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**Journal Title:** BRITISH JOURNAL OF SPORTS MEDICINE

**Volume:** Volume 56, Number 3

**Publisher:** BMJ PUBLISHING GROUP | 2021-08-13, Pages 151-+

**Type of Work:** Article

**Publisher DOI:** 10.1136/bjsports-2021-104333

**Permanent URL:** <https://pid.emory.edu/ark:/25593/vz8hp>

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Final published version: <http://dx.doi.org/10.1136/bjsports-2021-104333>

*Accessed January 30, 2023 9:04 PM EST*



Published in final edited form as:

*Br J Sports Med.* 2022 February ; 56(3): 151–157. doi:10.1136/bjsports-2021-104333.

## Association between race and maladaptive concentric left ventricular hypertrophy in American-style football athletes

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### Abstract

**Objectives**—American-style football (ASF) athletes are at risk for the development of concentric left ventricular hypertrophy (C-LVH), an established cardiovascular risk factor in the general population. We sought to address whether black race is associated with acquired C-LVH in collegiate ASF athletes.

**Methods**—Collegiate ASF athletes from two National Collegiate Athletic Association Division-I programmes were recruited as freshmen between 2014 and 2019 and analysed over 3 years. Demographics (neighbourhood family income) and repeated clinical characteristics and echocardiography were recorded longitudinally at multiple timepoints. A mixed-modelling approach was performed to evaluate acquired C-LVH in black versus white athletes controlling for playing position (linemen (LM) and non-linemen (NLM)), family income, body weight and blood pressure.

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**Contributors** JHK had access to all of the data in the study and takes full responsibility for the integrity of the data and the accuracy of the data analysis. JVT, CL and JHK performed statistical analyses and drafted the manuscript. All authors contributed with acquisition of the data, conceptual design, analyses and interpretation of the results. All authors contributed to draft the article, critical revisions for intellectual content and gave final approval for the version submitted.

**Competing interests** JHK receives compensation serving in his role as team cardiologist for the Atlanta Falcons. AB receives compensation serving in his role as team cardiologist for the New England Patriots. HAT is an Advisory Board Member for Pfizer and Educational Consultant for Novartis.

**Patient and public involvement** Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

**Ethics approval** The Emory Institutional Review Board (#IRB00071873) approved all aspects of this study and subjects provided written informed consent.

**Provenance and peer review** Not commissioned; externally peer reviewed.

**Results**—At baseline, black athletes (N=124) were more often NLM (72% vs 54%,  $p=0.005$ ) and had lower median neighbourhood family income (\$54 119 vs \$63 146,  $p=0.006$ ) compared with white athletes (N=125). While both black and white LM demonstrated similar increases in C-LVH over time, among NLM acquired C-LVH was more common in black versus white athletes (postseason year-1: N=14/89 (16%) vs N=2/68 (3%); postseason year-2: N=9/50 (18%) vs N=2/32 (6%); postseason year-3: N=8/33 (24%) vs N=1/13 (8%),  $p=0.005$  change over time). In stratified models, black race was associated with acquired C-LVH in NLM (OR: 3.70, 95% CI 1.12 to 12.21,  $p=0.03$ ) and LM was associated with acquired C-LVH in white athletes (OR: 3.40, 95% CI 1.03 to 11.27,  $p=0.048$ ).

**Conclusions**—Independent of family income and changes in weight and blood pressure, black race was associated with acquired C-LVH among collegiate ASF NLM and LM was associated with acquired C-LVH in white athletes.

## INTRODUCTION

Population-based studies have established that left ventricular (LV) hypertrophy with concentric geometric morphology predicts incident heart failure, stroke, coronary artery disease and cardiovascular (CV) mortality in the general population.<sup>1 2</sup> Conventionally, chronic conditions associated with haemodynamic pressure overload, such as hypertension, underlie the development of concentric left ventricular hypertrophy (C-LVH).<sup>3</sup> Among black individuals, after adjusting for hypertension, the prevalence of C-LVH is still higher compared with white individuals.<sup>4 5</sup> Although recent data suggest a heightened sensitivity to arterial afterload may contribute to this epiphenomenon,<sup>6</sup> there is recognition that the interaction between biology and environmental and social determinants of health must be considered as part of this pathophysiological cascade.<sup>7 8</sup> Among young individuals with LVH, the relationship between race, LV geometry and outcomes is uncertain.

Sport-specific exercise-induced haemodynamic alterations lead to adaptive LV remodelling referred to as the 'athlete's heart'.<sup>9</sup> For strength athletes, including American-style football (ASF) and rugby athletes engaged in isometric training intensification,<sup>10</sup> surges in systemic afterload mimic pathological pressure overload states and may promote the development of adaptive concentric LV remodelling.<sup>11</sup> However, emerging longitudinal data demonstrate that ASF athletes can progress and further develop maladaptive C-LVH, particularly in those who play at the lineman (LM) field position (athletes engaged in more intensive isometric training as compared with non-linemen (NLM)),<sup>12</sup> become hypertensive, or gain significant weight.<sup>13–15</sup> At present, whether race classification is also associated with acquired maladaptive C-LVH observed in ASF athletes is uncertain.

We sought to address this important knowledge gap and examine the impact of race on acquired C-LVH across the span of collegiate ASF participation. We hypothesised that C-LVH would progress throughout collegiate ASF participation and that black race would be associated with acquired C-LVH. To address this hypothesis, we conducted a longitudinal and multicentre analysis of collegiate ASF athletes, evenly stratified by self-identified race, across the collegiate ASF career with repeated measures using transthoracic echocardiography and vascular applanation tonometry.

## METHODS

ASF athletes were recruited from National Collegiate Athletic Association (NCAA) Division-I programmes at Georgia Institute of Technology (Atlanta, Georgia, USA) and Furman University (Greenville, South Carolina, USA) and studied between 2014 and 2019. Clinical characteristics, demographics, echocardiography and vascular applanation tonometry were longitudinally captured over the study period.

### Study population

ASF athletes were recruited in parallel with the academic calendar, beginning with the freshman season. Athletes were prospectively studied at prespecified longitudinal timepoints (figure 1). For freshmen, this included the preseason (baseline) and the immediate postseason (postseason year-1), approximately 5–6 months later. For sophomores, athletes were studied at postseason year-2, 1 year after postseason year-1, and for juniors, athletes were studied at postseason year-3, 1 year after postseason year-2. Two time points for year-1 were chosen because of the significant CV plasticity previously demonstrated among first-year college athletes in response to athletic training.<sup>16</sup> Because athletes at the participating institutions engage in consistent levels of ASF training throughout the calendar year, only annual postseason study time points were captured beyond year-1. Athletes with normal pre-participation clinical history and physicals (including a first-year 12-lead ECG),<sup>17</sup> normal NCAA-mandated drug testing, complete clinical data and echocardiographic images, who completed at least one full season of ASF and who self-reported as black or white were included in the final analysis (figure 1).

Descriptive information including age (years), height (cm), current medication use, self-reported race as black or white and family history of hypertension was collected at baseline. Socioeconomic status was reported as the imputed median household income based on individual hometown zip codes, each one ascribed based on US census data available for all cities and counties with populations >5000 individuals.<sup>18</sup> Clinical data including weight (kg), systolic (SBP) and diastolic blood pressure (DBP) were collected at all timepoints. Blood pressure was measured in the sitting position using a manual aneroid sphygmomanometer and an appropriately sized cuff and recorded as the average of three measurements. Player position was classified as either LM or NLM as previously proposed.<sup>12</sup> LM included players at the tackle, guard, centre or defensive end positions; NLM included quarterbacks, running backs, wide receivers, tight ends, linebackers, cornerbacks, safeties, kickers, and punters.<sup>12</sup>

### Patient and public involvement

No patient or public participants were involved in the design or analysis of this study.

### Two-dimensional transthoracic echocardiography

Transthoracic echocardiography was performed using a commercially available system (Vivid-I, GE Healthcare, Milwaukee, Wisconsin, USA) at all study time points. Two-dimensional and tissue-Doppler imaging from standard parasternal and apical positions were performed by experienced sonographers, who were consistent throughout this study. All

information was stored digitally and post-study offline data analysis (EchoPAC version 7, GE Healthcare) was performed. Definitions of normality for cardiac structure and function were adopted from the most recent guidelines.<sup>19</sup> Average LV wall thickness was calculated as the mean of the intraventricular septum (IVS) and posterior wall thickness (PWT). LV mass was calculated using the area-length method (accounts for LV morphology in both the short-axis and long-axis) and indexed to body surface area.<sup>19</sup> Relative wall thickness was calculated as: (IVS +PWT)/LV end-diastolic diameter. With LV mass calculated by the area-length method, C-LVH was defined as per American Society of Echocardiography guidelines: relative wall thickness >0.42 and LV mass index >102 g/m<sup>2</sup>.<sup>19</sup> Comprehensive assessment of cardiac diastolic function using tissue-Doppler imaging was performed and tissue velocities (E', A' and S') were measured from colour-coded images at the lateral and septal mitral annulus. E' was reported as the average between the two measurements.

### Vascular applanation tonometry

Arterial stiffness was measured at all study timepoints using high fidelity applanation tonometry (SphygmoCor, Atcor Medical, Australia), which records high-quality pressure waveforms at peripheral pulse sites. The primary measure of arterial function was the carotid-femoral pulse wave velocity (PWV), the gold standard index of arterial stiffness,<sup>20</sup> measured as previously described.<sup>13 21</sup>

### Statistical analysis

Continuous variables are presented as the mean (SD) or median (IQR) and categorical variables as numbers (percentages). Baseline characteristics were stratified by race and compared using the  $\chi^2$  test of homogeneity or Fisher's exact test for categorical variables, or a two-sample t-test or Kruskal-Wallis test for continuous variables based on normality and equality of variances via a folded F-test. Race-stratified athletes were analysed by mixed-effects linear regression to evaluate for longitudinal changes in clinical measurements, adjusting for player position. Clinical measurements were treated as the dependent variable and the study timepoint was treated as the independent categorical variable. Changes at each timepoint were compared with baseline. The primary outcome measure, analysed by mixed-effects binomial logistic regression, was acquired C-LVH across the study period. Acquired C-LVH was defined as C-LVH that developed at any longitudinal timepoint, but not present at baseline. To investigate factors associated with acquired C-LVH, we adjusted for race, player position, family history of hypertension, median hometown family income, and the change in weight, SBP, PWV and E' across the study period. An interaction term between race and player position was created to explore the different effects of race conditioned by player position. Stratified analyses by player position and race were constructed with the same covariates as the original model. Normally distributed participant-specific random intercepts were incorporated in mixed-effects models to account for within-participant correlation. Analyses were performed with SAS software (V.9.4). A p value of < 0.05 was considered significant.

## RESULTS

### Black and white ASF athlete characteristics including socioeconomic status

Three hundred freshman ASF athletes were serially enrolled at preseason between June 2014 and June 2019. At postseason year-1, 249 (N=124 (50%) black athletes and N=125 (50%) white athletes) remained eligible for final analysis (51 were excluded due to sport-related attrition or identifying as non-black or white). Due to the rolling nature of study enrolment (athletes not yet at the appropriate temporal longitudinal time point), sport attrition or incomplete echocardiographic studies (including lack of imaging due to COVID-19 restrictions), 140 (N=70 (50%) black and N=70 (50%) white) athletes were analysed at postseason year-2 and 81 (N=47 (58%) black and N=34 (42%) white) were analysed at postseason year-3 (figure 1). At baseline (table 1), white athletes were taller, heavier and more commonly LM. Based on US census data, the average median neighbourhood family income was lower for black versus white ASF athletes (\$54 119 vs \$63 146,  $p=0.006$ ).

### Longitudinal changes in C-LVH as a function of race and player position

At baseline, C-LVH was present in 2% (N=5/249) of athletes. There was a significant increase in ASF athletes who developed C-LVH at postseason year-1 (N=25/249 (10%)), postseason year-2 (N=21/140 (15%)) and postseason year-3 (N=19/81 (23%),  $p<0.001$  for the change over time). C-LVH progression was similar in black (13%–21%) versus white athletes (7%–26%, figure 2A). In the LM-stratified analysis, progression in acquired C-LVH was similar between black and white athletes ( $p=0.39$ ) and notable among white LM (12%–38%, figure 2B). When stratified by NLM, the development of C-LVH was increased in black versus white athletes ( $p=0.005$ ; postseason year-1: N=14/89 (16%) vs N=2/68 (3%); postseason year-2: N=9/50 (18%) vs N=2/32 (6%); postseason year-3: N=8/33 (24%) vs N=1/13 (8%), respectively (figure 2C).

### Maladaptive C-LVH in ASF athletes

Black and white ASF athletes gained weight, increased SBP and demonstrated similar alterations in other CV structural and functional measurements across the study period (table 2). No athlete demonstrated concentric LV wall thickness that raised clinical suspicion for hypertrophic cardiomyopathy ( $>15$  mm).<sup>22</sup> Regardless of race, athletes who manifest C-LVH were heavier, had higher SBP and relative arterial stiffening and had reduced  $E'$  (diastolic function) compared with those without C-LVH (figure 3).

### Association between race and acquired C-LVH

In the analysis of acquired C-LVH, there was significant interaction between race and player position ( $p=0.02$ ). In addition, decreased  $E'$  ( $E'$ , OR: 0.85, 95% CI 0.74 to 0.98,  $p=0.03$ ) was associated with acquired C-LVH (table 3). In stratified analysis (table 4), black race (OR: 3.70, 95% CI 1.12 to 12.21,  $p=0.03$ ) was associated with acquired C-LVH in NLM. Among just white ASF athletes, the LM position (OR 3.40, 95% CI 1.03 to 11.27,  $p=0.048$ ) was associated with acquired C-LVH.

## DISCUSSION

### Principal findings

To our knowledge, this is the first longitudinal and multi-year study of athletes that assessed acquired C-LVH in the context of sport exposure and race. Based on our observed effect modification between ASF player position and race, key findings are as follows. First, among NLM, who are generally considered less likely to develop C-LVH<sup>14 15 23</sup> and engaged in less isometric training compared with LM,<sup>12</sup> we found that the development of C-LVH was significantly increased for black versus white NLM and black race was independently associated with acquired C-LVH. Second, among LM and in contrast to NLM, we observed similar progression in acquired C-LVH in black and white LM across the study period. This finding, coupled with the association between LM and acquired C-LVH in white athletes, aligns with the body of evidence suggesting LM<sup>14 15</sup> are at risk to develop C-LVH. Finally, our data suggest that acquired C-LVH is maladaptive in ASF athletes, particularly given the association with reduced diastolic function (decreased E'). In aggregate, our findings demonstrate that among black collegiate ASF NLM, independent of traditional CV risk, there is increased likelihood of acquired C-LVH compared with white NLM and that LM, regardless of race, are at risk of developing C-LVH. Our results suggest that black collegiate ASF athletes may have a greater potential for both the development of concentric LV remodelling and maladaptive C-LVH during ASF participation.

### Race and health outcomes

The complex interaction between race and health outcomes remains controversial.<sup>7 24 25</sup> Fundamental to this relationship is the interplay between normal biological processes, environmental stimuli and social determinants, in part, because standardised race classifications are unreliable substitutions for underlying genetic differences.<sup>26 27</sup> At present, there is recognition that social disparities, including racism and socioeconomic status imbalance, are critical determinants of health outcomes.<sup>28 29</sup> As such, caution is required in the synthesis and interpretation of race-based clinical observational data.<sup>7 25</sup> Based on prior studies in ASF athletes, those who gain weight, develop hypertension, or are LM, are at increased likelihood for acquired C-LVH.<sup>13–15</sup> Our findings in NLM add further insight to this risk calculus and suggest that black ASF NLM are more likely to acquire C-LVH compared with white ASF NLM, independent of these established ASF CV risk factors. Although the explanatory physiological, environmental and social determinants are uncertain, we observed lower baseline neighbourhood socioeconomic status in the black ASF athletes and there was a trend towards an association between lower median family income and acquired C-LVH. As such, more rigorous assessments of the potential association between social determinants of health and acquired pathological CV phenotypes in ASF athletes are warranted. Further, in the context of established risks of C-LVH in the general population, ascertainment of long-term health outcomes in all youthful ASF athletes with C-LVH are also warranted.

### Self-identified black race and C-LVH in ASF athletes

Our findings add further depth to our understanding of established CV risk factors and corollary maladaptive CV phenotypes observed among collegiate ASF athletes.<sup>13–16 21 30</sup>

Importantly, in contrast to adaptive strength training-induced concentric LV remodelling,<sup>9</sup> prior longitudinal data have demonstrated that further progression to C-LVH in collegiate ASF athletes is maladaptive<sup>13–15</sup> and inconsistent with the classic ‘athlete’s heart’ because of the association with substantial weight gain, hypertension,<sup>15 30 31</sup> relative arterial stiffening<sup>21 32</sup> and reduced diastolic function. Indeed, in this analysis, we observed that acquired C-LVH is associated with reduced diastolic function, which contrasts with the typical finding of enhanced diastolic function in the athletic heart.<sup>11 13 16</sup> Risks are most evident among LM, who are heavier and less aerobically trained compared with NLM.<sup>23</sup> Among retired professional ASF LM, there is also increased risk for adverse later-life health outcomes.<sup>33–35</sup> In the present study, our data affirm that among LM, black and white LM are similarly at risk for acquired C-LVH, but for NLM, acquired C-LVH is independently predicted by black race. Conversely, among just white ASF athletes, the LM field position increases the likelihood of acquired C-LVH, but among black ASF athletes, field position (LM vs NLM) does not alter the probability of acquired C-LVH. Overall, these observations suggest that black race may represent an independent stimulus for the development of C-LVH among NLM. Understanding the specific elements associated with black race that underlie this novel observation will require carefully designed studies inclusive of biological variables, such as measurements obtained from individualised exercise testing, training and environmental variables, and social determinants.

To what degree inherent biological pathways, as a function of race, factor into our findings is uncertain. In cross-sectional data from the UK, increased LVH and concentric LV remodelling were observed in black versus white athletes of multiple sporting disciplines.<sup>36</sup> Prior studies, also cross-sectional in design, reported differences in cardiac phenotypes by country of origin<sup>37</sup> and international geographic region<sup>38</sup> in the comparison of black and non-black athletes. More recently, a large cross-sectional study of 3000 mixed-race athletes (defined as having one black and one white parent) reported increased concentric LV remodelling in the mixed-race athlete cohort.<sup>39</sup> In the general population, across the age spectrum, C-LVH is more commonly observed among black individuals.<sup>4 5 40</sup> Recent data taken from older, but healthy black Americans (N=1286, 75±5 years old, 34% male) suggest heightened sensitivity to arterial afterload and corollary increased LV filling pressures may be present in black individuals.<sup>6</sup> In the present study, strengthened by the prospective and longitudinal design over multiple years, our data suggest that a similar physiological stimulus may also be present among young black NLM ASF athletes.

The suggestion that accelerated C-LVH occurs in self-identified black ASF NLM calls attention to the complex relationship between race, biology and environmental stressors, and the critical need for equipoise in investigating these metrics in future athletic outcomes studies. Our findings suggest that the CV response to stressful exposures, including the demands of collegiate ASF training, may also be affected by early-life social influences. Potential environmental stimuli affected by social determinants, such as diet, lifestyle and scholastic or cultural stress, are uncertain and merit careful investigation. As the paradigm of defining the healthy athlete and risks associated with intense exercise evolves, it is imperative that we consider and control for social and environmental variables in future athletic CV outcomes studies.



## Limitations

Several limitations of this study are noteworthy. First, because of the rolling nature of study enrolment, athletic attrition and COVID-19 pandemic restrictions, we acknowledge incomplete cases beyond year-1, and it is uncertain how full retainment of the study cohort through all 3 years would have affected the results. Specifically, because race-stratified groups were not equally stratified by player position, we acknowledge there may be limitations in the interpretation of our race and player position stratified analyses. Athletes were also not followed through the conclusion of their senior season due to logistic difficulties in subject retainment. However, all data acquisition time points for athletes who completed at least one full ASF season were included in the mixed-effects analyses, which accounted for missing data. Further, we observed similar maladaptive CV phenotypes previously reported in ASF athletes who develop C-LVH.<sup>13</sup> Second, although LM and NLM are generally exposed to distinct isometric training regimens,<sup>12</sup> we were unable to record specific differences in training loads between groups. Third, we were unable to record the exercise blood pressure response for subjects. Fourth, despite the replication of findings from multiple prior studies,<sup>13 14</sup> we acknowledge the lack of matched control groups, both athletic and non-athletic, in this analysis. Fifth, while our primary outcome measure was selected a priori, we acknowledge the limitation of multiplicity in our analyses. We also acknowledge that mixed-effects modelling is subject to time-varying confounding affected by prior exposures.<sup>41</sup> Finally, we were limited in the characterisation of socioeconomic status in this study. We reported imputed family income based on US census data and therefore not actual family income per athlete. Although imputed income from census data is predictive of CV risk,<sup>42</sup> further detailed assessments of socioeconomic status are warranted.

## CONCLUSIONS

In addition to collegiate ASF athletes who exhibit traditional CV risk or are LM, self-identified black NLM also harbour increased likelihood for the development of C-LVH. Thus, black race may represent an independent stimulus associated with the development of C-LVH. Characterisation of the specific underlying epigenetic, biological and environmental factors, and the need to scrutinise other young athletic cohorts for similar race relationships represent essential arenas of future investigation.

## Acknowledgements

We continue to thank the athletic departments and the student athletes at Georgia Institute of Technology and Furman University for their ongoing support of this research and participation in our athletic registry. We also acknowledge Digirad and Athletic Heart for providing all echocardiographic imaging services. Digirad and Athletic Heart were both compensated for providing the sonographers who performed all echocardiograms a part of this study.

## Funding

This work was entirely supported by US National Institutes of Health/National Heart, Lung, and Blood Institute research grant K23 HL128795 (to JHK).

## Data availability statement

No data are available. No data are available. The data are deidentified participant data and not available for public use.

## REFERENCES

1. Bluemke DA, Kronmal RA, Lima JAC, et al. The relationship of left ventricular mass and geometry to incident cardiovascular events: the MESA (multi-ethnic study of atherosclerosis) study. *J Am Coll Cardiol* 2008;52:2148–55. [PubMed: 19095132]
2. Levy D, Garrison RJ, Savage DD, et al. Prognostic implications of echocardiographically determined left ventricular mass in the Framingham heart study. *N Engl J Med* 1990;322:1561–6. [PubMed: 2139921]
3. Lorell BH, Carabello BA. Left ventricular hypertrophy. *Circulation* 2000;102:470–9. [PubMed: 10908222]
4. Drazner MH, Dries DL, Peshock RM, et al. Left ventricular hypertrophy is more prevalent in blacks than whites in the general population: the Dallas heart study. *Hypertension* 2005;46:124–9. [PubMed: 15939807]
5. Drazner MH. The progression of hypertensive heart disease. *Circulation* 2011;123:327–34. [PubMed: 21263005]
6. Fernandes-Silva MM, Shah AM, Hegde S, et al. Race-Related Differences in Left Ventricular Structural and Functional Remodeling in Response to Increased Afterload: The ARIC Study. *JACC Heart Fail* 2017;5:157–65. [PubMed: 28017356]
7. Vyas DA, Eisenstein LG, Jones DS. Hidden in Plain Sight - Reconsidering the Use of Race Correction in Clinical Algorithms. *N Engl J Med* 2020;383:874–82. [PubMed: 32853499]
8. Borrell LN, Elhawary JR, Fuentes-Afflick E, et al. Race and Genetic Ancestry in Medicine - A Time for Reckoning with Racism. *N Engl J Med* 2021;384:474–80. [PubMed: 33406325]
9. Baggish AL, Wood MJ. Athlete's heart and cardiovascular care of the athlete: scientific and clinical update. *Circulation* 2011;123:2723–35. [PubMed: 21670241]
10. Levine BD, Baggish AL, Kovacs RJ, et al. Eligibility and Disqualification recommendations for competitive athletes with cardiovascular abnormalities: Task force 1: classification of sports: dynamic, static, and impact: a scientific statement from the American heart association and American College of cardiology. *Circulation* 2015;132:e262–6. [PubMed: 26621643]
11. Baggish AL, Wang F, Weiner RB, et al. Training-specific changes in cardiac structure and function: a prospective and longitudinal assessment of competitive athletes. *J Appl Physiol* 2008;104:1121–8. [PubMed: 18096751]
12. Croft LB, Belanger A, Miller MA, et al. Comparison of national football League linemen versus nonlinemen of left ventricular mass and left atrial size. *Am J Cardiol* 2008;102:343–7. [PubMed: 18638599]
13. Kim JH, Hollowed C, Liu C, et al. Weight gain, hypertension, and the emergence of a maladaptive cardiovascular phenotype among US football players. *JAMA Cardiol* 2019;4:1221–9. [PubMed: 31617867]
14. Lin J, Wang F, Weiner RB, et al. Blood pressure and LV remodeling among American-Style football players. *JACC Cardiovasc Imaging* 2016;9:1367–76. [PubMed: 27931524]
15. Weiner RB, Wang F, Isaacs SK, et al. Blood pressure and left ventricular hypertrophy during American-style football participation. *Circulation* 2013;128:524–31. [PubMed: 23897848]
16. Kim JH, Hollowed C, Patel K, et al. Temporal changes in cardiovascular remodeling associated with football participation. *Med Sci Sports Exerc* 2018;50:1892–8. [PubMed: 29634639]
17. Sharma S, Drezner JA, Baggish A, et al. International Recommendations for Electrocardiographic Interpretation in Athletes. *J Am Coll Cardiol* 2017;69:1057–75. [PubMed: 28231933]
18. United States Census Bureau. U.S. census bureau QuickFacts, 2020. Available: <https://www.census.gov/quickfacts/fact/table/US/PST045219> [Accessed 26 Jun 2020].

19. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of echocardiography and the European association of cardiovascular imaging. *J Am Soc Echocardiogr* 2015;28:1–39. e14. [PubMed: 25559473]
20. Willum-Hansen T, Staessen JA, Torp-Pedersen C, et al. Prognostic value of aortic pulse wave velocity as index of arterial stiffness in the general population. *Circulation* 2006;113:664–70. [PubMed: 16461839]
21. Kim JH, Sher S, Wang F, et al. Impact of American-style football participation on vascular function. *Am J Cardiol* 2015;115:262–7. [PubMed: 25465938]
22. Kim JH, Baggish AL. Differentiating Exercise-Induced Cardiac Adaptations From Cardiac Pathology: The “Grey Zone” of Clinical Uncertainty. *Can J Cardiol* 2016;32:429–37. [PubMed: 26875013]
23. Kim JH, Zafonte R, Pascuale-Leon A, et al. American-style football and cardiovascular health. *J Am Heart Assoc* 2018;7:e008620. [PubMed: 29618471]
24. Burchard EG, Ziv E, Coyle N, et al. The importance of race and ethnic background in biomedical research and clinical practice. *N Engl J Med* 2003;348:1170–5. [PubMed: 12646676]
25. Ioannidis JPA, Powe NR, Yancy C. Recalibrating the use of race in medical research. *JAMA* 2021;325:623–4. [PubMed: 33492329]
26. Maglo KN, Mersha TB, Martin LJ. Population genomics and the statistical values of race: an interdisciplinary perspective on the biological classification of human populations and implications for clinical genetic epidemiological research. *Front Genet* 2016;7:22. [PubMed: 26925096]
27. Bryc K, Durand EY, Macpherson JM, et al. The genetic ancestry of African Americans, Latinos, and European Americans across the United States. *Am J Hum Genet* 2015;96:37–53. [PubMed: 25529636]
28. Schultz WM, Kelli HM, Lisko JC, et al. Socioeconomic status and cardiovascular outcomes. *Circulation* 2018;137:2166–78. [PubMed: 29760227]
29. Lewis TT, Williams DR, Tamene M, et al. Self-Reported experiences of discrimination and cardiovascular disease. *Curr Cardiovasc Risk Rep* 2014;8:365. [PubMed: 24729825]
30. Churchill TW, Krishnan S, Weisskopf M, et al. Weight gain and health affliction among former national football League players. *Am J Med* 2018;131:1491–8. [PubMed: 30102909]
31. Karpinos AR, Roumie CL, Nian H, et al. High prevalence of hypertension among collegiate football athletes. *Circ Cardiovasc Qual Outcomes* 2013;6:716–23. [PubMed: 24221829]
32. Kim JH, Hollowed C, Irwin-Weyant M, et al. Sleep-Disordered breathing and cardiovascular correlates in college football players. *Am J Cardiol* 2017;120:1410–5. [PubMed: 28823486]
33. Baron S, Rinsky R. National football League players mortality study: report No. HETA 88–085 Atlanta, GA: National Institute for Occupational Safety and Health, 1994.
34. Baron SL, Hein MJ, Lehman E, et al. Body mass index, playing position, race, and the cardiovascular mortality of retired professional football players. *Am J Cardiol* 2012;109:889–96. [PubMed: 22284915]
35. Lincoln AE, Vogel RA, Allen TW, et al. Risk and causes of death among former national football League players (1986–2012). *Med Sci Sports Exerc* 2018;50:486–93. [PubMed: 29077640]
36. Basavarajaiah S, Boraita A, Whyte G, et al. Ethnic differences in left ventricular remodeling in highly-trained athletes relevance to differentiating physiologic left ventricular hypertrophy from hypertrophic cardiomyopathy. *J Am Coll Cardiol* 2008;51:2256–62. [PubMed: 18534273]
37. Di Paolo FM, Schmied C, Zerguini YA, et al. The Athlete’s Heart in Adolescent Africans. *J Am Coll Cardiol* 2012;59:1029–36. [PubMed: 22402076]
38. Riding NR, Sharma S, McClean G. Impact of geographical origin upon the electrical and structural manifestations of the black athlete’s heart. *Eur Heart J* 2018.
39. Malhotra A, Oxborough D, Rao P, et al. Defining the normal spectrum of electrocardiographic and left ventricular adaptations in mixed-race male adolescent soccer players. *Circulation* 2021;143:94–6. [PubMed: 33378235]
40. Lai C-C, Sun D, Cen R, et al. Impact of long-term burden of excessive adiposity and elevated blood pressure from childhood on adulthood left ventricular remodeling patterns: the Bogalusa heart study. *J Am Coll Cardiol* 2014;64:1580–7. [PubMed: 25301461]

41. Mansournia MA, Etminan M, Danaei G, et al. Handling time varying confounding in observational research. *BMJ* 2017;359:j4587. [PubMed: 29038130]
42. Alter DA, Naylor CD, Austin P, et al. Effects of socioeconomic status on access to invasive cardiac procedures and on mortality after acute myocardial infarction. *N Engl J Med* 1999;341:1359–67. [PubMed: 10536129]

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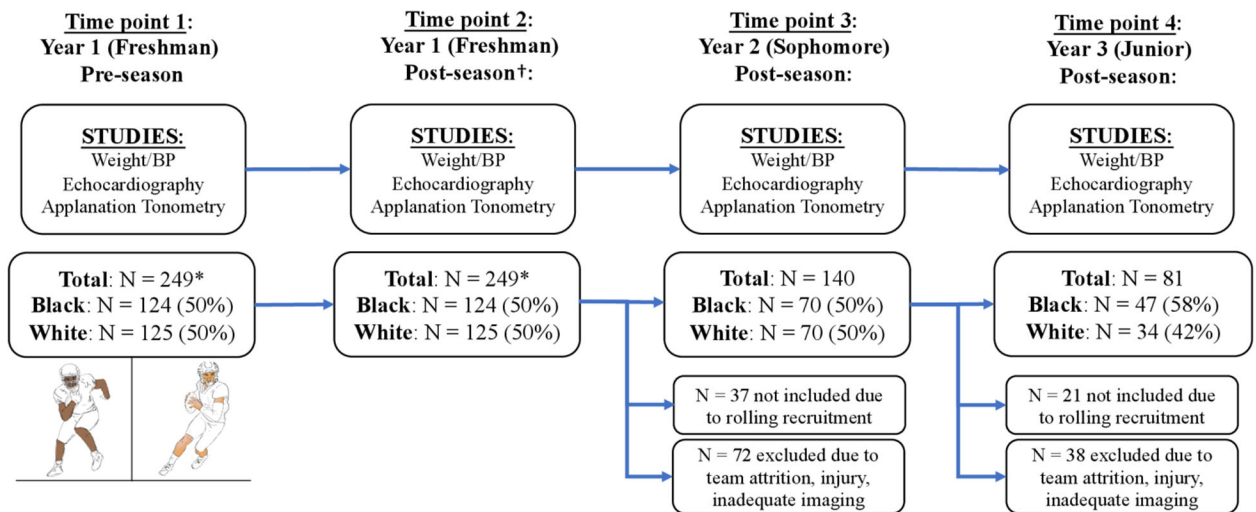
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**What are the findings?**

- Self-identified black race is independently associated with the development of maladaptive concentric left ventricular hypertrophy (C-LVH) in collegiate American-style football (ASF) non-linemen (NLM).
- Black and white ASF linemen (LM) similarly develop C-LVH throughout collegiate ASF participation affirming the higher likelihood of this cardiac phenotype in LM.

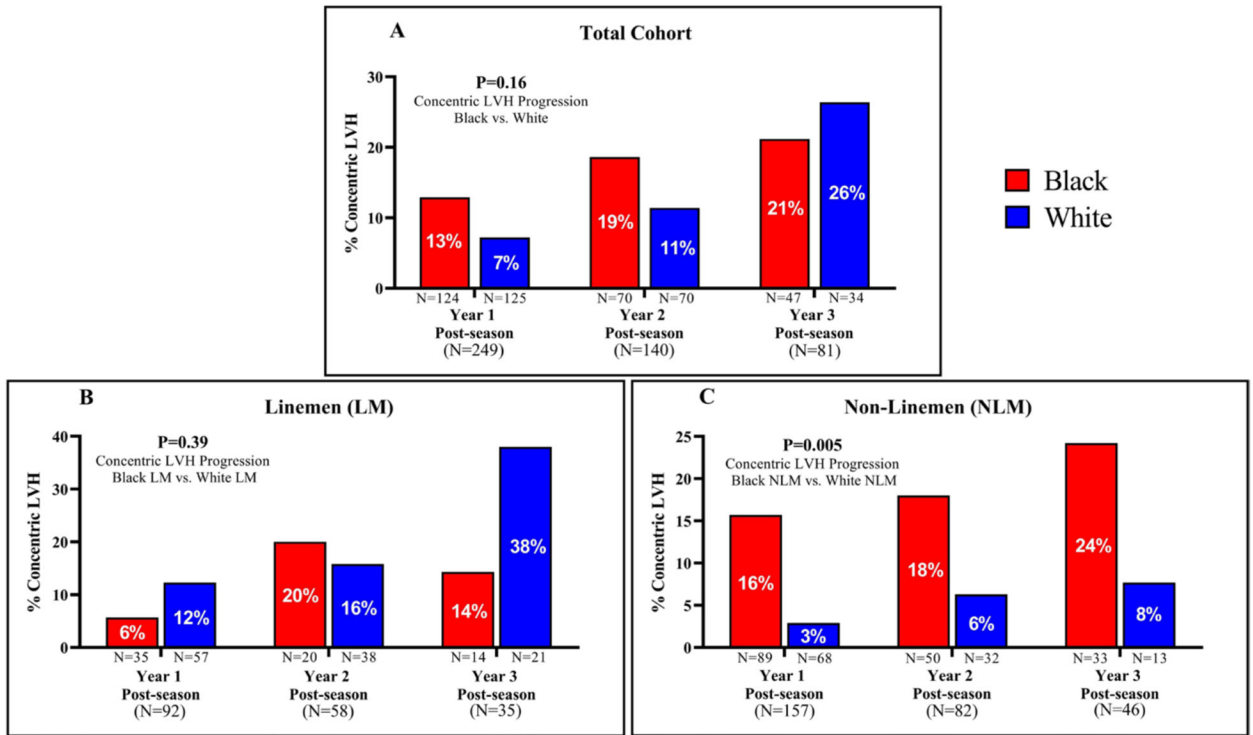
**How might it impact on clinical practice in the future?**

- Black race is associated with accelerated progression of C-LVH, an established cardiovascular risk factor, in ASF NLM. The identification and presence of this structural cardiac phenotype in any ASF athlete, regardless of race, warrants a more thorough cardiac preventive care assessment (ie, hypertension, weight gain) provided to this athletic population.
- Social determinants of health have been under-recognised in the clinical care of young athletes and should be considered in future athlete health outcomes investigations.



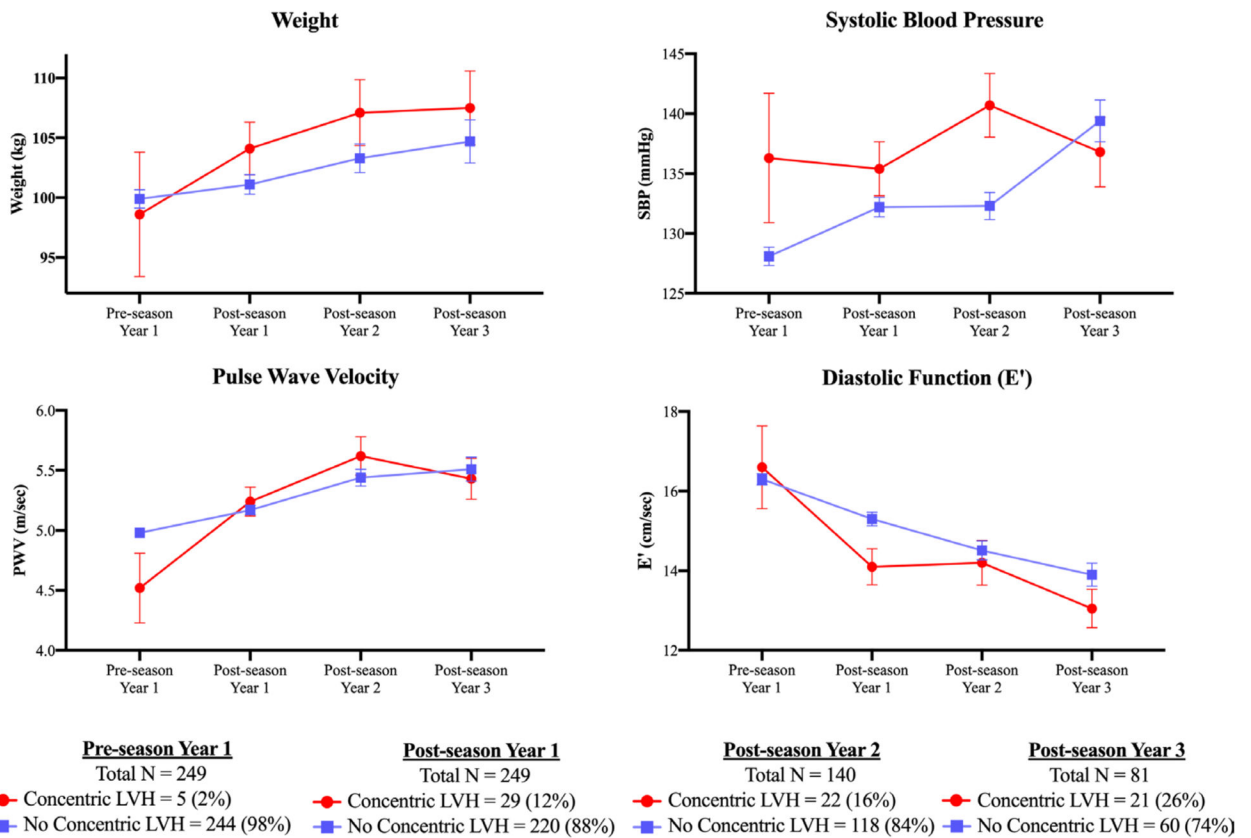
**Figure 1.**

Longitudinal study time points across the US collegiate ASF career, repeated measures studies obtained (clinical characteristics, transthoracic echocardiography and vascular applanation tonometry) and subject numbers and attrition across the study period. \*In year-1, N=51 subjects were excluded due to injury/leaving the team (includes N=2 not self-reported black or white). †In year-1, postseason was 5–6 months after the preseason; remaining time points were 1 full year apart. ASF, American-style football; BP, blood pressure.



**Figure 2.**

Acquired concentric left ventricular hypertrophy (LVH) throughout collegiate American-style football participation and stratified by race. (A) Total cohort: postseason year-1 (black: N=16 vs white: N=9); postseason year-2 (black: N=13 vs white: N=8); postseason year-3 (black: N=10 vs white: N=9). (B) LM: postseason year-1 (black: N=2 vs white: N=7); postseason year-2 (black: N=4 vs white: N=6); postseason year-3 (black: N=2 vs white: N=8). (C) NLM: postseason year-1 (black: N=14 vs white: N=2); postseason year-2 (black: N=9 vs white: N=2); postseason year-3 (black: N=8 vs white: N=1).



**Figure 3.** Comparison of key clinical and cardiovascular measurements (shown as mean (SE)) in ASF athletes with versus without concentric LVH across collegiate ASF participation. ASF, American-style football participation; LVH, left ventricular hypertrophy; PWV, pulse wave velocity; SBP, systolic blood pressure.



**Table 1**

Baseline ASF participant characteristics stratified by race

	<b>Black ASF athletes (N=124)</b>	<b>White ASF athletes (N=125)</b>	<b>P value</b>
Player position			<b>0.005</b>
Lineman	35 (28%)	57 (46%)	
Non-lineman	89 (72%)	68 (54%)	
Family history of hypertension	53 (43%)	34 (27%)	<b>0.005</b>
Height, cm	184 (6)	187 (6)	<b>&lt;0.001</b>
Weight, kg	95.5 (17.4)	104.3 (19.7)	<b>&lt;0.001</b>
Systolic blood pressure, mm Hg	128 (12)	129 (12)	0.30
Diastolic blood pressure, mm Hg	75 (10)	76 (10)	0.45
Concentric left ventricular hypertrophy	2 (2%)	3 (2%)	1
Median household income, \$	54 199 (40 301–60 120)	63 146 (44 654–70 997)	<b>0.006</b>

All values are N (%), mean (SD) or median (25–75 percentile).

Bold indicates p-values were of statistic significance. P 0.05.

ASF, American-style football.

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**Table 2**  
Longitudinal changes in select clinical and cardiovascular measurements stratified by race\*

Characteristic	Black American-style football athletes					White American-style football athletes					P value (overall black vs white)		
	Baseline N=124	Year-1 postseason N=124	Year-2 postseason N=70	Year-3 postseason N=47	Year-1 postseason vs baseline	Year-2 postseason vs baseline	Year-3 postseason vs baseline	Year-1 postseason N=125	Year-2 postseason N=70	Year-3 postseason N=34		Year-1 postseason vs baseline	Year-2 postseason vs baseline
LM (%) / NLM (%)	35 (28) / 89 (72)	35 (28) / 89 (72)	20 (29) / 71 (71)	13 (30) / 70 (70)	-	-	-	57 (46) / 68 (54)	38 (54) / 46 (46)	21 (62) / 13 (38)	-	-	-
Weight, kg	95.6 (93.3 to 97.8)	97.7 (95.4 to 100.0)	98.8 (96.4 to 101.1)	100.3 (98.0 to 102.7)	2.1 (1.4 to 2.8) p<0.0001	3.2 (2.3 to 4.0) p<0.0001	4.8 (3.8 to 5.8) p<0.0001	105.3 (103.4 to 107.2)	108.4 (106.4 to 110.4)	110.6 (108.3 to 112.8)	1.3 (0.3 to 2.2) p=0.010	3.1 (1.9 to 4.3) p<0.0001	5.3 (3.7 to 6.9) p<0.0001
Systolic BP, mm Hg	127 (125 to 130)	131 (129 to 133)	131 (128 to 134)	137 (134 to 140)	3 (1 to 6) p=0.009	3 (0 to 7) p=0.03	10 (6 to 13) p<0.0001	129 (127 to 132)	137 (134 to 139)	142 (138 to 145)	5 (3 to 7) p<0.0001	7 (4 to 10) p<0.0001	12 (8.4 to 16.0) p<0.0001
Diastolic BP, mm Hg	75 (74 to 77)	76 (74 to 77)	76 (74 to 78)	78 (75 to 81)	0 (-2 to 2) p=0.80	1 (-2 to 3) p=0.70	3 (0 to 6) p=0.09	77 (75 to 78)	79 (77 to 82)	78 (75 to 81)	0 (-2 to 2) p=0.89	3 (0 to 5) p=0.04	1 (-2 to 4) p=0.51
PWV, m/s	4.9 (4.8 to 5.0)	5.1 (4.9 to 5.2)	5.3 (5.2 to 5.5)	5.3 (5.2 to 5.5)	0.1 (0.02 to 0.3) p=0.02	0.4 (0.3 to 0.6) p<0.0001	0.4 (0.2 to 0.6) p<0.0001	5.1 (4.9 to 5.2)	5.5 (5.2 to 5.6)	5.5 (5.3 to 5.7)	0.3 (0.2 to 0.4) p<0.0001	0.4 (0.3 to 0.6) p<0.0001	0.4 (0.2 to 0.6) p<0.0001
LVIDD index, g/m <sup>2</sup>	23.5 (23.1 to 23.8)	23.7 (23.4 to 24.1)	23.8 (23.4 to 24.2)	24.0 (23.6 to 24.4)	0.3 (0.03 to 0.5) p=0.03	0.3 (0.01 to 0.6) p=0.04	0.5 (0.2 to 0.9) p=0.004	22.9 (22.6 to 23.3)	23.0 (22.6 to 23.4)	23.1 (22.6 to 23.6)	0.2 (-0.02 to 0.5) p=0.071	0.1 (-0.3 to 0.4) p=0.73	0.1 (-0.3 to 0.6) p=0.54
Average WT, mm	9.0 (8.8 to 9.1)	10.0 (9.8 to 10.1)	10.3 (10.1 to 10.6)	10.7 (10.5 to 11.0)	1.0 (0.8 to 1.2) p<0.0001	1.4 (1.2 to 1.6) p<0.0001	1.8 (1.5 to 2.0) p<0.0001	9.0 (8.9 to 9.2)	10.4 (10.2 to 10.6)	10.9 (10.6 to 11.1)	1.0 (0.8 to 1.1) p<0.0001	1.3 (1.1 to 1.5) p<0.0001	1.9 (1.6 to 2.1) p<0.0001
LV mass index, g/m <sup>2</sup>	90.5 (88.5 to 92.4)	101.5 (99.6 to 103.4)	104.5 (102.1 to 106.9)	111.3 (108.5 to 114.1)	11.0 (9.0 to 13.0) p<0.0001	14.0 (11.6 to 16.5) p<0.0001	20.8 (18.0 to 23.7) p<0.0001	89.9 (87.9 to 91.9)	100.9 (98.9 to 102.9)	110.1 (107.1 to 113.2)	11.0 (9.2 to 12.8) p<0.0001	14.6 (12.4 to 16.8) p<0.0001	20.2 (17.3 to 23.2) p<0.0001
Relative WT	0.36 (0.35 to 0.37)	0.39 (0.38 to 0.40)	0.40 (0.39 to 0.41)	0.41 (0.39 to 0.42)	0.03 (0.02 to 0.04) p<0.0001	0.04 (0.03 to 0.05) p<0.0001	0.05 (0.03 to 0.06) p<0.0001	0.35 (0.34 to 0.36)	0.38 (0.37 to 0.39)	0.40 (0.39 to 0.41)	0.03 (0.02 to 0.04) p<0.0001	0.04 (0.03 to 0.05) p<0.0001	0.05 (0.04 to 0.07) p<0.0001
E', cm/s	16.0 (15.6 to 16.5)	14.9 (14.5 to 15.3)	14.3 (13.8 to 14.8)	14.0 (13.3 to 14.6)	-1.1 (-1.6 to -0.7) p<0.0001	-1.7 (-2.3 to -1.2) p<0.0001	-2.1 (-2.7 to -1.4) p<0.0001	15.4 (15.0 to 15.8)	14.9 (14.4 to 15.4)	13.8 (13.1 to 14.5)	-1.1 (-1.5 to -0.6) p<0.0001	-1.5 (-2.0 to -1.0) p<0.0001	-2.7 (-3.4 to -2.0) p<0.0001

\* Analyses adjusted for player position, values are mean (95% CI).

BP, blood pressure; E', tissue-Doppler averaged mitral annular early diastolic velocities; LM, linemen; LV, left ventricular; LVIDD, left ventricular internal diameter in diastole; NLM, non-linemen; PWV, pulse wave velocity; WT, wall thickness.

Table 3

Factors associated with acquired concentric left ventricular hypertrophy

	<b>Acquired concentric left ventricular hypertrophy</b>	<b><math>\beta</math> (95% CI)</b>	<b>OR (95% CI)</b>	<b>P value</b>
Race		1.28 (0.14 to 2.41)	–	<b>0.03</b>
Player position		1.20 (0.02 to 2.38)	–	<b>0.047</b>
Race×player position interaction		–1.91 (–3.50 to 0.32)	–	<b>0.02</b>
Family history of hypertension		0.014 (–0.73 to 0.76)	1.01 (0.48 to 2.13)	0.97
Weight, kg		0.057 (–0.008 to 0.12)	1.06 (0.99 to 1.13)	0.09
Systolic blood pressure, mm Hg		0.014 (–0.01 to 0.04)	1.01 (0.99 to 1.04)	0.26
Tissue-Doppler E', cm/s		–0.16 (–0.30 to 0.02)	0.85 (0.74 to 0.98)	<b>0.03</b>
Pulse wave velocity, m/s		–0.17 (–0.65 to 0.32)	0.85 (0.52 to 1.37)	0.50
Household income, per \$10 000 increase		–0.18 (–0.38 to 0.02)	0.84 (0.68 to 1.02)	0.08

Factors associated with concentric left ventricular hypertrophy—stratified analyses by race and player position

Table 4

Acquired concentric LVH Player position	OR (95% CI)		P value	OR (95% CI)		P value
	Non-linemen	Linemen		Linemen	White	
Black race	<b>3.70 (1.12 to 12.21)</b>	<b>0.03</b>	<b>0.03</b>	0.53 (0.17 to 1.69)	0.29	
Family history of hypertension	0.83 (0.30 to 2.34)	0.73	0.73	1.21 (0.38 to 3.93)	0.75	
Weight, kg	1.06 (0.94 to 1.18)	0.34	0.34	1.04 (0.96 to 1.13)	0.33	
Systolic blood pressure, mm Hg	1.01 (0.98 to 1.04)	0.57	0.57	1.02 (0.98 to 1.05)	0.40	
Tissue-Doppler E', cm/s	0.86 (0.71 to 1.05)	0.14	0.14	0.85 (0.69 to 1.06)	0.15	
Pulse wave velocity, m/s	0.55 (0.28 to 1.10)	0.09	0.09	1.47 (0.69 to 3.16)	0.32	
Household income, per \$10 000 increase	0.79 (0.59 to 1.07)	0.14	0.14	0.88 (0.66 to 1.17)	0.37	
<b>Race classification</b>	<b>Black</b>			<b>White</b>		
Lineman position	0.46 (0.15 to 1.42)	0.18	0.18	<b>3.40 (1.03 to 11.27)</b>	<b>0.048</b>	
Family history of hypertension	0.92 (0.35 to 2.42)	0.87	0.87	1.21 (0.35 to 4.20)	0.77	
Weight, kg	1.07 (0.96 to 1.19)	0.20	0.20	1.05 (0.96 to 1.15)	0.27	
Systolic blood pressure, mm Hg	1.02 (0.99 to 1.05)	0.28	0.28	1.00 (0.96 to 1.04)	0.89	
Tissue-Doppler E', cm/s	0.87 (0.72 to 1.06)	0.16	0.16	0.82 (0.65 to 1.02)	0.07	
Pulse wave velocity, m/s	0.75 (0.39 to 1.44)	0.39	0.39	1.01 (0.47 to 2.15)	0.99	
Household income, per \$10 000 increase	0.90 (0.70 to 1.17)	0.44	0.44	0.73 (0.52 to 1.02)	0.07	

Bold indicates p-values were of statistic significance. P < 0.05.

LVH, left ventricular hypertrophy.