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Review

Are the Cardiovascular Benefits and Potential Risks of Physical Activity and Exercise Dependent on Race, Ethnicity, or Sex?

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ABSTRACT

Physical activity (PA) is established as a cornerstone of cardiovascular health, however, disparities in participation exist across sociocultural groups, which in turn affect cardiovascular outcomes. Evidence suggests that although the positive cardiovascular effects of exercise are consistent across populations, notable differences in the magnitude of these benefits exist for racial and ethnic minorities and the female sex. Women derive greater protection from PA compared with men, with reduced rates of sudden cardiac death. In this review we examine the complex interplay of race and/or ethnicity and sex on the cardiovascular benefits associated with PA and exercise, cardiovascular adaptations to exercise, risks of sudden cardiac death, and "excessive" volume of exercise. Understanding these factors is crucial for developing targeted interventions to promote cardiovascular health and offset disparities.

RÉSUMÉ

L'activité physique est considérée comme la pierre angulaire de la santé cardiovasculaire, mais il existe des disparités dans la participation entre les groupes socioculturels et celles-ci influent sur les résultats cardiovasculaires. Les données indiquent que si les effets bénéfiques de l'activité physique sur la santé cardiovasculaire sont uniformes dans toutes les populations, il existe des différences notables dans l'ampleur de ces bienfaits dans le cas des minorités raciales et ethniques et des femmes. L'activité physique confère une plus grande protection aux femmes qu'aux hommes, comme en témoigne la réduction des taux de mort subite d'origine cardiaque. Dans cet article, nous nous intéressons à l'interaction complexe entre la race et/ou l'origine ethnique et le sexe et les bienfaits cardiovasculaires associés à l'activité physique et à l'exercice, le phénomène d'adaptation cardiovasculaire à l'exercice, les risques de mort subite d'origine cardiaque et le degré « excessif » d'exercice. Il est impératif de comprendre ces facteurs pour concevoir des interventions ciblées, promouvoir la santé cardiovasculaire et aplanir les disparités.

The effect of physical activity (PA) on cardiovascular health is well established, with known cardioprotective effects, including risk modulation by improving blood pressure (BP) control, lipid profile, and insulin sensitivity.[1-3] This is reinforced by the fact that inactivity poses a threat to well-being, particularly in individuals with chronic, long-term health conditions.[4] Distinguishing PA from exercise is required to understand the effect of various modalities on the cardiovascular system. PA is defined as energy expenditure resulting from bodily movements produced by skeletal muscles, broadly incorporating activities relating to functional activities of daily living, household chores, one's occupation, recreational and competitive sport, and other incidental activities.[5] Comparatively, exercise refers to planned, structured, and repetitive activity, precisely prescribed using the FITT principles (frequency, intensity, time, type) with the goal of maintaining or enhancing specific components of an individual's physical fitness or sporting performance.[5] Exercise is generally classified on the basis of the predominant energy system and metabolic pathway involved, namely aerobic (endurance) or anaerobic, or on the basis of muscle contraction type in the case of resistance training (static or isometric or dynamic or isotonic).[6]

Exercise provides irrefutable benefits to an individual's health and well-being via established physiological effects and

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adaptations, in proportion to the volume, intensity, and modality that is undertaken. Exercise specifically plays a key role in the primary and secondary prevention of cardiovascular disease (CVD), including tailored prescription for treatment of specific conditions, in addition to reducing the burden of cardiovascular risk factors (RF) and associated metabolic conditions, as outlined in international guidelines.[2,6-9] Despite the extensive benefits, exercise is not without risk, such as sudden cardiac death (SCD) in individuals with and without established cardiac conditions, as well as in the context of excessive volume and intensity in the long-term.[6,10,11] Understanding factors that influence the benefits and risks of exercise is imperative, particularly in the era of precision medicine.

The social determinants of health (SDOH) as defined by the World Health Organization, encompass nonmedical elements pertaining to economic, social, environmental, and psychological factors that affect an individual's state of wellness or disease.[12-18] This incorporates any social situation or circumstance within which people are born, grow up, live (including exercise participation), learn, work, play, worship, and age.[12-18] Such social constructs are not only of importance on a sociocultural level, but are also highly individualized and dynamic, thus limiting generalizability. Understanding the effect of social processes via established frameworks, such as stigmatization, discrimination, and marginalization, is important in identifying vulnerable populations with specific needs who are prone to facing the extremes of disparities resulting from complex relationships between SDOH and specific outcomes of interest.[17,18] Race, ethnicity, and sex are among the SDOH that are more commonly encountered in the literature, including their effect on cardiovascular health.[12-18] They too are linked to associated SDOH, such as income and education, which must also be considered. This article is focused on addressing the question of whether race, ethnicity, and sex influence the positive and negative cardiovascular effects of PA and exercise.

The Sociocultural Influence of SDOH on Exercise

Distinguishing race, ethnicity, and sex in biomedicine

In biomedical research a social constructivist approach can be taken to describe race and/or ethnicity, traditionally referring to ancestry and physical characteristics, compared with modern conceptualization, which integrates self-identification and social affiliation, which are borne of sociopolitical processes.[12,19,20] One important limitation identified in the literature is the absence of providing a definition of each term and inconsistent application across numerous studies, which reflects the dynamic nature of the terms and fluidity of the boundary separating them.[20,21] Research in the PA and exercise realm has used the terms race and ethnicity interchangeably, using ethnicity to refer to specific racial groups, or combined the terms to encompass racial groups and relevant ethnic groups as a collective in SDOH.[22-24]

Race describes a group of people typified by shared physical traits on the basis of geographic origin. This has been well demonstrated across numerous studies, which use the key racial categories, such as those outlined by the Canadian Cardiovascular Society definition of ethnic groups, which include Aboriginal/Indigenous, Black, East-Southeast Asian, South Asian, Hispanic, Middle Eastern, White, and mixed.[25] Ethnicity is a broader construct, which is group-defined from a social perspective, and incorporates cultural tradition, history, values, religion, and shared genetic heritage.[20,21] Contrary to these definitions, a recent review implored that race itself be recognized as a sociopolitical construct that represents an important aspect of SDOH that must be recognized to overcome potential harm to implicated individuals, and shift toward race-conscious practice.[26] The authors share the same sentiment; therefore, this review is focused on distinguishing race from ethnicity using the aforementioned definitions, other than when studies have used the terms interchangeably or when specific cultural aspects pertaining to ethnicity influence the discourse. An important limitation is that not all racial and ethnic groups are equally represented in most studies, nor have all groups been studied in detail. The increasing racial, and thus ethnic, diversity for a given population reinforces the importance of understanding specific needs of respective groups and the insight population-based studies continue to provide. Accurate description of racial and ethnic groups is also important, which in part is limited by historical terminology used in previous studies. Nonetheless, terms applied in specific articles have been reported on in the same manner to best understand the population examined.

Sex is distinct from gender, defined on the basis of one's biologic make-up as determined by chromosomal and phenotypic traits, compared with an individual's social and behavioural expression of their sex.[12,16] Similarly to race and/or ethnicity, sex and gender are often used interchangeably in some aspects of biomedical research, which is often incorrectly conceptualized as a binary factor (ie, female or male) rather than incorporating the spectrum of gender identities and expressions that exist.[27] Although contemporary gender differences pose unique challenges from a social perspective, for example the gender role of primary caregiver predominantly taken up by women and associated stressors, this review is focused on the biological differences between female and male individuals. Gender is referred to for this same distinction solely for common concepts that have been previously defined as such in the literature. Because most of the work in this sphere has been undertaken in male individuals, a greater emphasis has been placed on distinguishing unique differences in female individuals.

Disparities related to participation in exercise and PA

Disparities in exposure to PA and exercise across sociocultural groups, each with different contributing factors, is likely to negatively affect cardiovascular outcomes. On a population level, participation in exercise as per national PA guidelines has been documented to be as low as approximately 24% in Australia[28] and the United States,[29-32] compared with almost 50% in Canada[33] and 63% in the United Kingdom (UK).[34] A consistent trend among all of the populations evaluated, is that female individuals were reported to have lower rates of participation compared with male individuals, with an all-inclusive group named "other gender" noted to have the lowest overall participation in the UK.[34] Of the reports focused on differences on the basis of race and/or ethnicity, individuals from ethnic minorities were reported to be less active than their White counterparts; noting again that female individuals from ethnic minorities were less active than male individuals.[30,34]

Disparities associated with exercise and the primary prevention of CVD

Participation in PA and exercise is greatly influenced by factors linked to broader SDOH of interest, which in turn affect cardiovascular outcomes (Fig. 1). These incorporate aspects of one's lived experience, including everyday discrimination in the form of racism and sexism, neighbourhood, environment perception, health literacy, social needs, stigma, and implicit bias.[13,16-18,35,36]

Racial and ethnic factors. Despite their increased cardiovascular risk and higher body mass index (BMI), indicating a greater need to prioritize PA, racial and/or ethnic minorities are less likely to engage in healthy lifestyle behaviours.[37] Ali et al.[1] outlined that the epidemic of physical inactivity in this cohort is influenced by cultural, socioecological, and sociocultural drivers. Their review identified important barriers, including cultural expectations, areas of deprivation being unsuitable environments for PA, and poor engagement with health professionals.[1] Morris et al.[37] identified education, BMI, and weight perception to be important factors associated with engaging in PA across White, Black, and Hispanic participants, whereas the effect of income was variable. Additional barriers identified include reduced availability of appropriate commercial facilities in low-income neighbourhoods, perception of safety, financial hardship, income, perception of social class, immigration status, lack of time, lack of familiarity, unease in taking the first step, language, racism, and sex.[22,37-39]

Sex-specific factors. Cardiovascular outcomes in female individuals are influenced by unique SDOH, including race and/or ethnicity, racism and discrimination, rurality, postal code, education, social support, communication barriers, cultural norms, and sexual orientation.[40,41] Cardiovascular RF unique to women also need to be considered, such as pregnancy-related factors including gestational hypertension, gestational diabetes, and preeclampsia.[40] The concept of a "gender gap," referring specifically to reduced levels of engagement with PA on the basis of female sex, is well established.[29] This has been shown to occur across the life span, with male participation exceeding that of women from the early years throughout adulthood.[23,29,42,43] Recent epidemiological data reinforces that this issue persists. Additional distinct barriers to account for this include race, income, safe exercise locations, increased stress, and reduced time associated with working multiple jobs and childcare responsibilities in individuals with low incomes.[22,40]

Cardiovascular Benefits of Exercise

The positive effects of exercise on cardiovascular health and outcomes are dose-dependent, with higher volumes proffering greater benefits.[6,11,44,45] International guidelines outline the minimum requirements to achieve cardiovascular health benefits, including primary and secondary prevention.[2,6-9,32] Recommendations include aerobic exercise of moderate intensity for 150 minutes per week, or vigorous intensity for 75 minutes per week.[2,6-9,32] Metabolic equivalents of task (METs), a measurement of energy expenditure on the basis of oxygen consumption, is used to grade exercise intensity. For example, slow walking is a mild-intensity activity equivalent to < 3 METs, compared with running at > 9 kilometres per hour, which is a vigorous activity equivalent to ≥ 6 METs.[6,32] The addition of at least 2 days of resistance training propagates these benefits because of skeletal muscle adaptations that lead to metabolic benefits, which improve one's cardiovascular risk profile.[6,7,29,32]

These guidelines summarize a generalized approach, which does not reflect specific needs on the basis of race and/or ethnicity, or sex; thus, limiting our understanding of how these SDOH affect benefits on cardiovascular health. A significant proportion of the literature on effects of exercise on the cardiovascular system, specifically focused on cardiovascular outcomes including mortality and major adverse cardiovascular events, incorporate PA levels in their findings. This in turn enables a range of activity levels to be distinguished from sedentary status. As such, this article will include the effect of PA on cardiovascular outcomes. Much of the research focused on cardiovascular adaptations to exercise is on the basis of cross-sectional comparisons, as shown in a recent meta-analysis.[46] Data on longitudinal outcomes would enable further insight, thus, the studies presented need to be interpreted in the context of this limitation.

Cardiovascular outcomes

Influence of race and/or ethnicity on all-cause mortality and cardiovascular outcomes. CVD and risk profiles vary according to race and/or ethnicity within a given population. Although not all groups are represented in each study, important disparities have been identified. A Canadian study that evaluated 371,154 individuals from 3 cross-sectional cycles, which adjusted for sociodemographic characteristics, showed that individuals from the most apparent minorities had lower levels of PA engagement and would also benefit from prioritizing detection and control of diabetes mellitus and hypertension.[47]

In the **M**ulti-**E**thnic **S**tudy of **A**therosclerosis (MESA) study, 6814 Black, White, Hispanic, and Chinese adults in the United States were recruited, and followed for approximately 15 years.[48] All-cause mortality was 34% higher for Black participants and 21% lower for Chinese participants compared with White participants, whereas there was no difference for Hispanic participants.[48] After adjusting for socioeconomic status, the excess mortality risk for Black compared with White participants reduced to 16%.

After further adjusting for additional RF and immigration history, Hispanic participants had a lower mortality risk than White participants, and Black and Chinese participants had risk similar to that in White participants. Cardiovascular mortality shared similar trends, however, adjusting for sex and age resulted in even greater risk (72%) for Black compared with White participants.[48]

PA remains a key behavioural strategy for reducing cardiovascular risk in diverse and underserved racial and

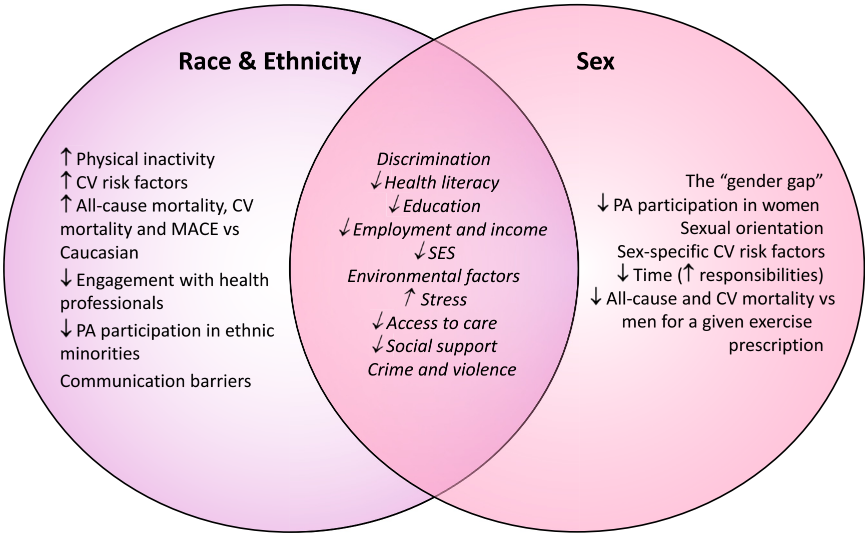

Figure 1. Sociocultural and physiological factors that influence participation and cardiovascular (CV) outcomes unique to race and/or ethnicity and sex; and factors that are common to both social determinants of health (in **italic**). Discrimination encompasses racism and sexism, bias (implicit and explicit), and victimization. Environmental factors include housing instability, homelessness, postal code, and areas of deprivation limiting suitability and safety for participation in physical activity (PA). CV risk factors specific to women include pregnancy-related considerations (preeclampsia, gestational hypertension, gestational diabetes, preterm delivery, delivery of small for gestation age infant, recurrent spontaneous miscarriage), polycystic ovarian syndrome, early menarche, premature menopause, hormone-based contraception, autoimmune disorders, chronic inflammatory conditions, and depression and other mental health comorbidities that accompany increased levels of stress. ↑, increased; ↓, decreased; MACE, major adverse cardiovascular events; SES, socioeconomic status.

ethnic groups, despite disparities in participation.[49] Mathieu et al.[50] investigated the effect of PA participation and health perceptions on cardiovascular mortality in 3018 African American, Hispanic, and White adults recruited as part of the Dallas Heart Study. African American and Hispanic participants were less likely to be active, despite accounting for income, education, sex, BMI, and other cardiovascular RF; however, beliefs surrounding the benefits of PA was not a contributing factor.[50] The authors proposed other factors established in the literature might be accountable, including the safety and quality of surrounding facilities, perceptions toward gender roles and exercise, and different peer support requirements.[50]

Disparities related to race have also been shown by rates of initiation, adherence, and completion of cardiac rehabilitation (CR). These disparities are also likely to influence disparities in cardiovascular mortality. Despite findings from a recent review, which indicated that both sexes elicit a similar physiological response to CR,[51] women have been shown to face greater barriers to participation in a cohort comprising 16 countries from all 6 World Health Organization regions, despite similarities between sexes.[52] A study involving 822 patients, composed of 52% non-White minority individuals consecutively referred to CR, showed that non-White individuals were 78% less likely to initiate CR than White participants. Reductions in mortality were shown for individuals who participated.[53]

Bell et al.[54] examined the effect of PA on CVD in 3707 African American and 10,018 Caucasian individuals, recruited as part of the (**A**therosclerosis **R**isk **i**n **C**ommunities (ARIC) study, who were followed for 21 years. An inverse relationship was reported for CVD, heart failure, and coronary artery disease (CAD) in both races, and with stroke in African American individuals, after adjustment for confounders.[54] Hazard ratio patterns correlated with PA levels for race, which indicated that regular PA reduces the risk of CVD in African American and Caucasian individuals.[54]

Vásquez et al.[55] studied the association between varying levels of PA and all-cause and cardiovascular mortality across different races and/or ethnicities in older adults. In their study 2520 female and 2398 male individuals aged 60 years or older were recruited and analyzed as part of the National Health and Nutrition Examination Survey III, and categorized as inactive (no PA), active (3-6 METs $\geq$ 5 times per week or $<$ 6 METs $\geq$ 3 times per week), or insufficiently active (not meeting either criterion). Overall, any level of PA was associated with a lower all-cause mortality across all racial and/or ethnic groups. Inactive non-Hispanic Black and Mexican American individuals had a reduced risk of all-cause mortality vs non-Hispanic White individuals, whereas insufficiently active Mexican American individuals were reported to have lower all-cause mortality vs non-Hispanic White individuals.[55]

Influence of sex on all-cause mortality and cardiovascular outcomes. Data on sex differences in cardiovascular RF and outcomes associated with exercise are limited. Sex is often a confounder, which is accounted for as part of study design. Some studies have selectively evaluated findings in a particular sex among a mixed cohort or have only recruited a single sex. This makes it difficult to draw generalized conclusions applicable at a population level. PA has been previously shown to have similar favourable effects on traditional cardiovascular RF, with no substantiative differences identified between female and male participants.[45] Sex-specific differences pertaining to PA and cardiovascular outcomes have been explored independently in most instances, with a focus placed on all-cause and cardiovascular mortality, dose-response, and minimum PA required to achieve these benefits.

One study of male participants, which used 2 medical examinations, comprising a maximal exercise test and health evaluation with a mean interval of 4.9 years, reported that participants who maintained or improved their fitness between assessments had reduced all-cause and cardiovascular mortality vs unfit participants.[56] Mortality risk decreased by 7.9% per minute increase in exercise time between initial and repeat evaluations.[56] Exercise capacity achieved during a maximal exercise test was also shown to be a more significant predictor of mortality in men compared with other established cardiovascular RF.[57] Survival increased by 12% for every 1 of METs increase in exercise capacity.[57]

Kokkinos et al.[58] studied the association between cardiorespiratory fitness and mortality in a cohort of 750,302 US veterans aged 30-95 years, and based their analysis on sex, age, and race. Male and female participants were shown to have the lowest risk in individuals who achieved 14.0 METs during a standardized exercise treadmill test, with no further benefit reported at the extremes of fitness.[58] Comparatively, individuals with the lowest fitness levels were shown to have a fourfold greater risk of mortality compared with extremely fit individuals, however, a 50% reduction in risk could be achieved by most individuals with moderate fitness relative to their age category.[58]

Similarly, PA has been shown to act as an independent predictor of all-cause mortality using the Framingham Risk Score-adjusted mortality risk measurement in asymptomatic female individuals, leading to a 17% reduction in mortality for every 1 of METs increase in exercise capacity.[59] Gulati et al.[60] showed predicted exercise capacity, determined using the Bruce protocol treadmill exercise stress test, should be readjusted with lowered values for asymptomatic and symptomatic female compared to male individuals with reference to all-cause and cardiovascular mortality. In this study exercise stress test data were used to determine a sex-specific nomogram in female participants across different age groups, and reported that predicted exercise capacity of $\geq$ 85%, determined using the "predicted METs" formula, halved the risk of death.[60]

Dose-response was explored by a prospective study involving 97,230 women aged 27-44 years at its inception, who were followed over 20 years.[61] Of the parameters evaluated, including frequency, type, and volume, total volume of exercise equating to $\geq$ 30 METs hours per week had a significantly reduced risk of CAD compared with those who were the least active, completing $<$ 1 METs hours per week.[61]

Similarities between sexes has been shown in a Taiwanese cohort aimed to establish the minimum amount of exercise required to reduce all-cause mortality. The results from a self-administered questionnaire for individuals at risk of developing CVD were evaluated.[62] It was reported that a 14% risk reduction could be achieved with either 15 min/d or 90 minutes per week of moderate-intensity exercise in female and male participants.[62] Sattelmair et al.[63] performed a meta-analysis, comprising 33 studies, and investigated the dose-response relationship between PA and the risk of CAD. Individuals who participated in 150 minutes per week of moderate-intensity PA achieved a 14% risk reduction compared with sedentary individuals, which increased to 20% for those who completed 300 minutes per week at the same intensity.[63] A significant sex interaction was identified, whereby this risk was twofold lower in women.[63]

Ji et al.[29] recently conducted a prospective study of 412,413 adults in the United States, of whom 55% were female with a mean age of 44 years, involving survey data on leisure-time PA. Sex-specific multivariable-adjusted associations were undertaken for measurements of PA with cardiovascular and all-cause mortality reported between 1997 and 2019.[29] Race and/or ethnicity was classified in the cohort characteristics, with no significant difference identified between men and women on the basis of this specific SDOH.[29] There were 11,670 cardiovascular deaths among the 39,935 deaths observed during follow-up. Regular PA compared with inactivity resulted in a 24% vs 15% lower all-cause mortality in women compared with men, respectively.[29]

Maximal survival benefit was reached at 300 minutes per week of moderate-to-vigorous exercise in men, whereas women not only achieved a similar benefit at less than half this volume (ie, 140 minutes per week) but these positive effects continued up to a maximum survival benefit achieved at 300 minutes per week. Similar sex-specific findings were observed for cardiovascular death, which was consistent across all measurements of aerobic activity and strength training. An age interaction was identified, with benefits being more pronounced for middle-aged women between 40 and 59 years of age, compared with older women in whom benefits were mitigated.[29] These provocative results indicate that women stand to benefit more than men for a given exercise prescription, across multiple measures of PA.[29] The authors have suggested that overcoming the "gender gap" might be achieved by shifting a focus on the engagement of women with exercise, opposed to matching exercise volume with men.[29]

The protection PA provides in relation to major adverse cardiovascular events has been shown to be reasonably well balanced between the sexes, with some sex-specific findings identified. Fransson et al.[64] studied the effect of aerobic exercise and PA and acute myocardial infarction, and reported a greater degree of protection in women with reduced relative risk shown for the most vs least PA and frequency of 3 times per week vs seldom.[64] Manson et al.[65] prospectively investigated the role of walking vs vigorous exercise in 73,743 postmenopausal women aged 50-79 years as part of the Women's Health Initiative study. Significant reductions in coronary events and a composite of total cardiovascular events were identified with increasing levels of PA, independent of intensity, which was similar according to race, age, and BMI.[65]

The inverse relationship between PA and stroke risk is similar for women and men. A Norwegian cohort of 34,868 women and 32,872 men were followed for 16 years, and cause-specific mortality relative to PA was documented.[66] The P trend for total activity and stroke mortality was $<$ 0.001 for women and 0.009 for men, with a significant relative risk reduction in both sexes for high levels of PA vs inactive (ie, "never active") status, namely a relative risk of 1.45 (95% confidence interval [CI], 1.14-1.83) for inactive women (vs 1.00 reference for highly active women) and an relative risk of 1.35 (95% CI, 1.05-1.74) for inactive men (vs 1.00 reference for highly active women). A Japanese study that recruited 42,242 women and 31,023 men, who were followed over a

Table 1. Electrical and structural cardiovascular adaptations in athletes on the basis of race and/or ethnicity

Race and/or ethnicity	Electrical remodelling	Structural remodelling
Caucasian (White)	• Sinus bradycardia • First-degree and Mobitz type 1 second degree AV block • ↑ QRS voltage • Incomplete RBBB • Early repolarization	• ↑ LV wall thickness • ↑ LV cavity dimensions • LV eccentric vs concentric hypertrophy • Atrial dilatation • Aortic root dilatation
African/Afro-Caribbean (Black)	• TWI anterior leads V_1-V_4 • TWI lateral leads • Early repolarization in inferior leads • RVH • Left atrial enlargement	• ↑ LV wall thickness • ↑ RV and RVOT dimensions • LV trabeculation
West Asian (Arabic and Middle Eastern)	• ↓ Prevalence of ECG changes vs Caucasian individuals • ST elevation with upward convexity and TWI in V_1-V_4	• ↑ in LV wall thickness • ↑ LV cavity dimensions (↓ BSA)
East Asian and South Asian	• ECG changes more common in Chinese vs South Asians • ↑ Prevalence of convex ST elevation and biphasic or inverted T waves in Japanese individuals	• ↑ in LV wall thickness • ↑ LV cavity dimensions (in Japanese individuals)
Mixed race	• ↑ TWI • ↑ Left axis deviation • ↑ LV hypertrophy • Atrial dilatation	• ↑ LV wall thickness
Pacific Islanders	• ↑ TWI	• ↑ LV mass • ↑ Relative wall thickness

↑, increased; ↓, decreased; AV, atrioventricular; BSA, body surface area; ECG, electrocardiogram; LV, left ventricular; RBBB, right bundle branch block; RV, right ventricular; RVH, right ventricular hypertrophy; RVOT, right ventricular outflow tract; TWI, T-wave inversion.

mean of 9.7 years, showed walking and sports participation were inversely related to cardiovascular mortality, all stroke, and ischemic stroke mortality.[67]

A US cohort partaking in the National Health and Nutrition Examination Survey Epidemiologic Follow-up Study did not show a significant relationship between PA and total or ischemic stroke in both sexes in the younger age group of 45-64 years compared with the 65-74 age year group, however, the study did show a strong association between PA and stroke for White women.[68] Sex-specific findings were also shown in the Nurses' Health Study, in which PA levels were analyzed using METs in hours per week in 72,488 female nurses aged 40-65 years.[69] During 8 years of follow-up, increasing PA levels lead to a strong inverse association with the risk of total stroke, with a considerable reduction in in total stroke and ischemic stroke even with lower levels of PA, such as moderate-intensity brisk walking, in a dose-dependent manner.

Cardiovascular adaptation

Race and/or ethnicity and cardiovascular adaptations to exercise. Regular exercise can result in significant electrical and structural cardiovascular changes, identified on the 12-lead electrocardiogram (ECG) and various imaging modalities. Although many studies on cardiac remodelling in athletes focus on adult, male, White athletes who participate in common sports, evidence suggests sporting discipline, age, sex, and race and/or ethnicity are important determinants of cardiovascular adaptations to exercise.[23,24,70] Growing interest and subsequent experience through preparticipation screening of an increasingly diverse population of athletes over the past 3 decades has significantly enhanced our understanding in this area. Table 1 shows a summary of electrical and structural adaptations observed across the racial and/or ethnic cohorts evaluated in the literature. Figure 2 shows examples of anterior T-wave inversion (TWI) on the 12-lead ECG, as influenced by race and/or ethnicity, sex, one's chosen sport, and age. African and Afro-Caribbean (Black) athletes represent the most distinct paradigm of cardiovascular adaptation, because they frequently demonstrate repolarization anomalies and left ventricular (LV) hypertrophy (LVH) compared with White athletes, making the differentiation between athlete's heart and cardiac conditions implicated in exercise-related SCD more challenging.[23,24,70,71]

These distinct adaptations observed in this population are labelled "Black athlete's heart" in the sports cardiology literature, thus, have been referred to as such in this section of the review. The discourse in recent work by Krishnan et al.[26] is an important one striving for equity and offsetting structural racism within the subspecialty, which has the protentional to drive a change in nomenclature of this phenotypic variation observed in "athlete's heart."

Electrical remodelling in Black athletes. Repolarization changes are more common in Black vs White athletes.[23,24,70,71] Papadakis et al.[72] reported on a cohort of 2842 individuals who underwent cardiac screening, comprised of 904 Black athletes, 1819 White athletes, and 119 Black control participants. Ethnicity was determined through self-reported questionnaires. The authors identified TWI in 22.8% of Black athletes compared with 3.7% in White athletes and 10.1% of Black control participants. A significant proportion of TWI in athletes (55%) were isolated to contiguous anterior leads (V_1-V_4) and were associated with J point elevation and convex ST-segment elevation. This pattern was not associated with disease in short-term follow-up. In contrast, lateral TWI (I, aVL, V_5, V_6), which was present in 4.1% of Black and 0.3% of White athletes, was associated with a diagnosis of hypertrophic cardiomyopathy (HCM) in a small number of athletes (n = 3) during subsequent follow-up, and was also the predominant TWI pattern in a cohort of 52 Black patients with HCM. Although the authors reported that the pattern of TWI in V_1-V_4 with associated J point elevation and convex ST-segment elevation

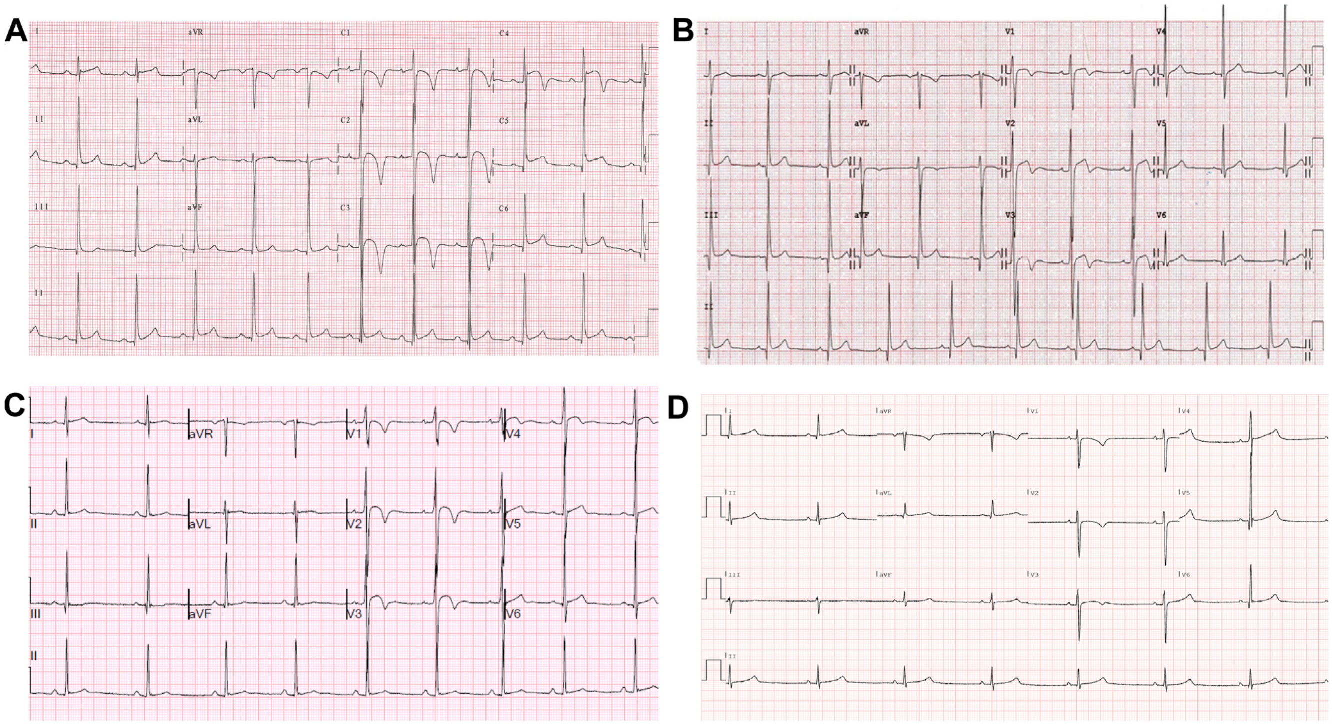

Figure 2. Examples of common causes of anterior T-wave inversion encountered during preparticipation screening, including Black (**A**), "juvenile" (**B**), endurance (**C**), and White female (**D**) athletes.

represented an ethnic variant of athlete's heart ("Black athlete's heart"), subsequent studies revealed that the same pattern can be present in athletes of other ethnicities and highly prevalent in adolescent endurance athletes.[24,73-77] A study by Riding et al.[78] further highlighted the challenges of using skin colour to attribute a particular characteristic, because they showed significant variation in the prevalence of TWI in Black athletes of different geographical origins ranging from 0 to 8.5%.

Other ECG changes identified include an increased prevalence of early repolarization in the inferior leads, which can be associated with an increased risk of SCD, right ventricular (RV) hypertrophy, and atrial enlargement.[24,72,79,80] Although these ECG changes have been observed to have a greater prevalence in this cohort, the literature indicates these findings are not exclusive to Black athletes. Thus, in time the focus of interpretation might shift toward understanding the broader spectrum of ECG findings specific to athletic adaptation relevant to individuals rather than considering them to be purely specific to racial and/or ethnic groups.

Structural remodelling in Black athletes. LV and RV cavity volumes are similar to those of White athletes.[23,24,81,82] Increased LV wall thickness (LVWT) is the most prominent adaptation in Black athletes, with 12.4% of male Black athletes shown to have an LVWT measuring between 13 and 16 mm vs 1.6% of White athletes.[81-83] Basavarajaiah et al.[81] evaluated echocardiogram data from 300 national Black male athletes to delineate LV remodelling from HCM, with findings compared with 150 Black and White sedentary individuals and 300 highly trained White athletes. Black athletes had greater LVWT vs White athletes (11.3 $\pm$ 1.6 mm vs 10 $\pm$ 1.5 mm; $P < 0.001$), of whom 18% of Black athletes had LVWT > 12 mm vs 4% of White athletes, and 3% of Black athletes had an LVWT $\geq$ 15 mm.[81] Consequently, Black athletes exhibit higher relative wall thickness and more frequent rates of concentric remodelling compared with White athletes.[84] This further highlights the challenges a physician might face when assessing athletes of Black ethnicity and the potential to falsely attribute disease or conversely offer false reassurance.

Rawlins et al.[83] studied 240 nationally ranked Black adolescent female athletes, who were shown to have greater LVWT compared with White female athletes (9.2 $\pm$ 1.2 mm vs 8.6 $\pm$ 1.2 mm; $P < 0.001$). Among these individuals, 3% of Black athletes were shown to have an LVWT > 11 mm (up to 13 mm) compared with none of the White athletes.[83] Sheikh et al.[76] showed similar adaptations in a group of adolescent Black athletes, consisting of 245 male and 84 female individuals, of whom 7% were identified to have an LVWT between 13 and 15 mm compared with 0.6% of White athletes. Di Paolo et al.[77] evaluated 154 soccer players who participated in the 2009 under 17 African Championship, and compared them with 62 Italian players, and reported that LVWT was 5% greater in the African vs Caucasian cohorts; of whom 4 African players had an LVWT exceeding 13 mm.

LV trabeculations are more prevalent in Black athletes compared with White athletes (28.8% vs 16.3%; $P = 0.002$) and control participants, posing a diagnostic challenge in relation to distinguishing this adaptation from a cardiomyopathy phenotype.[85]

Sex and cardiovascular adaptations to exercise. Female athletes typically exhibit less pronounced adaptations compared with male athletes, however, the quantitative changes compared with sedentary female individuals are

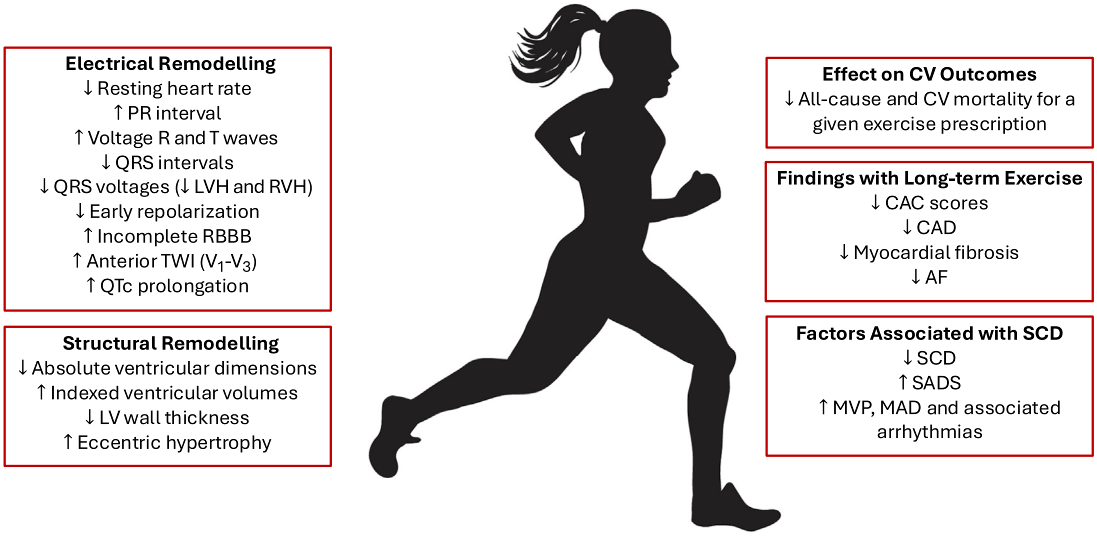

Figure 3. Sex-specific exercise-based considerations for females compared to males. ↑, increased; ↓, decreased; AF, atrial fibrillation; CAC, coronary artery calcium; CAD, coronary artery disease; CV, cardiovascular; LV, left ventricular; LVH, left ventricular hypertrophy; MAD, mitral annular disjunction; MVP, mitral valve prolapse; RBBB, right bundle branch block; RVH, right ventricular hypertrophy; SADS, sudden arrhythmic death syndrome; SCD, sudden cardiac death; TWI, T-wave inversion; QTc, heart-rate corrected QT interval.

proportionate to similar comparisons made between athletic and sedentary male participants.[71] These sex-specific differences (Fig 3) have been described to occur from early adolescence, with remodelling beyond pediatric reference values for both sexes seen more frequently for male individuals from the midadolescent years.[86]

Despite similarities in cardiac physiological adaptation in both sexes, female individuals have less lean body mass, a different hormonal profile, a lower sympathetic adrenergic response to exercise, peak systolic BP, stroke volume, and peak oxygen consumption, and are anthropometrically smaller.[87,88] Under-representation of female participants in research has limited our understanding of why these sex differences in adaptations and outcomes exist, in addition to whether the differences that occur later in life result from biological mechanisms or reduced access to sports across the lifespan in female individuals.[89,90]

Electrical remodelling in female athletes. ECG abnormalities have consistently been identified at a higher prevalence rate in male individuals.[71,73,87,89-97] Despite this, unique observations pertaining to ECG findings in female individuals have been made. These include: lower resting heart rate, longer PR interval, higher voltage R and T waves, shorter QRS intervals, and lower QRS voltages, which are less likely to meet LVH and RV hypertrophy criteria, reduced early repolarization, more frequent prevalence of incomplete right bundle branch block, anterior TWI, and greater QT interval corrected for heart rate prolongation.[71,73,87,89-97] Considerations that might explain some of these findings include differences in cardiac mass, chest size, breast tissue, electrical conductance with respect to anterior TWI, and hormonal factors resulting in QT prolongation.[71,73,87,89-97] Sporting discipline was not shown to affect ECG changes in female individuals in a recent review.[98]

Structural remodelling in female athletes. LV end diastolic volume $>$ 54 mm and LVWT are greater in female athletes vs control participants, with LVWT consistently shown to not exceed 11 mm, and reduced structural remodelling compared with male participants.[71,83,87,90,98-100] Indexed to body surface area, a dilated LV cavity (LV end diastolic volume $\geq$ 31 mm/m^2) is more prevalent in female athletes.[82,98,101] Female athletes tend to adapt with eccentric hypertrophy more frequently than male athletes in the setting of dynamic sport, therefore concentric remodelling should raise suspicion of cardiac pathology rather than physiology.[99]

Changes to cavity dimension in female individuals has been shown to involve both ventricles, understanding the right ventricle is more sensitive to volume loading.[87,98] Although overall RV dimensions are larger in men, similar to the left ventricle, indexed volumes are generally higher in female athletes.[87,102,103]

Cardiovascular Risks of Exercise

Sudden cardiac death

From an epidemiological perspective, SCD in young athletic populations is dependent on multiple factors, including age, sex, race and/or ethnicity, and sporting discipline.[6,10,11,104-106] SCD has recently been highlighted as an important racial disparity in sports cardiology.[26,107] National Collegiate Athletic Association (NCAA) data from the United States has provided important insights, showing a strong link between race and/or ethnicity and SCD. Black athletes have consistently been shown to experience a threefold higher incidence of SCD, with reduced rates of survival post cardiac arrest despite a similar incidence of inherited cardiac conditions, vs White athletes.[6,10,11,26,101,104-106,107-112]

Petek et al.[106] recently conducted a retrospective analysis of 143 SCD cases among 1102 total deaths in NCAA athletes between 2002 and 2022, and identified a greater incidence of SCD in Black (1:26,704 athlete-years) compared with White

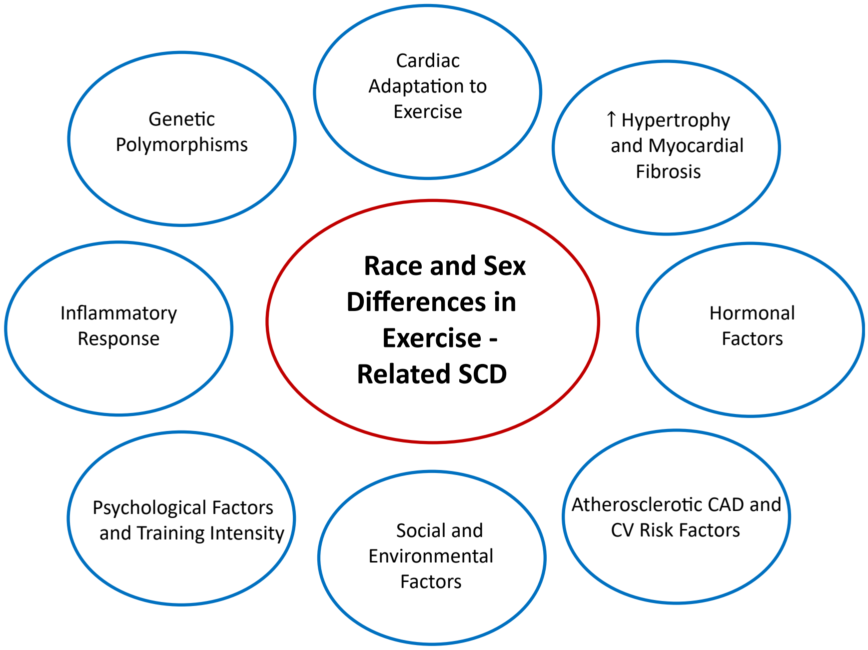

Figure 4. Factors contributing to differences in exercise associated sudden cardiac death based on sex and race. ↑, increased; CAD, coronary artery disease; CV, cardiovascular; SCD, sudden cardiac death.

(1:74,581 athlete-years) athletes. Peterson et al.[101] undertook a prospective analysis from 2014 to 2018 via the National Center for Catastrophic Sports Injury Research, using data from high school associations and the NCAA, which specifically showed African American male division I NCAA basketballers had the highest incidence of death (1:2087 athlete-years). Harmon et al.[108] reviewed deaths identified from an NCAA database between 2003 and 2013, similarly identifying Black athletes (1:21,491 athlete-years) were at higher risk of SCD than White athletes (1:68,354 athlete-years). These more recent studies follow the same trend identified in earlier work, which showed a disproportionately greater risk of SCD in Black athletes, quoted to be fivefold greater, inclusive of specified causes in some instances, including HCM and congenital coronary anomalies.[109-111]

Finocchiaro et al.[104] investigated the etiology of SCD in a UK cohort of 7880 cases referred to their national cardiac pathology centre for detailed autopsy by cardiac experts between 1994 and 2022, of whom 848 (11%) were athletes, with clinical data provided by the referring coroner. A total of 758 athletes were White (89%), 51 were Black (6%), and 39 were Asian (5%), with death occurring during exercise in 737 (87%) cases.[111] The cause of death in descending order of prevalence was sudden arrhythmic death syndrome (SADS) in 385 (45%) cases, myocardial disease CAD in 58 (7%) cases, and coronary artery anomalies in 29 (3%) cases.[104] Arrhythmogenic cardiomyopathy was a more prevalent cause of death in Black (25%) vs White (14%; $P = 0.03$) and Asian athletes (8%; $P = 0.04$). Moreover, the death was attributed to CAD more commonly in Asian (15%) vs White (7%; $P = 0.03$) and Black athletes (2%; $P = 0.02$).

A sex-based disparity for SCD has consistently been shown, whereby male individuals exhibit a greater incidence.[10,88,101,105,107-111,113,114] Finocchiaro et al.[113] reported on data from a large UK registry, which included 748 cases of SCD in individuals who participated in more than 3 hours of PA per week. Female individuals constituted 13% (n = 98) of all SCD cases, among whom 41 (42%) were competitive athletes and 57 (58%) were recreational athletes.[113] Overall, a greater proportion of deaths was identified in male individuals, noting a significantly lower incidence rate of death during intense exertion in female individuals (58% vs 83%; $P < 0.001$).[113] SADS accounted for 57% of all deaths in female compared with 43% in male individuals, determined by a structurally normal heart on autopsy.[113] Deaths due to myocardial disease, including HCM, myocarditis, and idiopathic LVH, were less common in female individuals.[113] These findings showed consistency over time compared with an earlier study undertaken by the same group.[115]

Corrado et al.[114] undertook a prospective study over 21 years, which identified 300 cases of SCD in 1,386,600 adolescents and young adults, among whom 112,790 were competitive athletes. The incidence of SCD among athletes was 2.6 per 100,000 person-years in male compared with 1.1 per 100,000 person-years in female individuals.[114] Maron et al.[109] identified that 11% of deaths occurred in female athletes in their evaluation of 1049 cases of SCD in young athletes, equating to a male-to-female SCD risk ratio of approximately 10:1. A greater proportion of SCD in male individuals has been identified in studies that quoted rates of 81%,[108] 83%,[101,106] and 90%.[1] With regard to etiology, a female predominance has been shown for mitral valve prolapse (MVP), in addition to the aforementioned relationship established for SADS.[92,115,116] Basso et al.[116] identified 43 cases among 650 young adults included in a cardiac pathology registry who had MVP listed as the cause of SCD, of whom 61% were female. The authors concluded that MVP is an understated cause of SCD and the leading structural cardiovascular cause in female individuals.[116]

Factors contributing to racial and sex-based differences in SCD are summarized in Figure 4. Postulated explanations behind these include differences in cardiac adaptation to exercise, such as increased hypertrophy and associated myocardial fibrosis, which might act as a substrate risk of life-threatening arrhythmias; hormonal differences, for instance the protective effects of estrogen, which can also introduce the risk of arrhythmias via QT prolongation; differences in atherosclerotic CAD, genetic polymorphisms, and inflammatory response; and psychological factors, which can influence the intensity of training and engagement with competitive performance.[99,105,113,117] Social and environmental factors, including low socioeconomic status, income, and education, have also been implicated on a population level.[118-121] Zhao et al.[121] highlighted the effect these factors might have, including unhealthy behaviours, poor health literacy, and limited access to health care.

The paradox of excessive exercise

Athletes routinely exercise at volumes and intensities that far exceed recommendations for cardiovascular health, posing the risk of diminishing cardiovascular benefit and potential harm, as described by a reverse U-shaped relationship. Concerning physiological effects associated with sustained elevations in cardiac pressure and volume-loading include LVH, elevated coronary artery calcium (CAC) scores, acute cardiac biomarker release, myocardial fibrosis, and cardiac arrhythmias.[6,11,89,122-124] This typically applies to master/veteran endurance athletes, aged 35 years and older.

Most studies that evaluated CAC, myocardial fibrosis, and atrial fibrillation (AF) in this cohort have been limited to male participants.[125] Despite this, female sex might be protective considering the reduced prevalence of elevated CAC level, atherosclerosis, myocardial fibrosis, and arrhythmias; thought to be mediated via estrogenic mechanisms.[89,92,126-129] Merghani et al.[128] assessed 152 masters athletes, consisting of 70% male athletes, who were shown to have a higher prevalence of atherosclerotic plaques of any luminal irregularity compared with their sedentary counterparts; with predominantly calcific vs mixed morphology.[128] Late gadolinium enhancement was identified on cardiac magnetic resonance imaging in 15 athletes, all of whom were male, with 7 showing a pattern indicative of previous myocardial infarction; 3 of whom possessed a luminal stenosis of 50% or greater in the corresponding coronary artery.[128] No female athletes were observed to have clinically significant coronary artery stenoses.[128]

CAC scores in female athletes vs sedentary counterparts are known to be similar, suggestive of protective hormonal benefits independent of a lifetime of exercise.[92,106,128] Similarly, reduced rates of myocardial fibrosis might be explained by lower peak BP attained during exercise and lower levels of testosterone, which is known to be responsible for myocardial inflammation.[89,92,130] Although some studies have shown female athletes also appear to be protected from developing AF, recent evidence suggests their risk might actually be enhanced as similarly observed in male athletes.[129,131] Mohanty et al.[129] conducted an extensive literature review and meta-analysis, which showed reduced rates of AF, a benefit that appeared to be propagated by increasing exercise intensity; with women participating in moderate PA shown to have an 8.6% reduced risk of AF vs 28% lower risk in women who performed intense exercise.[129] Comparatively, men engaging in vigorous exercise were reported to have a significantly increased AF risk.[129] Drca et al.[132] retrospectively evaluated a cohort of 228 high-level Swedish female endurance athletes, and compared them with 1368 control participants. National registry data enabled identification of AF in 10 athletes (4%) and 23 control participants (< 2%), with a hazard ratio of 2.56 (95% CI, 1.22-5.37), indicating that elite female endurance athletes in this study had an increased risk of AF compared with the general population.[132]

Dose-response and cardiovascular outcomes, with respect to harm associated with high volumes of exercise in a general rather than athletic population, was studied by German et al.,[133] who evaluated CAC score to determine cardiovascular risk and cardiovascular outcomes in 6777 participants recruited as part of the MESA study. Individuals with low cardiovascular risk within the highest quartile of PA had a significant reduction in the hazard ratio for cardiovascular outcomes and all-cause mortality, whereas solely a reduction in all-cause mortality was shown in individuals with high cardiovascular risk within the highest quartile of PA.[132] High levels of PA specifically showed a reduction in all-cause mortality for high-risk Black and White participants, as well as low-risk and high-risk Hispanic participants, whereas there was no relationship identified for Chinese participants.[132] There was no increased risk associated with cardiovascular outcomes or all-cause mortality in any racial or ethnic group engaging in the highest levels of PA, independent of CAC score.[132]

Conclusion

In this review we investigated the effects of SDOH, namely race, ethnicity, and sex, on the cardiovascular benefits and risks of PA and exercise. Despite consistent evidence to support the positive effects of exercise on cardiovascular health, disparities in PA participation exist among racial and/or ethnic minority and female populations, which influences cardiovascular outcomes that have been shown to correlate best with PA levels. Notably, female individuals appear to derive greater cardiovascular benefits from exercise compared with male individuals and have reduced rates of SCD. Black race and/or ethnicity and male sex is consistently linked with increased rates of SCD. Although the relationship between race, ethnicity, and sex and cardiovascular outcomes is complex and evolving, the need for further research to elucidate the interplay between SDOH and exercise is evident. Addressing these factors is crucial for promoting equitable PA participation and optimizing cardiovascular health across diverse populations.

Ethics Statement

This is a review article that is conceptual and does not rely on any original data analysis, therefore, no institutional review board approval was sought for the writing of this review.

Patient Consent

The author confirms that patient consent is not applicable to this article, because it is a review article that is conceptual and therefore does not rely on any original data analysis.

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