Risk Factors for Cardiometabolic Disease in Professional Firefighters

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Objective: Firefighters are plagued with cardiometabolic disease (CMD). Obesity, poor cardiorespiratory and muscular fitness, and blood lipids (low-density lipoprotein cholesterol, triglycerides, low high-density lipoprotein cholesterol) are risk factors for CMD. However, markers of oxidative stress, inflammation, and insulin resistance can provide further insight regarding CMD risk. **Methods:** This study investigated the relationships between fitness metrics (cardiorespiratory and muscular fitness, percent body fat, waist circumference), blood lipids, blood pressure, and years of experience as a firefighter to blood markers of insulin resistance: Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), oxidative stress: advanced oxidation protein products (AOPPs), and inflammation: C-reactive protein. **Results:** Waist circumference and blood concentrations of triglycerides were significantly related to AOPPs and HOMA-IR. Cardiorespiratory fitness was inversely related to AOPPs, HOMA-IR and C-reactive protein. **Conclusion:** These findings demonstrate the importance of high cardiorespiratory fitness and low waist circumference to reduce markers of CMD.

Keywords: cardiovascular, diabetes, fire, health, insulin resistance, oxidative stress, stress, tactical occupation

LEARNING OUTCOMES

- Firefighters are plagued with high rates of cardiovascular disease compared with the general population.
- High waist circumference was significantly related to increased blood markers of cardiometabolic disease in professional firefighters.
- Cardiorespiratory fitness was inversely related to blood markers of cardiometabolic disease.

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Sudden cardiac death is the leading cause of mortality that firefighters face in the line of duty, which accounts for approximately 50% of deaths.[1,2] Firefighters are exposed to numerous stressors including intense physical exertion, exposure to heat and smoke, and psychological stress, which leads to cardiovascular strain, impaired vascular function, oxidative stress, and inflammation, contributing to increased risk for developing cardiovascular disease (CVD).[3,4] Chronic exposure to these stressors can cause inflammation and oxidative stress, which are main contributors to CVD and other cardiometabolic diseases.[2,5] Firefighters have also been shown to demonstrate traditional risk factors for CVD such as high concentrations of blood triglycerides (TAGs), cholesterol, and lower concentrations of high-density lipoprotein cholesterol.[6] Moreover, firefighters have been shown to have a high prevalence of obesity,[6,7] which is another factor that contributes to chronic low-grade inflammation and oxidative stress and has been shown to be a primary cause of CVD, especially when the obesity is associated with excessive central adiposity (ie, elevated waist circumference).[8–10]

Oxidative stress and inflammation play a key role in the progression of cardiometabolic disease (ie, CVD and insulin resistance)[11] in addition to neurodegenerative diseases and cancer.[12–14] In fact, blood markers of oxidative stress and inflammation have been considered to be predictive biomarkers for CVD risk[15] and provide better insight related to cardiometabolic health as opposed to the use of traditional biomarkers alone (ie, blood lipids and glucose [GLU]).[16–18] For example, C-reactive protein (CRP) has been extensively evaluated by the Centers for Disease Control and Prevention and the American Heart Association in relation to its role in CVD progression.[18] Moreover, high-sensitivity detection of CRP is widely used among clinical laboratories as a supplementary tool to predict inflammatory status and subsequent risk for an adverse cardiovascular event.[19,20] Although CRP concentrations can vary significantly between demographics such as age, sex, race, and ethnic groups, the concentrations less than 1, 1 to 3, and greater than 3.0 mg/L have been identified as being indicative of low, moderate, and high risks for CVD, respectively,[18,21] and very high concentrations of CRP (>10 mg/L) have been identified as an independent predictor of CVD-related mortality.[16,22] Inflammatory markers, such as CRP, are useful tools in predicting cardiometabolic health[16]; however, more work is needed to further explore its usefulness among the fire community. Regardless, the combination of traditional markers of CVD (ie, blood lipids and GLU) and markers of oxidative stress and inflammation is most useful in gauging cardiometabolic health.[16]

In terms of fitness metrics, the minimum standard for aerobic capacity is 42 $mL \cdot kg^{-1} \cdot min^{-1}$ (or within a range of 39 to 45 $mL \cdot kg^{-1} \cdot min^{-1}$), which is recommended for firefighter to successfully perform and complete various occupational-specific tasks efficiently and with less fatigue.[23–25] Moreover, recent work has shown that fitness measures such as lower body strength and time to exhaustion (TTE) during a $\dot{V}O_{2peak}$ test were significant predictors to improved work efficiency during a simulated fire grounds test in professional firefighters.[26] However, it should also be noted that individuals with higher $\dot{V}O_{2max}$ values have a lower risk for developing CVD and demonstrate lower markers of inflammation and oxidative stress.[27–29] Firefighters are frequently required to perform tasks that require a significant amount of cardiorespiratory conditioning, in addition to muscular strength

TABLE 1. Sample Characteristics

	n	Mean	SD	Range
Age, y	95	36.26	9.08	20–60
Professional experience, y	88	11.81	8.00	0.83–35
$\dot{V}O_{2max}$, mL · kg^{-1} · min^{-1}	94	36.17	7.08	21.10–55.29
Waist circumference, cm	95	96.51	10.48	73.66–132.08
Height, cm	95	179.54	7.07	149.86–194.31
Body fat, %	95	24.39	5.21	13.8–39.6
AOPPs, μM	93	129.43	88.84	50.93–411.64
HOMA-IR	98	0.68	0.69	0.1–5.2
CRP, ng/mL	93	35,511.14	32,166.18	1,511.26–130,363.30

AOPPs, advanced oxidation protein products; CRP, C-reactive protein; HOMA-IR, Homeostatic Model Assessment for Insulin Resistance.

and/or endurance such as carrying heavy objects, advancing charged hose lines, climbing several flights of stairs with equipment, victim drag/carry, search and rescue, and forceful entry.[30] Interestingly, muscular strength and endurance are also inversely related to CVD risk.[31,32] Thus, it is important to note that improving aerobic capacity and muscular strength and endurance is ideal for execution of occupational-specific tasks, but also significant for improving cardiometabolic health. However, research is needed to examine this relationship in firefighters specifically.

A recent study by Strauss et al[33] examined the relationship between markers of cardiorespiratory fitness (CRF) and CVD risk factors in German firefighters. This study demonstrated an inverse relationship between increased CRF and body mass index, body fat percentage, blood pressure (BP), and blood concentrations of TAGs and cholesterol.[33] However, to our knowledge, no study has examined this relationship in conjunction with years of experience as a firefighter, as well as markers of inflammation, oxidative stress, and insulin resistance. Therefore, the purpose of this study was to examine the relationship (if any) between a variety of fitness metrics including $\dot{V}O_{2max}$, body composition, and muscular strength, endurance, and flexibility variables in relation to traditional markers of CVD (blood lipids, GLU), as well as markers of inflammation (CRP) and oxidative stress (advanced oxidation protein products [AOPPs]) and insulin resistance as quantified by the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR).

METHODS

Participants

Ninety-eight apparently healthy professional, male firefighters from a local fire department (Bryan, Texas) were studied (Table 1). Participants completed a health and lifestyle history questionnaire to identify potential risk factors for heart disease, skeletal muscle injury, orthopedic risk, and/or any contraindications to exercise. The assessment procedures used in this study were approved by the university's institutional review board, and all participants provided written and verbal informed consent before participation.

Testing Procedures

Anthropometric and demographic data including age, height, total body mass, percent body fat (%BF), fat-free mass, and waist and hip circumference measurements were collected in addition to other fitness metrics including muscular endurance and maximal oxygen uptake for all participants. Participants were 12-hour fasted before testing sessions. Resting heart rate (HR) and BP measurements were taken following the American College of Sports Medicine (ACSM) guidelines.[34] Before body composition assessment, participant height and body mass were measured using a medical-grade scale (Seca Model 700, Hamburg, Germany). Waist and hip circumference measurements were taken following the World Health Organization standards for waist circumference and waist-to-hip ratio.[35] Dual-energy x-ray absorptiometry scans (Hologic Horizon A, Marlborough, MA) were used to acquire body composition values including total body mass, %BF, and fat-free mass. In addition, a maximal graded exercise test (GXT) was performed using the Bruce protocol[36] on a standard treadmill (Quinton Q Stress System [Cardiac Science Corporation, Bothell, WA] with TM65 treadmill) to determine $\dot{V}O_{2max}$. Participants were instructed to follow a standard GXT protocol and continue the test until they reach exhaustion. Time to exhaustion was recorded and used to predict $\dot{V}O_{2max}$ via the Foster equation,[37] which can be found in the Supplemental Digital Content, http://links.lww.com/JOM/B227.

Participants followed the ACSM criteria and protocols subsequently described for fitness metrics including sit-ups, push-ups, and vertical jump.[34] For both the push-up and sit-up assessments, participants were instructed to perform as many repetitions as possible without stopping within 1 minute.[38] Next, participants performed three vertical jumps, and the highest distance in inches was recorded.[39] A pressure "Just Jump" Probotics mat (Warren, PA) was used to record the hang time and jump height in inches. Participants were instructed to squat to a position with the knees in a 90° angle and the hands by the side. Upon instruction, the participant performed three vertical jumps straight up as high as possible while reaching their hands up toward the ceiling, without tucking the legs, and land with both feet on the mat. Finally, the participants completed a sit-and-reach assessment following the ACSM criteria and protocols.[34]

Blood Collection Procedures

Venous blood samples approximately 8.5 mL were collected into serum separation tubes (BD Vacutainer®; Becton, Dickinson

TABLE 2. OLS Regression of Blood Markers by Demographics, HR, and BP

	AOPPs[a]			HOMA-IR[a]			CRP[a]		
	b	SE	*β*	*b*	SE	*β*	*b*	SE	*β*
Years of experience	0.01	0.01	0.19	0.00	0.01	0.04	0.00	0.02	0.01
RHR	0.00	0.01	0.01	−0.00	0.01	−0.03	−0.01	0.01	−0.09
Resting SBP	0.00	0.01	0.06	0.00	0.01	0.02	0.01	0.02	0.08
Resting DBP	0.01	0.01	0.09	0.03	0.01	0.37**	0.04	0.02	0.29*
Constant	3.16	1.06		−2.98	1.08		6.17	1.93	
Adjusted R^2	0.02			0.11			0.07		
n	84			87			82		

[a]Dependent variable log transformed.

$^*P \le 0.05$, $^{**}P \le 0.01$.

AOPPs, advanced oxidation protein products; BP, blood pressure; CRP, C-reactive protein; DBP, diastolic blood pressure; HOMA-IR, Homeostatic Model Assessment for Insulin Resistance; HR, heart rate; OLS, ordinary least-squares; RHR, resting heart rate; SBP, systolic blood pressure.

TABLE 3. OLS Regression of Blood Markers by Blood Markers

	AOPPs[a]			HOMA-IR			CRP		
	b	SE	*β*	*b*	SE	*β*	*b*	SE	*β*
Glucose	−0.01	0.00	−0.04	0.03	0.01	0.56***	621.68	311.93	0.21
Cholesterol	0.00	0.00	0.04	−0.01	0.00	−0.24	333.89	272.63	0.34
Triglycerides	0.01	0.00	0.80***	0.00	0.00	0.34**	0.84	70.35	0.00
HDL	0.00	0.01	0.04	0.01	0.01	0.10	−1,495.88	898.38	−0.53
Risk ratio LDL/HDL	0.04	0.15	0.06	0.19	0.21	0.26	−16,254.57	13,609.5	−0.47
Constant	3.84	0.55		−2.80	0.73		30,313.69	47,886.8	
Adjusted R^2	0.67			0.47			0.07		
n	92			93			89		

[a]Dependent variable log transformed.
**$P \le 0.01$, ***$P \le 0.001$.
AOPPs, advanced oxidation protein products; CRP, C-reactive protein; HDL, high-density lipoprotein; HOMA-IR, Homeostatic Model Assessment for Insulin Resistance; LDL, low-density lipoprotein; OLS, ordinary least-squares.

and Company, Franklin Lakes, NJ) via venipuncture in more than 8-hour fasted state from an antecubital vein. Blood samples were left at room temperature to clot for 30 minutes before they were centrifuged at 2500 revolutions/min for 15 minutes at 4°C. One aliquot of serum was immediately transported on ice to a clinical laboratory for analysis of total cholesterol, TAGs, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and GLU. A second aliquot of serum was frozen at −80°C and analyzed for concentrations of insulin, as well as a marker of oxidative stress and inflammation—AOPPs and ultrasensitive CRP. Analyses of concentrations of insulin, AOPPs, and CRP were conducted in duplicate and performed using commercial assay kits: CRP (Eagle Biosciences, Amherst, NH), insulin (IN374S; Calbiotech, El Cajon, CA), and AOPPs (Cell Biolabs, San Diego, CA). All procedures were adhered to as instructed by the kit manufacturer, and absorbance was detected using a BioTek colorimetric plate reader (Winooski, VT). Homeostatic Model Assessment for Insulin Resistance was calculated by fasting GLU (mg/dL) × fasting insulin (μU/mL)/405.[40]

Statistical Analysis

All statistical analyses were conducted via Stata v.16.1 (College Station, TX). The primary analysis technique to assess relationship for AOPPs, CRP, and HOMA-IR was ordinary least-squares (OLS) regression. Tables present both unstandardized (*b*) and standardized (*β*) coefficients across the models. All OLS models were tested for multicollinearity and heteroscedasticity by assessing variance inflation factor scores and Breusch-Pagan tests, respectively. All variance inflation factor scores were less than 4, indicating no multicollinearity present. Some models presented heteroscedastic values for the dependent variable. In these instances, we performed a logarithmic transformation on the dependent variable. Transformations are noted in the OLS results. Principal components analysis and bivariate correlation matrices are also presented where appropriate.

RESULTS

Samples were collected from all 98 participants. Outlier values (ie, values over 3 SDs from the mean) were removed. Other samples may have been undetectable (either too low or too high relative to the standard curve). This resulted in the removal of five observations from the CRP dependent variable and five observations from the AOPP dependent variable. In addition, a few of the participants were unable to provide demographic data. As such, each model presented indicates the sample size represented. Table 1 presents baseline characteristics of the sample.

Participant Demographics, HR, and BP

None of the presented demographic, HR, or BP measures had a significant impact on concentrations of AOPPs (Table 2). However, a higher resting diastolic BP was significantly related to higher levels of HOMA-IR and CRP when controlling for the presence of the other variables.

Blood Markers

Regarding the blood markers, TAGs were found to positively increase AOPP levels when controlling for the other blood markers in the model. The AOPP model also accounted for 67% of the variance present in the AOPP dependent variable (Table 3). The HOMA-IR values were increased by both GLU and TAGs. This model accounted for 47% of the variance present in the HOMA-IR dependent variable. Although there were significant findings for AOPPs and HOMA-IR, none of the blood markers seem to have a significant impact on CRP.

Fitness Metrics

Regarding the series of fitness-related tasks to including sit-ups, push-ups, vertical jumps, grip strength, and sit-and-reach exercises, a principal components analysis was used to determine how many, if any, of the fitness tasks were related to a larger construct of overall fitness. Three of the items were correlated with each other ($r > 0.5$). A subsequent principal components analysis with oblimin rotation showed these three items loaded into a single factor (Cronbach $\alpha = 0.81$). Table 4 presents the individual items and their respective loadings. The fitness scale was additive, and values ranged from 85.65 to 225.1 (mean, 153.71 [SD, 24.60]), with higher values representing higher levels of fitness.

Table 5 presents OLS regression models using the fitness scale and TTE during the GXT as independent variables. Increased TTE during the VO_{2max} test was significantly related to lowering AOPPs, HOMA-IR, and CRP levels when controlling for the fitness levels of participants. Note that both predicted $\dot{V}O_{2max}$ and TTE were analyzed as independent variables in separate models. However, results for TTE are shown in Table 5; the TTE explained slightly more of the model variance with regard to AOPPs, HOMA-IR, and CRP levels (although the direction was identical, the magnitude of the relationship was slightly larger than the $\dot{V}O_{2max}$ model). Adjusted R^2 for $\dot{V}O_{2max}$ ranged from 5% to 15% compared with 9% to 15% for TTE. The results for the

TABLE 4. Fitness Scale Principal Components Analysis

Items	Factor Loadings	Mean	SD
Sit-up	0.87	37.68	9.40
Vertical	0.85	19.49	4.00
Push-up	0.81	44.33	15.49

Cronbach $\alpha = 0.81$.

TABLE 5. OLS Regression of Blood Markers by Fitness Measures

	AOPPs			HOMA-IR[a]			CRP[a]		
	b	SE	*β*	*b*	SE	*β*	*b*	SE	*β*
Fitness	0.08	0.43	0.02	−0.00	0.00	−0.07	0.00	0.01	−0.05
Time to exhaustion	−18.71	6.60	−0.35**	−0.17	0.05	−0.42***	−0.27	0.08	−0.39**
Constant	317.25	66.85		1.48	0.48		13.19	0.83	
Adjusted R^2	0.09			0.19			0.15		
n	86			91			86		

[a]Dependent variable log transformed.
**$P \leq 0.01$, ***$P \leq 0.001$.
AOPPs, advanced oxidation protein products; CRP, C-reactive protein; HOMA-IR, Homeostatic Model Assessment for Insulin Resistance; OLS, ordinary least-squares.

$\dot{V}O_{2max}$ model can be found in the Supplemental Digital Content, http://links.lww.com/JOM/B227.

Body Composition

With respect to body composition, participant waist circumference and %BF were used to estimate changes in the blood markers. An increase in waist circumference was positively correlated with an increase in AOPP concentrations and HOMA-IR but not CRP concentrations. An increase in body fat percentage was significantly related to an increase in CRP concentrations (Table 6).

Blood Marker Correlations

While the previous results examined relationships between a series of independent variables and AOPPs, CRP, and HOMA-IR, Table 7 presents bivariate correlations between the blood markers. Homeostatic Model Assessment for Insulin Resistance was correlated with AOPPs and CRP at the 0.05 level of statistical significance. However, AOPPs and CRP did not exhibit a positive bivariate relationship.

DISCUSSION

The main findings of this study show significant relationships between waist circumference, body fat percentage, and CRF (demonstrated by TTE during the GXT and predicted $\dot{V}O_{2max}$) in relation to blood markers of oxidative stress, inflammation, and insulin resistance. More specifically, blood concentrations of oxidative stress as measured by AOPPs were significantly related to blood concentrations of TAGs, as well as waist circumference and CRF. The HOMA-IR and CRP concentrations were significantly related to CRF as well. This is the first study to our knowledge to examine the relationships of fitness metrics to blood markers of oxidative stress and insulin resistance. These findings underscore the importance of maintaining high CRF status among firefighters to reduce risk for CVD. Given these findings, certain blood biomarkers as measures here (eg, AOPPs, CRP, and HOMA-IR) could serve as a useful tool for clinically assessing cardiometabolic disease risk in firefighters.

The relationship between body fat and inflammation has been widely documented such that elevated body fat percentages and increased android adiposity (ie, belly fat) are linked to high levels of inflammation including biomarkers interleukin 6, and CRP.[41,42] In fact, adipose tissue acts as a highly active endocrine organ secreting numerous hormones and adipokines that have a profound impact on numerous aspects of physiology including, but not limited to, regulation of energy intake, metabolism, and inflammatory process.[43] Increased amounts of central adiposity have been shown to increase circulating concentrations of proinflammatory adipokines such as tumor necrosis factor α and resistin, which are known to cause insulin resistance.[44–46] Thus, it is not surprising that numerous studies have correlated increased amounts of android adiposity with chronic inflammation and insulin resistance.[43,47–49] Moreover, android fat has been previously identified as a more accurate predictor of inflammation than body mass index in patients with type 2 diabetes mellitus.[42] Although these relationships among firefighters have yet to be elucidated, the results from the present study suggest waist circumference and body fat percentage are related to markers of cardiometabolic health as measured by HOMA-IR, AOPPs, and CRP concentrations.

With respect to CRF, extensive work has suggested the usefulness of CRF in predicting CVD-related mortality.[50] In fact, CRF has been shown to be more useful in predicting adverse CVD-related events than alternate methods such as ST-segment depression and hemodynamic responses.[50–53] Cardiorespiratory fitness, as directly quantified by measuring respiratory gas exchange during GXTs (ie, $\dot{V}O_{2max}$/$\dot{V}O_{2peak}$), has been shown to be inversely related to markers of inflammation and oxidative stress[27] and is one of the most effective predictors of fatal and nonfatal CVD-related events.[50] Moreover, individuals who regularly exercise are less susceptible to oxidative stress and inflammation.[54,55] This is explained by the hormesis hypothesis, such that acute exposure to mild oxidative stress (via exercise) results in favorable adaptations in terms of increased antioxidant status to mitigate oxidative stress.[56,57] Chronic exercise training results in improvements to aerobic exercise capacity and numerous cellular changes (eg, mitochondrial biogenesis, increased concentrations of aerobic/lipolytic enzymes, and

TABLE 6. OLS Regression of Blood Markers by Fat Measures

	AOPPs[a]			HOMA-IR[a]			CRP		
	b	SE	*β*	*b*	SE	*β*	*b*	SE	*β*
Waist circumference	0.08	0.02	0.54***	0.05	0.02	0.32*	222.99	1,210.26	0.03
Fat percentage	−0.02	0.02	−0.17	0.03	0.02	0.24	2,155.82	938.98	0.33*
Constant	2.28	0.55		−3.49	0.61		−24,372.26	33,669.30	
Adjusted R^2	0.17			0.26			0.10		
n	92			95			90		

[a]Dependent variable log transformed.
*$P \leq 0.05$, ***$P \leq 0.001$.
AOPPs, advanced oxidation protein products; CRP, C-reactive protein; HOMA-IR, Homeostatic Model Assessment for Insulin Resistance; OLS, ordinary least-squares.

TABLE 7. Blood Marker Correlation Matrix

	AOPPs	HOMA-IR
HOMA-IR	0.34***	
CRP	0.12	0.28**

**$P \le 0.01$, ***$P \le 0.001$.

AOPPs, advanced oxidation protein products; CRP, C-reactive protein; HOMA-IR, Homeostatic Model Assessment for Insulin Resistance.

increased antioxidant capacity).[54] Recent work by Strauss et al[33] demonstrated significant inverse relationships between CRF levels and cardiovascular risk factors in firefighters. Specifically, the authors reported lower body mass index, waist circumference, body fat percentage, blood TAG and cholesterol concentrations, and resting BP and HR in firefighters.[33] The findings from the present study provide additional support to those reported by Strauss et al.[33] In addition, the present findings also show an inverse relationship with increasing levels of CRF and blood markers of inflammation, oxidative stress, and insulin resistance. These findings are important to the firefighter community given the high prevalence of CVD and metabolic syndrome.[2,58]

With respect to muscular fitness, the current findings do not suggest significant relationships between muscular fitness and blood markers of CVD risk. However, a review by Artero et al[32] provides an overview between the importance of muscular strength/fitness in relation to reduced risk for all-cause mortality, obesity, hypertension, and metabolic syndrome. Other work suggests that CRF and muscular strength are inversely related to long-term risk for heart failure in Swedish military service personnel.[59] In terms of proposed mechanisms, higher levels of muscular strength may be associated with reduced android adiposity, improved lipid profiles and insulin sensitivity, and reduced inflammation.[32]

It should be mentioned that the current study does have some limitations. First, the present study used a sample from a single department in Texas. Thus, future studies should examine a larger scale population sample to further extrapolate findings. In addition, one single time point was used for analysis (ie, blood sample, GXT). This could also be viewed as a limitation especially because acute changes in occupational factors (eg, call frequency, intensity, acute stressors) may impact some of the results. Future studies involving physiological metrics should attempt to record call frequency, type, and/or intensity.

In summary, our data demonstrate significant relationships between physical and biochemical measures of health in professional firefighters, with waist circumference and blood concentrations of TAGs significantly related to AOPPs and HOMA-IR. Moreover, CRF was inversely related to AOPPs, HOMA-IR, and CRP. Collectively, these findings highlight the importance of maintaining a high level of CRF and low waist circumference to reduce cardiometabolic risk, such as insulin resistance, oxidative stress, and inflammation. Future studies should seek to extend these findings, while also continuing to examine relationships between muscular fitness metrics and markers of cardiometabolic disease in firefighters and other tactical occupations.

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REFERENCES

1. Fahy RF, Petrillo JT, Molis JL. Firefighter fatalities in the US—2019. *NFPA Research*. 2019;1–26.
2. Soteriades ES, Smith DL, Tsismenakis AJ, Baur DM, Kales SN. Cardiovascular disease in US firefighters: a systematic review. *Cardiol Rev*. 2011;19:202–215.
3. Smith DL, Friedman NMG, Bloom SI, et al. Firefighting induces acute inflammatory responses that are not relieved by aspirin in older firefighters. *J Occup Environ Med*. 2019;61:617–622.
4. Barros B, Oliveira M, Morais S. Firefighters' occupational exposure: contribution from biomarkers of effect to assess health risks. *Environ Int*. 2021;156:106704.
5. D'Oria R, Schipani R, Leonardini A, et al. The role of oxidative stress in cardiac disease: from physiological response to injury factor. *Oxid Med Cell Longev*. 2020;2020:5732956.
6. Tsismenakis AJ, Christophi CA, Burress JW, Kinney AM, Kim M, Kales SN. The obesity epidemic and future emergency responders. *Obesity (Silver Spring)*. 2009;17:1648–1650.
7. Poston WS, Haddock CK, Jahnke SA, Jitnarin N, Tuley BC, Kales SN. The prevalence of overweight, obesity, and substandard fitness in a population-based firefighter cohort. *J Occup Environ Med*. 2011;53:266–273.
8. Ross R, Neeland IJ, Yamashita S, et al. Waist circumference as a vital sign in clinical practice: a consensus statement from the IAS and ICCR Working Group on Visceral Obesity. *Nat Rev Endocrinol*. 2020;16:177–189.
9. Flint AJ, Rexrode KM, Hu FB, et al. Body mass index, waist circumference, and risk of coronary heart disease: a prospective study among men and women. *Obes Res Clin Pract*. 2010;4:e171–e181.
10. Jha K MA, Marvel F, Ndumele CE, Martin SS, Blaha M, Blumenthal R. Waist circumference as a vital sign: is it ready for prime time? American College of Cardiology [Internet]. 2021. Available at: https://www.acc.org/latest-in-cardiology/articles/2021/05/07/17/27/waist-circumference-as-a-vital-sign. Accessed January 25, 2022.
11. García-Sánchez A, Miranda-Díaz AG, Cardona-Muñoz EG. The role of oxidative stress in physiopathology and pharmacological treatment with pro- and antioxidant properties in chronic diseases. *Oxid Med Cell Longev*. 2020; 2020:2082145.
12. Khansari N, Shakiba Y, Mahmoudi M. Chronic inflammation and oxidative stress as a major cause of age-related diseases and cancer. *Recent Pat Inflamm Allergy Drug Discov*. 2009;3:73–80.
13. Solleiro-Villavicencio H, Rivas-Arancibia S. Effect of chronic oxidative stress on neuroinflammatory response mediated by $CD4^+$ T cells in neurodegenerative diseases. *Front Cell Neurosci*. 2018;12.
14. Reuter S, Gupta SC, Chaturvedi MM, Aggarwal BB. Oxidative stress, inflammation, and cancer: how are they linked? *Free Radic Biol Med*. 2010;49:1603–1616.
15. Stoner L, Lucero AA, Palmer BR, Jones LM, Young JM, Faulkner J. Inflammatory biomarkers for predicting cardiovascular disease. *Clin Biochem*. 2013;46: 1353–1371.
16. Cozlea DL, Farcas DM, Nagy A, et al. The impact of C reactive protein on global cardiovascular risk on patients with coronary artery disease. *Curr Health Sci J*. 2013;39:225–231.
17. Blake GJ, Ridker PM. Inflammatory bio-markers and cardiovascular risk prediction. *J Intern Med*. 2002;252:283–294.
18. Pearson TA, Mensah GA, Alexander RW, et al. Markers of inflammation and cardiovascular disease. *Circulation*. 2003;107:499–511.
19. Ridker PM. High-sensitivity C-reactive protein, inflammation, and cardiovascular risk: from concept to clinical practice to clinical benefit. *Am Heart J*. 2004;148: S19–S26.
20. Tayefi M, Tajfard M, Saffar S, et al. hs-CRP is strongly associated with coronary heart disease (CHD): a data mining approach using decision tree algorithm. *Comput Methods Programs Biomed*. 2017;141:105–109.
21. Ridker PM, Cook N. Clinical usefulness of very high and very low levels of C-reactive protein across the full range of Framingham risk scores. *Circulation*. 2004;109:1955–1959.
22. Lagrand WK, Visser CA, Hermens WT, et al. C-reactive protein as a cardiovascular risk factor. *Circulation*. 1999;100:96–102.
23. O'Connell ER, Thomas PC, Cady LD, Karwasky RJ. Energy costs of simulated stair climbing as a job-related task in fire fighting. *J Occup Med*. 1986;28: 282–284.
24. Gledhill N, Jamnik VK. Characterization of the physical demands of firefighting. *Can J Sport Sci*. 1992;17:207–213.
25. Bilzon JL, Scarpello EG, Smith CV, Ravenhill NA, Rayson MP. Characterization of the metabolic demands of simulated shipboard Royal Navy fire-fighting tasks. *Ergonomics*. 2001;44:766–780.
26. Norris MS, McAllister M, Gonzalez AE, et al. Predictors of work efficiency in structural firefighters. *J Occup Environ Med*. 2021;63:622–628.
27. Rosado-Pérez J, Mendoza-Núñez VM. Relationship between aerobic capacity with oxidative stress and inflammation biomarkers in the blood of older Mexican urban-dwelling population. *Dose Response*. 2018;16:1559325818773000.
28. Kawamura T, Muraoka I. Exercise-induced oxidative stress and the effects of antioxidant intake from a physiological viewpoint. *Antioxidants (Basel)*. 2018; 7:119.
29. Wichitsranoi J, Ladawan S, Sirijaichingkul S, Settasatian N, Leelayuwat N. Relationship between aerobic capacity and cardiovascular disease risk factors in Thai men and women with normolipidemia and dyslipidemia. *J Phys Ther Sci*. 2015;27:3503–3509.
30. Abel MG, Palmer TG, Trubee N. Exercise program design for structural firefighters. *Strength Cond J*. 2015;37:8–19.

31. Magnussen CG, Schmidt MD, Dwyer T, Venn A. Muscular fitness and clustered cardiovascular disease risk in Australian youth. *Eur J Appl Physiol*. 2012;112: 3167–3171.
32. Artero EG, Lee DC, Lavie CJ, et al. Effects of muscular strength on cardiovascular risk factors and prognosis. *J Cardiopulm Rehabil Prev.* 2012;32:351–358.
33. Strauss M, Foshag P, Jehn U, Brzęk A, Littwitz H, Leischik R. Higher cardiorespiratory fitness is strongly associated with lower cardiovascular risk factors in firefighters: a cross-sectional study in a German fire brigade. *Sci Rep*. 2021;11:2445.
34. Ferguson B. ACSM's guidelines for exercise testing and prescription 9th ed. 2014. *J Can Chiropr Assoc*. 2014;58:328–328.
35. World Health Organization. Waist circumference and waist-hip ratio: report of a WHO expert consultation, Geneva, 8–11 December 2008. 2011.
36. Bruce RA. Methods of exercise testing. Step test, bicycle, treadmill, isometrics. *Am J Cardiol*. 1974;33:715–720.
37. Foster C, Jackson AS, Pollock ML, et al. Generalized equations for predicting functional capacity from treadmill performance. *Am Heart J.* 1984;107:1229–1234.
38. Liguori G, American College of Sports Medicine. *ACSM's Guidelines for Exercise Testing and Prescription*. Lippincott Williams & Wilkins; 2020.
39. National Fire Protection Association. *Comprehensive Occupational Medical Program for Fire Departments (NFPA 1582)*. Quincy, MA: National Fire Protection Association; 2003.
40. Bloomer RJ, Gunnels TA, Schriefer JM. Comparison of a restricted and unrestricted vegan diet plan with a restricted omnivorous diet plan on health-specific measures. *Healthcare (Basel)*. 2015;3:544–555.
41. Ellulu MS, Patimah I, Khaza'ai H, Rahmat A, Abed Y. Obesity and inflammation: the linking mechanism and the complications. *Arch Med Sci*. 2017;13:851–863.
42. Bawadi H, Katkhouda R, Tayyem R, Kerkadi A, Bou Raad S, Subih H. Abdominal fat is directly associated with inflammation in persons with type-2 diabetes regardless of glycemic control—a Jordanian study. *Diabetes Metab Syndr Obes*. 2019;12:2411–2417.
43. Kershaw EE, Flier JS. Adipose tissue as an endocrine organ. *J Clin Endocrinol Metabol*. 2004;89:2548–2556.
44. Kumari R, Kumar S, Kant R. An update on metabolic syndrome: metabolic risk markers and adipokines in the development of metabolic syndrome. *Diabetes Metab Syndr Clin Res Rev.* 2019;13:2409–2417.
45. Hivert M-F, Sullivan LM, Fox CS, et al. Associations of adiponectin, resistin, and tumor necrosis factor-α with insulin resistance. *J Clin Endocrinol Metabol*. 2008;93:3165–3172.
46. Hotamisligil GS, Spiegelman BM. Tumor necrosis factor α: a key component of the obesity-diabetes link. *Diabetes*. 1994;43:1271–1278.
47. Peterson MD, Al Snih S, Serra-Rexach JA, Burant C. Android adiposity and lack of moderate and vigorous physical activity are associated with insulin resistance and diabetes in aging adults. *J Gerontol A Biol Sci Med Sci*. 2015; 70:1009–1017.
48. Ponce-de-Leon M, Linseisen J, Peters A, et al. Novel associations between inflammation-related proteins and adiposity: a targeted proteomics approach across four population-based studies. *Transl Res*. 2021;242:93–104.
49. Hariri AA, Oliver NS, Johnston DG, Stevenson JC, Godsland IF. Adiposity measurements by BMI, skinfolds and dual energy x-ray absorptiometry in relation to risk markers for cardiovascular disease and diabetes in adult males. *Dis Markers*. 2013;35:753–764.
50. Laukkanen JA, Khan H, Kunutsor S. Cardiorespiratory fitness, muscle strength and risk of cardiovascular outcomes. *J Public Health Emerg*. 2017;1.
51. Kodama S, Saito K, Tanaka S, et al. Cardiorespiratory fitness as a quantitative predictor of all-cause mortality and cardiovascular events in healthy men and women: a meta-analysis. *JAMA*. 2009;301:2024–2035.
52. Myers J, Prakash M, Froelicher V, Do D, Partington S, Atwood JE. Exercise capacity and mortality among men referred for exercise testing. *N Engl J Med*. 2002;346:793–801.
53. Blair SN, Kampert JB, Kohl HW 3rd, et al. Influences of cardiorespiratory fitness and other precursors on cardiovascular disease and all-cause mortality in men and women. *JAMA*. 1996;276:205–210.
54. Sallam N, Laher I. Exercise modulates oxidative stress and inflammation in aging and cardiovascular diseases. *Oxid Med Cell Longev.* 2016;2016:7239639.
55. Samjoo IA, Safdar A, Hamadeh MJ, Raha S, Tarnopolsky MA. The effect of endurance exercise on both skeletal muscle and systemic oxidative stress in previously sedentary obese men. *Nutr Diabetes*. 2013;3:e88–e88.
56. Radak Z, Chung HY, Koltai E, Taylor AW, Goto S. Exercise, oxidative stress and hormesis. *Ageing Res Rev.* 2008;7:34–42.
57. Peake JM, Markworth JF, Nosaka K, Raastad T, Wadley GD, Coffey VG. Modulating exercise-induced hormesis: does less equal more? *J Appl Physiol*. 2015;119:172–189.
58. Smith DL, Fehling PC, Frisch A, Haller JM, Winke M, Dailey MW. The prevalence of cardiovascular disease risk factors and obesity in firefighters. *J Obes*. 2012;2012:908267.
59. Lindgren M, Åberg M, Schaufelberger M, et al. Cardiorespiratory fitness and muscle strength in late adolescence and long-term risk of early heart failure in Swedish men. *Eur J Prev Cardiol*. 2017;24:876–884.