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Muscular Strength and Incident Hypertension in Normotensive and Prehypertensive Men

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Abstract

The protective effects of cardiorespiratory fitness (CRF) on hypertension (HTN) are well known; however, the association between muscular strength and incidence of HTN has yet to be examined.

Purpose—This study evaluated the strength-HTN association with and without accounting for CRF.

Methods—Participants were 4147 men (20–82 years) in the Aerobics Center Longitudinal Study for whom an age-specific composite muscular strength score was computed from measures of a 1-repetition maximal leg and a 1-repetition maximal bench press. CRF was quantified by maximal treadmill exercise test time in minutes. Cox proportional hazards regression analysis was used to estimate hazard ratios (HRs) and 95% confidence intervals of incident HTN events according to exposure categories.

Results—During a mean follow-up of 19 years, there were 503 incident HTN cases. Multivariable-adjusted (excluding CRF) HRs of hypertension in normotensive men comparing middle and high strength thirds to the lowest third were not significant at 1.17 and 0.84, respectively. Multivariable-adjusted (excluding CRF) HRs of hypertension in baseline prehypertensive men comparing middle and high strength thirds to the lowest third were significant at 0.73 and 0.72 ($p=.01$ each), respectively. The association between muscular strength and incidence of HTN in baseline prehypertensive men was no longer significant after control for CRF ($p=.26$).

Conclusions—The study indicated that middle and high levels of muscular strength were associated with a reduced risk of HTN in prehypertensive men only. However, this relationship was no longer significant after controlling for CRF.

Keywords

physical fitness; blood pressure; cohort study; epidemiology

Introduction

Hypertension (HTN) is the most prevalent cardiovascular disease in the United States (US) affecting nearly one-third of adults aged 18 and older (9,35). Mortality due to HTN in 2002

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was estimated at 277,000 deaths and the overall death rate from HTN in 2004 was 17.9 per 100,000 persons (35). The estimated direct and indirect cost of HTN for 2007 was \$66.4 billion (35), a major burden on the US health care system. An estimated 62% of prehypertensive and hypertensive patients were unaware of their HTN status (39). Low awareness and inadequate management of HTN is a serious problem (39), specifically when prehypertension is a condition that likely leads to HTN. Therefore, it is very important to target prehypertensive individuals before they develop HTN. Lifestyle and behavioral modifications are stressed for the prevention, treatment, and control of HTN, with aerobic exercise being an integral component (30). Whether strength training can also provide protective benefits on HTN status remains uncertain.

Physical fitness is comprised of several components, including cardiorespiratory fitness (CRF) or aerobic fitness and muscular strength and endurance (10). The majority of studies that have examined the relationship between physical fitness and a variety of health outcomes have referred to physical fitness in the context of aerobic fitness (40). Similarly, studies that have examined physical activity (PA) have referred to PA as aerobic activity. These studies have established that aerobic fitness and PA are protective against many health outcomes, such as hypertension (1,2,4,7,8,11,14,15,17,24–28,32). However, little epidemiological research has been reported on the association of muscular strength on different health outcomes and none to report on the association between muscular strength and hypertension.

Traditionally, strength training has been seen as a means of improving muscular strength, power, endurance, lean body mass and bone mineral density for athletes and weight lifters, and for preservation of function in older adults; but strength training has not been seen as a means of improving health in the general population (31). The long-term biological mechanisms behind muscular strength and blood pressure are still not yet clearly defined and currently being studied. However, one possible mechanism that links strength training to hypertension status is through acute elevations in arterial blood pressure during weight-lifting that leads to long-term protective changes in the smooth muscle content of the arterial wall and to the long-term protective changes in the load-bearing properties of collagen and elastin that in turn leads to an overall decreased blood pressure during rest (3). Also, strength training may improve endothelial function and may upregulate the production of nitric oxide synthase and increase the release of endothelium-derived nitric oxide (32). This may be an important mechanism because hypertension may be associated with an impairment of endothelium-derived vasodilation related to a decrease in the upregulation of nitric oxide and a decrease in the release of endothelium-derived nitric oxide (32). Within the past decade, the modest amount of muscular strength research has shown that strength training influences health and disease prevention (6,13,16,19,20,20,34). Because only a few studies incorporate both strength and CRF, less is known on whether these health benefits of strength training are independent of, or additive to, those already established for CRF and PA (19). It may be possible that strength training can provide protection against developing hypertension beyond what is seen for physical activity and CRF (19).

This study examined the association between muscular strength and incidence of hypertension with and without accounting for CRF. To the best of our knowledge, this study is the first to examine the prospective association between muscular strength and incidence of hypertension in a cohort of healthy men initially free of hypertension.

Methods

Study Population and Design

Data for the current study are from the Aerobics Center Longitudinal Study (ACLS). The ACLS is a prospective study on the association of physical activity and physical fitness to health

outcomes in patients examined at the Cooper Clinic in Dallas, TX from 1970. Between 1980 and 1990, 9704 men aged 20–82 years received a comprehensive medical examination and muscular strength tests. Study participants came to the clinic for periodic preventive health examinations and for counseling regarding diet, exercise, and other lifestyle factors associated with increased risk of chronic disease. Many participants were sent for the examination by their employers, some were referred by their personal physicians, and others were self-referred. Inclusion criteria for the current analysis required participants to have a maximal treadmill exercise test at baseline, during which they must have achieved at least 85% of their age-predicted maximal heart rate ($220 - \text{age}(\text{years})$), completed both voluntary strength tests, and returned at least one follow-up health survey from 1982, 1986, 1990, 1995, 1999 or 2004. We excluded those with baseline hypertension defined as a history of physician diagnosis ($n=1293$) or resting blood pressure $\geq 140/90$ mmHg ($n=1864$), myocardial infarction ($n=117$), stroke ($n=12$), cancer ($n=55$), diabetes ($n=94$), abnormal resting or exercise ECG ($n=516$), and participants without a follow-up survey ($n=1,606$). In addition, we excluded 97 (2.3%) participants due to missing or incomplete data. These criteria resulted in 4147 initially normotensive men (age: 43 ± 9 , range: 20–82 years) who were followed up from the date of their baseline examination until their date of incident HTN, or June 30, 2004. Overall, the majority of the study participants was white, well-educated, from middle to upper socioeconomic strata, and employed in, or retired from, professional or executive positions. All participants provided written informed consent to participate in the follow-up study, and the Cooper Institute Institutional Review Board approved the study annually.

Baseline examination

Prior to the medical examination, the participants fasted for 12 hours and were asked not to smoke on the day of examination. The medical examination included: a physical exam; anthropometric assessments; blood pressure; blood chemistry tests; maximal treadmill exercise test; voluntary strength tests; and a questionnaire of personal and family medical history, demographic characteristics, and health habits. All procedures were administered by trained technicians who followed standardized protocols. Height and weight were measured using a stadiometer and standard physician's scale. Body mass index (BMI, kg/m^2) was computed as weight in kilograms divided by height in meters squared. Resting blood pressure was measured in the seated position and was recorded as the first and fifth Korotkoff sounds by auscultatory methods after at least 5 min of sitting quietly. Two or more readings separated by 2 min were averaged. If the first two readings differed by more than 5 mm Hg, additional readings were obtained and averaged. Serum samples were analyzed for lipids and glucose using standardized automated bioassays. Normotensive and prehypertensive participants were defined as having resting blood pressure $<120/80$ mmHg and as having either systolic blood pressure of 120–139 mmHg or diastolic blood pressure of 80–89 mmHg, respectively. Information on smoking habits (current smoker or not), alcohol intake (drinks per week), and family history of HTN were obtained from a standardized questionnaire.

CRF was assessed by a maximal treadmill test using a modified Balke protocol as previously described (5,20). CRF was defined as total minutes on the treadmill in all the multivariable adjusted models. However, CRF was divided into age-specific thirds (low, middle, and high fitness) with low fitness as the referent level for examining the joint effects of between muscular strength and CRF on incident HTN. Muscular strength was quantified from the results of a standardized strength assessment using variable-resistance Universal (Universal Equipment, Cedar Rapids, IA) weight machines (6,10,12,21). Upper body strength was assessed with a one-repetition maximum (1-RM) supine bench press. The initial load was 70% of body weight and increments of 2.27–4.54 kg were added and separated by short rest periods until maximal effort was achieved. Lower-body strength was assessed with a 1-RM seated leg press with an initial load set at 100% body weight and was increased 2.27–4.54 kg until maximum effort

was achieved. These assessment protocols have been previously been described more thoroughly (6,19). Test–retest reliability estimates were .90 for the bench press and .83 for the leg press indicating acceptable levels of true score and measurement error in the raw strength scores (20). It may be important to note that previous ACLS studies have investigated the degree to which muscular strength correlated with resistance exercise habits via self-report questionnaires and observed stronger participants also reported significantly higher levels of participation in resistance training activity using a questionnaire (20). This observation suggests that the measurements of muscular strength are an adequate reflection of the resistance exercise habits (20). A muscular strength index was computed by taking each individual's 1-RM score for the bench press and leg press, expressed as weight lifted per kilogram body weight, and dividing the scores into five age group distributions (20–29, 30–39, 40–49, 50–59, and 60+ yr). Within each age group, the individual's scores were subtracted from the age-specific mean and then divided by the age-specific standard deviation. These new scores for the bench press and leg press were averaged to express an age-specific standardized composite score for each individual. The age-specific composite strength scores were then divided into age-specific thirds (low, middle, and high strength) with the low strength third held as the referent level. Age-specific thirds were combined across age levels (i.e. all age-specific low strength thirds were combined into the overall low strength third).

Ascertainment of incidence of hypertension

The incidence of hypertension was ascertained from responses to mail-back health surveys in 1982, 1986, 1990, 1995, 1999, and 2004. The aggregate survey response rate across all survey periods in the ACLS is 65%. Non-response bias is a concern in epidemiological surveillance; however, this issue has been investigated in the ACLS and it was found that responders and non-responders were equally healthy at entry (23). Baseline health histories and clinical measures were similar between responders and non-responders and between early and late responders. The primary endpoint was defined as a participant's report of a physician diagnosis of HTN from the six mail-back surveys. The participants were asked if a physician has ever told them they had hypertension. If yes, respondents were asked to report the year of diagnosis. For participants who completed multiple surveys, the first survey in which hypertension was reported was used in analyses. This method of case ascertainment is similar to those used in other established epidemiologic studies on HTN (4,28). Sensitivity and specificity of self-reported, physician diagnosis of HTN in the ACLS cohort was previously verified at 98% and 99%, respectively (4).

Statistical Analysis

Descriptive statistics were computed for each variable using Chi-square tests, analysis of variance, and t-tests. The primary exposure variable was muscular strength and was defined categorically as low, middle, and high strength. Follow-up time among non-cases was computed as the difference between the date of the baseline examination and the date of the last returned survey. Follow-up time among cases was computed as the difference between the baseline examination date and the reported date of the HTN event. If a diagnosis date was not provided, the midpoint between the date of the case-finding survey and either the baseline examination date or the date of the last returned survey where the participant reported being free of HTN was used. We also conducted Pearson correlation analyses to examine the strength of the association among muscular strength composite score and CRF (minutes on treadmill), and the strength of the association among ungrouped components of muscular strength- one-repetition maximal bench press and leg press.

Incidence rates were computed as the number of incident hypertension cases divided by man-years of exposure for the population sample within categories of muscular strength. Cox proportional hazards regression analysis was used to estimate hazard ratios (HRs) and 95%

confidence intervals (CIs) of HTN events according to exposure categories. Ties were handled using the Efron method. The proportional hazards assumption was met in the comparison of the cumulative hazard plots grouped on exposure. Indicator variables (did not respond/ responded) for each of the 6 survey periods were constructed to account for differences in survey response frequency in order to reduce the influence of ascertainment bias. This is a typical approach to account for differences in survey response patterns among study participants. The indicator variables were entered into the models as covariates in order to standardize for surveillance and period and length of follow-up. In all men ($n=4147$), multivariable adjusted models first controlled for the potential confounding effects of baseline age (continuous), year of the baseline examination, and survey response frequency indicator variables (yes/no) (Model 1). Then multivariable adjusted models additionally controlled for current smoker (yes/no), alcohol intake (≥ 5 drinks/wk or not), family history of hypertension (present/absent for each), and baseline systolic blood pressure and diastolic blood pressure (continuous) (Model 2). Lastly, the association between muscular strength and hypertension after additional control for CRF (continuous minutes) was examined with and without control of BMI (continuous) because BMI may represent an intermediate variable lying in the causal pathway (Model 3, Model 4, respectively). To examine potential effect modification, stratum-specific Cox-regression analyses were performed according to baseline prehypertensive status. For baseline normotensive men ($n=1951$) and prehypertensive men ($n=2196$), multivariable adjusted models controlled for the same potential confounding effects as previously described. All these variables were left in the model regardless of significance to be consistent with previous ACLS publications and to remain accountable for adjusting for known hypertension risk factors. Contrast tests were performed to examine if the incidence of hypertension in the low strength third was significantly different than the average incidence of hypertension between the middle and high strength thirds after controlling for CRF, but not BMI. Finally, to examine additional potential effect modification, additional stratum specific Cox-regression analyses were performed on all men and prehypertensive men according to baseline age (<55 and ≥ 55 years), BMI (<25 vs. ≥ 25 kg/m²), and CRF (low, middle, and high fitness). We then calculated hypertension incidence rates and Cox proportional HRs for the joint associations of muscular strength (low, middle, and high) and CRF (low, middle, and high) in baseline prehypertensive men after adjusting for age, year of the baseline examination, current smoker, alcohol intake, family history of hypertension, and baseline systolic blood pressure and diastolic blood pressure. For all analyses, tests of linear trends were reported (unless otherwise noted) across exposure categories using ordinal scoring. All p values are two sided and all significant results had a p value below 0.05.

Results

A total of 503 incident cases (12.1%) of HTN occurred during a total of over 79,641 man-years (average of 19.2 years) of follow-up. Baseline characteristics of the study population by muscular strength group are shown in Table 1. There were statistically significant differences across muscular strength levels according to all variables except alcohol consumption and family history of hypertension. However, these statistically significant differences were comparable for most, but not all variables which is not surprising for such a large sample population. Baseline characteristics according to baseline prehypertensive status are shown in Table 2. There were statistically significant differences across baseline prehypertensive status for all variables except smoking, alcohol consumption, and kilograms per kg body weight for both upper and lower body strength. Similar to Table 1, these statistically significant differences were comparable for most, but not all variables. In addition, among normotensive men, there were significant differences in both systolic blood pressure and diastolic blood pressure between the low strength third and the middle strength third. In prehypertensive men, there was a significant difference in systolic blood pressure between the middle strength third and the high strength third (data not shown). The age-adjusted Pearson correlation between the

muscular strength composite score and the maximal treadmill exercise time was $r = 0.33$ ($p < 0.0001$). The Pearson correlation between bench press and leg press scores was $r = 0.58$ ($p < 0.0001$).

In all men ($n=4147$), there were 195, 161, and 147 incident hypertension cases in the low, middle, and high strength categories, respectively. Overall, we observed a significant relationship (p trend < 0.01) of hypertension incidence rates categories of muscular strength (Table 3). Incidence rates per 10,000 man-years in the high strength level and middle strength level were 27% and 20% lower than among men with low muscular strength, respectively. In baseline normotensive men ($n=1951$), there was no gradient (p trend $= 0.35$) across muscular strength levels. The incidence rate among men in the high strength level was 18% lower than among men with low strength, while incidence rates among men in the middle strength level were 14% higher than men in the low muscular strength level. In baseline prehypertensive men ($n=2196$), a significant relationship (p trend $= 0.012$) was observed across muscular strength levels with incidence rates among men with high strength and middle strength 26% and 28% lower than men with low strength, respectively.

HRs and 95% CIs were computed to quantify the strength of the association between muscular strength and incidence of hypertension in all men ($n=4147$) with the low strength group as the referent group (Table 3). After adjusting for age, examination year, and survey pattern response, we observed a significant inverse association (p trend $= 0.002$) between muscular strength and the risk of incident hypertension. Men in the high strength group had a 28% significant lower risk of developing hypertension than men in the low strength group. The association remained significant (p trend $= 0.009$) after additional adjustment for smoking, alcohol intake, family history of hypertension, and baseline systolic blood pressure and diastolic blood pressure. Men in the high strength group had a 25% lower risk of developing hypertension than men in the low strength group. The association was no longer significant after additional adjustment for CRF (p trend $= 0.63$), and after further additional adjustment for BMI (p trend $= 0.91$).

We next examined the influence of muscular strength on incidence of hypertension within baseline normotensive men and prehypertensive men (Table 3). No significant results were noted for baseline normotensive men. In baseline prehypertensive men, after adjusting for age, examination year, and survey pattern response, we observed a significant inverse association (p trend $= 0.007$) between muscular strength and the risk of hypertension. The risk of developing hypertension was 28% and 29% lower in the high strength level and middle strength level, respectively, compared with the low strength level. We also observed by the contrast test that the incidence of hypertension in the low strength group was significantly higher than the average incidence of hypertension between the middle and high strength groups ($p = 0.002$). The association remained significant (p trend $= 0.009$) after adjusting for smoking, alcohol intake, family history of hypertension, and baseline systolic blood pressure and diastolic blood pressure. In addition, we observed from the contrast test that the incidence of hypertension in the low strength third is significantly higher than the incidence of hypertension in the middle and high strength thirds ($p = 0.003$). After further adjustment of CRF, the association became not significant (p trend $= 0.3$); although not significant, the effects were still protective. After further adjustment of BMI, the association remained not significant (p trend $= 0.4$). In addition, the contrast test between low strength and the average of middle and high strength was no longer significant ($p = 0.13$).

We then examined the influence of muscular strength on incidence of hypertension within strata of known hypertension risk factors, such as age, BMI, and CRF in the entire study population and baseline prehypertensive men after adjusting for other potential confounders (data not shown). When stratified according to age ($< 55 / \geq 55$ years), BMI ($< 25 \text{ kg/m}^2 / \geq 25 \text{ kg/}$

m²), and CRF (low, middle, and high fitness) no significant effect modifiers or patterns were noted (data not shown).

Hypertension incidence rates according to the combination of muscular strength and CRF in baseline prehypertensive men are shown in Figure 1. Across levels of muscular strength, a protective trend in incidence of hypertension is suggested with progressively higher levels of CRF. Additionally, a protective trend between muscular strength and incidence of hypertension is suggested across levels of CRF. Figure 1 also shows the joint hypertension incidence HRs according to level of muscular strength and CRF in baseline prehypertensive men after adjustment for previously mentioned confounders, however, no significant trends across levels of muscular strength were noted. The overall trend suggests greater inverse HRs across muscular strength and CRF from low levels to high levels of each; although not significant, the effects were still protective.

Discussion

The primary findings from this study were: 1) In only baseline prehypertensive men (not normotensive men), muscular strength was inversely associated with incidence of hypertension (in models not including CRF); 2) the association between muscular strength and incidence of hypertension in baseline prehypertensive men was no longer significant after extensive control for CRF; however the association remained protective; 3) the inverse association seen only in baseline prehypertensive men and not in normotensive men focuses our attention on prehypertensive men, and this inverse association may suggest a threshold relationship, rather than a dose-response relationship in these men; and 4) although the joint associations were not significant, there was evidence of a protective trend for across levels of muscular strength in incidence of hypertension with progressively higher levels of CRF, and a protective trend for levels of CRF in incidence of hypertension with progressively higher levels of muscular strength.

Past studies have reported protective associations between CRF or aerobic activity on incidence of hypertension (2,4,11,14,15,17,25–29), and others have reported protective associations between muscular strength and adverse health outcomes (6,10,18,19,36), but we believe the data reported here are the first to demonstrate that muscular strength is inversely associated with the development of hypertension in models that do not consider CRF.

The relative importance of baseline blood pressure status was shown in Table 3. There was an inverse relationship across strength levels among prehypertensive men that was not seen in normotensive men, which suggests that baseline BP status (normotensive/prehypertensive) is an effect modifier and may provide evidence that the risk of hypertension by muscular strength categories is only seen in or is greatest for prehypertensive individuals. The risk in the prehypertensive men suggested a threshold relationship where men with any level higher than a low level of strength have the same reduced risk of hypertension compared to men with a low level of strength. This argument was somewhat strengthened with the results from the contrast tests. On average, men with at least middle strength had a lower risk of developing hypertension compared with men with low strength. Because this study is the first of its kind, we cannot compare our results with other similar studies.

Our analysis in this study shows that in baseline prehypertensive men, the association between muscular strength and incidence of hypertension is no longer significant after controlling for CRF. In our population, muscular strength and CRF measures were modestly correlated (age-adjusted Pearson $r=0.33$). This possibly adds strength to our results since correlation between exposures makes it more difficult to discern independent associations, therefore making it less likely that we would be able to observe significant results.

We ran three additional Cox proportional hazard regressions for comparisons with the primary analyses. First, we ran the regression with only lower body strength; second, we ran the regression with only upper body strength; and third, we ran the regression with both muscular strength and CRF as continuous variables (data not shown). All three regressions showed that baseline blood pressure status was the only effect modifier and also showed similar results compared with the analyses presented in the results section.

Strengths of the current study include the availability of an objective assessment of both muscular strength and CRF on a large sample of men. Muscular strength, assessed by one-repetition maximum bench press and leg press, is most likely stronger than self-reported strength training as a predictor of adverse health outcomes because this assessment may provide a more objective measure of recent strength training than the self-report data. Generally, studies examining the association between muscular strength and adverse health effects have focused on strength assessed in one muscle group (i.e. grip strength). Grip strength may have good reliability, but there is no consistent protocol across studies, which could ultimately result in significant differences in grip strength scores (22,38). To measure overall muscular strength, strength assessments should include more than one major muscle group (10). One repetition maximum bench and leg press are two common tests that have shown good reliability ($r=0.90-0.96$) (37) and these tests show better consistency in protocols across studies.

Since middle and high levels of muscular strength could represent a more active lifestyle (10), the ability to adjust for CRF allowed us to focus specifically on the relation of muscular fitness to incidence of hypertension. In addition, our muscular strength composite score was age-adjusted and standardized to allow for better comparisons between individuals. Similar to the analysis of Mora et al., BMI may lie or partially lie on the causal pathway between muscular strength and incident hypertension. By adjusting for BMI in model 4, we are able to examine the independent and joint effects of muscular strength and CRF on incident hypertension before consideration of BMI (24). This is the first study to our knowledge to report on the prospective association between muscular strength and incidence of hypertension.

One limitation of this study is that women could not be analyzed, due to their small sample sizes and low hypertension incidence rates. Because of the homogenous population, external validity is limited to healthy white men with mid- to high-socioeconomic status. However, the homogeneity of our sample on education, occupation, gender, and income enhances internal validity of our findings because the potential for confounding by these variables is reduced. The generalizability of the findings to women and other ethnicities are unknown. These participants were volunteers to the Cooper Clinic, which may result in a degree of self-selection bias. However, previous analyses in the ACLS cohort have investigated this bias and have found ACLS participants are very similar on key clinical measures such as lipids, glucose, and blood pressure to participants in other large epidemiological studies in the United States (2). Because strength testing was also offered on a volunteer basis, differences in baseline characteristics between those who volunteered for the strength tests and those who did not volunteer during 1980–1990 were investigated (data not shown), and there were no appreciable differences between baseline characteristics for these two groups. Lack of sufficient data on diet intake prohibits us from adjusting for potential nutritional confounders that may be risk factors for hypertension.

Our prospective findings in a large cohort of healthy men initially free of hypertension show a significant relationship between muscular strength and incidence of hypertension in baseline prehypertensive men only; however, this relationship is no longer significant after controlling for CRF. In these baseline prehypertensive men, higher levels of CRF alone may show greater protective associations against developing hypertension compared to higher levels of muscular strength alone, but our study suggests the possibility that higher levels of muscular strength in

each CRF level could provide small additive protective associations against developing hypertension. Future studies should continue to examine the relation between muscular strength and incidence of hypertension in larger populations that include women and minorities as well.

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FIGURE 1.

Age-adjusted hypertension incident rates and joint incident hypertension hazard ratios, adjusted for potential confounders, across levels of muscular strength and cardiorespiratory fitness in baseline prehypertensive men (n=2196), Aerobics Center Longitudinal Study, Dallas, Texas, 1980–2004. Incident rates per 10,000 person years and joint incident hazard ratios are shown above bars. Joint sample sizes are shown in table.

Table 1

Baseline characteristics according to muscular strength thirds, Aerobics Center Longitudinal Study, 1980–2004.

Characteristic	Muscular Strength			P-value *
	Low	Middle	High	
	(n=1379)	(n=1384)	(n=1384)	
Age (years)	43.4 ± 9.0	42.9 ± 8.8	42.3 ± 9.1	0.005
Follow-up (person-years)	18.9 ± 4.4	19.4 ± 3.8	19.3 ± 3.9	<0.001
Body Mass Index (kg/m ²)	26.1 ± 3.5	24.9 ± 2.7	24.6 ± 2.4	<0.001
Cardiorespiratory fitness (minutes)	19.1 ± 4.7	21.0 ± 4.5	22.6 ± 4.3	<0.001
Blood pressure (mmHg)				
Systolic	115.4 ± 9.5	113.8 ± 9.6	114.6 ± 9.6	<0.001
Diastolic	76.8 ± 6.5	75.8 ± 6.8	75.9 ± 6.8	<0.001
Current Smoker (%)	14.7	13.8	10.8	0.007
Alcohol consumption (≥5 drinks/wk) (%)	48.0	47.5	47.7	0.970
Family history of HTN (%)	4.4	3.7	4.0	0.616
Upper body strength				
Kilograms	61.0 ± 11.5	69.5 ± 12.3	82.8 ± 18.6	<0.001
Kilograms per kg body wt	0.7 ± 0.1	0.9 ± 0.1	1.1 ± 0.2	<0.001
Lower body strength				
Kilograms	122.6 ± 22.2	134.0 ± 21.7	148.2 ± 25.0	<0.001
Kilograms per kg body wt	1.4 ± 0.2	1.7 ± 0.2	1.9 ± 0.2	<0.001
Muscular Strength Index Score	−0.9 ± 0.4	−0.1 ± 0.2	1.0 ± 0.6	<0.001

* Pearson chi-square tests for categorical variables, ANOVA f-tests for continuous variables.

Data shown as Means ± Standard Deviation unless specified otherwise.

HDL-C= high density lipoprotein cholesterol.

Table 2

Baseline characteristics according to HTN status

Characteristic	HTN Status at baseline		P-value *
	Normal (n=1951)	Pre-HTN (n=2196)	
Age (years)	42.3 ± 8.5	43.4 ± 9.4	<0.001
Follow-up (person-years)	19.9 ± 3.2	18.6 ± 4.6	<0.001
Body Mass Index (kg/m ²)	24.8 ± 2.7	25.5 ± 3.2	<0.001
Cardiorespiratory fitness (%)	20.6 ± 4.8	21.2 ± 4.6	<0.001
Blood pressure (mmHg)			
Systolic	108.3 ± 6.7	120.2 ± 8.1	<0.001
Diastolic	71.4 ± 4.8	80.4 ± 5.1	<0.001
Current Smoker (%)	14.0	12.3	0.096
Alcohol consumption (≥5 drinks/wk) (%)	48.9	46.7	0.161
Family history of HTN (%)	2.6	5.4	<0.001
Upper body strength			
Kilograms	70.8 ± 16.7	71.3 ± 17.3	0.006
Kilograms per kg body wt	0.9 ± 0.2	0.9 ± 0.2	0.357
Lower body strength			
Kilograms	134.2 ± 24.6	135.5 ± 25.8	<0.001
Kilograms per kg body wt	1.7 ± 0.3	1.7 ± 0.3	0.093
Muscular Strength Index Score	0.0 ± 0.8	0.0 ± 0.9	0.011

* Pearson chi-square tests for categorical variables, t-tests for continuous variables.

Data shown as Means ± Standard Deviation unless specified otherwise.

HDL-C= high density lipoprotein cholesterol.

Table 3

Incidence rates and hazard ratios for incident hypertension by muscular strength thirds.

	Baseline Muscular Strength Third			P-value [†]	P for linear trend [‡]
	Low	Middle	High		
All men (n=4147)	1379	1384	1384		
No. of hypertension cases	195	161	147		
Rate [‡]	75.0	59.8	55.0	0.009	0.004
Model 1 [‡] HR (95% CI)	1.0	0.78 (0.63–0.96)	0.72 (0.58–0.89)	0.006	0.002
Model 2 [‡] HR (95% CI)	1.0	0.84 (0.68–1.03)	0.75 (0.61–0.93)	0.029	0.009
Model 3 [‡] HR (95% CI)	1.0	0.94 (0.76–1.16)	0.95 (0.76–1.19)	0.824	0.629
Model 4 [‡] HR (95% CI)	1.0	0.98 (0.79–1.22)	0.99 (0.79–1.24)	0.986	0.909
Normotensive men (n=1951)	610	669	672		
No. of hypertension cases	45	57	41		
Rate [‡]	37.4	42.6	30.7	0.281	0.346
Model 1 [‡] HR (95% CI)	1.0	1.09 (0.73–1.61)	0.81 (0.53–1.23)	0.338	0.314
Model 2 [‡] HR (95% CI)	1.0	1.17 (0.79–1.73)	0.84 (0.55–1.29)	0.282	0.434
Model 3 [‡] HR (95% CI)	1.0	1.29 (0.87–1.92)	1.16 (0.75–1.82)	0.446	0.467
Model 4 [‡] HR (95% CI)	1.0	1.41 (0.82–1.61)	1.20 (0.78–1.91)	0.243	0.343
Prehypertensive men (n=2196)	769	715	712		
No. of hypertension cases	150	104	106		
Rate [‡]	107.2	76.8	79.3	0.010	0.012
Model 1 [‡] HR (95% CI)	1.0	0.71 (0.55–0.91)	0.72 (0.56–0.92)	0.007	0.007
Model 2 [‡] HR (95% CI)	1.0	0.73 (0.56–0.93)	0.72 (0.56–0.93)	0.011	0.009
Model 3 [‡] HR (95% CI)	1.0	0.81 (0.63–1.05)	0.88 (0.68–1.05)	0.262	0.303
Model 4 [‡] HR (95% CI)	1.0	0.83 (0.64–1.07)	0.90 (0.69–1.17)	0.352	0.398

[‡] Rate per 10,000 person-years.[†] Adjusted for age, examination year, and survey response pattern.

§ Additionally adjusted for smoking (yes/no), alcohol intake (≤ 5 (drinks/wk)), family history of hypertension (present/absent), and baseline resting systolic blood pressure and diastolic blood pressure (mmHg).

// Additionally adjusted for CRF (minutes).

° Additionally adjusted for BMI (kg/m^2).

¶ P-value for H_0 : low strength=middle strength=high strength.

P-value for linear trend across strength levels.

HR= hazard ratio, CI= confidence interval, BMI= body mass index, CRF= cardiorespiratory fitness