**Impact of Geographical Origin Upon the Electrical and Structural Manifestations of the Black Athlete’s Heart**

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**One-sentence summary**

Repolarisation anomalies and increased left ventricular wall thickness are common manifestations of athletic adaptation in black male athletes, however, the magnitude of these alterations is highly dependent on geographical origin; athletes from West Africa and Middle Africa reveal the most profound changes.

**INTRODUCTION**

Sudden cardiac death (SCD) in young athletes is rare but represents the leading medical cause of death in this population. Certain high-risk subgroup have been further identified such as athletes of African or Caribbean descent [1:18,000 per year], basketball players [1:11 000 per year] and Division I male basketball players with an incidence of 1: 3,000 per year.1 Pre-participation examination (PPE) including a 12-lead electrocardiogram (ECG) is an effective strategy for detecting athletes with potentially life-threatening cardiac disorders.2 If identified in a timely fashion these disorders can be managed through lifestyle modifications, pharmacotherapy, and/or implantable cardioverter-defibrillators.

Increasing globalisation has provided athletes from all parts of the world the opportunity to compete at the highest level and has accordingly informed our understanding of the influence of ethnicity on cardiac adaptation to exercise. From this perspective, the group most intensely studied are athletes of African or Caribbean descent (universally termed black athletes). Data from adult black athletes demonstrate a 6-fold increase in the prevalence of marked repolarisation anomalies, notably T wave inversion3 and a near 5-fold increase in the prevalence of left ventricular hypertrophy (LVH) on echocardiography compared with white athletes.4 Taken together with the increased incidence of SCD in this population,1 such phenotypes often complicate the differentiation between physiological remodelling and morphologically mild hypertrophic cardiomyopathy (HCM).5

International recommendations for ECG interpretation in athletes have partly accounted for the black athlete and the high frequency of repolarisation changes observed in this population.6 The above recommendation is based however on the broad assumption that the term ‘black’ is applicable to all black athletes irrespective of their geographical descent. Although black race is entrenched in scientific writings, it is currently unclear whether such a blanket approach to this ethnic group is appropriate, as substantial regional differences do exist in anthropometric constitution, skeletal muscle physiology and cardiovascular disease patterns.7, 8 Therefore, the aim of this study was to examine the impact of geographical origin upon the electrical and structural manifestations of the black athlete’s heart.

**METHODS**

**Participants**

Between November 2010 and April 2018, 1,013 black, 418 Arabic and 267 white male athletes registered with the Qatar Olympic Committee [exercising ≥6 h/week, aged between 16 and 35 years] presented at our organisation for PPE. Ethical approval was provided by the Anti-Doping Laboratory Qatar (IRB# E2013000003) and all athletes provided informed consent.

Athletes were sub-divided by geographical origin by applying the United Nations “*Standard Country or Area Codes for Statistical Use*” coding system,9 through ascertaining the athlete’s country of origin (Figure 1). For comparison, 2 non-black athletic populations who shared close or connected geographical borders with Africa were also recruited. To limit the potential bias of sporting discipline, athletes were classified according to EACPR position statement recommendations.10 Only athletes engaged in ‘mixed’ sports were included for study; where football (48.5%), basketball (18.7%), handball (18.3%), and volleyball (12.1%) predominated.

**Pre-participation cardiovascular screening**

All athletes were screened using a cardiovascular pre-competition medical assessment pertaining to family history and personal symptoms. Measurements of height, body mass and left brachial artery blood pressure were recorded. A physical examination was carried out by a sports medicine physician.

**Resting 12-lead ECG**

All athletes underwent a 12-lead ECG after a period of 5 minutes of rest in the supine position using a GE Mac 5500 (New York, USA). ECGs were interpreted using recent international recommendations for ECG interpretation in athletes.6

**Echocardiography**

Routine PPE echocardiography was performed on 1,222 athletes (72%), as requested by the athlete’s respective sporting organisation. Echocardiographic examination was performed using a commercially available ultrasound system (ie33, Philips, USA), and interpreted using previously described criteria.11 Relative wall thickness (RWT) and LV geometry were calculated using American Society of Echocardiography guidelines.12

**Further evaluation and follow-up**

Athletes presenting with symptoms, a family history of SCD, ECG abnormalities and/or echocardiographic abnormalities considered indicative of possible cardiac pathology were investigated further. Further investigations included 24h ECG Holter monitoring, cardiopulmonary exercise testing and cardiac magnetic resonance imaging (cMRI). As necessary, CT coronary angiography (CTCA) and electrophysiological study were also available. A diagnosis of cardiac disease associated with SCD was established using published criteria.13

**Statistical analysis**

Data were expressed as mean ± SD or percentages as appropriate and were analysed using SPSS (v.21.0, 2012, IBM Corporation). Demographic differences and continuous ECG data between race and geographical region were assessed using a one-way analysis of variance (ANOVA) with Tukey adjustment when main effects were significant. To compare absolute and allometrically scaled echocardiographic parameters to geographical origin, a univariate general linear model was adopted, including training load per week and age as covariates. To compare the prevalence of positive family history, high blood pressures, normal/abnormal ECG patterns, and training load between geographical regions, Chi-square testing was adopted with Bonferroni correction and an equivalent non-parametric Fisher’s exact test was used when expected cell count was low.

All LV structural variables were scaled to individual differences in BSA, as the data was not supported by the concept of geometric similarity. Scaling was performed allometrically using the general form (*y*/*xb*). Using the natural logs of *y* (cardiac variable) and *x* (body surface area) linear regression was performed to generate standard *b* exponents. If the allometric model successfully accounted for body size the Pearson’s correlation between the scaled variable and body surface area should be no different from zero, demonstrating no significance (p>0.05). This held true for all cardiac parameters, and was thus adopted within the present study (Supplementary table 1). A p-value <0.05 was considered statistically significant.

**RESULTS**

**Demographics**

African-American/Caribbean athletes in our cohort were significantly older than athletes from all other regions except those from South America. They were also taller and heavier than all other athletes except European white athletes (p<0.001; Table 1). East and North African athletes were significantly smaller than Africans from other regions, athletes of African-American/Caribbean origin and South American athletes (p<0.01). Sixty-three (6.2%) black athletes had a resting blood pressure of ≥140/90mmHg. African-American/Caribbean and white Southern European athletes demonstrated significantly greater systolic blood pressure readings compared with East African, North African and West Asian athletes (p<0.01) (Table 1). When BSA was included as a covariate within univariate analysis however, there were no significant differences in blood pressure between athletes of any regions.

The prevalence of personal symptoms recorded during the PPE was high and varied significantly by geographical origin (Table 1). Further, 60% of West-Asians reported a family history of a CV disease in a first-degree relative, with 4% also reporting a family history of cardiac related death under 50 years of age in a first-degree relative.

**Prevalence of serious cardiac pathology**

Nine (0.5%) athletes were considered to have serious cardiac pathology and excluded from further analysis. Of these, 7 were disqualified due to the diagnosis or high clinical suspicion of HCM confirmed with echo and/or cMRI. One athlete was disqualified for an anomalous coronary artery, originating from the opposite sinus of Valsalva confirmed by CTCA. One athlete revealed the Wolff Parkinson White ECG pattern and returned to play following radiofrequency ablation. One athlete was symptomatic, 1 reported a history of a cardiac murmur and 1 had a family history of premature SCD (Supplementary table 2).

**The ECG of the black athlete**

**Normal physiological ECG findings**

Training related ECG findings were common across all regions (96.9%), with no differences between athletes of any region (Supplementary table 3). Sokolow-Lyon voltage criteria for LVH was significantly more frequent in Middle and West African athletes compared to those of African- American/Caribbean, North African and South American descent (p<0.05); whilst Sokolow criteria for right ventricular hypertrophy was more common in Middle African athletes (28%) compared to other African athletes. The prevalence of repolarisation anomalies in black athletes varied by region. Benign TWI patterns were significantly more frequent in Middle Africans (11.8%) than all other athletes (p<0.05), except West Africans (5.3%) (Table 2). The presence of a benign TWI pattern was not associated with any absolute or scaled cardiac structural parameters. There were no differences in cardiac dimensions between athletes with benign TWI and those without TWI.

The presence of two (or more) borderline findings was uncommon (n=10, 0.6%) and mostly due to bi-atrial enlargement (n=8, 0.5%), and with no regional significance (Figure 2).

**Abnormal ECG findings**

The combined average of black athletes demonstrating an abnormal ECG was 4.7%, compared to 2.2% of Arabic North Africans (p<0.05) and 0% in white athletes (p<0.001). The prevalence of an abnormal ECG differed significantly between black athletes (Figure 2). Abnormal TWI was significantly more frequent in Middle Africans (n=5, 8.5%) than all athletes except West Africans (p<0.05). There were no significant differences in the prevalence of abnormal TWI within the other regions (Table 2).

Abnormal TWI was associated with a greater absolute and scaled end diastolic interventricular septal thickness and LV posterior wall thickness, LV mass, and RWT (p<0.01); but not associated with a greater LV end diastolic diameter or LV volume. Finally, there were no significant differences in left atrial or aortic root diameter between athletes with abnormal TWI and those without abnormal TWI. The presence of other abnormal ECG findings beyond TWI were rare (0.8%). The prevalence of an abnormal ECG did not correlate with the number of hours of training.

**The cardiac structure of the black athlete**

**Left ventricular wall thickness**

Absolute LV wall thickness was significantly greater among African-American/Caribbean, Middle African and West African athletes compared with other black athletes. (p<0.001; Table 3). A LV wall thickness ≥ 12mm (LVH) was identified in 9.5% African-American/Caribbean, 5.5% Middle African and 4.9% West African athletes compared with none of the North and East African athletes (p<0.01). Among the other regions, LVH was present in 2.9% of South American, 2% of South European white, and 0.8% of West Asian athletes (Figure 1). After accounting for body size by allometric scaling, LV wall thickness remained significantly greater in Middle and West Africans compared to other black athletes (p<0.05) (Table 3).

**Left ventricular cavity size**

East Africans and West Asians revealed a smaller LV internal diameter compared with other athletes, however upon scaling for body size, LV cavity dimensions were similar between all athletes except North Africans, who presented with significantly larger LV cavity dimensions than African-American/Caribbean, East African and West Asian athletes (p<0.05).

**Left ventricular geometry**

Athletes from East and North Africa, Southern Europe and West Asia had a significantly greater prevalence of normal geometry than other regions (p<0.05) (Figure 3). Middle and West African and African-American/Caribbean athletes demonstrated significantly greater RWT than East and North Africans athletes (p<0.05). Among black athletes, only those from West Africa (n=6, 2.1%) and Middle Africa (n=1, 1.7%) demonstrated concentric hypertrophy (Figure 3).

There were no differences in cardiac function between black athletes from any region. The number of hours spent training was not associated with any cardiac variable after indexing for body size.

**Football specific analysis**

All our athletes engaged in sports with a mixture of strength and endurance components, however we recognised the heterogeneous nature of the various sporting disciplines included. Given that football was the most popular sport among all our black athletes, we performed a separate analysis including only football players (n = 823; 48.5%). Consistent with the results of the entire cohort, we observed a higher prevalence of TWI and greater LV wall thickness measurements in Middle and West African athletes compared with other black athletes (Supplementary tables 4-5 and supplementary figures 1-2).

**DISCUSSION**

It is recognised that there is widespread genetic, phenotypic, and cultural diversity within the global black population, with over 2,000 unique ethno-linguistic groups in Africa alone.14 Over the past 2 decades, black race has emerged as an important determinant of cardiac adaptation to exercise. It is also recognised that African-American athletes are at a greater risk of SCD than non-black athletes.1, 15 Whilst the term ‘black’ is embedded in the medical literature to classify an athlete’s racial identity, it is unclear whether such a blanket approach to interpreting the 12-lead ECG or echocardiogram of a black athlete is appropriate. This unique study, comprising a large black athlete cohort from four separate continents reports several pertinent findings to improve the cardiovascular assessment of black athletes. The prevalence of an abnormal ECG, in particular T wave inversion, differs significantly between black athletes depending on geographical origin, with a distinct predilection towards those of West and Middle African descent. Black athletes from West and Middle Africa, and African-American/Caribbean athletes have a greater LV wall thickness and LV mass compared with black athletes from East Africa and West Asia. Following allometric scaling, athletes from West and Middle Africa continue to demonstrate a significantly greater LV wall thickness and mass compared with other black athletes. Finally, there appears to be a significant relationship between abnormal T-wave inversion and increased LV wall thickness, LV mass and RWT in black athletes.

**The ECG in black athletes; impact of geographical origin**

To our knowledge, Di Paolo *et al* 16 and Schmied *et al* 17 are the only other groups to investigate regional and/or tribal differences in ECG patterns amongst black athletes; however both focused solely on relatively small cohorts of adolescent African athletes. Similar to our data, Di Paolo *et al* revealed that soccer players from North Africa have smaller R/S wave voltages, and a reduced frequency of ST segment elevation and repolarisation anomalies than other sub-Saharan African athletes. Likewise, Schmied *et al* 17 observed a greater prevalence of abnormal ECG patterns in adolescent African soccer players of Mande ethnicity than those of Bantu or Semitic-Hamitic ethnicities. Whilst interpretational care is warranted due to small sample sizes and the crude approximation of an athlete’s origin, both studies confirm that there are distinct geographical differences in ECG patterns amongst black athletes from Africa.

**Repolarisation Abnormalities**

Repolarisation abnormalities have been highlighted among the general black population since the 1950s. 18 While the studies of reference have typically focused on anterior TWI, the prevalence of inferior and lateral TWI is also more common in the black population than white athletes and controls.3 Few studies have investigated the prevalence of these repolarisation anomalies with respect to geographical origin. In the present study, there was no significant variation in the frequency of abnormal TWI between African-American/Caribbean, South America and West Asian athletes, however within black athletes from Africa, the differences were stark. The distribution of abnormal TWI in African athletes was heavily distributed towards athletes of Middle and West African origin, versus those of East and North African origin. This finding was also mirrored in the distribution of benign TWI. The precise mechanism(s) underlying these differences is unknown, but the present study also observed that abnormal TWI was significantly associated with greater absolute and scaled LV wall thickness, LV mass, and RWT; suggesting a potential relationship between, cardiac muscle enlargement and repolarisation changes. The association between SCD and the detection of an isolated increase in cardiac mass > 500g at autopsy (idiopathic LVH) especially in black athletes suggests that black athletes originating from Middle and West Africa may be capable of developing substantial exercise induced LVH, which may potentiate fatal arrhythmias. When one takes into consideration however, the high prevalence of abnormal TWI in Middle and West African athletes, it is improbable that all such athletes harbour pathological LVH. Nevertheless, it is clear that abnormal TWI is almost universal in black patients with HCM,19 signifying the need for comprehensive investigation and longitudinal follow-up of black athletes exhibiting such repolarisation anomalies.

**Practical implications; ECG interpretation in Black athletes**

Recent efforts have focused on improving the specificity of criteria for ECG interpretation for use within athletes. Using the international ECG interpretation recommendations, the mean prevalence of an abnormal ECG for our black cohort was 5.5% and was much lower than previous large studies using the refined criteria.20 Recent data from the USA suggests that the international ECG recommendations still inflict a high percentage of abnormal ECGs among African American basketball players (15.8%)21; yet it is worth noting that the prevalence of an abnormal ECG in the present study differed significantly between our black athletes [Middle African (11.9%), West African (8.9%), West Asian (4.8%), African-American/Caribbean (4.1%), South American (2.4%) and East African (2.5%)]. Consequently, the higher prevalence of ECG abnormalities observed in papers from the UK and US may reflect a much higher proportion of black athletes of West African origin.

Our data suggest that TWI in leads V1-V4 when preceded by J-point elevation and convex ST segment elevation is part of the normal ‘black athlete’s heart continuum’, however this particular pattern was identified in only 1.5% East African athletes compared with 12% black athletes from Middle Africa suggesting that larger studies among certain subgroups of African athletes are necessary before such conclusions can be accepted with greater certainty.

**The cardiac structure of the black athlete; impact of geographical origin**

It is well known that West African and African-American/Caribbean athletes present with disproportionate wall thickening compared to white athletes.4, 16 Consequently, Basavarajaiah *et al* 4 proposed that the upper limits of LV hypertrophy in black athletes should be raised from 12mm to 15mm in all athletes of African or African-American/Caribbean descent. However, Basavarajaiah *et al* also reported that 18% of their West African athletes had a maximal wall thickness ≥12mm, compared to just 4% in their East African athletes; a prevalence 5-times lower. Whilst our data demonstrates a significantly lower prevalence of LV hypertrophy in both West and East African athletes compared to Basavarajaiah *et al*, a similar trend was observed. Consequently, our data questions the proposal that the physiological upper limits of LV hypertrophy in all black athletes should be raised to 15mm, as we observed significant disparity in LVH between regions.

In line with the literature in hypertension22, concentric remodelling was more common in the black athletes of West and Middle Africa. In Africa the prevalence of hypertension varies by region, being twice as common in West compared with East Africa (23% vs. 10.3%).23 Whilst our data do not support this finding after scaling for BSA, the persistent concentric hypertrophy observed in West and Middle African athletes, particularly those with abnormal TWI, may perhaps be alternatively explained by different vascular adaptations, such as increased aortic stiffness, endothelial dysfunction, and an overall different hemodynamic profile.24

**Limitations**

This study did not employ genetic genealogy to determine an athlete’s true geographical origin. Secondly, while our cohort of Middle African athletes was smaller than the other groups (n =59), we believe it reflects a fair representation compared with other studies in black athletes. Thirdly, ‘mixed race’ is an understudied population, however we did not have the sample size required to compare mixed raced athletes to those of African origin. The same situation also applied to black female athletes, since few present at our institution for PPE. Whilst all athletes with inferolateral TWI are annually followed-up, we recognise that a small proportion of athletes with HCM may not have been identified given the heterogeneity and aged-related penetrance of the condition. Finally, we acknowledge that the use of banned substances has the potential to cause LVH and concentric hypertrophy, however as part of the medical history, the use of medication and supplementation is noted. None of the athletes within the present study reported the use of any illicit substances, however we did not specifically test for illicit drugs.

**Conclusion**

The collective term ‘black’ should not imply that the hearts of all black athletes are universally comparable. There is considerable variability in the cardiac electrical and structural remodelling response to exercise that appears to be governed in part by geographic origin. Black athletes from West and Middle Africa have a significantly higher prevalence of abnormal TWI and greater LV wall thickness, and LV mass than black athlete of East African and West Asian origin. While the aetiology of the TWI in black athletes is unclear, there was a link between abnormal TWI and LV wall thickening and concentric LV remodelling. The precise significance of these observations requires a prospective multi centre longitudinal study.

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**Figure 1**. Global representation of athletes included within the study and the key findings, split by geographical region.

**Figure 2.** Percentage of borderline and abnormal ECG findings per geographical region.

**Figure 3.** Percentage of athletes with eccentric hypertrophy, concentric remodelling or concentric hypertrophy per geographical region.

**Table 1.** Comparison of demographic, personal symptoms, family history and ECG parameters between athletes of the varying regions.

**Table 2.** Comparison of T-wave inversion prevalence between athletes from the varying regions.

**Table 3.** Comparison of absolute and body size indexed structural and functional parameters between athletes from the varying regions.

**Supplementary Table 1.** Body size *b* exponent derived from log linear allometric, and theory of geometric similarity (ToGS) models.

**Supplementary Table 2.** Features and outcomes of athletes considered to have serious pathology.

**Supplementary Table 3.** Comparison of training related ECG changes between athletes from the varying regions

**Supplementary Table 4.** Comparison of T-wave inversion prevalence between football players from the varying regions.

**Supplementary Table 5.** Comparison of absolute and body size indexed structural and functional parameters between football players from the varying regions.

**Supplementary Figure 1.** Percentage of borderline and abnormal ECG findings per geographical region (%).

**Supplementary Figure 2.** Percentage of athletes with eccentric hypertrophy, concentric remodelling or concentric hypertrophy per geographical region (%).

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