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Cardiorespiratory Fitness, Alcohol Intake, and Metabolic Syndrome Incidence in Men

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Abstract

Purpose—To prospectively examine the independent and joint effects of alcohol consumption and cardiorespiratory fitness on the incidence of metabolic syndrome in a cohort of men.

Methods—A prospective examination of 3,411 apparently healthy men at baseline, who came to the Cooper Clinic (Dallas, Texas) for at least 2 preventive visits (1979–2010). Primary exposure variables were cardiorespiratory fitness and alcohol intake; the outcome measure was metabolic syndrome (MetS) and the components thereof. Cox proportional hazard models were computed to assess the relationship between the exposure variables and the incidence of MetS while adjusting for confounders.

Results—Over a mean follow-up period of 9 years (SD=7.8), 276 men developed MetS. In multivariable analysis, a dose-response relationship was observed between increased levels of fitness and reduced MetS risk (moderate fitness: HR=0.60, 95%CI 0.43–0.82; high fitness: HR=0.49, 95%CI 0.35–0.69). When examining the independent effects of alcohol, light drinking increased the risk for MetS by 66% (HR=1.66, 95%CI 1.11–2.48). No statistically significant interaction effect was observed between alcohol and fitness in relation to MetS ($P = 0.32$). When assessing the relation between each exposure and the components of MetS, higher fitness consistently reduced the risk of all components; whereas lower alcohol intake reduced the risk of elevated glucose and blood pressure and increased the risk for low HDL-c.

Conclusions—Among this cohort of men, higher fitness levels reduced the risk for MetS and its components. The relation between alcohol intake levels and metabolic risk was more complex and not reflected when examining MetS as a whole.

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Keywords

CARDIORESPIRATORY FITNESS; ALCOHOL INTAKE; METABOLIC SYNDROME; COHORT STUDY

Metabolic syndrome (MetS) is defined by the American Heart Association/ National Heart, Lung, and Blood Institute as a clustering of metabolic abnormalities, including elevated plasma glucose, central obesity, dyslipidemia, and hypertension (6, 13). This constellation of metabolic risk factors is associated with increased risk for developing type 2 diabetes and cardiovascular diseases (CVD) (16), and once diabetes has developed, the risk for CVD increases even further (13, 25). The age-adjusted prevalence of MetS in the U.S. is 24% in men, with an increased prevalence with age (8). Physical activity and cardiorespiratory fitness (fitness) have been linked independently to MetS. Higher levels of physical activity and fitness increase insulin sensitivity, improve endothelial function, and are inversely associated with MetS (12). For example, Ribisl et al., in a cross-sectional study, and Hassinen et al., in a prospective cohort study, found a dose response effect between increased fitness and a decrease in prevalence and incidence of MetS (14, 29). In comparison to the inverse association between fitness and MetS, the direction of the alcohol-MetS relationship has not been consistent; with some studies finding an inverse association while others a positive or null association (2, 23, 30).

Though the independent effects of fitness and alcohol on MetS have been examined, the combined impact has rarely been investigated. A number of studies have assessed the effects of alcohol intake in conjunction with physical activity on type 2 diabetes and coronary heart disease; finding moderate alcohol intake to be protective against these conditions while controlling for physical activity and other lifestyle factors (e.g. weight and smoking status) (17, 24). These studies, however, have not focused on MetS as an endpoint, and have examined self-report physical activity, which might be subject to recall and information bias (31). The effects of objectively measured fitness, a physiological consequence of physical activity behavior (26), and alcohol on MetS have yet to be investigated. Hence in the current study we investigate the effects of both fitness and alcohol consumption (independently and jointly) on the incidence of MetS in a large cohort of healthy men.

METHODS

Design and Participants

We prospectively examined patients from the Cooper Clinic (Dallas, Texas), who were enrolled in the Cooper Center Longitudinal Study (CCLS), to assess the associations between fitness, alcohol intake, and the incidence of MetS. The CCLS, a continuation of the Aerobics Center Longitudinal Study(3) with additional morbidity and mortality data, is an ongoing cohort study aimed primarily at examining the health-promoting effects of physical activity and cardiorespiratory fitness on cardiovascular health and longevity (7). The Cooper Institute's Institutional Review Board approves the CCLS annually; the current study received exempt status from the Institutional Review Board of the University of Texas Health Science Center at Houston. Participants were included in these analyses if they were adult men, came to the Cooper Clinic for at least 2 preventive service visits between 1979 and 2010, and had complete measurements pertaining to the primary exposure variables (i.e., fitness and alcohol intake), the outcomes (i.e., MetS and its components), and covariates (e.g., smoking status). Of the 17,884 men meeting these inclusion criteria, participants with prevalent or potential underlying diseases were excluded: 11,928 with MetS or at least one of its components; 1,105 with abnormal ECG; 860 reporting a personal history of cancer, myocardial infarction, or stroke; 442 who did not reach 85% of their

maximal heart rate (220-age) on the treadmill test at baseline; and 138 men with <1 year of follow up. These criteria resulted in 3,411 apparently healthy men, aged 20 – 79 years for analysis, who were primarily non-Hispanic white (>95%) and well-educated.

Primary Exposure Variables

Cardiorespiratory fitness was measured using the modified-Balke protocol (18). In this protocol, the treadmill speed is set at 88 meters/minute for 25 minutes. The treadmill's incline is set at 0% for the first minute, 2% for the second minute, and is followed by 1% increases each minute up to 25 minutes. After the 25-minute juncture, the incline remains constant and the speed is increased by 5.4 meters/minute each minute until the end of the test. From the final treadmill speed and grade, which is highly correlated with maximal oxygen uptake (28), we calculated maximal metabolic equivalents (METs) (1 MET = 3.5 ml O_2 uptake · kg body mass^{-1} · min^{-1}). Participants were assigned to fitness categories based on their age groups (20–39, 40–49, 50–59, and ≥60 years) and maximal METs from the treadmill test. Quintiles of METs were determined for each age group. Men in the first quintile within each age group were assigned to the low fitness category. Those in the second and third quintiles were assigned to the moderate fitness category and those in the fourth and fifth to high fitness strata.

Alcohol consumption was measured through responses to a medical history questionnaire, asking participants how many times per week they consumed a 12-ounce (355 ml) beer, a 3-ounce (89 ml) glass of wine, or a 1.5-ounce (44 ml) drink of hard liquor (33). Based on these responses, participants were categorized into 1 of 4 mutually exclusive categories: 1) nondrinkers, if not consuming alcohol; 2) light drinkers (≤3 drinks per week); 3) moderate drinkers (>3 to 14 drinks per week); and 4) heavy drinkers (>14 drinks per week) (32, 33).

Primary Outcome Measures

MetS was defined as meeting 3 or more of the following 5 criteria measured during a subsequent clinical examination: 1) elevated waist circumference ≥102 cm; 2) triglycerides ≥150 mg/dL; 3) low high-density lipoprotein cholesterol (HDL-c) <40 mg/dL; 4) blood pressure ≥130 mm Hg systolic or ≥85 mm Hg diastolic; and 5) fasting glucose ≥100 mg/dL (13). Trained staff measured blood pressure at the first and fifth Korotkoff sounds using auscultation methods. Serum samples were analyzed for lipids and glucose using automated bioassays based on the Centers for Disease Control and Prevention Lipid Standardization Program. Waist circumference was measured at the umbilicus level with a tape measure. In the analysis, MetS was a dichotomous variable (yes/no) and each component of the syndrome (yes/no) was assessed independently using the criteria listed above.

Covariates

The covariates, selected based on the literature (18), were derived from responses to the medical history questionnaire and adjusted for in multivariable analysis. Covariates included age, smoking status (never, former, current), and family history of CVD (yes/no). Additionally, the date (i.e., month and year) of the baseline clinic visit was ascertained for each participant and adjusted for in the analysis. Furthermore, participants' height and weight, measured during the clinic visit, was utilized to calculate body mass index (BMI) using the standard formula (kg/m^2), which was controlled for in the analysis.

Statistical Analysis

The incidences of MetS and its components for cases were calculated as the follow-up interval between the date of participants' baseline visit (free of MetS and its components) and the date of the next clinical visit in which the criteria for MetS and its components were

met (18). The follow-up period for the non-cases was calculated as the difference between the date of the baseline visit (free of MetS and its components) and the last clinic visit (also free of MetS and its components). Incidence rates (per 10,000) were calculated as the number of cases divided by the person years of exposure in cases and non-cases according to fitness levels and alcohol intake. Cox proportional hazard models were computed to estimate hazard ratios (HR) and 95% confidence intervals (CI) of MetS incidence according to fitness (low, moderate, high) and alcohol intake (nondrinkers, low, moderate, heavy). Low fitness and heavy alcohol intake, both detrimental to cardiometabolic risk, were utilized as the reference categories. We assessed the proportional hazards assumption using the method of Lin et al. based on cumulative sums of Martingale residuals (21). Four regression models were computed for each exposure separately (i.e., fitness and alcohol) with MetS (yes/no) as the outcome while adjusting for the following covariates: 1) age; 2) age, baseline examination year, smoking status, and family history of CVD; 3) all variables in models 1 and 2 plus either fitness or alcohol intake; and 4) variables in model 3 plus BMI. Effect modification was tested using the cross products of alcohol and fitness as the interaction variable in the regression models. We used the Wald test to examine the linear and quadratric trends across ordered levels of fitness and alcohol. Additionally, we assessed the joint effects of alcohol and fitness on MetS incidence in the same model while controlling for all covariates. We also examined the effects of the exposures (separately) on the incidence of each of the 5 components of MetS, while adjusting for age, examination year, smoking status, BMI, family history of CVD, and either alcohol intake or fitness. *P* values were two sided with an alpha of 0.05. All statistical analysis was conducted using SAS (version 9.2, SAS Institute, Cary, NC).

RESULTS

Of the 3,411 participants followed for a mean duration of 9 years (SD=7.8), 276 developed MetS. At baseline, participants' mean age was 42.3 (SD=8.6) years, they were of normal weight (i.e., mean BMI=24.7, SD=2.3 kg/m^2), and 87% were not current smokers. Their estimated oxygen uptake at baseline was 46.2 mL/kg/minute (SD=7.3), and slightly less than half (48%) were moderate drinkers. Baseline characteristics of participants, stratified by drinking levels, are depicted in Table 1. Most notably, men who were heavy drinkers smoked more and had higher total cholesterol and HDL-c levels than light drinkers.

The independent associations between fitness, alcohol intake, and the incidence and risk of developing MetS appear in Table 2. The age-adjusted incidence rate of MetS decreased linearly with the increase in fitness ($P < 0.001$), that is, the incidence rates per 10,000 person years were 142.7 in the low fit, 97.7 in the moderately fit, and 62.9 in the high fit strata. A similar relationship was found in multivariable analysis. There was a 40% reduced risk in the moderately fit as compared to the reference group of the low fit (HR=0.60; 95%CI 0.43–0.82) and a 51% reduced risk in the high fit strata (HR=0.49, 95%CI 0.35–0.69), while controlling for age, examination year, smoking, BMI, family history of CVD, and alcohol consumption. When examining the association between alcohol and MetS incidence, the age-adjusted incidence rates per 10,000 person years were: 94.2 (non-drinkers), 89.5 (light drinkers), 82.7 (moderate drinkers), and 110.1 (heavy drinkers); however, this U-shaped curve was not statistically significant (curvilinear $P = 0.52$). In multivariable analysis, light drinking in comparison to the reference category of heavy drinking increased the risk for MetS by 66% (HR=1.66; 95%CI 1.11–2.48) after adjusting for fitness, BMI, and other covariates. Additionally, the joints effects of both alcohol intake and fitness on MetS (adjusted for potential confounders) appear in Table 3. The interaction between alcohol intake and fitness in relation to MetS was not statistically significant ($P = 0.32$). Light and non-drinking in the low fitness strata significantly increased the risk for MetS (HR=3.38,

95%CI 1.56–7.28; HR=3.08, 95%CI 1.34–7.09, respectively); in the moderate and high fitness strata these detrimental associations were not found.

In addition to examining the effects of alcohol intake and fitness on MetS, the effect of each of these exposures on the 5 components of MetS was assessed (Table 4). A significant linear relation was found between increased fitness and reduced risk for all metabolic components with the exception of elevated blood pressure. When this relation was examined categorically, both moderate and high fitness (in comparison to low fitness) significantly reduced the risk for elevated waist circumference, elevated levels of triglycerides, and low levels of HDL-c (e.g., for waist circumference: HR=0.57; 95%CI 0.42–0.78; HR=0.42 95%CI 0.29–0.61 in the moderate and high fitness strata, respectively). High fitness significantly reduced the risk for elevated fasting glucose (HR=0.77; 95%CI 0.63–0.95), whereas the association between moderate fitness and glucose (HR=0.83; 95%CI 0.68–1.02) did not reach statistical significance ($P = 0.07$). Moderate and high fitness did not significantly reduce the risk for elevated blood pressure.

In comparison to the fitness-MetS relationship, analyses with alcohol intake revealed variability across MetS components in both the direction and the statistical significance of the relationships (Table 4). Thus, significant and positive linear relationships were found between alcohol consumption and risks for elevated blood pressure ($P < 0.01$) and high glucose levels ($P = 0.04$), whereas for low HDL-c the relationship was significant and negative (i.e., increased alcohol intake reduced the risk, $P < 0.001$). When examining these relationships categorically (Table 4), low levels of alcohol consumption significantly increased the risk for low HDL-c by 71% in comparison to high levels of alcohol intake (HR=1.71, 95%CI 1.15–2.55). No statistically significant associations were found in categorical analysis between alcohol intake, waist circumference, triglycerides, or blood pressure.

DISCUSSION

Current study findings pertaining to the protective effects of fitness against MetS risk are consistent with a large body of research, including a previous study examining this cohort for a shorter duration (14, 18). This study expands upon the previous study by following this cohort an additional 7 years, while assessing the effects of two primary behavioral exposures on MetS and examining the impact on MetS components. Results emphasize the dose-response effect between fitness and reduced metabolic risk (i.e., 40% and 57% reduced risk in the moderate and high fitness strata respectively). Additionally, significant risk reductions were observed in all MetS components, with the exception of blood pressure, which was reduced (7% and 12% in the moderate and high fitness groups, respectively) without statistical significance. Most studies to date (both prospective observational studies and randomized controlled trials) have found higher levels of fitness to reduce hypertension incidence (27). Higher levels of cardiorespiratory fitness are associated with reduced resting heart rate, increased cardiac output, and reduced vascular resistance, all of which impact blood pressure (22, 27). The impact of exercise interventions, aimed at reducing blood pressure, is greater when the study population includes hypertensive patients (27). Hence, the lack of a statistically significant association between fitness and blood pressure is most probably due to the fact that participants in the present study were a normotensive sample with little variation in their blood pressure.

When looking at the independent effect of alcohol on MetS incidence, current findings indicate that higher levels of alcohol consumption were protective of MetS incidence. This finding is in contradiction with a meta-analysis by Alkerwi et al. and other studies finding a J-shape association; that is, lower, but not higher, levels of alcohol are protective against

MetS (2, 5, 20) In contrast, both Freiberg et al., in a cross-sectional study, and Gigleux et al., in a cohort study, determined that higher levels of alcohol decrease the risk of MetS, which is consistent with current findings (9, 10). In the present study, when assessing the impact of alcohol on the various components of MetS, the direction of association varied. Thus, of the 3 components significantly associated with alcohol intake, lower alcohol consumption was linked to the decreased risk of two (elevated blood pressure and glucose) and the increased risk of one (low HDL-c). The stronger association between alcohol and HDL-c levels appeared to have a paramount role in the alcohol-MetS association found in the current results. The positive effects of alcohol intake on HDL-c is consistent with previous studies, which found a dose-response relationship between increased alcohol and higher circulating levels of HDL, even in excess of 21 drinks per week (1). Proposed biological mechanisms for this association include increased transport rates of lipoprotein and lipoprotein lipase activity (4).

Interpretation of the study's findings should be tempered by its limitations. The sample consists of mostly white well-educated participants, which limits our ability to generalize findings to more diverse populations. This, however, enhances the study's internal validity by minimizing confounding based on the heterogeneity of the sample in terms of race/ethnicity and socio-economic status. Additionally, though information was obtained on the quantity of alcohol intake, drinking patterns (e.g., binge drinking), which also have important health implications, were not assessed (23). Further, the nondrinking category in the study included both current and lifetime abstainers who may have different cardio-metabolic risks (19). As a result, the nondrinking category was not used as the reference group and results pertaining to nondrinkers should be interpreted with caution. An additional limitation to the study pertains to not accounting for participants' medication use, an inherent limitation of this dataset. This might have resulted in non-differential misclassification of the outcome (i.e., MetS and its components). Therefore, the measures of association presented might be 'diluted', that is, biased towards the null (11). Similarly, sufficient dietary information on this sample was unavailable, thus hindering our ability to control for dietary intake in multivariable analysis. Furthermore, though fitness is highly correlated with physical activity, genetics make a significant contribution to aerobic capacity (22). Despite these limitations, the study has significant strengths. To our knowledge this is the first study to assess the combined effects of both fitness, a physiologic attribute of physical activity, and alcohol consumption on MetS risk. Additionally, the large cohort of patients free of CVD risk factors and a follow-up period is significant enough to assess the effects of both exposures on MetS incidence. Moreover, the prospective cohort study design enables calculating risk and determining a temporal relationship between exposures (fitness and alcohol) and outcome (MetS), a criteria enroute to causality (15).

In summary, current study findings emphasize the independent protective effects of cardiorespiratory fitness on the development of metabolic risk factors, irrespective of alcohol intake. Achieving higher levels of cardiorespiratory fitness should be regarded as preventive therapy in the reduction of risk for MetS and its components, and the subsequent development of type 2 diabetes and CVD. The alcohol intake-MetS risk relationship appears to be more complex: lower levels of alcohol intake are protective against elevated glucose and blood pressure, whereas higher alcohol intake protects against low HDL-c. These nuances are not reflected when assessing the effects of alcohol on MetS (as a whole) in this cohort. Future research should continue to explore the joint effects of fitness (and/or objectively measured physical activity) and alcohol intake (while taking into account both quantity and patterns of drinking) on MetS risk in additional, more diverse samples of healthy adults.

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TABLE 1

Baseline characteristics of all men and stratified by alcohol intake, Cooper Center Longitudinal Study, 1979 – 2010.

	Alcohol Intake [a]				
	All	**Non-drinkers**	**Light Drinkers**	**Moderate Drinkers**	**Heavy Drinkers**
N	3,411	504	782	1,643	482
Age (years)	42.3 ± 8.6	43.7 ± 8.6	41.4 ± 8.6	42.4 ± 8.4	41.9 ± 8.8
Follow-up (years)	9.0 ± 7.8	9.5 ± 7.8	8.4 ± 7.4	9.0 ± 7.8	9.6 ± 8.7
BMI (kg/cm^2)	24.7 ± 2.3	24.6 ± 2.5	24.6 ± 2.4	24.7 ± 2.2	24.7 ± 2.2
Waist circumference (cm)	87.6 ± 6.6	87.3 ± 6.9	86.9 ± 6.7	87.7 ± 6.5	88.3 ± 6.4
Glucose (mg/dL)	91.7 ± 5.4	90.9 ± 5.9	91.4 ± 5.3	91.9 ± 5.3	92.4 ± 5.4
Cholesterol (mg/dL)	195.5 ± 33.5	195.4 ± 33.4	191.2 ± 33.4	196.0 ± 32.9	200.7 ± 34.7
Triglycerides (mg/dL)	81.6 ± 27.8	82.6 ± 27.6	79.1 ± 28.1	82.1 ± 27.9	82.8 ± 27.1
HDL (mg/dL)	53.0 ± 10.1	49.1 ± 7.6	51.5 ± 9.6	54.0 ± 10.1	55.9 ± 11.3
Resting systolic blood pressure (mmHg)	113.2 ± 8.0	112.3 ± 8.0	113.1 ± 7.9	113.2 ± 8.2	113.8 ± 7.6
Resting diastolic blood pressure (mmHg)	75.2 ± 6.0	74.9 ± 6.1	74.8 ± 6.3	75.2 ± 6.1	75.7 ± 5.4
MTs	13.2 ± 2.1	12.9 ± 2.1	13.2 ± 2.1	13.3 ± 2.1	13.4 ± 2.1
Smoking (n, %)					
Never smoker	2124 (62.3)	365 (72.4)	157 (20.1)	998 (60.7)	210 (43.6)
Former smoker	842 (24.7)	98 (19.4)	551 (70.5)	418 (25.4)	169 (35.1)
Current smoker	445 (13.1)	41 (8.1)	4 (9.5)	227 (13.8)	103 (21.4)
Fitness [b]					
Low	564 (16.5)	103 (20.4)	139 (17.8)	251 (15.3)	71 (14.7)
Moderate	1436 (42.1)	226 (44.8)	317 (40.5)	696 (42.4)	197 (40.9)
High	1411 (41.4)	175 (34.7)	326 (41.7)	696 (42.4)	214 (44.4)

Abbreviations: BMI, body mass index; HDL, high density lipoprotein cholesterol; METs, maximal metabolic equivalent (1 MET = 3.5 ml O_2 uptake · kg body mass^{-1} · min^{-1}).

Values are mean ± SD unless otherwise stated.

[a] Alcohol intake categories consisted of either ‘nondrinkers’ or to one of the following categories: (1) ‘light drinker’ (≤3 drinks per week); (2) ‘moderate drinker’ (>3 to 14 drinks per week); and (3) ‘heavy drinker’ (>14 drinks per week).

[b] Age-specific cardiorespiratory fitness categories were defined by the age-specific quintile distribution of METs for this sample of men in the Cooper Center Longitudinal Study utilizing the following age groups: 20–39, 40–49, 50–59, and ≥60 years. Low fitness represents quintile 1 of the age-specific fitness distribution, moderate fitness is quintiles 2 and 3 of the distribution, and high fitness is comprised of quintiles 4 and 5.

TABLE 2

Incidence of metabolic syndrome stratified by cardiorespiratory fitness and alcohol intake in men, Cooper Center Longitudinal Study, 1979 – 2010.

	N	Cases	Follow-up (per 10,000 PY)	Age adjusted rate per 10,000 PY	HR (95% CI)[a]	HR (95% CI)[b]	HR (95% CI)[c]	HR (95% CI)[d]
Fitness[e]								
Low	564	66	4,618	142.7 (111.5–182.5)	1.0	1.0	1.0	1.0
Moderate	1436	124	12,585	97.7 (81.8–116.7)	0.56 (0.41–0.76)	0.62 (0.45–0.85)	0.60 (0.44–0.83)	0.60 (0.43–0.82)
High	1411	86	13,532	62.9 (50.6–78.1)	0.38 (0.28–0.53)	0.45 (0.32–0.63)	0.43 (0.31–0.61)	0.49 (0.35–0.69)
Linear trend, *P* value				<0.0001	<0.0001	<0.0001	<0.0001	0.0002
Alcohol Intake[f]								
Non-Drinker	504	45	4,795	94.2 (70.4–126.2)	1.19 (0.79–1.79)	1.33 (0.86–2.05)	1.48 (0.96–2.30)	1.51 (0.97–2.36)
Light	782	59	6,569	89.5 (69.0–116.2)	1.32 (0.90–1.92)	1.41 (0.95–2.08)	1.56 (1.05–2.32)	1.66 (1.11–2.48)
Moderate	1643	121	14,733	82.7 (69.2–98.8)	1.11 (0.79–1.54)	1.08 (0.76–1.52)	1.21 (0.85–1.72)	1.29 (0.90–1.85)
Heavy	482	51	4,637	110.1 (83.5–145.2)	1.0	1.0	1.0	1.0
Curvilinear trend, *P* value				0.52 (linear: 0.54)	0.44 (linear: 0.28)	0.62 (linear: 0.10)	0.37 (linear: 0.04)	0.19 (linear: 0.04)

Abbreviations: HR, Hazards Ratio; CI- confidence interval; PY, person years.

[a]Hazard ratios were calculated via a Cox proportional hazard model, while adjusting for age.

[b]Hazard ratios were calculated via a Cox proportional hazard model, while adjusting for age, examination year, smoking status, and family history of cardiovascular disease.

[c]Hazard ratios were calculated via a Cox proportional hazard model, while adjusting for age, examination year, smoking status, family history of cardiovascular disease, and alcohol or fitness in respective models.

[d]Hazard ratios were calculated via a Cox proportional hazard model, while adjusting for age, examination year, smoking status, family history of cardiovascular disease, BMI, and alcohol or fitness in respective models.

[e]Age-specific cardiorespiratory fitness categories were defined by the age-specific quintile distribution of METs for this sample of men in the Cooper Center Longitudinal Study utilizing the following age groups: 20–39, 40–49, 50–59, and ≥60 years. Low fitness represents quintile 1 of the age-specific fitness distribution, moderate fitness is quintiles 2 and 3 of the distribution, and high fitness is comprised of quintiles 4 and 5.

[f]Alcohol intake categories consisted of either ‘nondrinkers’ or to one of the following categories: (1) ‘light drinker’ (≤3 drinks per week); (2) ‘moderate drinker’ (>3 to 14 drinks per week); and (3) ‘heavy drinker’ (>14 drinks per week).

TABLE 3

Joint effects of alcohol consumption and cardiorespiratory fitness on the incidence of metabolic syndrome in men, Cooper Center Longitudinal Study, 1979 – 2010.

	Cardiorespiratory Fitness[a]					
	Low		Moderate		High	
	Cases	HR (95% CI)	Cases	HR (95% CI)	Cases	HR (95% CI)
Alcohol Intake[b]						
Nondrinkers	12	3.08 (1.34–7.09)	19	1.10 (0.52–2.34)	14	1.08 (0.49–2.40)
Light	16	3.38 (1.56–7.28)	29	1.39 (0.69–2.78)	14	0.89 (0.41–1.95)
Moderate	25	1.63 (0.80–3.31)	55	1.24 (0.66–2.36)	41	0.84 (0.44–1.63)
Heavy	13	1.0	21	0.73 (0.36–1.47)	17	1.16 (0.55–2.49)

Abbreviations: HR, Hazards Ratio; CI- confidence interval

Hazard ratios (HR) were calculated via a Cox proportional hazard model, while adjusting for age, examination year, smoking status, family history of cardiovascular disease, and BMI.

[a]Age-specific cardiorespiratory fitness categories were defined by the age-specific quintile distribution of METs for this sample of men in the Cooper Center Longitudinal Study utilizing the following age groups: 20–39, 40–49, 50–59, and ≥60 years. Low fitness represents quintile 1 of the age-specific fitness distribution, moderate fitness is quintiles 2 and 3 of the distribution, and high fitness is comprised of quintiles 4 and 5.

[b]Alcohol intake categories consisted of either ‘nondrinkers’ or to one of the following categories: (1) ‘light drinker’ (≤3 drinks per week); (2) ‘moderate drinker’ (>3 to 14 drinks per week); and (3) ‘heavy drinker’ (>14 drinks per week).

TABLE 4

Incidence of the 5 components of metabolic syndrome stratified by cardiorespiratory fitness and alcohol intake in men, Cooper Center Longitudinal Study, 1979 – 2010.

	Metabolic Syndrome Components[a]									
	Elevated Waist Circumference		Elevated Triglycerides		Elevated Blood Pressure		Elevated Fasting Glucose		Low HDL-c	
	Age-adjusted incidence rate	HR (95% CI)	Age-adjusted incidence rate	HR (95% CI)	Age adjusted incidence rate	HR (95% CI)	Age-adjusted incidence rate	HR (95% CI)	Age-adjusted incidence rate	HR (95% CI)
Fitness[b]										
Low	151.3 (119.3–191.8)	1.0	206.3 (168.4–252.8)	1.0	329.8 (280.9–387.2)	1.0	312.4 (265.0–368.5)	1.0	166.9 (132.9–209.5)	1.0
Moderate	88.4 (73.3–106.5)	0.57 (0.42–0.78	128.7 (110.3–150.1)	0.64 (0.49–0.83)	310.7 (281.2–343.4)	0.93 (0.77–1.12)	267.1 (239.9–297.5)	0.83 (0.68–1.02)	112.8 (95.7–133.0)	0.64 (0.48–0.85)
High	40.9 (31.3–53.6)	0.42 (0.29–0.61)	94.0 (78.8–112.2)	0.52 (0.40–0.69)	266.1 (239.7–295.4)	0.88 (0.72–1.07)	242.4 (217.3–270.4)	0.77 (0.63–0.95)	67.7 (54.6–83.9)	0.44 (0.32–0.59)
Linear trend, *P* value	<0.0001	<0.0001	<0.0001	<0.0001	0.03	0.43	0.01	0.05	<0.0001	<0.0001
Alcohol Intake[c]										
Non-Drinker	79.3 (57.6–109.1)	1.20 (0.74–1.94)	115.3 (88.4–150.5)	1.00 (0.69–1.46)	253.1 (211.5–303.0)	0.71 (0.56–0.91)	220.9 (182.2–267.7)	0.81 (0.63–1.04)	160.9 (128.6–201.3)	2.38(1.59–3.57)
Light	83.4 (63.8–109.0)	1.50 (0.97–2.32)	112.0 (88.6–141.7)	1.01 (0.72–1.43)	293.8 (254.8–338.8)	0.87 (0.70–1.08)	218.2 (184.8–257.5)	0.82 (0.65–1.04)	115.2 (91.3–145.4)	1.69 (1.14–2.52)
Moderate	71.0 (58.5–86.0)	1.19 (0.80–1.76)	127.8 (110.7–147.5	1.04 (0.77–1.39)	295.6 (268.9–324.9)	0.86 (0.71–1.04)	276.7 (251.0–305.1)	0.99 (0.82–1.20)	83.5 (69.9–99.8)	1.17 (0.81–1.70)
Heavy	86.0 (62.7–118.1)	1.0	135.1 (104.8–174.2)	1.0	333.0 (284.1–390.3)	1.0	328.1 (279.7–384.8)	1.0	82.5 (59.8–113.9)	1.0
Curvilinear trend, *P* value		0.31 (Linear: 0.17)		0.85 (Linear: 0.98)		0.77 (Linear: 0.009)		0.97 (Linear: 0.04)		0.46 (Linear: <0.0001)

Abbreviations: HR, Hazards Ratio; CI- confidence interval.

Hazard ratios were calculated via a Cox proportional hazard model, while adjusting for age, examination year, smoking status, family history of cardiovascular disease, alcohol or fitness in respective models, and BMI.

[a]Elevated waist circumference: ≥102 cm; 2. Elevated triglycerides of ≥150 mg/dL; 3) Low HDL cholesterol of <40 mg/dL; 4) Elevated blood pressure of ≥130 mm Hg systolic or ≥85 mm Hg diastolic; and 5) Elevated fasting glucose ≥100 mg/dL.

[b]Age-specific cardiorespiratory fitness categories were defined by the age-specific quintile distribution of METs for this sample of men in the Cooper Center Longitudinal Study utilizing the following age groups: 20–39, 40–49, 50–59, and ≥60 years. Low fitness represents quintile 1 of the age-specific fitness distribution, moderate fitness is quintiles 2 and 3 of the distribution, and high fitness is comprised of quintiles 4 and 5.

[c]Alcohol intake categories consisted of either ‘nondrinkers’ or to one of the following categories: (1) ‘light drinker’ (≤3 drinks per week); (2) ‘moderate drinker’ (>3 to 14 drinks per week); and (3) ‘heavy drinker’ (>14 drinks per week).