y (range 3-25). AC: 24y (range 11-35); 522km/wk (range 240-580), 16h/wk.	Bradycardia ?; Prominent U-waves ?; RBBB incomplete ?; Terminal inversion of T-waves ?; 1st Degree AV block
	8/0	CON; non-athletic (no competitive sport ever)	43.3±2.3	178±3	77±5	33±3	ND; <3h/wk	
Case-Control/Cohort Studies (Cyclists versus athletes; n=10)								
Agati et al. 1985 [57]	12/0	PC; competitive national team members	ND	ND	ND	ND	ND	$LVID_d$?
	12/0	HH; same as PC	ND	ND	ND	ND	ND	
	18/0	MHLM; same as PC	ND	ND	ND	ND	ND	
Azevedo et al. 2014 [50]	11/0	PC; highly trained	26±1	ND	ND	79±2	10±1y; 7days/wk; 826km/wk. Top 5 in national competition last 5y	LA =; $LVID_d$ =; LVM =; PWT_d ↑ Bradycardia ?
	13/0	LMMH; highly trained	29±1	ND	ND	80±2	7±1y; 7days/wk; 185 km/wk. Top 10 in national	

							competition last 5y	
Hong et al. 2015 [51]	40/0	PC; competing in the Tour de France	27±5	182±5	71±6	ND	ND	$IVST_d$ =; LA_s ↑; LVEDV ↑; LVESV ↑; $LVID_d$ ↑; $LVID_s$ ↑; LVM ↑; PWT_d ↑; $RVID_d$ ↑ Bradycardia =; Early repolarisation ↑; RBBB incomplete ↑; T-wave abnormalities =; 1st Degree AV block =
	40/0	LMMH; Danish National team or Danish Premier league	26±6	181±14	80±7	ND	ND	
Jordaens et al. 1994 [28]	15/0	PC; healthy PC	26±4	183±9	74±7	ND	ND	$LVID_d$ ↑; $LVID_s$ =; $LVID_d$ ↑; LVM =
	10/0	LMMH; 1st Belgian National team	25±5	195±8	92±12	ND	ND	
Lattanzi et al. 1992 [53]	14/0	PC; PC	22±3	ND	71±4	ND	ND	LVM =
	10	MHLM; weightlifters	25±6	ND	83±7	ND	ND	
Milliken et al. 1988 [52]	8/0	AC; highly trained & competitive (US Cycling Federation cat 2/3)	25±1	182±3	77±2	72±2	6±1y; ND	LVM ?
	18/0	LMMH; Nationally ranked in Top25 or 10km in ≤32min	26±2	180±2	71±5	76±2	8±1y or 13±1y; ND	
Palatini et al. 1985 [54]	20/0	AC; highly trained, partake in competitions	21±7	BSA = 1.8±0.1		ND	≥3y; 317km/wk	$IVST_d$ =; $LVID_d$ =; LVM =; PWT_d =; $RVID_d$ = Ventricular ectopic beats ?; 1st Degree AV block ?
	20/0	LMMH; same as AC	19±7	BSA = 1.8±0.1		ND	≥3y; 75km/wk	
Pellicca et al. 1991 [47]	49/15	PC; Italian National team members	20±3	BSA = 1.9±0.1		ND	ND	LVM ?
	158/11	HH; same as PC	21±4	BSA = 2.0±0.1		ND	ND	
	356/114	LMMH; same as PC	21±4	BSA = 1.9±0.1		ND	ND	
	175/42	MHLM; same as PC	26±4	BSA = 1.9±0.1		ND	ND	
Pellicca et al. 1999 [55]	45/4	PC; Italian National team members	24±4	BSA = 1.9±0.2		ND	ND	$LVID_d$?
	122/12	HH; same as PC	23±4	BSA = 2.0±0.2		ND	ND	
	485/209	LMMH; same as PC	24±5	BSA = 1.9±0.2		ND	ND	
	166/94	MHLM; same as PC	23±6	BSA = 1.7±0.3		ND	ND	

Spataro et al. 1985 [49]	44/0	PC; top-class international athletes	20±2	BSA = 1.9±0.1	ND	ND	AR_d ?; $IVST_d$?; LA_d ?; $LVID_d$?; $LVID_s$?; LVM ?; PWT_d ?
	43/0	HH; same as PC	20±3	BSA = 1.9±0.9	ND	ND	
	123/0	LMMH; same as PC	24±5	BSA = 2.0±0.2	ND	ND	
	61/0	MHLM; same as PC	24±5	BSA = 2.0±0.2	ND	ND	

Indicates that the outcome was; not statistically compared '?', significantly larger/higher in cyclists '↑', significantly lower/smaller in cyclists '↓', or nor different '='. Note: AC = Amateur cyclists; AR_d = Aortic root diameter at end-diastole; AV = Atrioventricular; CCA = Common carotid artery cross sectional area; CON = Non-athletic controls; CV = Cardiovascular; HH = High static & high dynamic sports; $IVST_d$ = End-diastolic interventricular septal diameter; LV = Left ventricle; LMMH = Low-moderate static & moderate-high dynamic sports; $LVID_d$ = Left ventricular internal diameter at end-diastole; $LVID_s$ = Left ventricular internal diameter at end-systole; LVM = Left ventricular mass; MHLM = Moderate-high static sports & low-moderate dynamic sports; ND = Not described; PC = Professional cyclists; PWT_d = End-diastolic posterior wall thickness; RBBB = Right bundle branch block; $RVID_d$ = Right ventricular internal diameter at end-diastole; SV = Stroke volume.

Table 2. Cyclist structural and functional outcomes reported in units relative to body size.

Parameter (relative units)	Descriptive details	Cyclist data (Mean ± SD)	Mean Difference (95% CI)	Quality of evidence (GRADE)
CO ($L/m^2/min$)	3; n=317 [CON; n=77]	3.2 ± 0.6	0.2 (-0.0 to 0.4)	⨁⊖⊖⊖ Very low[a]
$IVST_d$ (mm/m^2)	2; n=64 [CON; n=90]	6.28 ± 1.02	0.90 (0.58 to 1.23)*	⊖⊖⊖⊖ Very very low[a,b]
	3; n=104 [All; n=307]	5.92 ± 1.06	0.31 (-0.17 to 0.79)	⊖⊖⊖⊖ Very very low[a,b,d]
	3; n=104 [LMMH; n=203]	5.92 ± 1.06	0.13 (-0.73 to 1.00)	⊖⊖⊖⊖ Very very low[a,b,d]
LA_d (mm/m^2)	3; n=95 [All; n=280]	20.38 ± 1.89	0.87 (-0.37 to 2.10)	⊖⊖⊖⊖ Very very low[a,d]
	3; n=95 [LMMH; n=176]	20.38 ± 1.89	1.03 (-1.01 to 3.07)	⊖⊖⊖⊖ Very very low[a,d]
LVEDV (ml/m^2)	2; n=36 [CON; n=27]	97.1 ± 22.1	17.2 (12.0 to 22.5)*	⨁⊖⊖⊖ Very low[b]
$LVID_d$ (mm/m^2)	7; n=401 [CON; n=186]	30.33 ± 2.65	3.53 (2.05 to 5.01)*	⊖⊖⊖⊖ Very very low[a,d]
	7; n=191 [All; n=1428]	30.27 ± 2.49	2.04 (1.12 to 2.95)*	⨁⊖⊖⊖ Very low[d]
	4; n=125 [HH; n=209]	30.08 ± 2.67	1.2 (0.18 to 2.21)*	⊖⊖⊖⊖ Very very low[a,d]

	5; n=159 [LMMH; n=880]	30.20 ± 2.30	2.07 (0.34 to 3.81)*	⨁⊖⊖⊖ Very low[d]
	3; n=93 [MHLM; n=339]	30.59 ± 2.50	3.22 (0.92 to 5.53)*	⨁⊖⊖⊖ Very low[d]
$LVID_s$ (mm/m^2)	2; n=59 [CON; n=65]	19.9 ± 2.6	1.2 (0.4 to 1.9)*	⊖⊖⊖⊖ Very very low[a,b]
	3; n=99 [All; n=277]	19.82 ± 2.48	1.67 (1.06 to 2.28)*	⨁⊖⊖⊖ Very low[a]
	3; n=99 [LMMH; n=173]	19.82 ± 2.48	1.94 (1.31 to 2.57)*	⨁⊖⊖⊖ Very low[a]
LVM (g/m^2)	16; n=686 [CON; n=477]	151.7 ± 39.7	59.8 (45.0 to 74.6)*	⨁⊖⊖⊖ Very low[a]
	8; n=216 [All; n=1194]	139.55 ± 40.07	17.44 (7.12 to 27.77)*	⊖⊖⊖⊖ Very very low[a,d]
	2; n=108 [HH; n=212]	145.41 ± 41.29	0.47 (-2.89 to 3.84)	⊖⊖⊖⊖ Very very low[a,c]
	7; n=202 [LMMH; n=721]	138.24 ± 36.18	14.23 (-0.24 to 28.70)*	⊖⊖⊖⊖ Very very low[a,d]
	3; n=122 [MHLM; n=288]	152.73 ± 45.01	35.24 (10.32 to 60.16)*	⊖⊖⊖⊖ Very very low[a,d]
PWT_d (mm/m^2)	2; n=64 [CON; n=90]	5.8 ± 0.8	1.03 (0.61 to 1.45)*	⊖⊖⊖⊖ Very very low[a,d]
	3; n=104 [All; n=287]	5.76 ± 0.84	0.31 (-0.10 to 0.71)	⊖⊖⊖⊖ Very very low[a,d]
	3; n=104 [LMMH; n=183]	5.76 ± 0.84	0.39 (0.02 to 0.77)*	⊖⊖⊖⊖ Very very low[a,d]
$RVID_d$ (mm/m^2)	2; n=60 [LMMH; n=60]	16.2 ± 4.4	1.4 (-2.2 to 4.9)	⊖⊖⊖⊖ Very very low[a,d]
SV (ml/m^2)	3; n=51 [CON; n=75]	59.1 ± 11.0	10.9 (7.7 to 14.1)*	⊖⊖⊖⊖ Very very low[a,b]

Descriptive details are: number of studies; number of cyclists [non-cyclist description; number of non-cyclists]. * Mean difference was significant ($p < 0.05$). Note: CO, Cardiac output; CON, control; HH, High static & high dynamic sports; $IVST_d$, End-diastolic interventricular septal diameter; LA_d, End-diastolic left atrium diameter; LVEDV, Left ventricular end diastolic volume; LMMH, Low-moderate static & moderate-high dynamic sports; $LVID_d$, End-diastolic left ventricular internal diameter; $LVID_s$, end-systolic left ventricular internal diameter; LVM, Left ventricular mass; MHLM, Moderate-high static sports & low-moderate dynamic sports; PWT_d, End-diastolic posterior wall thickness; $RVID_d$, end-diastolic right ventricular internal diameter; SD, standard deviation; SV, Stroke volume.

GRADE working group grades of evidence.
High quality: Further research is very unlikely to change our confidence in the estimate of effect.
Moderate quality: Further research is likely to have an important impact on our confidence in the estimate of effect and may change the estimate.
Low quality: Further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate.

Very low quality: We are very uncertain about the estimate.
[a] Downgraded for selection bias.
[b] Downgraded for imprecision (inadequate sample size).
[c] Downgraded for indirectness (differences in population, indirect comparisons).
[d] Downgraded for inconsistency (CI overlap, heterogeneity and I^2).
[e] Downgraded for publication bias.

Table 3. Odds of cardiac abnormalities in cyclists compared to control and LMMH athletes.

		Total number		With condition		
Abnormality	**Studies**	**Controls**	**Cyclists**	**Controls**	**Cyclists**	**OR (95% CI)**
Cyclists compared to control						
Overall	5	1340	1366	213	294	1.45 (1.19 to 1.76)
Rate & rhythm	4	520	516	104	143	1.47 (1.10 to 1.96)
-Bradyarrhythmia	3	140	156	15	36	2.50 (1.30 to 4.80)
-Tachyarrhythmia	1	124	124	24	37	1.77 (0.98 to 3.19)
-Supraventricular arrhythmia	1	186	186	42	54	1.40 (0.88 to 2.24)
-Ventricular arrhythmia	2	70	50	22	14	0.87 (0.42 to 1.80)
ECG abnormalities	4	184	198	11	23	2.07 (0.98 to 4.37)
Coronary/ Structural abnormalities	1	124	124	1	5	5.17 (0.59 to 44.89)
Symptoms	1	372	372	97	119	1.33 (0.97 to 1.83)
Cyclists compared to LMMH athletes						
Overall	3	253	251	52	54	1.06 (0.69 to 1.63)
Rate & rhythm	3	273	271	40	36	0.89 (0.55 to 1.45)
-Bradyarrhythmia	3	113	111	30	26	0.85 (0.46 to 1.55)
-Tachyarrhythmia	1	20	20	15	13	0.62 (0.16 to 2.43)
ECG abnormalities	1	120	120	7	15	2.31 (0.90 to 5.88)

Note: LMMH = Low-moderate static & moderate-high dynamic sports; OR = Odds ratio.