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A career in sport does not eliminate risk of cardiovascular disease; A systematic review and meta-analysis of the cardiovascular health of field-based athletes.

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Abstract

Objective: To determine the prevalence of cardiovascular disease (CVD) risk factors in current field-based athletes.

Design: Meta-analysis.

Methods: This review was conducted and reported in accordance with PRISMA and pre-registered with PROSPERO. Articles were retrieved via online database search engines, with no date or language restriction. Studies investigating current field-based athletes (>18years) for CVD risk factors according to the European Society of Cardiology and American Heart Association were screened. Full texts were screened using Covidence and Cochrane criteria. Eligible articles were critically appraised using the

AXIS tool. Individual study estimates were assessed by random-effect meta-analyses to examine the overall effect.

Results: This study was ascribed a 1b evidence level, according to the Oxford Centre for Evidence-based Medicine. 41 studies were identified, including 5,546 athletes from four sports; American football; soccer; rugby and baseball (mean ages:18-28). Despite participation in sport, increased body mass was associated with increased total cholesterol, low-density lipoprotein, triglycerides, hypertension, systolic blood pressure, and decreased high-density lipoprotein. Linemen had increased prevalence of hypertension compared to non-athletes. Conflicting findings on fasting glucose were prevalent. There were inconsistencies in screening and reporting of CVD risk factors. Sport specific anthropometric demands were associated with elevated prevalence of CVD risk factors, most notably: elevated body mass; dyslipidemia; elevated systolic blood pressure and; glucose

Conclusions: There are elevated levels of risk for CVD in some athletes, primarily football players. Lifestyle behaviours associated with elite athleticism, particularly football linemen potentially expose players to greater metabolic and CVD risk, which is not completely offset by sport participation.

Keywords: Cardiovascular; athlete; risk-factors; evidence-based review; heart disease

Introduction

While clinical cardiovascular disease (CVD) is rare among young, highly active athletes, they are exposed to known risk factors such as increased body size, elevated blood pressure (BP) and abnormal lipoprotein profiles.[1,2] Athletes represent a unique cohort of adults who engage in known healthy behaviours to maximise performance. However, certain behaviours are associated with CVD risk factors, particularly in sports where size is important, such as American football and rugby.[1,2] In sports where body size is integral to successful participation, athletes often pursue extreme solutions to gain a competitive advantage that can jeopardise their long-term cardiovascular health. This contributes to existing concern surrounding the cardiovascular implications of elite athletes with a

playing time body mass index (BMI) above 30 kg.m^2,[3] and morphologic adaptations of an athlete's heart.[4] Despite American football players having a lower overall mortality risk, the NIOSH study revealed that linemen had a 52% greater risk of dying from CVD than the general population.[5] Increasing player size and sporadic deaths of active young retired professional athletes [6] warrants timely investigation into the cardiovascular health of current field-based athletes.

Therefore, the purpose of this paper was to systematically review the evidence on the cardiovascular health and risk factors for CVD in current sportsmen and sportswomen, and to investigate the influence of other factors associated with CVD including, obesity, hypertension, dyslipidemia, insulin resistance and cardio-metabolic syndrome.

Methods

This review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-analysis (PRISMA) statement.[7] (www.prisma-statement.org) and was registered with PROSPERO, a registry of systematic reviews. Registration is available at https://www.crd.york.ac.uk/prospero/; registration number: CRD42017077885.

Articles were retrieved via online database search engines, including; CINAHL, EMBASE, Pubmed, and WOS. The reference lists of all reviews and meta-analysis related to the cardiovascular health and A systematic literature search was conducted using the online databases of CINAHL, EMBASE, Pubmed, and WOS from their inception to November 2019. No search restrictions were imposed. The search strategy key words, MeSH terms and combinations of these words included, CVD, cardiovascular health, blood pressure, lipids, cholesterol, cardio-metabolic syndrome, hypertension, glucose intolerance, body composition, BMI, body fat percentage, low-density lipoprotein, high-density lipoprotein, triglycerides, total cholesterol, sleep-disordered breathing, field-based athlete, American football, baseball, field hockey, rugby, GAA and soccer. Studies include human subjects only. Studies were identified that could provide information on the prevalence of known CVD risk factors according to the European Society of Cardiology[1] and American Heart

Association.[2] All study designs were included. Participants were currently engaged in a field-based sport and over 18 years. The electronic database search was supplemented by a manual search of the reference lists of all reviews and meta-analysis related to the cardiovascular health and articles meeting the eligibility criteria. The authors of studies that presented data incorporated with components from inclusion criteria were contacted for further information relevant to this review.

The search methodology and process are described in Figure 1. The title and abstracts of the retrieved studies were independently screened in accordance with the pre-defined inclusion criteria. Following this, two reviewers independently assessed full texts. A third reviewer was available to make a final decision if consensus was not reached. Eligible articles were screened in a full text using Covidence (https://www.covidence.org/home) and the AXIS tool was used for critical appraisal.[8] Data extraction from selected studies was conducted using STROBE guidelines (Appendix A).[9]

A meta-analysis was deemed appropriate to examine the overall effect. Heterogeneity between studies was determined by the I^2 statistic, [10] as an indicator of the proportion of total variation in estimates that is caused by heterogeneity. I^2 values of 25%, 50% and 75% correspond to low, moderate and high degrees of heterogeneity. Sensitivity analysis was implemented where high levels of heterogeneity ($I^2 > 75\%$) were detected.

Results

The results from the literature search and selection of articles are summarised in Figure 1. Overall, the search retrieved 1,828 publications. A title screening for duplicates left 1,816 papers for abstract review. Review of abstracts left 233 papers for full text screening. Of 233 studies, 152 were excluded as study outcomes were not relevant to traditional cardiovascular health assessment, for example; electrocardiogram and/or echocardiogram. Thirty-two studies were excluded because participants included cohorts beyond inclusion criteria; data amalgamated with participants less than 18 years or athletes were retired. Three studies were removed due to incompatible study design. Authors of five studies were contacted for further information and data applicable to this study. Overall, 41 studies met the criteria.

Of relevant studies, 28 were cross sectional, 6 descriptive, 2 observational, 2 prospective-longitudinal, and 1 randomised control, pre- and post- test and retrospective study design. Thirty-nine studies included male participants and 2 included female participants. Thirty studies included American football athletes (29/41), 8 from soccer (8/41), 3 from rugby (3/41) and 1 from baseball (1/41). Of the 30 American football studies, 13 included professional football athletes and 17 included collegiate athletes. Athletes were compared with age-sex-BMI matched non-athletic individuals and/or cohorts from the Coronary Artery Risk Development in Young Adults CARDIA [11] and the National Health and Nutrition Examination Survey 2000 (NHANES).[12] Analysis was carried out to compare risk factors based on playing position, race and the presence of cardio-metabolic syndrome (Appendix A). The primary aim of these studies was to assess the prevalence of CVD risk factors, dissimilarity in the prevalence and severity of risk factors based on race and playing position, and the role of body composition on players CVD risk factor profile.

Body Composition:

Thirty-three studies measured body composition, 30 of which assessed BMI. Football and rugby players had a greater mean BMI than comparators.[13-20] Sub-group analysis of football players found that linemen position was associated with a significantly greater BMI than non-linemen.[17,19,21-28] Ninety percent of studies reported that linemen had BMI ≥30 kg.m^2; many of which reported a BMI exceeding 32 kg.m^2.[21,22,24,27] Baseball players had a lower percentage of athletes with BMI ≥30 kg.m^2 compared to controls and football players (Appendix B).[29] Soccer players had a similar mean BMI to controls.[30,31]

Rugby players had a significantly greater body fat percentage than race-walkers but lower than sedentary controls.[13,14] Mixed findings were reported in football when compared to controls; two studies reported lower and one study reported a greater body fat percentage for players (Appendix B).[32-34] Nine studies reported a greater body fat percentage for linemen compared to non-linemen (Appendix B).[17,23-25,32,35,36] Mean body fat percentage values for collegiate athletes was greater than

25%.[37] One study reported lower mean body fat percentage in female soccer players compared to controls.[30]

Fourteen studies included waist circumference as a measure of body composition. Baseball players had a significantly lower percentage of athletes with waist circumference ≥100cm compared to football players and controls.[29] Three studies reported higher waist circumference values for football players than controls (Appendix B).[16,17,38] All studies reported that linemen had a significantly greater waist circumference than non-linemen (Appendix B). [17,22,24,25,27,35,38] In collegiate football players, Division III players had a significantly lower waist circumference than players from division I and II.[37] Five studies included waist-to-hip ratio as an outcome measure. A similar waist-to-hip ratio was reported for soccer players compared to sedentary controls (Appendix B).[31] Baseball players had significantly lower percentage of athletes with waist to hip ratio >0.5 compared to footballers and controls.[29] Three studies assessed waist to hip ratio in football players, all reporting a higher value for players compared to controls.[16,17,38]

All studies on football reported a greater prevalence of BMI >30kg.m^2, WC >100cm, WHR >0.5 and BF% >25% compared to other athletes and controls. Sub-group analysis found elevated measures of body composition for linemen compared to non-linemen. Baseball and rugby had similar measures of body composition to controls, whereas, soccer athletes had lower body fat percentage than controls.

Hypertension:

Ten studies reported a prevalence of hypertension ranging from 13.8% to 53% across all field-based athletes. A higher prevalence of hypertension for football players,[16,18,28,38] and baseball players was reported compared to controls.[29] Rates of pre-hypertension were significantly greater for athletes compared to controls, except for one study that reported a lower prevalence (61.9% v 64.4%).[18] Linemen had higher rates of hypertension than non-linemen in all studies.[25,28,38] Analysed by race, black college football players had a prevalence of hypertension at 78% compared to 63% for white players.[39]

In summary, the prevalence of hypertension and pre-hypertension was greater for baseball and football players compared to non-athlete controls. Linemen had a similar higher prevalence of hypertension and pre-hypertension compared to non-linemen.

Blood Pressure:

Most studies measuring BP were on a football cohort. Football players had higher BP than controls in four studies;[17,18,20,34] although one study reported lower BP than BMI matched controls (Appendix C).[33] When the influence of football playing position was analysed, higher BP for linemen compared to non-linemen was reported (Appendix C).[17,19,22,23,25,27,28,32,35] Soccer players were found to have significantly higher systolic BP,[40] and lower prevalence of optimal BP than controls (Appendix C).[41] In players where cardio-metabolic syndrome was present, resting systolic BP and diastolic BP was greater.[42,43] Race was not associated with elevated BP amongst football players.[16,19]

In summary, the studies in our review predominately measured BP in American football and soccer athletes, who showed significant BP elevation compared to controls. BP increased with body mass.

Lipid Profiles:

Twenty-nine studies assessed measures of lipid profile. Soccer, football and rugby players had lower or equivalent measures of *total cholesterol* compared to controls (Appendix C).[13-15,19,30,31,33,34,44,45] *HDL* was measured in baseball and football. Baseball had a lower percentage of players with high HDL levels (>40mg.dl) compared to controls.[14,15,29] Football players had similar HDL values as controls in four of six studies.[17,32,33,46]

Studies examining football found elevated *LDL* values comparable with controls.[16,17,19,34] In contrast, rugby players,[14,15] and male soccer players,[31,45] had lower mean LDL values compared to control groups; 93.5mg.dl and 102.95mg.dl, respectively. Similar values for female soccer players and controls was reported (Appendix C).[44]

Mixed findings were reported when mean *triglyceride* levels were measured in football; three studies reported lower values [19,34,38] and three reported higher values compared to control groups.[17,32,33] Baseball players had lower prevalence of high triglycerides compared to controls and football players.[29] In the presence of cardio-metabolic syndrome, athletes had significantly higher triglyceride values.[42]

Comparison of position of play in football showed that linemen position was reported with higher total cholesterol in 3 studies [19,22,35] and similar values in two studies compared to non-linemen.[16,17] Nine studies reported higher HDL values for non-linemen compared to linemen (Appendix C).[17,19,22,23,25,27,32,35,38] Six studies reported higher values of elevated LDL[17,19,22,23,25,35] and six reported higher triglyceride values for linemen compared to non-linemen.[17,19,21,22,35,38]

When race was analysed, black players had increased total cholesterol compared to white players but lower than Asian players (Appendix C).[19,47] Black race was associated with higher HDL values than white and Asian players.[16,19,47]

In summary, athletes from baseball, soccer and rugby were found to have a more favourable lipid profile than football players and non-athlete controls. The studies in our review reported an inverse relationship with HDL and a direct relationship with total cholesterol, LDL and triglycerides as body mass increased.

Glucose:

Conflicting findings were found within and between sports. Significantly lower mean fasting glucose (FG) and lower prevalence of impaired FG for football athletes compared to controls were reported.[16,17,33] Although, other studies reported higher FG levels for football players compared to controls (Appendix C).[32,34] In the same sport, a higher percentage of players with FG ≥100mg.dl was reported compared to controls.[33,38] Baseball players had a decreased prevalence of FG ≥100mg.dl compared to controls and football players.[29] Rugby players had similar fasting glucose to controls.[15] When player position was analysed, higher FG levels were reported for linemen compared to non-

linemen (Appendix C). [23,25,27,32,35] When cardio-metabolic syndrome was present, significantly higher FG was reported for football players.[42,43]

In summary, findings for FG for football and rugby players were inconsistent. As body mass for football players increased high FG levels were found.

Cardio-metabolic Syndrome and Sleep-disordered Breathing:

Prevalence of 19-22% for cardio-metabolic syndrome for football players was reported. [27,29,38,42] When football playing position was analysed, studies reported a higher prevalence of cardio-metabolic syndrome in linemen compared to non-linemen.[22,29,35,36,38,42] The most prevalent components of cardio-metabolic syndrome reported in athletes were elevated waist circumference/BMI, increased BP and low HDL values.[35,37] When between sport comparison was made, baseball players were found to have a lower prevalence of cardio-metabolic syndrome compared to controls and football linemen, but higher prevalence than non-linemen.[29] Two studies reported a prevalence of mild sleep-disordered breathing of 8% and 19%, respectively which was not influenced by playing position in football athletes.[48]

In summary, cardio-metabolic syndrome was predominately assessed in football players. As body mass increased a greater prevalence of cardio-metabolic syndrome was reported. Linemen position was not found to influence the prevalence of sleep-disordered breathing.

Critical Appraisal and Level of Evidence:

This study was ascribed a 1b level of evidence, according to the criteria of the Oxford Centre for Evidence-based Medicine.[49] Each study was attributed a level of evidence by measuring the reliability and quality of evidence for key outcomes across comparisons was evaluated according to the AXIS tool criteria.[8] The AXIS tool identifies twenty domains to determine the quality of a study. Overall, studies in this review were of moderate quality with common issues in several domains. Studies did not justify sample size as they were generally pilot, cross-sectional or observational in nature. Samples of convenience were sought, and studies were not clear as to how representative these

samples were to the true population, likely to be an elite population. Studies did generally not identify funding sources, although it is unlikely to influence outcomes where there was no intervention. Where studies were assigned 'unsure' was generally due to incomplete reporting and where authors did not respond to clarify information (Appendix D).

Meta-analysis:

Implementation of meta-analysis using random-effects indicated that the overall effect of engagement in elite sport across all participants for systolic BP, glucose and HDL was not homogenous (I^2 – 98%, 95% and 91%, respectively). Heterogeneity for FG remained high (I^2 -79%) for soccer and rugby studies following the removal of American football athletes through sensitivity analysis. There was an insufficient availability of studies to implement this sensitivity analysis for HDL and systolic BP. Several studies that analysed triglyceride levels between athletes and controls found a significant mean decrease of -3.78mg.dl (95% CI: -12.21, -4.65, I^2=62%) in athletes (Appendix E). Studies that analysed American football players based on playing position; linemen and non-linemen found a significant mean decrease in FG of 3.34mg.dl (95%CI: 0.62, 6.06, I^2=60%), systolic BP of 6.02mmHg (95%CI: 4.41, 7.63, I^2=31%) (Figure 2), LDL of 7.54mg.dl (95%CI: 3.10, 11.99, I^2=1%) (Figure 3), and triglycerides of 19.12mg.dl (95%CI: 9.66, 28.57, I^2=60%) in non-linemen (Appendix E). Greater HDL concentrations were found for non-linemen, with mean difference of -6.93mg.dl (95%CI: -8.78, -5.08, I^2=15%) (Appendix E).

Discussion

In this review, studies predominately measured American football athletes, with limited studies from other field-based sports. Several elevated risk factors in active field-based athletes were identified, primarily in American football players, [16,33,36,37,42] with reduced prevalence in players from other sporting disciplines.[13-15,29-31,40,41,44,45,47] Despite reduced risk in athletes from rugby, soccer and baseball, athletes with larger body mass, display higher prevalence of CVD risk factors, possibly reflecting the established relationship with increased BMI.[1,2] However, this postulation is based on general population where presumption of greater adiposity, not lean mass. Research is conflicted on

the cardio-protective benefits of exercise where elevated BMI is present; although beneficial, exercise does not eliminate risk of future cardiovascular events.[50] It is apparent that CVD risk factors are present and there is a need for a greater amount of research.

There is a predilection of cardiovascular related research on athletes to concentrate on American football athletes. American football is graded as a class 2B sport; moderate static and dynamic stress,[51] and is a heterogenous group and can be dichotomised by playing position; linemen and non-linemen. There appears to be greater concern for linemen, given their size and the repetitive blunt trauma due to high impact collisions and tackling. Elite athletes often engage in extreme lifestyle behaviours to gain a competitive advantage. In sports, such as American football and rugby where size is pivotal, these behaviours can include, deliberate body mass gain, through use of high-caloric diets.[52] Although this is not generalisable to all field-based sports and indeed all athletes, the long-term cardiovascular implications of prolonged engagement in these behaviours of those who require a large body size has not been established. Furthermore, the use of non-steroidal anti-inflammatory drugs, opioid-based analgesics and surreptitious use of performance-enhancing drugs remain incompletely understood in relation to cardiovascular health.[3] A recent systematic review of the cardiovascular health of retired field-based athletes suggested the prevalence and severity of CVD risk factors in retired athletes is influenced by their playing time body mass and playing position. [53]

Body Composition:

Epidemiological research has consistently reported increased risk of cardiovascular death with increased BMI in the general population.[54] Players with playing-time BMI of ≥35kg.m^2 have a significantly greater incidence of CVD mortality than the general population.[3] Elevated BMI (≥ 30 kg.m^2) was more prevalent in football players,[16-20,28] and particularly linemen. [17,19,21-28] Athletes engaged in contact collisions; linemen in NFL and props in rugby tend to have higher body mass. Position specific body mass increases has the potential to expose these players to cardiovascular health risks in the long-term as they may reach a point where increased body mass is not caused by

increased lean muscle mass but rather body fat. Furthermore, football athletes reported a greater prevalence of waist circumference ≥100cm, body fat percentage ≥25% and waist to hip ratio ≥0.5.

Eleven studies found a positive association between increasing BMI and body fat percentage for linemen and non-linemen and inter-divisional at collegiate level.[13,16,17,22-25,27,34,35,48] Interestingly, four studies indicated that despite increasing body fat with increasing BMI, body fat percentage in athletes was lower than expected.[13,17,25,55] Findings suggest that exercise, although beneficial may not prevent heavier players from developing CVD risk factors. Precision of body fat outcomes are dependent on the methods implemented, allowing for speculation on accuracy when comparing findings.[56] Mean waist circumference for all football players and larger players (99.24cm and 107.9cm, respectively) exceed proposed cut-off points.[1] Furthermore, 14% of football players and 71% of linemen with body fat percentage % ≥25% [16,27] and the 38% of football players and 95% of linemen with waist circumference ≥100 cm.[29,38] It remains unknown if athletes with measures exceeding proposed cut-off points are exposed to the same CVD implications seen in the general population.

Overeating is necessary for increasing body mass, potentially increasing the risk of elevated body fat and visceral fat which can negatively impact the metabolic health of the athlete.[36] Due to the vast number of cofounding factors it is not possible to indicated that the presence of CVD risk is exclusively caused by excess weight. It is assumed that elite athletes are attuned to their overall well-being. However, the demands of elite sports often cause additional stresses. Nattiv et al reported that collegiate athletes had a significantly higher proportion of maladaptive lifestyle behaviours, including overeating, steroid use, use of alcohol and drugs.[57] Given the high level of alcohol and substance use reported in collegiate athletes, and elevated use in retired NFL players,[58] it is not appropriate to eliminate these as a possible causes of cardiovascular mortality in this population.

Blood Pressure:

There is a strong relationship between elevated BP in early adulthood and CVD in later life;[59] however, this association is less clear in athletes. This review identified a greater prevalence of

hypertension and pre-hypertension for football players compared to other athletes and controls. A high prevalence of pre-hypertension; a recognised risk factor for CVD,[1] was consistently reported, particularly for collegiate football players.[18,32,33,42,60] An association between current NFL players and increased prevalence of hypertension (13.8%) compared to age-and-sex matched controls (5.5%) was identified.[16] The direct comparison of football players with endurance-based athletes indicates that development of hypertension and increased BP is not a uniform response to all forms of high-intensity exercise (Appendix C).[28] It is plausible that increased BP is a by-product of high-intensity strength-based training and therefore, reversible during retirement.

Reporting of higher mean systolic BP for football and soccer players compared to controls was common. Elevated systolic BP may be due to increased resting stroke volume and cardiac output associated with elite athleticism.[51,59] It is possible that athletes' body composition plays a role in elevated resting systolic BP, irrespective of playing position.[18,25,26,35] However, linemen playing position was predominately associated with increased BP and hypertension.[16,17,19,23,27,28,32,33,36,38] A multitude of factors may explain this; including, long term use of non-steroidal anti-inflammatory drugs, strength and resistance training, stimulant use, and pre-existing cardiovascular risk factors.[3,4] Findings from the meta-analysis indicate more favourable systolic BP for non-linemen (Figure 2), highlighting negative implications associated with position specific demands. Players of different races experience elevated measures of BP and higher rates of hypertension and pre-hypertension compared with age-and-race equivalent controls from the CARDIA study.[16,26] The recent re-classification of hypertension from140/90mmHg to 130/80mmHg dramatically increases the number of athletes with elevated BP and hypertension.[61] Although the pathophysiology of hypertension differs from the general population, long-term exposure may lead to similar negative effects on arterial function and increased risk of premature CV mortality.

Lipid Profiles:

Increased measures of body mass were found to be associated with an elevated prevalence of dyslipidemia; a direct relationship with total cholesterol, LDL, triglycerides and an inverse

relationship with HDL.[16,21,33,36] The Canadian Heart Health Surveys Research Group supports our finding that dyslipidemia primarily affects linemen, possibly due to increased body size.[54] Athletes with optimal body fat percentages were reported with a more favourable lipid profile compared to other athletes,[22,23,25,35] and controls, despite higher BMI.[13,14] Controls were predominately matched for BMI; potentially underestimating the beneficial effects of exercise and justification for lack of significant differences.

The majority of studies found no differences in prevalence of elevated LDL between football players and controls.[16,17,19] However, controls had significantly higher prevalence of LDL above recommended cut-off levels than athletes.[2,33,36] Linemen have higher LDL values than non-linemen, with a mean value of 111.7mg.dl,[17,19,22,23,25,35] suggesting although players are engaged in high-intensity exercise, elevated body mass may counteract benefits of exercise on plasma LDL.[21] The Forest plot for LDL (Figure 3) identified a common positive effect of non-linemen position on LDL levels, suggesting elite athletes competing at lower body masses have lower LDL levels. Despite similar total cholesterol values for linemen and non-linemen,[16,17] non-linemen had greater mean HDL values (Appendix C).[17,19,22,23,25,27,32,35,38] This supports the claim that increased BMI has an inverse relationship with HDL.[2,54] Despite conflicting results concerning triglyceride values, there is a strong association between increased BMI and triglyceride levels.[16,17,19,21,22,32,33,35,38] Large confidence intervals are observed for triglycerides between athletes and controls; however, there was a significant mean difference with athletes having lower values. Studies where athletes were found to have elevated triglyceride levels include football players and those with lower triglyceride levels than controls were predominately soccer players.

Glucose:

Findings on glucose are conflicting. It is unclear as to why non-linemen have similar or marginally lower mean FG values as linemen [23,25,27,32] and higher prevalence of players with FG ≥100mg.dl than controls, given their significantly lower BMI (Appendix B; Appendix C).[16] A possible explanation for similar or marginally lower FG levels despite significant difference in body composition is

similarities in dietary lifestyles of players during playing career. The increased BMI and high-caloric diet in the cohort poses a risk for hyperglycemia leading to insulin resistance, an underappreciated factor in CVD development.[1]

Cardio-metabolic syndrome:

A major finding of this review was the lower mean HDL values and lower percentage with HDL ≥40mg.dl in football players. [16,29,33,38] Buell and Mansell reported that elevated waist circumference/BMI, increased BP and low HDL values were the most prevalent components of cardio-metabolic syndrome.[35,37] Standard metabolic dysfunctions which typically coincide with obesity cannot be presumed to be present in athletes with elevated BMI. However, this appears to not be the case from findings in this review. Football linemen predominately aged between 20-30 years, exhibit multiple metabolic dysfunctions compared to non-linemen and age-sex-matched controls.[22,29,35,36,38,42] Persistent reporting of elevated waist circumference, body fat percentage and waist to hip ratio is significant given the role of obesity in development of cardio-metabolic syndrome and CVD. Thus, can engagement in sport offset the risk of the CV related health risks associated with elevated body mass? C-reactive protein (CrP) is a moderate predictor of cardiovascular health, [62] yet only two studies within this review analysed it. Given the association between high CrP, elevated BMI and elevated triglycerides,[62] both evident in this review, further investigation is warranted.

This review is predominated by American football athletes; therefore, it is important to mention the reported harmful behaviours associated, particularly the use of stimulants.[63] Speculation of stimulant use among athletes has long persisted. A recent meta-analysis found that the global prevalence rate of anabolic-androgenic steroids (AAS) use in elite athletes was 13.4%. [64] There is a notable absence of research reporting the level of AAS use in athletes given their illegal status. Horn et al., indicated that 9.1% of retired players self-reported using AAS during their career. [63] Growing evidence indicates negative effects of AAS on CVD risk factors. Studies have reported that AAS users have increased resting and exercise systolic BP; [65] negative alterations in lipid profiles; decreased HDL, increased LDL; [66] significant increase in CRP. [67]

Limitations:

This review is limited by several factors. Studies did not analyse the same cardiovascular measures, and incorporated multiple methods of investigation, most notably for body fat percentage. Most studies included were cross sectional, limiting ability to infer causality, therefore, findings should be viewed as hypothesis generating only. Studies predominately included male American football athletes, limiting generalisability. Therefore, caution is needed when applying findings to other current field-based athletes and female athletes. There is a lack of longitudinal and follow-up research tracking current athlete's cardiovascular health into retirement. Finally, there are several possible co-founding measures that were not assessed, including cardiovascular health and body composition prior to playing, years playing, diet, alcohol use, AAS use, socioeconomic status, education, genetics and/or use of medications.

Conclusion:

Many current athletes exhibit multiple risks for future CVD, confirming a need for further research. Elevated levels of risk have been clearly identified in active athletes, primarily football players, with reduced prevalence in players from other sporting disciplines. Lifestyle behaviours associated with elite athleticism, particularly football linemen, potentially expose players to an increased metabolic and CVD risk. Athletes at increased CVD risk have elevated body mass and/or BMI, which is similar to research findings in the general population. Attention to larger athletes is needed for preparing them for retirement in terms of education on dietary habits and remaining engaged in physical activity.

Declarations of interest: none

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Contributors: CM was first author. FW and KH are the two co-authors to this review. FW was involved with selection process of articles. She conducted methodology quality assessment for each study and edited the review. KH was involved in the study design and edited this review. DD edited the review. JC was involved in risk of bias analysis and quality assessment.

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Figure Legends

Figure 1: PRISMA flow diagram depicts the flow of information through each stage of the screening process of articles retrieved from online databases and the selection of eligible articles.

Figure 2: Forest plot examining the overall effect on systolic BP between football players based on playing position, categorised as linemen and non-linemen.

Figure 3: Forest plot examining the overall effect on LDL between football players based on playing position, categorised as linemen and non-linemen.

Figure 4: Forest plot examining the overall effect on triglycerides between athletes and non-athlete controls.

Figure 5: Forest plot examining the overall effect on Glucose between football players based on playing position, categorised as linemen and non-linemen.

Figure 6: Forest plot examining the overall effect on HDL between football players based on playing position, categorised as linemen and non-linemen.

Author	Study design	Aims	Setting	Participants	Variables	Risk factor prevalence
Tucker et al., 2009	Cross sectional	• To assess CVD risk factors in NFL players and compare with the CARDIA study. • To assess the association of risk factors with player size and race.	12 NFL athletic training facilities - April and July 2007.	NFL, n=504 Males Mean Age: 26.7	BMI, BF%, WC, WHR, SBP, DBP, HT, TC, TG, HDL, LDL, IFG, Smoking	Lower prevalence of IFG (6.7% v 15.5%) in NFL. Greater prevalence of HT (13.8% v 5.5%, p<0.001) and pre HT (64.5% v24.2%, p<0.001) in NFL. No difference in lipid profile.
Selden et al., 2009	Cross sectional	• To assess the prevalence of CMS in current NFL players.	University of Missouri-Kansas City.	NFL, n=69 Males Mean Age: 25	BMI, WC, WHR, TC, HDL, LDL, TG, TG/HDL ratio, IFG, BP, CMS	Equal prevalence of CMS between NFL and controls. CMS higher in LM than NLM. Higher FG in NFL (p<0.001). Increased BMI and HT prevalence in NFL players.
Borchers et al., 2009	Cross sectional	• Evaluate a cohort of division 1 collegiate football players to estimate prevalence of obesity, CMS and IR. • Evaluate the relationship between obesity CMS and IR – the risk for LM compared to other positions for these clinical entities.	Ohio State University (OSU) Clinical Research Center - August and October 2007	NFL, n=90 Males Mean Age: 20.10	BMI, WC, BF%, SBP, DBP, Fasting insulin, FG, TC, HDL, LDL, TG, HbA1C, QUICKI	100% with CMS and obese were LM. SBP (p= 0.0011), Insulin (p< 0.0001), Cholesterol (p< 0.0001), HDL-C (p< 0.0001), LDL-C (p< 0.0001), TG (p= 0.0029), WC (p<0.0001), were significantly associated with BF%.
Dobrosielski et al., 2010	Cross sectional	• To compare CVD risk factors, CV structure, function and parameters stratified by position. • To examine a vascular index of subclinical CVD-providing a comprehensive risk factor profile	Wake Forest University, North Carolina, 2010.	NFL, n=26 Males Mean Age:21	WC, BF% SBP, DBP, TG, HDL, Glucose, LV mass	50% of the LM and no NLM met CMS criteria. All LM exceeded WC threshold >90 cm and 11 LM were either preHT or HT.
Garry et al., 2001	Observational	• To evaluate the lipid-lipoprotein profiles in a group of professional football players. • To determine what association exists between these profiles and the players' BMI. • To assess the relationship between BMI and lipoprotein profiles and playing position.	The East Carolina University.	NFL, n=70 Males Mean Age: 26.9	BMI, ECG, HR, TC, HDL, LDL, TG and HDL/TC ratio.	BMI-NLM: 27.2; 31.2 kg.m2 V 38.1 kg.m2 LM (p<0.001). Players with BMI ≤ 28 kg.m2 and >28 kg.m2 demonstrated a difference for HDL (p<0.01) and TC/HDL ratios (p<0.01).

Mansell et al., 2011	Cross sectional	• To determine if college football LM exhibit characteristics of CMS. • To compare caloric intake levels based on playing position.	University of Saskatchewan, 2009.	NFL, n=39 Males Mean Age: 21.4	BMI, BF%, WC, TC, LDL, HDL, TG, SBP, DBP, FBG	14% of LM and no NLM met CMS criteria. Compared to NLM, LM had higher WC (108 Vs 28.9cm, p<0.001), higher BF% (26.4% vs 11.2%, p<0.001), lower mean HDL (0.93 VS 1.12 mmol/L, p=0.021) and higher FBG (5.22 Vs 4.77 mmol/L, p<0.001). No significant difference for BP, TC or TG.
Steffes et al., 2013	Cross sectional	• To determine the prevalence of CMS risk factors in High school and college football players. • To determine if this prevalence varies according to BF%.	Miami University.	NFL, n=82 Males Mean Age: 19.8	WC, BF%, BMI, TC, HDL, LDL, TG, BG, BP, MAP, MS	BF% was a significant predictor of mean arterial BP, HDL and WC. MS exists in collegiate players, with almost all in athletes with the highest %BF. Significant differences were observed by playing position for SBP, HDL and WC.
Allen et al., 2010	Cross sectional	• Analysis of NFL players by size for CVD risk factors. • Analysing the occurrence of CVD risk factors based on playing position.	University of Oklahoma- off season mini-camp, April-July 2007.	NFL, n=504 Males Mean Age: 26.65	BMI, BF %, WC, WHR, SBP, DBP, HDL, LDL, TC, TG, Glucose	SBP and DBP were significantly higher in the NFL group. LM had significantly lower HDL, higher TG than CARDIA. LM significantly elevated BP, lower HDL and higher TG than NLM.
Berge et al., 2013	Case control	• To identify the prevalence of high BP in male professional football players • To examine the players' compliance to recommended follow-up of high BP. • To investigate if indicators of sympathetic activity were increased in players with high BP.	La Manga, Spain - October 2010 until February 2011.	Soccer, n=26 Males Mean Age: 25.9	BMI, HR, OBP, ABP, MAP, HR and PP.	No differences in percentage with optimal BP. Controls had higher DBP (68.3±6.2 v 65.5±6.4 mmHg; p<0.05) and MAP (83.5±4.7 v 81.2±5.2 mmHg; p<0.05).
Buell et al., 2008	Cross sectional descriptive	• To identify the incidence of CMS in football LM at the NCAA Division I (DI), II (DII) and III (DIII) levels. • To document fasting insulin, CRP and glycosylated haemoglobin (HbA1c) level.	Ohio State University. Pre-season training camp, 2006.	NFL, n= 70 Males Mean Age: 20.2	WC, %BF, FBG, Fasting insulin	Body size increases with NCAA division. %BF of DII players was less lean than DI and DIII. FBG was lower in the DIII group than DII. DIII lower fasting insulin levels than DI and DII. 34 of 70 qualified for CMS.
Haskins et al., 2011	Cross sectional observational	• To investigate obesity, T2DM and hypercholesterolemia in a cohort of obese-classified collegiate	University of Wisconsin.	NFL, n=30 Males Mean Age: 19.9	BMI, BF%, BP, LDL, HDL, TG, TC, Glucose, Insulin.	LM had lower SBP and DBP but did not differ in other continuous variables, such as LDL, HDL and TG. BF% significantly correlated with every risk factor except glucose.

		football LM compared with sedentary age-matched and size-matched controls. • To investigate the relationship between fitness, obesity, and the risk factors of T2DM.				
Ahrensfield et al., 2012	Cross sectional	• To assess CIMT as an integrated index of CV risk.	MedStar Health Research Institute, Washington, DC. Mini training camps held between April and June 2007.	NFL, n=124 Males Mean Age:27.5	BMI, BF%, HDL, LDL, TG, TC, Glucose, SBP, DBP, IMT (mm), RCAA (mm).	CIMT higher in LM than NLM (0.65 v 0.62). Modest association between CIMT and; BMI (r=0.29; p=0.001), Weight (r=0.21; p=0.020) and WC (r=0.29; p=0.049). CIMT was not correlated with other traditional CV risk factors such as BF%, WHR and BP.
Arsic et al., 2011	Cross sectional	• To investigate FA profiles in plasma and erythrocytes phospholipids in elite female football players in comparison with sedentary women.	University of Belgrade.	Soccer, n=19 Females Mean Age: 21.19	BMI, BF%, Glucose, TG, TC,	Footballers significantly lower BF% than controls (19.92 v 25.38, p<0.05). Footballers had lower TC, TG and IFG, not significant.
Apostolidis et al., 2014	Cross sectional	• To examine changes in the lipid profile of male elite basketball and soccer players following a game compare it with that of inactive individuals.	Start of the regular season of the national-level soccer championships. Athens, Greece.	Soccer, n=21 Males Mean Age:25.8	TG, TC, LDL, HDL	Soccer players lower TG, TC and LDL than controls.. No difference in the baseline value of HDL between groups.
Brites et al., 2004	Cross sectional	• To explore the first 3 steps of reverse cholesterol transport. • To compare a group of well-trained soccer players to sedentary controls, with similar anthropometric parameters. • To characterise of the lipoprotein, apolipoproteins and lipoprotein particle environment concerned in this atherogenic pathway.	University of Buenos Aires.	Soccer, n=35 Males Mean Age: 18.2	BMI, WHR,TG, TC, HDL, HDL2, HDL3, HDL-Phospholipids, HDL-TG, Non-HDL, LDL, VLDL, APO B, APO A-I, APO A-II, LpA-I, LpA-I;A-II	No significant differences in TG, TC, HDL-phospholipids, HDL-TG, Non-HDL, LDL and VLDL concentrations. Average HDL was 12.5% higher in soccer players, larger because of greater HDL2 concentration.
Berge et al., 2010	Cross sectional	• To investigate male Norwegian elite football	Oslo Sports Trauma	Soccer, n=594 Males	BMI, SBP, DBP, HT,	High BP (>140/90) was detected in 39 players (6.6%), including two with Grade 2 Systolic HT

			players' BP and prevalence of HT.	Research Center.	Mean Age: 25	Daily snuffing.	and left ventricular concentric remodelling.
Crouse et al., 2016	Descriptive study	•	To describe echocardiogram characteristics and frequency of elevated BP in first year collegiate ASF athletes and compare to normal values where possible.	Department of Health and Kinesiology, Texas.	NFL, n=80 Males Mean Age:18	BMI, BF%, BSA, HR, SBP, DBP	DBP was significantly higher (7%) in black compared with nonblack athletes. Systolic and diastolic HT was present in 12% and 3% of the athletes, respectively; additionally, 64% and 27% were preHT. 78% were overweight or obese by BMI, but only 28% were >20% BF%.
Dobrosielski et al., 2016	Cross sectional	•	To estimate the prevalence of SDB in collegiate football players.	Townson University Division 1AA college. Pre-season mini-camp, August 2014.	NFL, n=51 Males Mean Age:19.6	BMI, NC (cm), Viscreal fat %, ESS, STOP-BANG Questionnaire.	8% with at least mild SDB.. Players with SDB had higher fat mass (31.8 ± 9.5 kg v 21.2 ± 11.2kg, p=.12), and total BF% (SDB: 26.7 ± 4.9% v non SDB: 20.3 ± 7.5%, p=.07).
		•	To evaluate the relationship between markers of SDB and body composition parameters using DEXA imagery.				
DiCesare et al., 2017	Descriptive study	•	To examine the relationship between muscle fiber type distribution and resting BP in collegiate-level football players.	Public university in the mid-American conference of the National Collegiate Athletic Association.	NFL, n=80 Males Mean Age: 19.8	BMI, BF%, WC, MAP.	BMI, BF% and WC were significantly greater for BIG group ($p \leq 0.001$). Players with significantly higher BF% and BMI had BP in the pre-HT range.
Feairheller et al., 2016	Cross sectional	•	To compare vascular health between football players and controls.	Ursinus College NCAA DIII football team - preseason and postseason camps.	NFL, n=23 Males Mean Age:19.8	BF%, SBP, DBP, Glucose, TC, HDL, LDL, TG, FMD%, FMD/sheer, IMT (mm)	NFL had higher FG (91.6 ± 6.5 v 86.6 ± 5.8, P<0.05), higher BF% (29.2 ± 7.9 % v 23.2 ± 7^, P<0.05), and lower fasting HDL (36.5 ± 11.2 V 47.1 ± 14.8, P<0.05) compared to controls. SBP was higher in NFL (p<0.05). LM had higher BW, BF% and lower HDL..
		•	To examine changes in CV health over a season.				
Haluzik et al, 1999	Cross sectional	•	To study the relation of serum leptin to blood viscosity and selected spiroergometric parameters of endurance capacity in a group of top rugby players and race walkers.	University Hospital, Prague	Rugby, n=13 Males Mean Age:23.8	BMI, BF%, Lean BM(kg), Leptin, IFG, Cholesterol, TG	BMI, BF% Lean BM and serum leptin levels were significantly higher in rugby players than in race walkers. Serum IFG, TC, TG did not differ significantly between the groups.
Helzberg et al., 2010	Cross sectional	•	To compare the risk of CV and metabolic diseases in professional baseball players and compare to professional football players and the general population.	Saint Luke's Hospital of Kansas City.	Baseball, n=155 Males Mean Age: 23 NFL, n=69 Males Mean Age: 25	BMI, WC, WHR, IFG, BP. TG, ALT, CMS,	Baseball players had a lower prevalence of CMS and CV risk factors compared to the NHANES. Baseball decreased obesity, insulin resistance, HDL cholesterol ≤ 40 mg/dl and TG ≥ 150mg/dl. Baseball significantly decreased prevalence of obesity, IFG, and CMS. CMS in baseball matched NFL but significantly more prevalent in LM (22% v 6%).

Karpinos et al., 2013	Retrospective cross sectional	• To determine the prevalence of HT among collegiate football athletes. • To compare HT among football athletes and non football athletes. • To compare the change in SBP of these two groups of athletes over the course of their collegiate career.	A private NCAA D1 university in the South-eastern Conference.	NFL, n=323 Males Mean Age:18.6	BMI, Smoking, HT, Pre HT, SBP, DBP	Prevalence of HT among footballers was 19.2%. Compared to controls, prevalence of HT was higher in initial year (19.2% v 7%, p<0.001) and final year (19.2% v 10.2%, p=0.005). BMI was an important cofounder.
Hurst et al., 2012	Cross sectional	• Not specified	Mayo Clinic, Scottsdale, Arizona – 13th-14th September 2009.	NFL, n=75 Males Mean Age: 27	Max RCAA (mm), Max LCAA (mm), BMI, HR, SBP, DBP, TC, HDL, LDL, TG, DM, HT, smoking	LM was larger than NLM and had higher SBP, TG and LDL. The control group had TC and TG statistically similar to the football group, but SBP and DBP, BMI, LDL, HDL and age were significantly higher for NFL group.
Evelson et al., 2002	Cross sectional	• To evaluate the lipid profile and the antioxidant status in a group of well-trained rugby players and compare with sedentary controls.	School of Pharmacology and Biochemistry, University of Buenos Aires.	Rugby, n=15 Males Mean Age: 23	BMI, LDL, HDL, TG, TC, IDL, VLDL, Glucose	Rugby significantly greater HDL (21% increase). No difference in TC, TG or LDL between well-trained athletes and age-BMII matched controls.
Kim et al., 2015	Prospective Longitudinal case controlled study	• To evaluate arterial elasticity and central BP in collegiate ASF participants.	Division 1 rugby team, Buenos Aries, Argentina.	NFL, n=32 Males Mean Age: 18.4	BMI, HT, Tobacco, HR, SBP, DBP, CAPP, Pulse Wave Velocity (m/sec)	28% of ASF had pre HT. After completing a single season of ASF, participants demonstrated significant increases in CPP, SBP, DBP with a resultant increase in the percentage with pre HT or HT (preseason-28% v postseason-59%, p=0.02).
Kirwan et al., 2012	Pre-test-post-test experimental design	• To determine dietary, anthropometric, blood lipid, and performance pattern of university-level American football players attempting to increase BM during 8 weeks of training.	Montana State University.	NFL, n=15 Males Mean Age: 18.5	BMI, TC, HDL. LDL, TG, VLDL	Increase in TC and LDL is likely due to overfeeding to gain weight. High levels of HDL - may provide a buffer against the negative effects of the rise in cholesterol.
Maso et al., 2002	Cross sectional	• To assess the distribution of lipoprotein particles in sportsmen. • To compare particles with other lipid factors including a further lipoparticle, Lp (a) and to compare to a control group of	French championship rugby club team.	Rugby, n=21 Males Mean Age: 26.6	Fat (%), BMI, TC, TG, HDL, LDL, Phospholipids, HDL-Phospholipids, Apo AI, Apo B, Apo E, APO CIII.	Rugby players were leaner, although they had a higher BMI. TC of Rugby lower than controls (p<0.01). LDL was not significantly different between groups, whereas HDL lower in rugby players (p<0.05). No difference in TC/HDL ratio or TC/LDL ratio. TG (p<0.05) and the phospholipids (p<0.0001) were significantly lower in Rugby.

			young adults in order to check anti-atherogenic effect of regular training in high level sportsmen.				
Oliver et al., 2015	Longitudinal study	•	To examine changes in blood lipids and lipoproteins over the course of a season.	NCAA Division 1 team-Pre-season and post-season (separated by 7 months)	NFL, n=14 Males Mean Age:18	BMI, TC, LDL, HDL, TG, TC/HDL ratio	TC was moderately correlated with fat mass (r=0.604, p=0.049). A moderate correlation between LDL and fat mass(r=0.528, p=0.095). TG was correlated with fat mass and BMI (r=0.833, p=0.001; r=0.752, p=0.002).
Rice et al., 2020	Cross sectional	•	To characterise the cross-sectional burden of SDB in active NFL athletes and its association with CV risk.	Sleep testing-mini-camp between April and July 2007.	NFL, n=137 Males Mean Age:27	BMI, BF%, WC, NC, SBP, DBP, , HT, pre HT, FBG, LDL, HDL, SDB	LM significantly higher BMI, BF%, WC and NC. No difference in other CV risk factors, beyond DBP. There was a 17.5% prevalence of HT, with 67.9% pre HT. Observed apnoeas were reported by 23.9. At least mild SDB was present in 19%.
Tucker et al., 2015	Cross sectional	•	To determine whether race is associated with differences in BP and prevalence of Pre-HT and HT among a large sample of professional football players.	Mandatory annual physical examination for active NFL players during team mini camps, April-August 2009.	NFL, n= 1,484 Males Mean Age: 26	BMI, SBP, DBP	LM group were the largest. A significant difference in BMI was found. No differences in BP based on race in any position groups. Black (n=1007) v White (n=477) players, no difference in the prevalence of HT (9.8% v 8.2%; p=0.353) or pre-HT (55.3% v 55.3%; p=1.0).
Wilkerson et al., 2010	Cross sectional	• •	To document the prevalence of CMS among collegiate football players. To develop a clinical prediction rule that does not require blood analysis to identify players who may possess a high level of CMS risk.	University of Tennessee, NCAA D1 NFL team.	NFL, n=62 Males Mean Age: 19.9	BMI, BF%, WC, SBP, DBP, TC, HDL, TG, FBG	Prevalence of CMS was 19.2% of players; 46% of the LM and 14% of NLM. The CMS risk in African-American players was underestimated. WC was a better discriminator than BF% or BMI.
Weiner et al., 2013	Prospective, longitudinal, observational study	•	To examine the hypothesis that collegiate ASF participation leads to clinically and statistically significant increases in resting BP.	The Harvard Athlete Initiative, 2006-2011	NFL, n=132 Males Mean Age:19	BMI, BSA, HR, SBP, DBP,	61% ASF had normal SBP and DBP, whereas the remaining 39% were pre HT. LM had significantly higher SBP and DBP and were more likely to meet criteria for pre HT (52% v 22%, p=0.002) than NLM.
Wilson et al., 2012	Cross sectional	•	To examine the CV risk of domestic and international professional football players of West-African descent.	7 Gulf states-and six Middle-Eastern countries. Doha, Qatar.	Soccer, n=190 Males Mean Age: Absent	SBP, DBP	No significant differences between ethnicities in either SBP or West Asian players had significantly higher TC (p=0.025) and significantly lower HDL (p=0.004). TC > 4.5mmol.L was more common in West-Asian players (43% v 37%, p=0.038). All lipid levels were within normal limits for both ethnicities.
Wright et al., 2017	Cross sectional	•	To assess the CVD risk profile of NCAA DIII	Pre-season physical exams - University Health Center,	NFL, n=89 Males Mean Age:19.6	BMI, BF%, WC, WHR, SBP, DBP,	No significant difference in DBP for LM or NLM (p>0.05). LM had higher BMI, SBP than NLM (p<0.05). 19% LM had CMS.

		•	intercollegiate football athletes. To collect pre-season data of physical characteristics of DIII athletes.	Whitworth University.		HDL, LDL, TG, TC, IFG	9.5% LM had HT, 42.9 % low HDL, and 6.7% high TG.
Yates et al., 2009	Rando mised control trial	•	To determine if Omega-3 essential fatty acids improve CV lipid risk factors.	Testing during 2 month period of active 1006-07 season. Pittsburugh Steelers Football Club.	NFL, n=36 Males Mean Age: 28.03	TC, LDL, HDL, VLDL, TG, Non-HDL	TG (98.72) and VLDL (21.59) below desired cut-off points. HDL (44.91mg/dl) was above desired min values (≥ 40mg/dl). VLDL-3 was found to be elevated above desired levels (13.04mg/dl).
Powers et al., 2016	Cross section al	•	To assess if positive energy balance and oxidative stress lead to vascular dysfunction in black football players.	Vanderbilt Medical Center-during offseason	NFL, n=33 Males Mean Age: Absent	SBP, DBP, HL	Elevated BP common black and white players (78% v 63%, p=0.34). Black players significantly better lipid profiles, body composition, and comparable insulin resistance.
Powers et al., 2015	Cross section al	•	To determine if CMS in football players is driven by oxidative stress and positive energy.	Vanderbilt Medical Center-during offseason	NFL, n=33 Males Mean Age: Absent	BMI, WC, HDL, TG, SBP, DBP, FG, CrP, Glucose AUC (mg/dl), Insulin AUC (mg/dl)	Prevalence of CMS was 33%. Elevated WC, HDL and elevated BP were present together in 73% of players. Players had increased oxidative stress (F2-isoprostanes and inflammation (CRP).
Carbuhn et al., 2008	Cross section al	•	To establish a position-by-position performance and BP profile of first-year players entering an NCAA D1 football program. To compare their profiles to professional, NCAA DI, II and III and junior college football athletes.	Huffines Institute for Sports Medicine and Human Performance, Texas A&M University.	NFL, n= 85 Males Mean Age:18.4	SBP, DBP	SBP was significantly and positively correlated (0.270) with BM. 23.5% of players had HT, 54% were pre HT, and only 22.5% had normal BP.
Randers et al., 2013	Cross section al	•	To determine if playing football on an elite level leads to significant improvements in the overall health profile.	Preseason period for the Danish women's national team.	Soccer, n=27 Females Mean Age: 24.4	TC, HDL, LDL, TG, LDL/HDL ratio	BMI was lower in athletes (21.7 v 24.0, p=0.035). no difference between groups in SBP or DBP (118 v 115 mmHg and 68 v 72 mmHg respectively). Haemoglobin was 4% higher in athletes. TC, LDL and TG levels were not different between groups, whereas athletes had 20% higher levels of HDL (p=0.047).

Abbreviations: *LM - linemen; NLM - non-linemen; BMI- body mass index; WC- waist circumference; WHR- waist-hip-ratio; BF% - body fat percentage; HT- hypertension; SBP- systolic blood pressure; DBP- diastolic blood pressure; MAP- mean arterial pressure; HL- hyperlipidemia; HDL- high-density lipoprotein; LDL- low- density lipoprotein; LDL-P- low-density lipoprotein particle number; TG-triglycerides; TC- total cholesterol; VLDL- very low density lipoprotein; APO- apolipoprotein; ALT- alanine aminotransferase; CMS- cardio-metabolic syndrome; DM - diabetes mellitus; IFG- impaired fasting glucose; IR: insulin resistance; hsCRP- high sensitive C reactive protein; FA- fatty acid; CAC- carotid artery calcium; CAP- carotid artery plaque; ASCVD - atherosclerotic*

cardiovascular disease; IMT- Intima-media thickness; RCA- right coronary artery NFL- National Football League; NCAA- National Collegiate Athletic Association.

Table 1: Body composition measures

Author	BMI kg.m^2	BF%	WC/NC	WHR
Tucker et al., 2009	NFL v CARDIA: 31.4 v 25.9***	Mean: 16.1%. Offensive LM - 25.8; Defensive LM - 20.8	NFL v CARDIA: 97 (97-98) v 86 (86-87) ***	NFL v CARDIA: 0.88 v 0.85***
Selden et al., 2009	NFL > NHANES ***		**WC ≥ 100cm:** Team v Nhanes:38% (26) v 26% LM v Nhanes: 95% (18) v 26%, *** NLM v Nhanes: 16% (8) v 26% LM v NLM: 95% (18) v 16% (8) ***	**WHR >0.5** Team v Nhanes:52% (36) v 55% LM V Nhanes:95% (18) v 55%, *** NLM v Nhanes:36% (18) v 55%* LM v NLM: 95% (18) Vs 36% (18) ***
Borchers et al., 2009	Mean: 29.93 ± 4.32	All: 17.29 ± 7.37 Group A (OLM, DLM) -25.62 ±7.37 Group B (WR, DB)- 11.73 ± 3.68 Group C (TE, LB QB, K)- 14.42 ± 3.77	Mean: 95.28 ± 13.22	
Dobrosielski et al., 2010		LM v Skill v Controls: 24.9 ± 4.3 v 11.7 ± 1.8* v 26.8 ± 13.4*		
Garry et al., 2001	**Skilled;** BMI <28 =69%, BMI 28-32 =31%, BMI >32= 0% **DE/LB/TE;** BMI <28= 10%, BMI 28-32 =57%, BMI >32 =33% **LM;** 100% LM had BMI >32			
Mansell et al., 2011	LM v NLM: 35.6 (3.5) v 26.4 (2.4) ***	LM v NLM: 26.4 (4.5) v 11.2 (3.5) ***	LM v NLM: 108.0 (9.1) v 82.9 (3.8) ***	
Steffes et al., 2013	Mean: 28.6 ± 3.7.	Mean: 15.5 ± 6.4.	Mean: 103.2 ± 57.0.	

	Big v Athletic v Skilled: 32.9 ± 2.7 v 27.9 ± 2.5 v 25.8 ± 1.9	Big v Athletic v Skilled: 22.9 ± 4.0 v 14.7 ± 4.5 v 10.1 ± 3.6	Big v Athletic v Skilled: 100.6 ± 6.3 v 87.9 ± 5.5 v 81.3 ± 3.4	
Allen et al., 2010	IL v AO v CARDIA: 38 v 29.5 v 25.9. IL > AO and CARDIA*; AO > CARDIA *	IL v AO v CARDIA: 25.2 (24.4-26) v 13.4 (12.9-14) v NA	IL v AO v CARDIA: 116 (114-118) v 92 (91-93) v 86 (86-87) IL > AO + CARDIA*; AO > CARDIA *	IL v AO v CARDIA: 0.92 (0.91-0.93) v 0.87 (0.86-0.88) v 0.85 (0.84-0.85). IL > AO and CARDIA*; AO > CARDIA *
Berge et al., 2013	Soccer v Controls: 23.7 (1.1) v 23.2 (0.9) *			
Buell et al., 2008		DI v DII v DIII: 26.2 ± 2.48 v 28.3 ± 2.80 v 25.5 ± 3.92** DI + DIII >DII***	DI v DII v DIII: 111.8 ± 8.32 v 115.3 ± 11.03 v 104.7 ± 9.46*** DI + DII > DIII ***	
Haskins et al., 2011	Football v Controls: 35 v 34.9	Football Players v Controls:21.8 v 27.1**		
Ahrensfield et al., 2012	All: 32.5 LM v NLM:37.6 v 29.1***	Mean: 17.5 LM v NLM: 24.2(22.4-25.8) v 13 (11.9-14) ***		
Arsic et al., 2011	Soccerl v Sedentary; 22.42 ± 1.33 v 22.10 ± 1.43	Football v Sedentary: 19.92 ± 3.25 v 25.38 ± 4.20*		
Brites et al., 2004	Soccer v Controls: 22.9 ± 0.2 v 24.1 ± 0.9			Soccer v Controls: 0.81 ± 0.01 v 0.81 ± 0.01
Berge et al., 2010	Mean: 23.7 kg.m2			
Crouse et al., 2016	Mean: 28.7 ± 5.0	Mean: 16.5± 9.7		
Dobrosielski et al., 2016	High Risk v Low Risk:33 ± 5.4 v 27.6 ± 3.6***		NC: High Risk v Low Risk: 44.6 ± 2.2 v 41.4 ± 2.8***	
DiCesare et al., 2017	Skill v Big: 26.9 ± 2.5 v 32.6 ± 2.9***	Skill v Big: 12.6 ± 4.8 v 22 ± 4.1***	Skill v Big: 84.7 ± 5.6 v 100 ± 6.6***	
Feairheller et al., 2016		Football v Controls: 29.2 ± 7.9 v 23.2 ± 7.0*		
Haluzik et al, 1999	Rugby v Race walkers: 26.7 ± 1.85 v 20.7 ± 1.88*	Rugby v Race walkers: 15.95 ± 3.15 v 9.68 ± 3.56*		

Helzberg et al., 2010	**BMI ≥ 30:** Baseball v NHANES:7 (5%) v 67 (21%) *** Baseball v Football: 7 (5%) v 35 (51%) *** Baseball v LM: 7 (5%) v 19 (100%) *** Baseball v NLM: 7 (5%) v 16 (32%) ***		WC > 100cm Baseball v NHANES: 11 (7%) v 85 (26%) *** Baseball v Football: 11 (7%) v 26 (38%) *** Baseball v LM: 11 (7%) v 18 (95%) *** Baseball v NLM: 11 (7%) v 8 (16%)	WHR > 0.5 Baseball v NHANES: 37 (23%) v 176 (55%) *** Baseball v Football: 37 (24%) v 36 (52%) *** Baseball v LM: 37 (24%) v 18 (95%) *** Baseball v NLM: 37(24%) v 18 (36%)
Karpinos et al., 2013	Football v Non-football: 28.4 ± 4.3 v 23.8 ± 2.6, ***			
Hurst et al., 2012	Mean: 32 ± 5 White Players v White Controls: 32 ± 4 v 29 ± 5 *** Black Players v Black Controls: 31 ± 5 v 29 ± 7*** NLM v LM: 29 ± 3 v 35 ± 5***			
Evelson et al., 2002	Rugby v Controls: 26.6 ± 2.2 v 25.1 ± 2.2			
Kim et al., 2015	ASF v Controls: 30 ± 4.3 v 24 ± 4***			
Maso et al., 2002	Sportsmen v Controls: 27.4 (3.1) v 23.5 (3.9) ***	Sportsmen v Controls: 15.5 (3.1) v 17		
Oliver et al., 2015	Mean: 26.9 ± 4.2			
Rice et al., 2020	Mean: 32.4 ± 4 LM v NLM: 37.3 ± 2.v 30 ± 3***	Mean: 17.9 ± 6.6 LM v NLM: 24.7 ±3.3 v 14.3 ± 4.9***	WC: Mean: 101 ± 14 LM v NLM: 116 v 94*** NC: Mean: 44.5 ± 3.3 LM v NLM: 47.4 v 43***	

Tucker et al., 2015	LM v DE/LB/RB/TE v QB/K/WR: 37 v 31 v 27***			
Wilkerson et al., 2010	Mean: 29.09 ± 4.54 MS-Negative v MS-Positive: 28.40 ± 3.97 v 31.98 ± 5.76	Mean: 15.38 ± 7.02 MS-Negative v MS-Positive: 14.39 ± 6.25 v 19.50 ± 8.76	Mean: 90.55 ± 10.84 MS-Negative v MS-Positive: 88.63 ± 9.87 v 98.53 ± 11.43, p =0.004.	
Weiner et al., 2013	ASF v Controls: 27.6 ± 3.3 v 24.4. ± 1.9 LM v NLM: 28.7 ± 3.4 v 26.2 ± 2.7			
Wright et al., 2017	LM v NLM: 33.9 v 26.6, p <0.001 All v OLM v DLM: 33.8 v 37.8 v 35.7	All v OLM v DLM: 29.9 v 25.8 v 20.8 LM with BF% > 25% = 71.4%	All v OLM v DLM: 102 v 117 v 107	All v OLM v DLM: 0.90 v 0.92 v 0.89
Powers et al., 2015	MS negative v MS positive: 31.20 ± 3.01 v 34.72 ± 2.50***		MS negative v MS positive: 98.6 ± 7.1 v 110.6 ± 6.6***	

*Abbreviations: LM - linemen; NLM - non-linemen; AO – all others; OLM- offensive linemen; DML- defensive linemen; DE- defensive ends; LB- line-backers; RB- running backs; TE- tight ends; WR- wide receivers; K- kickers BMI- body mass index; WC- waist circumference; WHR- waist-hip-ratio; BF% - body fat percentage; NFL- National Football League; NCAA - National Collegiate Athletic Association; ASF – American style football; CMS - cardio-metabolic syndrome. *= p<0.05; ** = p<0.01; ***= p<0.001.*

Table2: Blood pressure and lipid profiles.

	Blood Pressure (mmHg)	Lipids			
		TC (mg.dl)	HDL(mg.dl)	LDL (mg.dl)	TG (mg.dl)
Tucker et al., 2009	NFL v CARDIA: SBP: 127 v 112 *** DBP: 75 v 72 ***	NFL v CARDIA: 179 v 181	NFL v CARDIA: 48 v 49	NFL v CARDIA: 112 v 113	NFL v CARDIA: 96 v 95
Borchers et al., 2009	SBP:126.7 ± 12.49, DBP- 70.24 ± 8.55	16.87 ± 25.78	39.36 ± 8.97	106.08 ± 23.9	82.56 ± 46.34
Dobrosielski et al., 2010	LM v Skill v Controls: SBP: 134 ± 12.0 * v 121 ± 5.0 v 123 ± 10 DBP: 79 ± 6 v 73 ± 7 v 77 ± 6		LM v Skill v Controls: HDL: 38*± 8 v 49 ± 10 v 43 ± 11		LM v Skill v Controls: TG: 111 ± 50 v 129 * ± 72 v 75 ± 36
Garry et al., 2001		BMI < 28 (mmol): 4.95 BMI 28-32 (mmol): 5.00 BMI > 32(mmol): 5.10	BMI < 28 (mmol): 1.40 BMI 28-32 (mmol): 1.25 BMI > 32(mmol): 1.10	BMI < 28 (mmol): 3.10 BMI 28-32 (mmol): 3.25 BMI > 32(mmol): 3.25	BMI < 28 (mmol): 1.03 BMI 28-32 (mmol): 1.15 BMI > 32(mmol): 1.63
Mansell et al., 2011	LM v NLM: SBP: 109.2 (10.1) v 106.1 (9.0) DBP: 64.6 (8.5) v 63.6 (5.5)	LM v NLM (mmol): 3.86 (0.54) v 3.65 (0.70)	LM v NLM (mmol): 0.93 (0.22) v 1.12 (0.28) *	LM v NLM (mmol): 2.53 (0.49) v 2.05 (0.41) **	LM v NLM (mmol): 1.05 (0.60) v 0.83 (0.17)
Steffes et al., 2013	SBP: 122.4 ± 8.3; DBP: 79.4 ± 5.6 Big v Athletic v Skilled: SBP: 127.1 ± 9.0 v 121.9 ± 8.3 v 118.8 ± 5.4	168.2 ± 28.1 Big v Athletic v Skilled: 172.6 ± 27.7 v 170.4 ± 30.8 v 161.4 ± 24.6	46.0 ± 13.1 Big v Athletic v Skilled: 38.4 ± 12.1 v 47 ± 13 v 51.3 ± 11.2	106.2 ± 23.3 Big v Athletic v Skilled: 108 ± 26.6 v 106.9 ± 23.8 v 102.8 ± 18.5	103.2 ± 57.0; Big v Athletic v Skilled: 130.9 ± 71.2 v 103 ± 51.1 v 78.9 ± 36.3

	DBP: 81.2 ± 6.5 v 79.3 ± 5.5 v 78 ± 4				
Allen et al., 2010		IL v AO v CARDIA: 181 (175-187) v 178 (175-182) v 181 (179-182)	IL v AO v CARDIA: 43 (41-45) v 49 (48-51) v 49 (48-50). IL significantly < AO and CARDIA.	IL v AO v CARDIA: 117 (11-123) v 111 (107-115) v 113 (111-114)	IL v AO v CARDIA: 121 (107-135) v 89 (83-94) v 95 (91-99) IL significantly > AO and CARDIA *
Berge et al., 2013	Football v Controls: SBP: 144.1 (7.5) v 114 .2 (3.8) DBP: 76.9 (9.0) v 68.7 (6.4)				
Haskins et al., 2011	Football v Controls: SBP: 135.6 (13.3) v 148.1 (13.8) ** DBP: 74.9 (7.2) v 84.1 (4.7) ***	Football v Controls: 165 (33.6) v 181.7 (41.7)	Football v Controls: 44 (8.0) v 43.3 (10.9)	Football v Controls: 90.9 (27.1) v 116.3 (37.3) *	Football v Controls: 150.7 (85.5) v 110.9 (53.8)
Ahrensfield et al., 2012	Mean: 127/77 LM v NLM: SBP: 131 (128-133) v 125 (122-127) ** DBP: 79 (77-81) v 75 (73-77) **	Mean: 184 LM v NLM: 179 (170-189) v 187 (179-196)	Mean: 48 LM v NLM: 46 (42-50) v 50 (48-52) **	Mean: 116 LM v NLM: 118 (110-127) v 115 (105-124)	Mean: 95 LM v NLM:93 (81-106) v 96 (82-112)

Apostolidis et al., 2014		Soccer v Inactive: 179.3 ± 10.7 v 201.2 ± 10.5 **	Soccer v Inactive: 47.4 ± 4.1 v 44.2 ± 6.6	Soccer v Inactive: 110.9 ± 8.9 v 136.7 ± 11.3 **	Soccer v Inactive: 78.3 ± 6.7 v 177.6 ± 18.6 **
Brites et al., 2004		Soccer v Controls: 164 ± 4 v 170 ± 6	Soccer v Controls: 48 ± 1 v 42 ± 2 *	Soccer v Controls: 95 ± 4 v 108 ± 7	Soccer v Controls: 89 ± 6 v 95 ± 11
Crouse et al., 2016	SBP: 126 ± 10 DBP: 73 ± 9				
Feairheller et al., 2016	Football v Controls: SBP: 128.2 ± 6.4 v 122.4 ± 6.8 * DBP: 74.8 ± 4.1 v 73.9 ± 6.3	Football v Controls: 136.6 ± 23.9 v 157.1 ± 36.8	Football v Controls: 36.5 ± 11.2 v 47.1 ± 14.8 *	Football v Controls: 83.2 ± 18.2 v 97.3 ± 33.9	Football v Controls: 98.2 ± 55.2 v 102.1 ± 60.5
Halzuik et al., 1999		Rugby v Race Walkers (mmol): 4.04 ± 0.5 v 3.95 ± 0.79			Rugby v Race Walkers (mmol): 1.39 ± 0.7 v 1.15 ± 0.54
Karpinos et al., 2013	Football v Non-football SBP: 126.4 ± 11 v 122.5 ± 9.8 *** DBP: 75.3 ± 9.9 v 72.3 ± 9 ***	Mean: 189 ± 46 NLM v LM: 183 ± 39 v 197 ± 54	Mean: 53 ± 15 NLM v LM: 59 ± 13 v 47 ± 15 **	Mean: 110 ± 41 NLM v LM: 107 ± 38 v 114 ± 46	Mean: 138 ± 112 NLM v LM: 86 ± 44 v 205 ± 136 ***
Hurst el., 2012	Mean: SBP:123 ± 13; DBP: 75 ± 10 NLM v LM: SBP: 118 ± 9 v 130 ± 14 *** DBP: 74 ± 9 v 77 ± 10	LM V NLM: 197 v 183	LM V NLM: 47 v 59**	LM V NLM: 114 v 107	LM V NLM: 205 v 86 ***

Evelson et al., 2002		Rugby v Controls: 175 v 180	Rugby v Controls: 60 v 50 *	Rugby v Controls: 90 v 100	Rugby v Controls: 70 v 80
Kim et al., 2015	ASF v Controls: SBP: 123 ± 9 v 118 ± 13; DBP: 71 ± 9 v 72 ± 11				
Kirwan et al., 2012		Mean: 164 ± 88.3	Mean: 68 ± 16.2	Mean: 92.7 ± 32.7	Mean: 193.5 ±32.4
Maso et al., 2002		Sportsmen v Controls: (mM) 25 (0.76) v 4.85 (0.87) **	Sportsmen v Controls: (mM) 1.10 (0.22) v 1.23 (0.28) *	Sportsmen v Controls: (mM) 2.51 (0.68) v 2.55 (0.69)	Sportsmen v Controls: (mM) 0.80 (0.40) v 1.02 (0.32) *
Rice et al., 2010	Mean: SBP: 129 ± 11; DBP: 77 ± 8 LM v NLM: SBP: 131 v 128, p =0.12; DBP: 79 v 75 **		Mean: 47 ± 12 LM v NLM: 43 ± 11 v 49 ± 12 **	Mean: 111 ± 28 LM v NLM: 116 ± 34 v 109 ± 25	
Tucker et al., 2015	Group 1: Black v White: SBP: 126 (120, 135) v 126 (120, 134) DBP: 76 (70, 82) v 76 (72, 80) Group 2: Black v White: SBP: 122 (116, 128) v 122 (116, 128) DBP: 72 (67, 78) v 71 (68, 76)				

	Group 3: Black v White: SBP: 122 (114, 129) v 122 (115, 128) DBP: 71 (67, 76) v 70 (66, 76)				
Wilkerson et al., 2010	Mean: SBP: 129.65 ± 6.21; DBP: 82 ± 5.50 MS-Negative v MS-Positive: SBP: 128.66 ± 5.59 v 133.75 ± 7.20 ** DBP: 81.54 ± 5.20 v 83.92 ± 6.47		Mean: 48.92 ± 15.03 MS-Negative v MS-Positive: 51.52 ± 13.39 v 38.08 ± 17.19 **	Mean: 169.48 ± 38.0 MS-Negative v MS-Positive: 163.88 ± 36.19 v 192.83 ± 38.31 **	Mean: 110.06 ± 58.18 MS-Negative v MS-Positive: 91.42 ± 34.34 v 187.75 ± 73.19 **
Weiner et al., 2013	ASF v Controls: SBP: 116 ± 8 v 114 ± 9; DBP: 64 ± 8 v 60 ± 9 LM v NLM: SBP: 119 ± 8 v 113 ± 8 *; DBP: 66 ± 8 v 62 ± 9 *				
Wilson et al., 2012		West-Asian v Black-African (mmol): 4.4 ± 0.8 v 4.18 ± 0.8 *	West-Asian v Black-African(mmol): 1.3 ± 0.2 v 1.4 ± 0.2 **	West-Asian v Black-African (mmol): 2.6 ± 0.7 v 2.6 ± 0.7	West-Asian v Black-African (mmol): 0.97 ± 0.8 v 0.86 ± 0.1

Wright et al., 2017	OLM v DLM SBP: 130.6 v 132 v 127; DBP: 76.2 v 79 v 75 LM v NLM: SBP: 130.6 v 124.1 **; DBP: 76.2 v 74.2	All v OLM v DLM: 169.5 v 179 v 185	All v OLM v DLM: 39.9 v 43 v 47	All v OLM v DLM: 116.1 v 115 v 116	All v OLM v DLM: 93.9 v 119 v 111
Yates et al., 2009	SBP: 125.6; DBP: 74.7 LM v NLM: SBP: 130.6 v 124.1 **; DBP: 76.2 v 74.2		Mean: 44.91		Mean: 98.72
Powers et al., 2015	MS negative v MS positive: SBP: 133.6 ± 8.8 v 135.1 ± 7.3 DBP: 69.1 ± 5.6 v 71.7 ± 7.6		MS negative v MS positive: 45 ± 10 v 35.8 ± 8.42 **		MS negative v MS positive: 66.7 ± 77.8 v 118.4 ± 96.5
Carbuhn et al., 2008	SBP: 127 DBP: 79.7				
Wegmann et al., 2016	SBP: 138 ± 15; DBP: 88 ± 8				
Arsic et al., 2011		Football v Sedentary (mmol): TC: 3.94 ± 0.60 v 4.35 ± 0.67			Football v Sedentary (mmol): TG: 0.58 ± 0.20 v 0.82 ± 0.29
Randers et al, 2013		Elite football V Untrained: (mM): 4.5 ± 0.9 v 4.43 ± 4	Elite football V Untrained: (mM): 1.8 ± 0.3 v 1.5 ± 0.4 *	Elite football V Untrained: (mM): 2.4 ± 0.7 v 2.5 ± 0.7	Elite football v Untrained: (mM): 0.82 ± 0.1 v 0.99 ± 0.4

Abbreviations: LM - linemen; NLM - non-linemen; AO – all others; HT- hypertension; SBP- systolic blood pressure; DBP- diastolic blood pressure; MAP- mean arterial pressure; HDL- high-density lipoprotein; LDL- low-density lipoprotein; LDL-P- low-density lipoprotein particle number; TG-triglycerides; TC- total cholesterol; FG –

*fasting glucose; IFG- impaired fasting glucose; IR: insulin resistance; NFL- National Football League; NCAA- National Collegiate Athletic Association; ASF – American style football; CMS - cardio-metabolic syndrome, *- p<0.05, **-p<0.01, ***- p<0.001.*

Table 3: Critical appraisal of studies using AXIS

(1 of 3)	Ahrensfield et al., 2012	Allen et al., 2010	Arsic et al., 2011	Apostolidis et al., 2014	Berge at al., 2010	Berge et al., 2013	Borchers et al., 2009	Brites et al., 2004	Buell et al., 2008	Carbuhn et al., 2008
Introduction										
Were the aims/objectives of the study clear?	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Methods										
Was the study design appropriate for the stated aim(s)?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Was the sample size justified?	No	No	No	No	No	No	No	No	No	No
Was the target reference population clearly defined?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Was the sample frame taken from an appropriate population base so it closely represented the target/reference population under investigation?	Unsure	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Was the selection process likely to select subjects/participants that were representative of the target target/reference population under investigation?	Unsure	Unsure	Yes	Unsure	Unsure	Unsure	Unsure	Unsure	Unsure	No
Were measures undertaken to address and categorise non-responders?	No	Unsure	No	No	No	Unsure	No	No	No	No
Were the risk factor and outcome variables measured appropriate to the aims of the study?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Were the risk factor and outcome variables measured correctly using instruments/measurements that had been trialled, piloted or published previously?	Unsure	Yes	Yes	Yes	Unsure	Yes	Yes	Yes	Yes	Yes
Is it clear what was used to determine statistical significance and/or percision estimates? (e.g. Values , CI's)	Yes	Yes	Yes	Yes	Unsure	Yes	Yes	Yes	Yes	Yes

Were the methods (including statistical methods) sufficiently desribed to enable them to be repeated?	**Yes**	**Yes**	**Yes**	**Yes**	**Unsure**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**
Results										
Were the basic data adequately described?	**No**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**
Does the response rate raise concern about non-response bias?	**No**	**No**	**Unsure**	**No**	**No**	**No**	**No**	**No**	**No**	**No**
If appropriate, was information about non-responders described?	**No**	**No**	**No**	**No**	**No**	**No**	**No**	**No**	**No**	**No**
Were the results internally consistent?	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**
Were the results for the analyses described in methods, presented?	**Unsure**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**No**	**Yes**
Discussion										
Were the authors' discussions and conclusions justified by the results?	**Unsure**	**No**	**Yes**	**Yes**	**Unsure**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**
Were the limitations of the study discussed?	**No**	**Yes**	**Yes**	**Yes**	**No**	**Yes**	**Yes**	**No**	**Yes**	**No**
Other										
Were there any funding sources or conflicts of interest that may affect the authors' interpretation of the results?	**Unsure**	**Yes**	**No**	**Unsure**	**Unsure**	**Unsure**	**No**	**Unsure**	**Unsure**	**Unsure**
Was ethical approval or consent of participants attained?	**Unsure**	**Yes**	**Yes**	**Yes**	**Unsure**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**

(2 0f 3)	Evelson et al., 2002	Feairheller et al., 2016	Garry et al., 2001	Halzuik et al., 1999	Haskins et al., 2011	Helzberg et al., 2010	Hurst et al., 2012	Karpinos et al., 2013	Kim et al., 2015	Kirwan et al., 2012	Mansell et al., 2011	Maso et al., 2002	Oliver et al., 2015	Powers et al., 2015
Introduction														
Were the aims/objectives of the study clear?	Yes	No	Yes	Yes	Yes	No	Yes	No	No	Yes	Yes	Yes	No	No
Methods														
Was the study design appropriate for the stated aim(s)?	Yes	Yes	Yes	Yes	Yes	No	No	Yes	Yes	Yes	Yes	Yes	Yes	Unsure
Was the sample size justified?	No	No	No	No	No	No	No	Yes	No	No	No	No	No	No
Was the target reference population clearly defined?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Was the sample frame taken from an appropriate population base so it closely represented the target/reference population under investigation?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Was the selection process likely to select subjects/participants that were representative of the target	No	No	No	No	No	No	No	Yes	No	No	Unsure	No	No	Unsure

target/reference population under investigation?														
Were measures undertaken to address and categorise non-responders?	**No**	**No**	**Unsure**	**No**	**No**	**No**	**No**	**Unsure**	**No**	**No**	**No**	**No**	**No**	**Unsure**
Were the risk factor and outcome variables measured appropriate to the aims of the study?	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Unsure**
Were the risk factor and outcome variables measured correctly using instruments/measurements that had been trialled, piloted or published previously?	**Yes**	**Yes**	**Yes**	**Yes**	**No**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Unsure**
Is it clear what was used to determine statistical significance and/or percision estimates? (e.g. Values , CI's)	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Unsure**
Were the methods (including statistical methods) sufficiently	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**No**	**Yes**	**Yes**	**Yes**	**No**	**Yes**	**Yes**	**Yes**	**No**

desribed to enable them to be repeated?														
Results														
Were the basic data adequately described?	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**
Does the response rate raise concern about non-response bias?	**No**	**No**	**No**	**No**	**No**	**No**	**No**	**No**	**No**	**Yes**	**No**	**No**	**No**	**Unsure**
If appropriate, was information about non-responders described?	**No**	**No**	**No**	**No**	**No**	**No**	**No**	**Unsure**	**No**	**No**	**No**	**No**	**No**	**Unsure**
Were the results internally consistent?	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Unsure**
Were the results for the analyses described in methods, presented?	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**No**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**No**
Discussion														
Were the authors' discussions and conclusions justified by the results?	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Unsure**
Were the limitations of the study discussed?	**No**	**Yes**	**Yes**	**No**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**No**	**No**	**No**
Other														
Were there any funding sources or conflicts of interest that may affect the	**Unsure**	**No**	**No**	**Unsure**	**Unsure**	**Unsure**	**Unsure**	**Unsure**	**No**	**Unsure**	**Unsure**	**Unsure**	**No**	**Unsure**

authors' interpretation of the results?														
Was ethical approval or consent of participants attained?	**Unsure**	**Unsure**	**Unsure**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Unsure**

(3 of 3)	**Powers et al., 2016**	**Randers et al., 2013**	**Rice et al., 2010**	**Selden et al. 2009**	**Steffes et a., 2013**	**Tucker et al., 2009**	**Tucker et al., 2015**	**Turner et al., 2003**	**Weiner et al., 2010**	**Wilkerson et al., 2010**	**Wilson et al., 2012**	**Wright et al., 2017**	**Yates et al., 2009**
Introduction													
Were the aims/objectives of the study clear?	**No**	**No**	**Yes**	**No**	**Yes**	**Yes**	**Yes**	**Yes**	**No**	**Yes**	**Yes**	**Yes**	**No**
Methods													

Was the study design appropriate for the stated aim(s)?	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**
Was the sample size justified?	**No**	**No**	**No**	**No**	**No**	**Yes**	**No**	**No**	**No**	**No**	**No**	**No**	**No**
Was the target reference population clearly defined?	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**
Was the sample frame taken from an appropriate population base so it closely represented the target/reference population under investigation?	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**
Was the selection process likely to select subjects/participants that were representative of the target target/reference population under investigation?	**Unsure**	**Yes**	**No**	**Unsure**	**Yes**	**Yes**	**Yes**	**No**	**No**	**Unsure**	**Yes**	**No**	**Unsure**
Were measures undertaken to address and categorise non-responders?	**Unsure**	**No**	**No**	**No**	**No**	**Yes**	**No**	**No**	**No**	**No**	**No**	**No**	**No**
Were the risk factor and outcome variables measured appropriate to the aims of the study?	**Unsure**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**

Were the risk factor and outcome variables measured correctly using instruments/measurements that had been trialled, piloted or published previously?	**Unsure**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**
Is it clear what was used to determine statistical significance and/or percision estimates? (e.g. Values , CI's)	**Unsure**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Unsure**
Were the methods (including statistical methods) sufficiently desribed to enable them to be repeated?	**No**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Unsure**
Results													
Were the basic data adequately described?	**No**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**
Does the response rate raise concern about non-response bias?	**Unsure**	**No**	**No**	**No**	**No**	**No**	**No**	**No**	**No**	**No**	**No**	**No**	**No**
If appropriate, was information about non-responders described?	**Unsure**	**No**	**No**	**No**	**No**	**Yes**	**No**	**No**	**No**	**No**	**No**	**No**	**No**
Were the results internally consistent?	**Unsure**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**
Were the results for the analyses described in methods, presented?	**No**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**No**

Discussion													
Were the authors' discussions and conclusions justified by the results?	**Unsure**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**
Were the limitations of the study discussed?	**No**	**No**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**No**	**No**	**Yes**	**Yes**
Other													
Were there any funding sources or conflicts of interest that may affect the authors' interpretation of the results?	**Unsure**	**No**	**No**	**No**	**No**	**No**	**No**	**No**	**No**	**Unsure**	**No**	**No**	**Unsure**
Was ethical approval or consent of participants attained?	**Unsure**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**

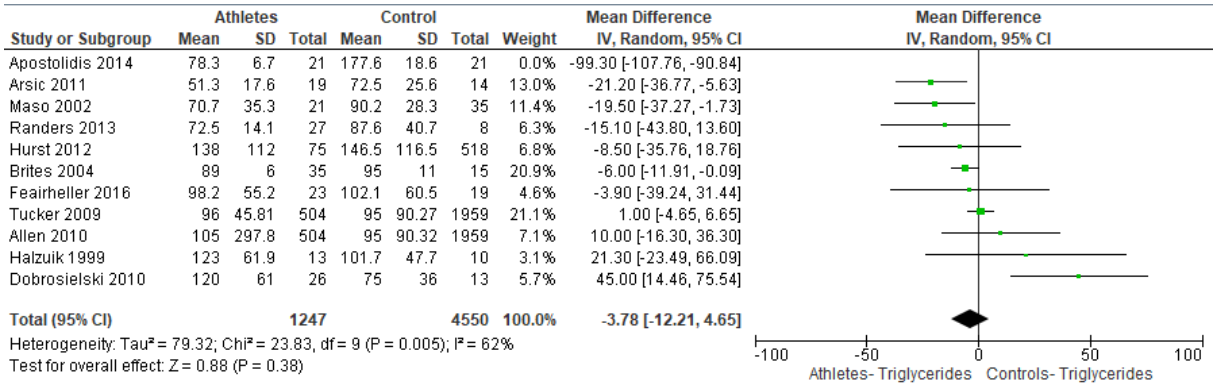


Figure 4: Forest Plot of Triglyceride levels for Athletes v Controls

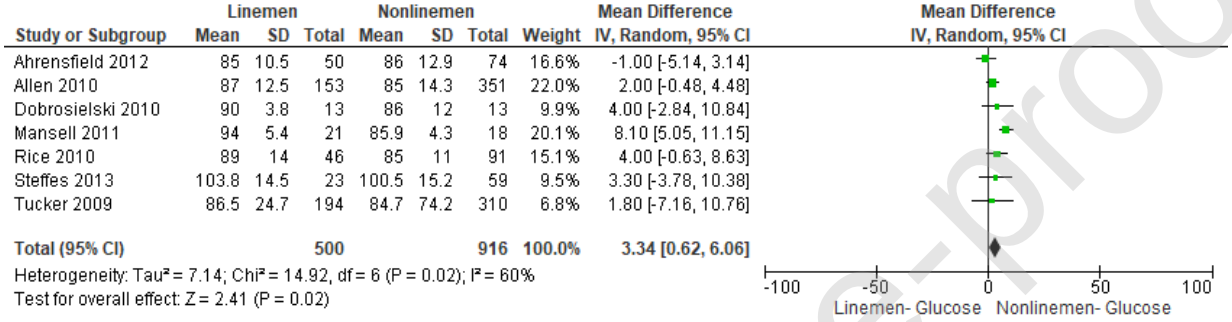


Figure 5: Forest Plot of Glucose for Linemen v Non-linemen

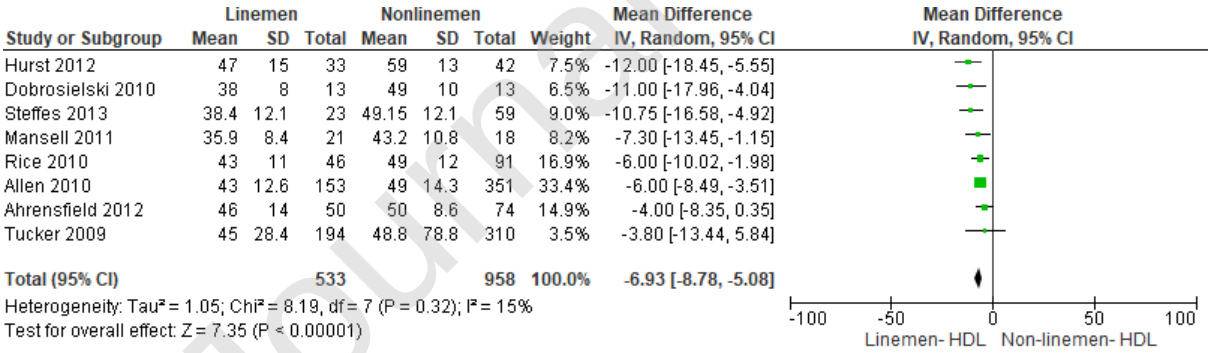
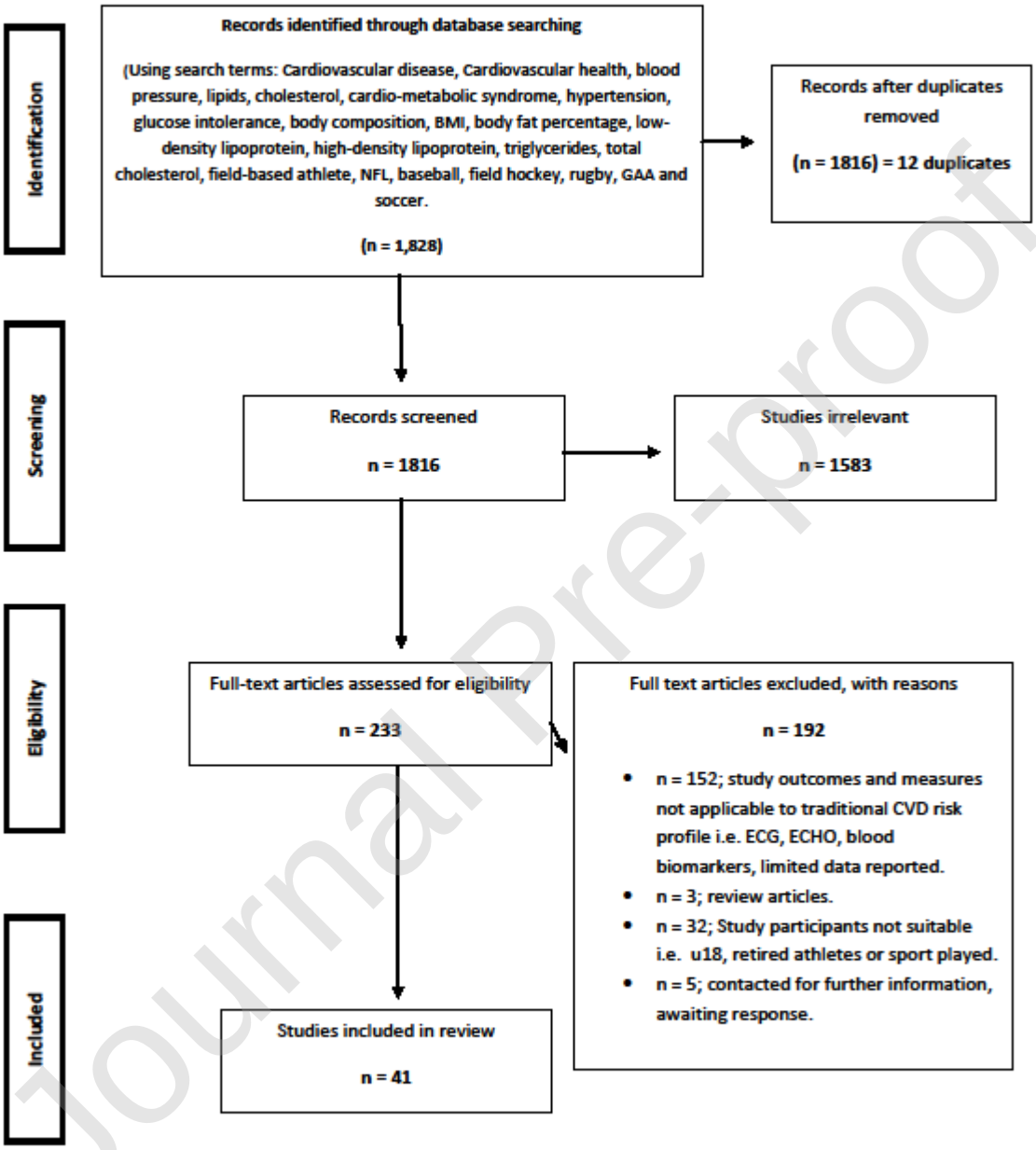


Figure 6: Forest Plot of HDL values for Linemen v Non-linemen



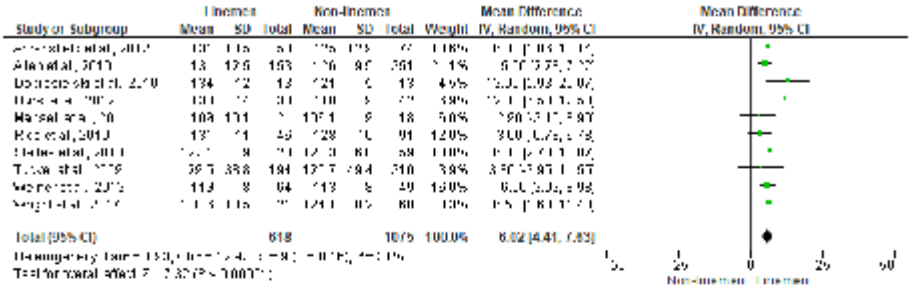


Figure 2: Forest plot of comparison: Linemen v Non-linemen, outcome: Systolic Blood Pressure

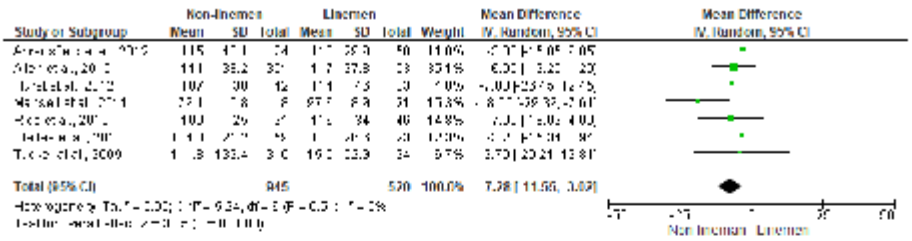


Figure 3: Forest plot of comparison: Non-linemen v Linemen, outcome: Low-density Lipoprotein