Clustered Cardiometabolic Risk and Arterial Stiffness of Recreational Adult Tennis Players

Running title: Clustered cardiometabolic risk: tennis

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ABSTRACT

BACKGROUND: Recent evidence highlights racquet sports as being associated with a substantially reduced risk of CVD mortality. The purpose of this investigation was to evaluate clustered cardiometabolic risk (CMR) and arterial stiffness in recreational adult tennis players.

METHODS: Forty-three recreational tennis players (T) and a matched group of 45 healthy, active non-tennis (NT) players, mean age (± SEM) 41.6 ± 1.8 years participated in this cross-sectional comparative study. Measurements included emerging and traditional CMR factors with pulse wave analysis/velocity utilised to assess indexes of arterial stiffness. Clustered cardiometabolic risk was calculated using two composites: CMR1 (central aortic systolic blood pressure, carotid-femoral pulse wave velocity, percentage body fat, HDL-C and maximal oxygen uptake) and CMR2 (brachial systolic blood pressure, triglycerides, TC:HDL-C, percentage body fat, HbA_{1c} and maximal oxygen uptake).

RESULTS: Analysis of covariance, controlling for age, revealed T had significantly lower (healthier) CMR1 scores than NT (EMM ± SEM, T: -0.48 ± 0.3 vs NT: 0.50 ± 0.3, $P = 0.03$). Similarly, T also demonstrated lower clustered CMR2 scores (EMM, T: -0.66 ± 0.4 vs NT: 0.59 ± 0.4, $P = 0.04$). Augmentation index of the pulse pressure wave, normalised to heart rate 75 bpm (AIx75), was lower in T vs NT (EMM, T: 10.7 ± 1.7% vs NT: 12.7 ± 1.6%; $P = 0.03$), when controlling for age and gender.

CONCLUSIONS: Tennis appears to be a suitable and effective physical activity modality for targeting cardiometabolic and vascular health and should be more frequently advocated in physical activity promotion strategies.

Key words: Tennis, Vascular Stiffness, Cardiovascular Diseases, Pulse Wave Analysis, Metabolic Syndrome

Introduction

Cardiovascular disease (CVD) remains the leading cause of death globally, with metabolic disorders such as type 2 diabetes, obesity and the metabolic syndrome continuing to pose significant and ever-increasing threats to morbidity and mortality rates world-wide.[1] The likelihood of developing these diseases is underpinned by a clustering of cardiometabolic abnormalities,[2,3] which encompass traditional and emerging risk factors, including dyslipidemia, hypertension, dysglycaemia, central obesity and inflammatory profile.[4]

Increased time spent sedentary is independently associated with clustered cardiometabolic risk (CMR)[3,5] and vascular endothelial dysfunction,[6] the latter a precipitating event in atherosclerosis.[6] Conversely, increased levels of physical activity and cardiorespiratory fitness are extensively supported in the research literature as being mediating mechanisms for both reducing CMR factors[7,8] and by directly altering vascular structure and function.[9,10] CVD mortality[11] and CVD risk[7] can thus be reduced by an active lifestyle, but despite this, a substantial proportion of adults do not meet the current physical activity recommendations.[12] Modes of activity that are appealing and effective are highly desirable for public health promotion. Past research has determined that different sports and activities proffer varying cardiometabolic and vascular health benefits, due to the divergent physiological demands inherent within each. Recently, a large, population-based cohort study[13] determined that participation in racquet sports (tennis, badminton and squash), in males and females aged >30 years, was associated with a substantially reduced risk (-56%) of CVD mortality, the highest risk reduction of any sporting type listed in the study. This finding was congruent with the earlier work of Pluim *et al.*[14] which established, through a systematic review, that regular tennis participation was associated with more favourable key traditional CVD risk factors and also overall relative risk of CVD. Quantitively, a 28% reduction in overall CVD risk has been attributed to playing tennis for ≥ 5 hours a week compared to not participating in this activity, through the large-scale, prospective male cohort study of Chomistek *et al.*[15] Coupled with the worldwide

popularity of tennis,[14] these studies have highlighted the sport as an ideal mode of physical activity for reducing CVD risk in the wider population.

Nevertheless, to date there has been limited research that has assessed the impact of tennis in recreational players,[16-18] with many studies instead focusing on elite.[19] Of those who have focussed on recreational tennis, some positive alterations to adiposity, lipid and lipoprotein subfractions have been reported, but these studies have tended to be small, used adapted versions of tennis training or have used comparison groups of significantly disparate activity levels to those of the tennis players.[16,17,20] Furthermore, previous studies have all assessed CMR factors as singular components when it is established that CMR factors cluster in a large proportion of the adult population[3] and that prognostically, CMR clusters significantly elevate the risk of CVD development.[2] Current physical activity guidelines for adults recommend both moderate and/or vigorous-intensity aerobic activity and strengthening exercise,[21] which tennis play satisfies. Nonetheless, research into the effects of recreational tennis on clustered CMR in healthy adults, to our knowledge, has not previously been undertaken.

In order to fully evaluate the cardiometabolic benefits of tennis however, it is important to be cognisant that modification of traditional CMR factors does not account fully for the magnitude of the CVD risk reduction attained through regular exercise and that in fact, direct effects on the vasculature play a substantial role.[10,22] Incorporation of direct structural and functional vascular measures are thus important indices that could significantly extend our existing knowledge of the health benefits of the sport. Both carotid-femoral pulse wave velocity and central blood pressure have been gaining widespread prominence as markers capable of refining CVD risk prediction.[23,24] Analysis of pulse pressure waveforms for indices such as central aortic systolic blood pressure (CAorSP) and augmentation index (AIx) permit insights into forward and reflective pulse pressure wave characteristics[25] and therefore systemic vascular resistance. Pulse wave velocity is representative of vascular stiffness[23,24] and relates to CVD risk.[24] To our knowledge, no direct measures of either index of vascular stiffness have been undertaken to date, in recreational tennis players. The purpose of this investigation was therefore to

evaluate whether recreational tennis participation is an effective mode of activity for reduced clustered cardiometabolic risk and arterial stiffness in adults.

Materials and methods

Study design and participants

Eighty-eight healthy adult participants (male: n = 42; female: n = 46), aged between 18 and 65 years (overall mean age ± SEM; 41.6 ± 1.8 years), volunteered to participate and consisted of tennis players (T) and non tennis (NT) players. The study was conducted in accordance with the recommended guidelines for ethical practice set out by the Declaration of Helsinki. Ethical approval for all procedures was granted by the University Ethics Committee and all procedures were performed in accordance with the institutional and national guidelines. Rights to confidentiality, withdrawal and the benefits/risks of the protocols were explained prior to participation. All participants provided written, informed consent.

Sample size was determined using G*Power Software12 and 80% power with an α error probability of 0.05 (2-tailed), based on previous findings by Swank *et al*.,1998[19] of significantly lower percentage body fat in tennis players versus age-matched moderately active non-players. Power calculation determined that a sample size of ≥42 participants per group was required to observe a statistically significant difference.

Participants were recruited into either the T or NT groups so that physiological variables could be directly compared between tennis players and non-players. For T, forty-three tennis players (18 males, 25 females) were recruited, primarily through the Liverpool and District Tennis Group, and had an average of 20.0 ± 2.5 years (mean ± SE) of playing experience. Inclusion criteria for the T group were a) playing year-round doubles and/or singles tennis on b) a regular weekly basis and c) being classed as ‘recreational players’ (played competitively in local leagues) but not currently or historically ‘elite’. A comparison group of age-matched healthy, habitually active non tennis players (n = 45; 24 males, 21 females) were recruited from within the same locality to form the NT group. Inclusion criteria for the NT-matched

comparison group included being a) classed as habitually physically active i.e. at a minimum, meeting the recommended 150 minutes per week of moderate-intensity physical activity[21] with the caveat of b) not playing any tennis as part of their habitual physical activity. The physical activity levels of both T and NT participants was established via the International Physical Activity Questionnaire–Short Form (IPAQ-SF) (data presented in Table 2). All participants were healthy, non-smokers, with no history of clinically diagnosed metabolic or cardiovascular diseases or co-morbidities including hypertension, diabetes mellitus, dyslipidaemia, CHD, and had no other contraindications, as indicated by their responses to the PAR-Q+ pre-participation health screening questionnaire.[26] None of the participants were taking any regular vasoactive, antihypertensive or lipid lowering medication.

Experimental procedures

In this cross-sectional study, all physiological testing took place during one laboratory visit ensuring the recommended minimum of 3 hrs refrain from food ingestion was observed, according to expert consensus guidelines.[23,26,27] Participants were instructed not to consume alcohol or caffeine or perform strenuous exercise within the 24 hours preceding their test visit. All test procedures took place in a well-ventilated laboratory that was maintained between 18-21^{o}C.

Anthropometry

Participants' height was measured using a wall-mounted stadiometer (Stable stadiometer Seca, Birmingham, UK) to the nearest 0.1 cm. Body mass was measured with mechanical measuring scales (Seca, Hamburg, Germany) to the nearest 0.1 kg. Waist (at the navel, while relaxed) and hip (largest hip girth around buttocks) circumferences were measured, to the nearest 0.1 cm, using ergonomic measuring tape (Seca, Hamburg, Germany). Body mass index (BMI; kg.m^{-2}) and waist:hip ratio (WHR) were calculated from these four base measurements.

Body composition was non-invasively assessed using a four-terminal, multi-frequency bioelectrical impedance analyser (Bioscan 920-2, Maltron International Ltd., Essex, UK). Whole-body composition

analysis was performed with participants in the supine position following 5 minutes of rest, with electrodes placed on the right side of the body on: the dorsal aspect of the hand, (over the third metacarpal and between the styloid process of the radius and ulnar), and on the dorsal aspect of the foot (over the third metatarsal and between the medial and lateral malleolus). Whole-body composition values were computed via BioScan 920 v1.1 software (Maltron International Ltd).

Pulse wave analysis

Non-invasive pulse wave analysis (PWA) was employed to assess central aortic pressure waveform parameters and carotid femoral pulse wave velocity (cf-PWV) of both T and NT participants, in accordance with expert consensus guidelines and manufacturers instructions.[23] Both PWA and cf-PWV (SphygmoCor XCEL, AtCor Medical Pty Ltd, NSW, Australia) were conducted on subjects in a supine position in a temperature-controlled room, following a 10 minute period of supine rest. Both PWA and cf-PWV measured on the SphygmoCor XCEL are accepted as being valid and highly reliable measures.[28] Briefly, a brachial cuff was placed around the upper left arm for the conventional measurement of brachial systolic and diastolic pressures.[28] Following this, the same brachial cuff was re-inflated to a sub-diastolic level of pressure for the derivation of the central aortic pressure waveform parameters from the peripheral pressure pulse.[28] Waveform parameters of interest were: central aortic diastolic pressure (CAorDP), central aortic systolic pressure (CAorSP) and central aortic pulse pressure (CAorPP) and were automatically generated via the SphygmoCor software. CAorDP is calculated by the lowest pressure value recorded in a pulse; CAorSP is calculated as the maximum pressure during aortic injection; CAorPP is calculated as central aortic systolic pressure minus the central aortic diastolic pressure. Augmentation index (AIx), an indirect, surrogate measure of vascular stiffness, was determined from the central pressure waveform via the pulse wave analysis software and is defined as the augmentation of the central pressure wave above that of the incident central pressure wave. AIx is thus calculated as the difference between the first and second systolic peaks (augmentation pressure) of the central arterial waveform divided by the central pulse pressure (SBP-DBP) x 100, at the participant's resting heart rate.[23,29] AIx was subsequently normalised to a heart rate of 75 beats per minute (AIx75) to standardise

the measure across participants. All arterial stiffness index measures were made in duplicate and repeated if the first two values differed beyond 0.5 m/s.[29]

Carotid-femoral pulse wave velocity

Immediately following the pulse wave analysis and still using the SphygmoCor XCEL, carotid-femoral pulse wave velocity (cf-PWV) was ascertained by simultaneously acquiring the common carotid pulse waveform, through a tonometer pressure sensor, and the femoral pulse waveform, by placing a cuff around the femoral artery. This enabled the calculation of the pulse transit time, which is the time taken for the pulse to travel from the common carotid artery to the femoral artery. To calculate cf-PWV, the distance between the two recording sites was measured on each participant (d: linear distance from suprasternal notch to top of femoral cuff minus distance from suprasternal notch to carotid pulse tonometry location),[28] so that cf-PWV could be automatically derived by dividing this distance by transit time. Heart rate and mean arterial pressure were recorded at the time of all measurements.[29]

Blood sampling

A 1.5µl volume of fingertip capillary blood was collected and assessed for glycated haemoglobin (HbA_{1c}) content (Alere Afinion AS100 Analyzer, Abbott Laboratories, Abbott Park, Illinois, USA). A further 30µl capillary sample was taken and assessed for: total cholesterol (TC), high density lipoprotein cholesterol (HDL-C) triglycerides (TRG), low density lipoprotein cholesterol (LDL-C), TC:HDL ratio, non-HDL cholesterol and blood glucose (Alere Cholestech LDX Analyzer, Abbott Laboratories).

Maximal oxygen uptake

Maximal oxygen uptake ($\dot{V}O_{2max}$) was assessed through a treadmill test to volitional exhaustion (h/p/cosmos, Munich, Germany) with a standard Bruce protocol.[30] Gas exchange and ventilatory variables were measured breath-by-breath continuously throughout the test using a metabolic cart (Ergostik, Geratherm Respiratory, GmbH, Bad Kissingen, Germany). Participants wore a silicone facemask (Hans Rudolph, Kansas City, KS, USA) of known dead-space attached to a low-dead space

flow sensor (Geratherm Respiratory, GmbH). A capillary line connected the flow sensor (on participant) to the metabolic cart. Gas analysers were calibrated prior to each test using gas mixtures of known concentrations and flow sensors were calibrated using a 3 L syringe (Hans Rudolph, Kansas City, MO, USA). Heart rate was sampled every 1 s throughout the test using short-range radiotelemetry (Garmin FR70, Garmin Ltd, Southampton, UK). $\dot{V}O_{2max}$ was defined as the highest $\dot{V}O_2$ value measured over 30s and was considered to have been attained at either the observation of a plateau in $\dot{V}O_2$ or the point of volitional exhaustion. If a plateau was not reached $\dot{V}O_{2max}$ was considered where either respiratory exchange ratio > 1.15, heart rate within 10 beats of age predicted maximum (220 – age) or rating of perceived exertion > 19.

Data analysis and statistics

The calculation of clustered CMR using z-scores is a statistical technique that permits the unitless summation of individual 'risk factors' that are known to influence cardiometabolic disease. A growing body of literature supports the use of clusters of continuous CMR scores[5,31-34] and the widespread recognition of the value of emerging risk factors suggest that the use of new indexes may be helpful predictive tools.[32] To this end, clustered CMR was calculated using 2 different composites (CMR1 and CMR2), that were indicative of risk of both cardiovascular disease and metabolic syndrome.[2]

Prior to the clustered CMR calculations, non-normally distributed data were firstly log-transformed. All individual risk factor variables were then converted to gender-specific z-scores using SPSS version 25 (IBM, New York, USA). Z-scores, otherwise known as standardised scores represent how many standard deviations above or below the population mean a raw score is. HDL-C and aerobic fitness z-scores were inverted for appropriate directional weighting (given higher scores in these 2 variables reflect reduced risk). The gender-specific z-scores of individual risk factors were then summed using 2 different composite equations: CMR1 comprised a novel cluster of traditional and emerging CVD risk factors and indices of arterial stiffness and central pressure namely: % body fat, CAorSP, cf-PWV, inverted HDL-C and inverted aerobic fitness. The inclusion of the two emerging vascular risk factors, CAoSBP and cf-

PWV, in this cluster was due to CAoSBP being a stronger predictor of CV events/atherosclerosis compared to brachial blood pressure.[24] Similarly, cf-PWV was included as it is considered the gold standard for the assessment of arterial stiffness[23] and is a powerful predictor of cardiovascular and all-cause mortality.[35,36] CMR2, commonly seen in the research literature, represents risk of cardiovascular disease and type 2 diabetes,[3,31,33] and included the traditional clustered CMRs of: systolic blood pressure, triglycerides, total cholesterol/HDL-C ratio, percentage body fat, HbA_{1c} and inverted aerobic fitness. For both these composites, a higher clustered CMR score indicated a higher cardiometabolic risk.

CMR1 = Σz scores for log % fat, CAorSP, log cf-PWV, inverted HDL-C and inverted $\dot{V}O_{2max}$

CMR2 = Σz scores for log SBP, log TRG, log TC:HDL-C, log % Fat, log HbA_{1c} and inverted $\dot{V}O_{2max}$

Differences in descriptive characteristics and physical activity of T and NT participants overall and by gender were established using independent t-tests, controlling for familywise error rate (Bonferroni-Holm). Analysis of covariance (ANCOVAs) assessed the differences in clustered CMR1 and CMR2 between T and NT groups, with age acting as a covariate. Two-way ANCOVAs, again controlling for age, were employed to explore whether any differences in clustered cardiometabolic risk 1 and 2, by tennis playing status, were gender specific. Analysis of covariance was further employed on indices of pulse wave analysis (AIx75, CAorSP, CAorDP, CAorPP) and pulse wave velocity (cf-PWV) to compare T and NT players. All PWA and cf-PWV analyses controlled for resting heart rate, age, gender and tennis status*age interaction with the exception of AIx75 which did not control for heart rate as it is already heart rate-normalised. Data are presented as means ± standard error of the mean (SEM) and 95% CIs where appropriate. Statistical significance was accepted at $P < 0.05$. All analyses were performed in SPSS version 25 (IBM, New York, USA).

Data availability

The data associated with the paper are not publicly available but are available from the corresponding author on reasonable request.

Results

No differences in aerobic capacity ($\dot{V}O_{2max}$) were detected between the T and NT players, confirming their successful matching as physically active groups (Table 1). No other differences in the descriptive characteristics or singular CMR markers of the T and NT participants were evident overall, or by gender. Table 2 displays the physical activity data collected from the IPAQ-SF of both the T and NT groups. No significant differences were evident in the total weekly physical activity levels between T and NT groups. Tennis players reported a greater amount of time spent performing moderate-intensity activity per week than NT (T: 1118 ± 130 vs NT: 495 ± 74 MET-min/wk, P <0.05) whilst no differences between groups were found for weekly vigorous-intensity activity or weekly accumulated MET-min/wk spent walking. Importantly, time spent sitting, as an estimate of sedentary behaviour, did not significantly differ between groups.

Clustered cardiometabolic risk

Results of the two clustered risk analyses are illustrated in Figure 1, in which the two separate clustered risk scores (CMR1, CMR2) were compared by means of analysis of covariance between T and NT whilst controlling for age. As shown in Figure 1, which displays estimated marginal means for the model (EMM ± SEM), clustered CMR1 was significantly lower in T compared to NT (EMM T: -0.48 ± 0.3 (95% CI -1.1 – 0.1) vs NT: 0.50 ± 0.3 (95% CI -0.1 – 1.1), P = 0.018), signifying a lower clustered cardiometabolic risk in the tennis players. Clustered CMR2 was also significantly lower in T compared to NT players (EMM T: -0.66 ± 0.4 (95% CI -1.5 – 0.2) vs NT: 0.59 ± 0.4 (95% CI -0.3 – 1.4), P = 0.043). Subsequent analyses through two-way ANCOVA evaluated whether gender moderated the differences in CMR1 and CMR2 according to tennis playing status, whilst controlling for age. No significant differences in CMR1 by gender (P = 0.77) or via a gender x tennis interaction (P = 0.53) were found, Figure 2A, (EMM Male, T: - 0.94 ± 0.6 (95% CI -2.1 – 0.3); NT: 0.71 ± 0.5 (95% CI -0.3 – 1.7); EMM Female, T: -0.44 ± 0.5 (95% CI -1.4 – 0.5); NT: 0.53 ± 0.5 (95% CI -0.5 – 1.6). Likewise, CMR2 differences according to tennis playing status were not moderated by gender (P = 0.67) nor a tennis x gender interaction (P = 0.65),

Figure 2B, (EMM Male, T: - 0.99 ± 0.7 (95% CI -2.3 – 0.4); NT: 0.60 ± 0.6 (95% CI -0.6 – 1.8); EMM Female, T: -0.44 ± 0.6 (95% CI -1.6 – 0.7); NT: 0.58 ± 0.6 (95% CI -0.7 – 1.8).

Pulse wave analysis/velocity

Differences between the T and NT groups for indices from the pulse wave analysis and pulse wave velocity are shown in Table 3. Analysis of covariance, with age and gender as covariates, determined that AIx75 was significantly lower in T vs NT (EMM T: 10.7 ± 1.7 % (95% CI 7.3 – 14.1) vs NT: 12.7 ± 1.6 % (95% CI 9.5 – 16.0), P = 0.03, η^2 = 0.06), Table 3. Significant main effects also occurred for age and gender in this model (age, P = 0.001, η^2 = 0.45; gender, P = 0.001, η^2 = 0.20) in addition to a significant interaction effect of tennis status x age (P = 0.04, η^2 = 0.052). No other indices of PWA (central pressures) or PWV (controlling for age, gender and heart rate) reached statistical significance for differences between T and NT. Significant main effects of age were also evident in the remaining PWA (central pressure) and PWV measures (cf-PWV, P = 0.001, η^2 = 0.57; CAorSp, P = 0.001, η^2 = 0.27; CAorDP, P = 0.001, η^2 = 0.22 and CAorPP, P = 0.005, η^2 = 0.09;) with larger (poorer) values in higher ages.

Discussion

This study set out to evaluate clustered cardiometabolic risk and arterial stiffness in recreational adult tennis players and compare against a group of matched healthy, physically active non-players. The principle finding of the investigation was that clustered CMR, calculated using two different clusters, was significantly lower in the T players compared to the NT participants, signifying lower clustered CMR risk, and this observation was not affected by gender. Additionally, we determined that augmentation index (AIx75) of the pulse pressure waveform, an indicator of arterial stiffness, was lower in T versus NT when controlled for age and gender.

Clustered cardiometabolic risk

The first cardiometabolic cluster utilised in this study, clustered CMR1, incorporated 2 novel and emerging risk factors of both cf-PWV and central aortic systolic blood pressure, alongside select traditional CVD risk factors (% fat, HDL-C and $\dot{V}O_{2max}$). Clustered CMR2 focused solely on traditional CMR factors of: peripheral SBP, TRG, TC:HDL-C ratio, % Fat, HbA_{1c} and $\dot{V}O_{2max}$, as used in previous research[3,33] as representative of the risk likelihood of T2DM and CVD.[2] Both clusters were significantly lower in T compared to matched, NT who acted as healthy physically active controls and this was unaffected by gender or gender x tennis interaction.

Whilst clustered CMR has not previously been employed in adult tennis players, these findings are compatible with those previously published that have reported a reduced risk of developing both singular CVD risk factors[14,16,17,19,20] and clinically diagnosed CVD[14] in tennis players, and reduced CVD mortality risk in players of racquet sports.[13] Specifically, positive alterations in the singular CMR factors adiposity/BMI[19,20] and cardiorespiratory fitness[14,19,20] have been identified in past studies in tennis players of various standards. In a similar population to the present study, some favourable adaptations in lipid and lipoprotein subfractions were also noted in middle-aged male and females tennis players,[16,17] although unlike the present study, the comparison group was of sedentary controls[16] or an adapted tennis training programme was used.[17] Our study however is the first to our knowledge to assess *clustered* CMR in recreational tennis players with a view to elucidate its potential for reducing risk of chronic disease. Using a clustered risk calculation approach, we have been able to detect differences in clustered CMR in healthy active adult men and women differentiated solely by tennis playing status, that were not evident in singular risk factor comparisons. This is an important finding given CVD and type 2 diabetes are underpinned by a clustering of CMR factors[2,3] and CVD risk increases progressively with an increased number of CMR risk factors.[32] Of particular interest is the finding that the noted differences in cardiometabolic risk according to tennis playing status were not affected by gender and therefore these findings relate to both biologic sexes. Consideration of biologic sex on chronic training responses is key

when prescribing exercise to optimise training adaptations and this findings supports tennis for health benefits in both sexes.

Arterial stiffness

The assessment of cf-PWV, central aortic pressures and pulse pressure waveforms (AIx75) in this population was a novel aspect to this study, as was the inclusion of central and peripheral vascular indices (CAorSP and cf-PWV) in the clustered CMR1 composite. The inclusion of both CAorSP and cf-PWV in CMR1, which was lower in T versus NT, is a positive finding, as it is established that elevated central pressure is associated with degree of atherosclerosis[24] and future CV events.[35] Furthermore, cf-PWV is considered the gold standard for the assessment of arterial stiffness[23] and is a powerful predictor of cardiovascular and all-cause mortality.[35,36] More specifically, from the pulse wave analysis, we demonstrated that AIx75 was lower in T versus NT (when covarying for age and gender and age/tennis playing status interaction). Analysis of the pressure waveforms in this way permitted insight into wave reflection and augmentation characteristics. During the second phase of systole, the forward pressure wave is augmented by the reflected pressure wave, which is generated by changes in impedance.[25] The magnitude of AIx75 is quantified as the augmented pressure relative to central pulse pressure,[25] thus a high AIx75 may in part be due to increased systemic vascular resistance.[23] From these results, is evident therefore that T display attenuation in this index of arterial stiffness (AIx75) in comparison to NT, although it should be noted that both age and gender are also impacting on this outcome as is the interaction between tennis playing status and age. Notwithstanding, the significant main effects of tennis play on AIx75 overall has positive ramifications given larger augmentation indexes have been found to coexist with augmented common carotid artery intima-media thickness and left ventricular mass[37] and, in males, AIx correlates with cardiovascular mortality.[35]

The mechanistic physiological underpinnings of lower AIx75 in T versus NT cannot be fully discerned from this study but reduced CMR and/or directly altered arterial calibre are plausible contributors. Exercise induces haemodynamic alterations, notably alterations in arterial wall shear stress, which initiate

anti-atherogenic functional adaptations and structural remodelling, via means of transduction signalling of vascular endothelial cells.[10,22,38] Further, it has long been recognised that distinct patterns of shear stress arise from different types of exercise stimului and beneficial changes in arterial structure have been reported previously in racquet sports and tennis players.[38-40]

Our findings thus offer support that tennis is suitable mode of activity for targeting cardiometabolic and vascular health benefits, in that T matched, and for some indices, surpassed those seen in NT who also attained the general weekly physical activity recommendations. One possible explanation is that tennis is an intermittent activity that challenges all energy systems.[41,42] It provides anaerobic/muscular strength and endurance stimuli through high intensity efforts (4-10 seconds) interspersed with lower level aerobic activity periods (10-20 seconds) during episodic recoveries, typically giving a work:rest ratio of between 3:1 and 5:1.[41,43] Although recreational level players are under-studied, a study by Bernardi *et al.*[44] in middle-level, non-elite male tennis players, determined match play elicits oxygen uptake values of 46-59 % of maximum, although there is significant HR variability due to the game's intermittent nature and fluctuating intensity.[43] Demands of match play can be varied and are specific to level, sex, singles or doubles, but typically, 300 to 500 intensity efforts are completed within a match.[41] High intensity exertion phases within sets corresponding to 97% maximal oxygen uptake and 100% of maximal heart rate.[42] Consequently, the metabolic and haemodynamic excursions of episodic exposures to such volumes and intensities of activity characterise tennis play as intermittent in nature.[42] Tennis is also of sufficient stimulus to meet the current global physical activity recommendations for[21] which recommend that adults should undertake 150-300 minutes a week of moderate-intensity, or 75-150 minutes a week of vigorous-intensity aerobic physical activity (or a combination of both) and perform muscle-strengthening activities, involving all major muscle groups, 2 or more days a week.[21] From a clinical exercise promotion perspective, tennis is provides an intermittent hybrid training stimulus of at least moderate-intensity.[41,42]

One acute bout of tennis is sufficient to cause a cascade of salutary metabolic and physiological responses, including augmented lipolysis.[17] As a mode of longer-term training, the value of intermittent

exercise on metabolic and vascular health indices has recently been highlighted.[45,46] Research has demonstrated that high-intensity interval training can ameliorate the severity of metabolic syndrome,[46] and shows a greater tendency to improve CVD risk factors than moderate-intensity continuous training.[45] Strong evidence exists that aerobic exercise training significantly improves indices of arterial stiffness (cf-PWV and AIx) and higher absolute and relative intensities of exercise procure the best training response in AIx.[9] Notably, a systematic review by Ramos *et al.*[45] determined that high intensity interval training is more potent at improving brachial artery vascular function than moderate-intensity continuous training. Taken together, it is plausible that the intermittent nature of tennis and the challenge it provokes in all energy systems may be important in distinguishing the reasons for the difference we are seeing between tennis players and healthy active NT counterparts for the clusters CMR1, CMR2 and AIx75 in this study.

An alternative explanation is that T participants in this study tended to spend more time within a given week performing activity of moderate-intensity than NT, which hypothetically, might have provided a greater relative workload and thus adaptive response. However, no statistically significant differences were detected in the cumulative total weekly physical activity levels (MET-min/wk) of T versus NT (incorporating moderate- and vigorous-intensity activity plus walking), nor in the objectively assessed cardiorespiratory fitness between groups or in their levels of sedentary behaviour (sitting time). Furthermore, moderate intensity physical activity was not statistically correlated with either clustered cardiometabolic risk composite or any vascular variable under investigation. With our use of a healthy, habitually active comparison group in this study therefore, the superior clustered CMR exhibited in T point towards being tennis being an effacious mode of activity for cardiometabolic health benefits in both men and women. Our findings thus extend those of others, in demonstrating superior CMR profiles in tennis players, and supports the possible health potential of recreational tennis as a viable, effective and social mode through which to meet physical activity guidelines.

The following limitations to this study should be considered alongside the findings. In a cross-sectional study such as this, we are only able to assess comparative differences in the CMR clusters and vascular

stiffness indices between T and NT rather than retrospective causality. Whilst other studies have previously reported salutary health effects of tennis, it is also feasible that individuals who play tennis were initially in better health. Whilst effort was made to control for potential confounders such as age, sex, physical activity level, BMI and locality/postcode, socioeconomic data was not collected which may have acted as a confounder. However, our findings align well with published research that vascular function can improve through particular exercise stimuli,[10,22] and past research demonstrates that tennis is a mode of exercise which is associated with reductions in some CVD risk factors.[14,16,17,19,20] Physical activity levels of the participants in this study were assessed through self-report which confers greater measurement error than objective assessment methods, due to recall issues and that both under- and over-reporting. However, the IPAQ-SF widely accepted for its high reliability[47,48] and stronger agreements between the IPAQ and accelerometer are noted in the literature for categorizing individuals as active (ie meeting physical activity guidelines),[47] as was done in this study. Whilst levels of moderate-intensity physical activity differed between the T and NT groups in this study, there was no significant difference reported between T and NT in total weekly physical activity (MET-min/wk) or sitting time. Furthermore, moderate-intensity physical activity did not linearly relate to the dependent variables CMR1, CMR2 or any dependent vascular variable under investigation. As a result, moderate-intensity physical activity was not a permissable covariate in the ANCOVA analyses as it would violate the assumption that a covariate should be linearly related to the dependent variable at each level of the independent variable. We are confident therefore in attributing the differences in clustered CMRs and AIx75 between T and NT in this study to the recreational tennis play stimulus and not to differences in total weekly physical activity or inactivity.

Conclusions

In conclusion, we have demonstrated that two different CMR clusters are significantly lower in a group of male and female tennis players compared to age-matched, habitually physically active, non-tennis playing counterparts. These clusters incorporated traditional and emerging risk factors that are predictive

of risk of atherosclerosis and also metabolic diseases such as type 2 diabetes. We also noted a lower vascular stiffness index in T vs NT, as evidenced by a reduced AIx75. Tennis participation therefore appears to proffer physiological benefits that could target metabolic and cardiovascular disease risk and these appear to go beyond those secured through meeting the current global physical activity guidelines.[21] Future research should now elucidate the relative importance of the components of the different physical activity exposures in tennis (frequency, intensity, total volume and mode) in relation to cardiometabolic and vascular health benefits alongside epidemiological outcomes. Notwithstanding, tennis tends to be a social sport suitable for both genders of all ages, in which the intermittent intensity can be varied by playing either singles or doubles, making it an appropriate mode for meeting the global physical activity guidelines. Its potential for health benefits should be considered in future physical activity strategies aiming to reduce cardiometabolic disease risk.

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Conflict of interest

The authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript.

Authors' Contributions

Denise M. Roche: study conception and design; analysis and interpretation of data; drafting of manuscript; critical revision; Matthew Jackson: study conception and design; acquisition and processing of data; critical revision; Farzad Amirabdollahian: study conception and design; critical revision; Omid Khaiyat: study conception and design; critical revision. All authors read and approved the final version of the manuscript.

Acknowledgements

The authors would like to thank the volunteers who participated in the study.

TABLES

Table 1 Descriptive characteristics of the study participants and cardiometabolic risk markers

	All		Male		Female	
	T	**NT**	**T**	**NT**	**T**	**NT**
n	43	45	18	24	25	21
Age (years)	44.3 (2.6)	39.5 (2.5)	47.5 (3.8)	37.0 (3.4)	42.1 (3.4)	41.5 (3.6)
Height (m)	1.69 (0.01)	1.69 (0.01)	1.74 (0.02)	1.74 (0.02)	1.65 (0.01)	1.63 (0.02)
Weight (kg)	68.2 (1.2)	72.4 (1.7)	70.2 (1.9)	76.1 (1.9)	66.8 (1.6)	67.8 (0.03)
BMI ($kg.m^{-2}$)	23.8 (0.4)	25.2 (0.5)	23.1 (0.6)	25.1 (0.6)	24.4 (0.6)	25.3 (0.9)
WHR	0.82 (0.01)	0.84 (0.01)	0.86 (0.01)	0.87 (0.01)	0.79 (0.01)	0.80 (0.01)
% Fat (%)	23.4 (1.2)	23.2 (1.2)	16.9 (1.2)	18.3 (1.4)	28.2 (1.0)	29.2 (1.1)
SBP (mmHg)	123.1 (1.9)	126.1 (1.8)	124.8 (2.9)	127.8 (2.4)	121.8 (2.5)	123.9 (2.5)
DBP (mmHg)	74.6 (1.4)	74.9 (1.5)	76.1 (2.1)	74.4 (2.4)	73.5 (1.9)	75.5 (2.0)
HbA_{1c} ($mmol.mol^{-1}$)[a]	34.2 (0.02)	33.8 (0.02)	34.8 (0.04)	33.6 (0.04)	33.8 (0.03)	34.1 (0.04)
HbA_{1c} (%)[b]	5.3 (0.04)	5.3 (0.04)	5.3 (0.06)	5.2 (0.07)	5.2 (0.05)	5.3 (0.06)
TC ($mmol.L^{-1}$)	4.8 (0.2)	4.5 (0.1)	4.8 (0.3)	4.5 (0.2)	4.8 (0.2)	4.4 (0.2)
HDL-C ($mmol.L^{-1}$)	1.7 (0.1)	1.5 (0.1)	1.5 (0.1)	1.3 (0.7)	1.8 (0.1)	1.7 (0.1)
LDL ($mmol.L^{-1}$)	2.6 (0.1)	2.4 (0.1)	2.7 (0.3)	2.5 (0.2)	2.5 (0.2)	2.3 (0.1)
TRG ($mmol.L^{-1}$)	1.2 (0.1)	1.3 (0.1)	1.3 (0.2)	1.4 (0.2)	1.2 (0.1)	1.2 (0.1)
TC:HDL-C	3.1 (0.2)	3.2 (0.2)	3.5 (0.4)	3.6 (0.3)	2.8 (0.2)	2.7 (0.1)
$\dot{V}O_{2max}$ ($mL.kg^{-1}.min^{-1}$)	39.2 (1.6)	41.5 (1.5)	44.6 (2.4)	46.7 (2.0)	35.2 (1.8)	35.4 (1.5)

Data are provided as means ± (SEM). No significant differences were evident between T and NT overall or between T and NT by gender.

Abbreviations: BMI, Body mass index; DBP, diastolic blood pressure; HbA_{1c}, glycosylated haemoglobin; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; NT, non-tennis players; SBP, brachial systolic blood pressure; T, tennis players; TC, total cholesterol; TRG, triglycerides; $\dot{V}O_{2max}$, maximal oxygen uptake; WHR, waist:hip ratio.

[a] IFCC (International Federation of Clinical Chemistry) units ($mmol.mol^{-1}$)

[b] DCCT (Diabetes Control and Complications Trial) units (%)

Table 2 International Physical Activity Questionnaire–Short Form responses for tennis players (T) and nonplayers (NT)

	T (n = 43)	**NT (n = 45)**
Vigorous-intensity, MET-min/wk	1218 ± 240	1409 ± 141
Moderate-intensity, MET-min/wk	1118 ± 130	495 ± 74*
Walking, MET-min/wk	862 ± 120	927 ± 98
Total, MET-min/wk	3198 ± 301	2831 ± 200
Sitting, min/d	352 ± 24	448 ± 28

Data are presented as mean ± SE.

*Indicates significant difference ($P < 0.05$) between groups when compared by independent t test.

Abbreviations: MET, metabolic equivalent; NT, non-tennis players; T, tennis players;

Data processing and cleaning guidelines available at www.ipaq.ki.se.

Table 3 Estimated marginal means of indices of pulse wave analysis and pulse wave velocity between T and NT

Variable		EMM ± SEM	95% CI	Sig.	Partial Eta Squared
AIx75 (%)[†]	T	10.7 ± 1.7	7.3 – 14.1	0.03	0.06
	NT	12.7 ± 1.6	9.5 – 16.0		
Cf-PWV ($m.s^{-1}$)	T	6.3 ± 0.2	6.0 – 6.6	0.36	0.01
	NT	6.5 ± 0.1	6.2 – 6.8		
CAorSP (mmHg)	T	110.9 ± 1.6	107.6 – 114.1	0.27	0.02
	NT	114.1 ± 1.6	111.0 – 117.2		
CAorDP (mmHg)	T	75.3 ± 1.2	72.8 – 77.8	0.83	0.001
	NT	76.3 ± 1.2	73.9 – 78.8		
CAorPP (mmHg)	T	35.5 ± 1.0	33.4 – 37.5	0.07	0.04
	NT	37.6 ± 1.0	35.6 – 39.5		

EMM, estimated marginal means with 95% CI.

Covariates in the ANCOVA model: gender, age = 41.8 years, HR = 59.9 bpm, age*tennis.

[†] HR excluded as a covariate.

Abbreviations: AIx75, augmentation index at heart rate 75 bpm; cf-PWV, carotid-femoral pulse wave velocity; CAorSP, central aortic systolic blood pressure; CAorDP, central aortic diastolic blood pressure; CAorPP, central aortic pulse pressure

TITLES AND LEGENDS OF FIGURES

Figure 1 Clustered cardiometabolic risks of T and NT players; CMR1 (left), CMR2 (right)

Bars represent estimated marginal means (± SEM). Black bars represent tennis players (T), grey bars non-tennis players (NT). * $P < 0.05$ (ANCOVA); covariates in the model are evaluated at the following values: age = 41.8 years.

CR1 = Sum of z scores for % Fat, CAorSP, cf-PWV, HDL-C and $\dot{V}O_{2max}$

CR2 = Sum of z scores for SBP, TRG, TC:HDL-C, % Fat, HbA_{1c} and $\dot{V}O_{2max}$

Abbreviations: CAorSP, central aortic systolic blood pressure; cf-PWV, carotid-femoral pulse wave velocity; HbA_{1c} glycosylated haemoglobin; HDL-C, high density lipoprotein cholesterol; SBP, systolic blood pressure; TC:HDL-C, total cholesterol to HDL-C ratio; TRG, triglycerides; $\dot{V}$O2max, maximal oxygen uptake; % Fat, percentage body fat

Figure 2 Gender differences in clustered cardiometabolic risk by tennis playing status.

Bars represent estimated marginal means (± SEM). Diagonal hatched grey bars represent males, light dotted bars represent females; covariates in the model are evaluated at the following values: age = 41.8 years. Panel A: CMR1; Panel B: CMR2.

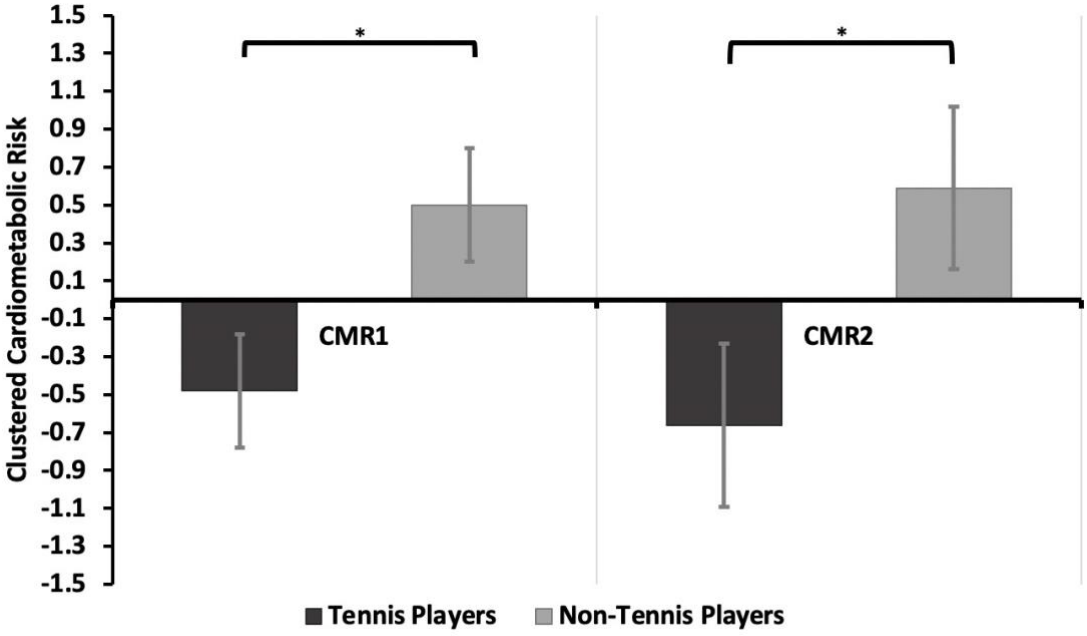

Figure 1 Clustered cardiometabolic risks of T and NT players

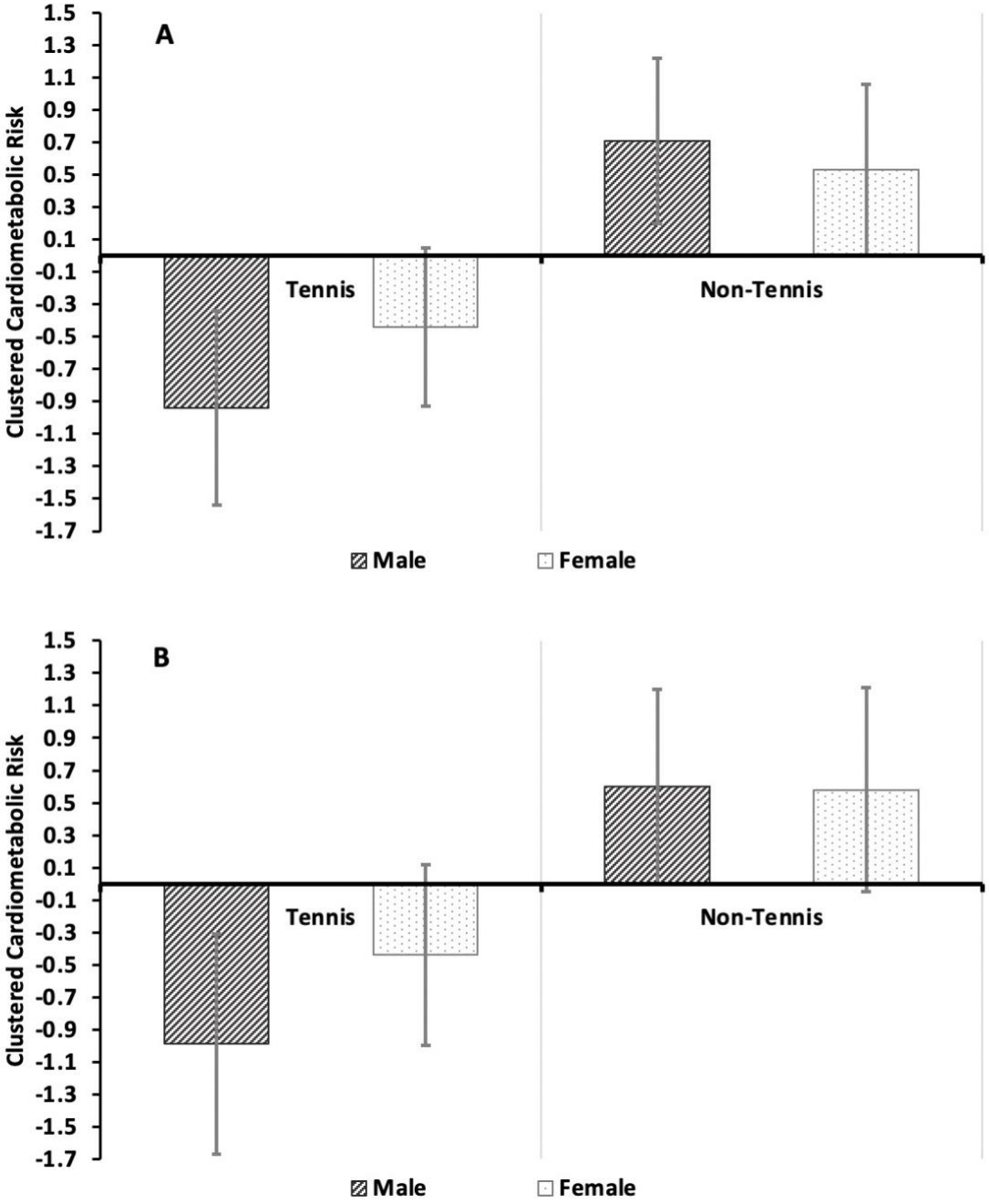

Figure 2 Gender differences in clustered cardiometabolic risk by tennis playing status.