HHS Public Access

Author manuscript

Ann Intern Med. Author manuscript; available in PMC 2016 November 03.

Published in final edited form as:

Ann Intern Med. 2016 May 3; 164(9): 577–584. doi:10.7326/M15-2002.

PHYSICAL FITNESS AMONG SWEDISH MILITARY CONSCRIPTS AND LONG-TERM RISK OF TYPE 2 DIABETES: A COHORT STUDY

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Abstract

Background—Early-life physical fitness rarely has been examined in relation to type 2 diabetes mellitus (T2DM) in adulthood, because of the lengthy follow-up required. Elucidation of modifiable risk factors at young ages may help facilitate earlier and more effective interventions.

Objective—We examined aerobic capacity and muscular strength at age 18 in relation to T2DM risk in adulthood.

Design—National cohort study.

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Statistical analysis: Crump, J. Sundquist.

Obtained funding: J. Sundquist, K. Sundquist.

REPRODUCIBLE RESEARCH STATEMENT

Protocol, statistical code, and data: Not available

Setting—Sweden.

Participants—All 1,534,425 military conscripts during 1969-1997 (97-98% of all 18-year-old males nationwide) without prior T2DM.

Measurements—Aerobic capacity and muscular strength measured in Watts and Newtons per kg of body weight, respectively, were examined in relation to T2DM identified from outpatient and inpatient diagnoses during 1987-2012 (maximum age 62 years).

Results—34,008 men were diagnosed with T2DM in 39.4 million person-years of follow-up. Low aerobic capacity and low muscular strength were independently associated with increased risk of T2DM. Comparing lowest vs. highest tertiles of both aerobic capacity and muscular strength, the absolute difference in cumulative incidence of T2DM was 0.22% at 20 years of follow-up (95% CI, 0.20-0.25), 0.76% at 30 years (0.71-0.81), and 3.97% at 40 years (3.87-4.06). Overall, the combination of low aerobic capacity and low muscular strength was associated with a 3-fold risk of T2DM (adjusted hazard ratio, 3.07; 95% CI, 2.88-3.27; $P<0.001$), with a positive additive interaction ($P<0.001$). These associations were observed even among men with normal BMI.

Limitations—This cohort did not include women or physical fitness measurements at older ages.

Conclusions—In this large cohort of Swedish male military conscripts, low aerobic capacity and low muscular strength at age 18 were associated with increased long-term risk of T2DM, even among those with normal BMI.

Primary funding source—NIH.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) affects more than 300 million people worldwide and has more than doubled in prevalence over the past 3 decades, concurrently with increasing rates of obesity and sedentary lifestyle (1). US economic costs of T2DM and its complications exceed $200 billion annually (2). Although physical inactivity is a well-established risk factor, few studies have examined objective measurements of physical fitness in relation to T2DM. Physical fitness (which includes both aerobic capacity and muscular strength) may be a more informative risk factor, because it can be measured more objectively and is a better indicator of habitual physical activity than self-reported activity (3). Most studies of physical fitness have examined aerobic but not muscular fitness, and have focused on adults but lacked data at younger ages with sufficient follow-up to examine the long-term risk of T2DM. As a result, the relative effects of aerobic capacity and muscular strength, and their effects at younger ages on long-term T2DM risk, are still unknown. Elucidation of these risk factors at young ages may help facilitate earlier and more effective preventive interventions.

We analyzed data from a national cohort of military conscripts to examine aerobic capacity and muscular strength at age 18 years in relation to T2DM risk in adulthood. Aerobic capacity and muscular strength were assessed using standardized tests in ~1.5 million male military conscripts in Sweden who were followed up to a maximum age of 62 years. Our aims were to examine whether low aerobic capacity and low muscular strength at age 18 are associated with long-term risk of T2DM in this cohort.

METHODS

Study Population

We identified 1,547,478 men (age 18 years) who underwent a military conscription examination in Sweden during 1969-1997. This examination was compulsory for all 18-year-old men nationally each year except for 2-3% who either were incarcerated or had severe chronic medical conditions or disabilities documented by a physician. We excluded all 13,053 (0.8%) men who had a prior inpatient or outpatient diagnosis of diabetes. A total of 1,534,425 (99.2% of the original cohort) remained for inclusion in the study.

Physical Fitness Ascertainment

Aerobic capacity and muscular strength measurements were obtained using the Swedish Military Conscription Registry, which contains information from a 2-day standardized physical and psychological examination required for all conscripts starting in 1969. Aerobic capacity was measured as the maximal aerobic workload in Watts, using a standard well-validated electrically-braked stationary bicycle ergometer test, as previously described (4). Maximal aerobic workload is highly correlated with maximal oxygen uptake (VO_2 max; correlation ~0.9) (5), and its measurement using this bicycle ergometer test is highly reproducible, with a test-retest correlation of 0.95 (6). Muscular strength was measured as the weighted sum of maximal knee extension (weighted × 1.3), elbow flexion (weighted × 0.8), and hand grip (weighted × 1.7), each measured in Newtons, using standard well-validated isometric dynamometer tests (7). Each dynamometer test was performed three times and the maximum value recorded for analysis, except when the last value was highest, in which case testing was repeated until strength values stopped increasing. All testing equipment was calibrated daily (7). In the present study, aerobic capacity and muscular strength were standardized per kg of body weight, and were examined alternatively as continuous linear variables, categorical variables in tertiles (aerobic capacity in Watts per kg of body weight: low [<3.58], medium [3.58-4.18], high [≥4.18]; muscular strength in Newtons per kg of body weight: low [<28.23], medium [28.23-32.13], high [≥32.13]), and using cubic spline curves.

T2DM Ascertainment

The study cohort was followed up for T2DM from the time of the military conscription examination through December 31, 2012. T2DM was identified using *International Classification of Diseases* (*ICD*) diagnosis codes in the Swedish Hospital and Outpatient Registries. The Swedish Hospital Registry contains all primary and secondary hospital discharge diagnoses from six populous counties in southern Sweden starting in 1964, and with nationwide coverage starting in 1987; and the Swedish Outpatient Registry contains outpatient diagnoses nationwide starting in 2001. Diagnoses in the Hospital Registry are currently >99% complete and have a reported positive predictive value of 85-95% (8). Because earlier *ICD* versions did not distinguish between type 1 and type 2 diabetes, we ascertained T2DM using *ICD*-9 code 250 (excluding codes 250.X1 and 250.X3) during 1987-1996, and *ICD*-10 code E11 during 1997-2012. A sensitivity analysis was performed which further included all diabetes diagnoses during 1969-1986 using *ICD*-8 code 250 from hospital discharge records (before outpatient data were available). Among men without

T2DM diagnoses during 1987-2012, 542 were diagnosed with diabetes during 1969-1986, of which the majority would be expected to be type 2 (e.g., among men with the same age distribution, 75% of inpatient diabetes diagnoses during 1987-2012 were type 2).

Adjustment Variables

Other variables that may be associated with T2DM were obtained from the Swedish Military Conscription Registry and national census data, which were linked using an anonymous personal identification number. The following were used as adjustment variables: year of the military conscription examination (modeled simultaneously as a continuous and categorical [1969-1979, 1980-1989, 1990-1997] variable); body mass index (BMI = [weight in kg]/[height in m]^2; modeled simultaneously as a continuous and categorical variable using Centers for Disease Control and Prevention [CDC] definitions for children and adolescents aged 2 to 19 years: "overweight or obesity" is defined as ≥85th percentile on the CDC's 2000 sex-specific BMI-for-age growth charts, which corresponds to BMI ≥25.6 for 18-year-old males (9)); family history of diabetes in a parent or sibling (yes or no, identified from diagnoses in the Swedish Hospital Registry during 1964-2012 and the Swedish Outpatient Registry during 2001-2012, not self-reported, thus enabling unbiased ascertainment); highest education level attained during the study period (<12, 12-14, ≥15 years); and neighborhood socioeconomic status at baseline (SES, included because neighborhood SES characteristics have been associated with T2DM (10, 11) and with physical activity and BMI (12); comprised of an index that includes low education level, low income, unemployment, and social welfare receipt, as previously described (13), and categorized as low [<−1 SD from the mean], medium [≥−1 SD and ≤1 SD], or high [>1 SD]). As alternatives to BMI, we also examined height and weight in a separate model, which were modeled simultaneously as continuous and categorical (height: <175, 175-184, ≥185 cm; weight: <60, 60-79, ≥80 kg) variables (9).

Missing data for each variable were imputed using a standard multiple imputation procedure based on the variable's relationship with all other covariates (14). Missing data were relatively infrequent for aerobic capacity (5.7%), muscular strength (5.0%), height (7.2%), weight (7.3%), education level (0.4%), and neighborhood SES (9.1%). As an alternative to multiple imputation, sensitivity analyses were performed after restricting to individuals with complete data for all variables (N=1,361,083; 88.0%).

Statistical Analysis

Absolute time-to-event measures were calculated using the cumulative incidence function for T2DM. Covariate-standardized cumulative incidence curves for T2DM were generated using the method of Simon and Makuch (15). We also used Cox proportional hazards regression to estimate the relative hazard of T2DM for different levels of aerobic capacity and muscular strength. The Cox model time scale was elapsed time since the military conscription examination (which also corresponds to attained age because baseline age was the same [18 years] for all conscripts). Individuals were censored at emigration (n=112,158; 7.3%) or death (n=58,835; 3.8%). The proportional hazards assumption was evaluated by graphical assessment of log-log plots and was met in all models. Interactions between aerobic capacity and muscular strength were examined on either the additive or

multiplicative scale. Additive interactions were assessed using the "relative excess risk due to interaction" (RERI), which is computed for binary variables as: $RERI_{HR} = HR_{11} - HR_{10} - HR_{01} + 1$ (16, 17). Multiplicative interactions were assessed using the ratio of HRs: $HR_{11} / (HR_{10} HR_{01})$. We also examined interactions graphically using cubic spline curves.

Sensitivity analyses were performed that included only men with at least 30 years of follow-up (N=686,964; 44.8%), or that evaluated the effect of unmeasured confounders (e.g., smoking) using external adjustment (18). In the analysis of smoking, we performed 10,000 model simulations assuming two uniform independent distributions for smoking prevalences among exposed and unexposed between 0.2 and 0.4 (19), and a lognormal distribution for the smoking-T2DM relative hazard that implies a mean relative hazard of 1.5 and SD of 0.4 (20). All statistical tests were 2-sided and used an α-level of 0.05. All analyses were conducted using Stata version 13.0 (21).

Role of the Funding Source

This work was supported by the National Heart, Lung, and Blood Institute at the National Institutes of Health (R01 HL116381); the Swedish Research Council; and ALF project grant, Region Skåne/Lund University, Sweden. The funding agencies had no role in the study design, conduct, or reporting. This study was approved by the Regional Ethics Committee of Lund University.

RESULTS

Among the 1,534,425 men in this cohort, 34,008 (2.2%) were subsequently diagnosed with T2DM in 39.4 million person-years of follow-up (mean follow-up, 25.7 years). The median age at the end of follow-up was 46.1 years (mean 45.9, SD 8.9, range 19.0 to 62.0), and at T2DM diagnosis was 46.8 years (mean 44.7, SD 9.9, range 18.0 to 62.0). Table 1 shows aerobic capacity, muscular strength, and other characteristics among 18-year-old men who did or did not subsequently develop T2DM.

Table 2 shows the covariate-standardized cumulative incidence of T2DM at 10, 20, 30, and 40 years of follow-up by aerobic capacity and muscular strength at age 18. Low aerobic capacity was associated with significantly increased cumulative incidence at each of these follow-up times, irrespective of muscular strength level. Low muscular strength was associated with increased cumulative incidence after 40 years of follow-up among men with low or medium (but not high) aerobic capacity. The combination of low aerobic capacity and low muscular strength was associated with highest cumulative incidence, which reached 4.45% at 40 years of follow-up (risk difference relative to high aerobic capacity and high muscular strength, 3.97%; 95% CI, 3.87-4.06; $P<0.001$). Cumulative incidence curves for T2DM by aerobic capacity and muscular strength are shown in Figure 1.

Table 3 summarizes the adjusted relative hazards of T2DM across the entire follow-up period by aerobic capacity and muscular strength at age 18. Low aerobic capacity and low muscular strength were independently associated with higher risk of T2DM, although low aerobic capacity was the stronger risk factor ($P_{heterogeneity}<0.001$). The combination of low aerobic capacity and low muscular strength was associated with highest T2DM risk (HR,

3.07; 95% CI, 2.88-3.27; $P<0.001$). Comparing lowest vs. highest tertiles, aerobic capacity and muscular strength had a positive interaction on the additive ($P_{interaction}<0.001$) but not multiplicative ($P_{interaction}=0.62$) scale. The same additive interaction was found when examined at 30 or 40 years of follow-up ($P_{interaction}<0.001$), but not at earlier times ($P_{interaction}>0.05$).

The univariate effects of aerobic capacity, muscular strength, BMI, and other variables in association with T2DM are shown in Supplemental Table 1. In secondary analyses, we found positive additive (but not multiplicative) interactions between either low aerobic capacity or low muscular strength and high BMI in relation to T2DM ($P<0.001$; Supplemental Tables 2 and 3). Low aerobic capacity and low muscular strength were associated with higher risk of T2DM even among men with normal BMI. In sensitivity analyses that included diabetes diagnoses from 1969-1986 (for which type 1 and type 2 could not be distinguished), that were restricted to men with no missing data, or that included only men with at least 30 years of follow-up, all risk estimates were very similar to the main results (data not shown). External adjustment for smoking yielded risk estimates for association between low aerobic capacity or low muscular strength and T2DM that were 9% lower and remained highly significant ($P<0.001$), suggesting that unmeasured confounding had little influence on our main findings.

DISCUSSION

In this large national cohort study, low aerobic capacity and low muscular strength in 18-year-old men were associated with higher risk of developing T2DM in adulthood, independent of BMI, family history, or socioeconomic factors. A combination of low aerobic capacity and low muscular strength was associated with highest risk, although aerobic capacity had the stronger influence. Furthermore, both of these factors were associated with increased risk of T2DM even among men with normal BMI. Positive additive interactions were found between low aerobic capacity and low muscular strength, suggesting that, if the associations are causal, interventions to improve aerobic capacity would have the greatest public health impact on T2DM among men with low muscular strength.

Most previous studies have examined physical fitness only in adulthood (22-30). The largest of these was a US study of 46,979 middle-aged adults with median follow-up of 5 years, which reported that higher physical fitness based on a treadmill stress test was independently protective against diabetes ($P_{trend}<0.001$) (27). Fewer studies have examined physical fitness early in life, and none examined physical fitness in adolescence in relation to the long-term risk of T2DM. Our findings suggest that low aerobic capacity at age 18 is strongly associated with higher risk of developing T2DM later in life, irrespective of baseline muscular strength or BMI, after follow-up to a maximum age of 62 years.

We also found that low muscular strength was an independent risk factor for T2DM later in life, although was less influential than aerobic capacity. These findings are broadly consistent with previously reported associations between muscular strength among adults and reduced risk of metabolic syndrome (31, 32), between resistance training among adults and reduced risk of T2DM (33, 34), and between resistance training and improved glycemic

control among adults with T2DM (35). The overall evidence to date suggests that high muscular strength or resistance training improves glycemic control and is protective against T2DM, independent of aerobic capacity. However, the combination of high muscular strength and high aerobic capacity is associated with the greatest protective benefit (33, 34).

Obesity is a well-established strong risk factor for T2DM (36-38). Importantly, we found that low aerobic capacity and low muscular strength were long-term risk factors for T2DM even among men with normal BMI. Other cohort studies have reported that low aerobic capacity is associated with T2DM even among non-obese adults, without examining muscular strength (22, 27, 30). Overall, these findings suggest that physical fitness has important health benefits for all, even those who are not overweight or obese.

There are several mechanisms by which aerobic and muscular fitness may enhance glycemic control (39, 40). Aerobic exercise is known to increase mitochrondrial density and oxidative enzyme activity, which promotes fatty acid oxidation and insulin sensitivity (41). Strength training augments type II muscle fiber growth, increasing glucose use capacity (41), and may up-regulate proteins in the insulin-signaling cascade, increasing insulin activity and further enhancing glucose utilization (42). Both aerobic exercise and strength training help reduce adiposity, a known risk factor for T2DM (43).

Strengths of the present study include its large national cohort design with prospective ascertainment of aerobic capacity, muscular strength, BMI, and T2DM. The national cohort design prevented selection bias, and the use of registry data with prospectively measured exposures prevented bias that may result from self-reporting. We examined objective, well-validated measures of aerobic capacity and muscular strength, which are likely better indicators of habitual physical activity than self-reported activity (3). We were able to adjust for other strong risk factors for T2DM, including BMI, family history, and socioeconomic factors, which also were prospectively ascertained and not self-reported.

Limitations include the measurement of physical fitness and BMI at only one age (18 years), and hence we were unable to examine changes in these factors over time. Because this study was based on Swedish military conscripts, the cohort consisted entirely of men. Other studies have reported similar associations between low physical fitness and risk of T2DM among women (22, 29). Outpatient diagnoses in the present study were available only starting in 2001, and hence T2DM prior to this period was underreported. This underreporting is expected to be non-differential with respect to physical fitness and therefore to influence results toward the null hypothesis. In addition, diagnoses prior to 1987 were excluded from the main analyses because they did not distinguish between type 1 and type 2 diabetes. However, sensitivity analyses that included all diabetes diagnoses prior to 1987 (of which most are expected to be type 2) yielded very similar results as our main findings. Last, this was a relatively young cohort in Sweden. Additional studies will be needed in other populations, diverse ethnic groups, and with follow-up to older ages.

In summary, we found that low aerobic capacity and low muscular strength at age 18 were independently associated with higher risk of developing T2DM in adulthood, among men with either normal or high BMI. These findings suggest that interventions to improve

aerobic and muscular fitness early in life could help reduce T2DM risk in adulthood. Additional studies with longitudinal measurements of fitness will be needed to delineate the most important windows of susceptibility and further inform preventive interventions.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

ACKNOWLEDGMENTS

GRANT SUPPORT

This work was supported by the National Heart, Lung, and Blood Institute at the National Institutes of Health (R01 HL116381); the Swedish Research Council; and ALF project grant, Region Skåne/Lund University, Sweden. The funding agencies had no role in the design and conduct of the study; in the collection, analysis, and interpretation of the data; or in the preparation, review, or approval of the manuscript.

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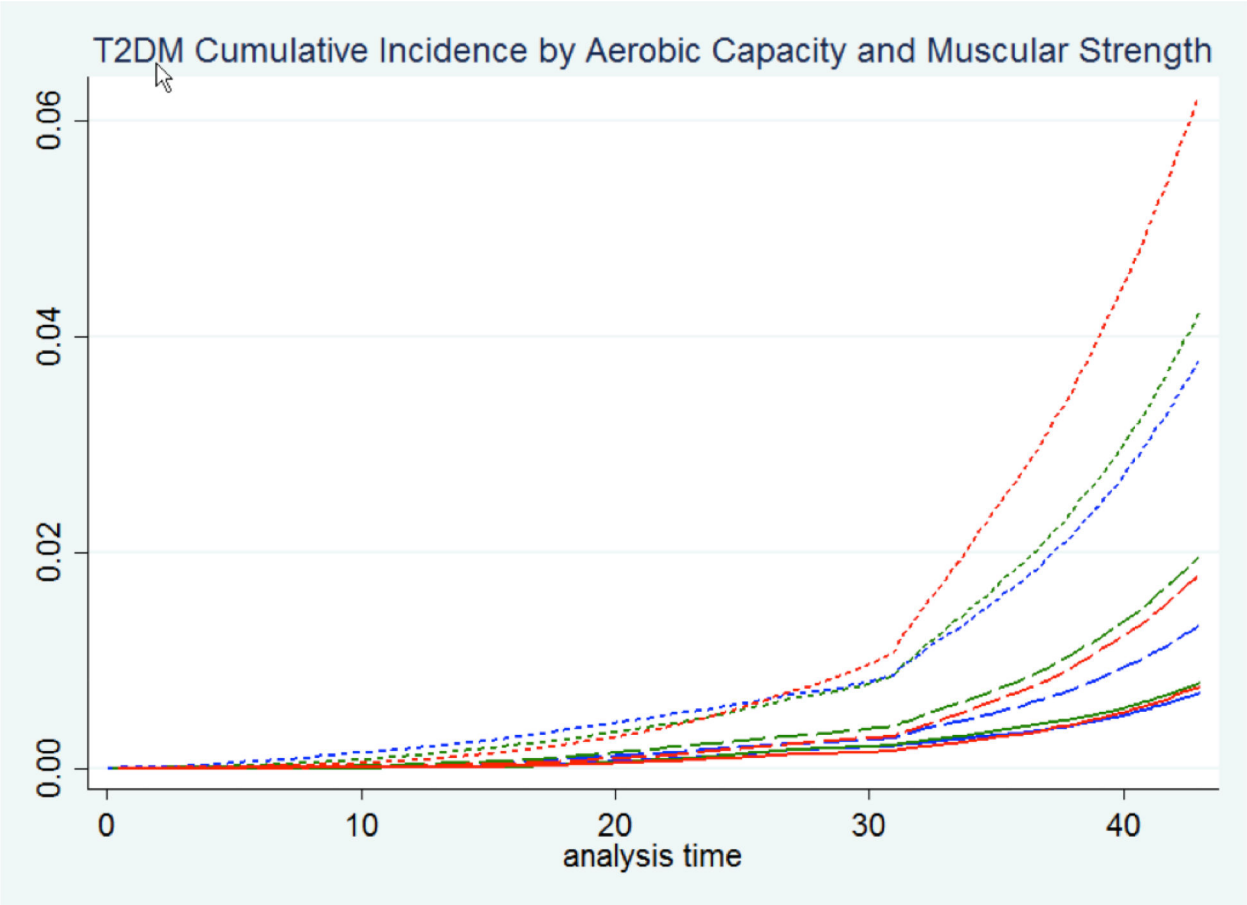

Figure 1.
Cumulative incidence of type 2 diabetes by aerobic capacity and muscular strength in 18-year-old men with maximum follow-up of 44 years (aerobic capacity, tertiles: solid line = high, long dash = medium, short dash = low; muscular strength, tertiles: blue = high, green = medium, red = low).

Table 1

Physical fitness and other characteristics among 18-year-old men who did or did not develop type 2 diabetes.

	Type 2 Diabetes	
	Yes (N=34,008)	**No (N=1,500,417)**
Aerobic capacity per kg body weight (tertiles)		
Low, n (%)	22,878 (67.3)	489,539 (32.6)
Medium, n (%)	7,604 (22.4)	499,672 (33.3)
High, n (%)	3,526 (10.4)	511,206 (34.1)
Mean (SD)	3.3 (0.7)	3.9 (0.7)
Muscular strength per kg body weight (tertiles)		
Low, n (%)	15,887 (46.7)	498,406 (33.2)
Medium, n (%)	10,751 (31.6)	501,151 (33.4)
High, n (%)	7,370 (21.7)	500,860 (33.4)
Mean (SD)	28.4 (5.0)	29.2 (6.5)
Body mass index		
Normal, n (%)	25,918 (76.2)	1,388,856 (92.6)
Overweight or obese, n (%)	8,090 (23.8)	111,561 (7.4)
Mean (SD)	23.3 (4.3)	21.6 (2.8)
Height (cm)		
<175, n (%)	9,552 (28.1)	336,657 (22.4)
175-184, n (%)	18,684 (54.9)	883,191 (58.9)
≥185, n (%)	5,772 (17.0)	280,569 (18.7)
Mean (SD)	177.6 (7.4)	178.0 (7.5)
Weight (kg)		
<60, n (%)	3,694 (10.9)	187,755 (12.5)
60-79, n (%)	20,543 (60.4)	1,115,921 (74.4)
≥80, n (%)	9,771 (28.7)	196,741 (13.1)
Mean (SD)	73.5 (14.3)	68.8 (10.3)
Family history of diabetes		
No, n (%)	18,251 (53.7)	1,164,670 (77.6)

	Type 2 Diabetes	
	Yes (N=34,008)	**No (N=1,500,417)**
Yes, n (%)	15,757 (46.3)	335,747 (22.4)
Education (years)		
<12, n (%)	8,247 (24.2)	225,154 (15.0)
12-14, n (%)	16,177 (47.6)	662,094 (44.1)
≥15, n (%)	9,584 (28.2)	613,169 (40.9)
Neighborhood socioeconomic status		
Low, n (%)	7,864 (23.1)	231,265 (15.4)
Medium, n (%)	21,166 (62.2)	988,613 (65.9)
High, n (%)	4,978 (23.2)	280,539 (18.7)

Table 2

Cumulative incidence of type 2 diabetes by aerobic capacity and muscular strength in 18-year-old men.

Follow-up time	Muscular strength (tertiles)	Aerobic capacity (tertiles)								
		High			Medium			Low		
		Cumulative incidence (%)	Risk difference	*P* value	Cumulative incidence (%)	Ris k difference	*P* value	Cumulative incidence (%)	Ris k difference	*P* value
10 years	High	0.01 (0.00, 0.01)	Reference	--	0.01 (0.01, 0.02)	0.01 (0.00, 0.01)	0.05	0.14 (0.12, 0.17)	0.14 (0.11, 0.16)	<0.001
	Medium	0.00 (0.00, 0.01)	0.00 (−0.01, 0.00)	0.06	0.02 (0.01, 0.03)	0.01 (0.01, 0.02)	<0.001	0.08 (0.07, 0.09)	0.07 (0.06, 0.09)	<0.001
	Low	0.00 (0.00, 0.01)	0.00 (−0.01, 0.00)	0.53	0.01 (0.00, 0.01)	0.00 (0.00, 0.01)	0.46	0.05 (0.04, 0.05)	0.04 (0.03, 0.05)	<0.001
20 years	High	0.06 (0.05, 0.07)	Reference	--	0.11 (0.10, 0.12)	0.05 (0.03, 0.07)	<0.001	0.41 (0.37, 0.45)	0.35 (0.31, 0.39)	<0.001
	Medium	0.05 (0.04, 0.06)	−0.01 (−0.03, 0.00)	0.15	0.14 (0.12, 0.16)	0.08 (0.06, 0.10)	<0.001	0.33 (0.31, 0.36)	0.27 (0.24, 0.30)	<0.001
	Low	0.04 (0.03, 0.06)	−0.02 (−0.03, 0.00)	0.03	0.09 (0.07, 0.11)	0.03 (0.01, 0.05)	0.003	0.28 (0.26, 0.31)	0.22 (0.20, 0.25)	<0.001
30 years	High	0.19 (0.18, 0.22)	Reference	--	0.26 (0.24, 0.28)	0.06 (0.03, 0.09)	<0.001	0.80 (0.75, 0.85)	0.61 (0.55, 0.66)	<0.001
	Medium	0.20 (0.18, 0.22)	0.01 (−0.02, 0.03)	0.70	0.36 (0.33, 0.39)	0.16 (0.13, 0.20)	<0.001	0.78 (0.74, 0.82)	0.58 (0.53, 0.63)	<0.001
	Low	0.15 (0.13, 0.17)	−0.05 (−0.08, −0.02)	0.001	0.28 (0.25, 0.31)	0.08 (0.05, 0.12)	<0.001	0.96 (0.92, 1.00)	0.76 (0.71, 0.81)	<0.001
40 years	High	0.48 (0.45, 0.52)	Reference	--	0.92 (0.88, 0.97)	0.43 (0.37, 0.48)	<0.001	2.69 (2.59, 2.79)	2.21 (2.10, 2.31)	<0.001
	Medium	0.55 (0.52, 0.59)	0.07 (0.02, 0.11)	0.006	1.35 (1.29, 1.41)	0.88 (0.81, 0.95)	<0.001	2.98 (2.89, 3.06)	2.50 (2.41, 2.59)	<0.001
	Low	0.51 (0.48, 0.55)	0.03 (−0.02, 0.08)	0.39	1.22 (1.16, 1.28)	0.74 (0.67, 0.80)	<0.001	4.45 (4.36, 4.54)	3.97 (3.87, 4.06)	<0.001

Table 3

Interactions between aerobic capacity and muscular strength among 18-year-old men in relation to subsequent risk of type 2 diabetes.[a]

	Aerobic capacity (tertiles)						HRs for medium aerobic capacity within strata of muscular strength	HRs for low aerobic capacity within strata of muscular strength
	High		Medium		Low			
	No. cases/total	HR (95% CI)	No. cases/total	HR (95% CI)	No. cases/total	HR (95% CI)		
Muscular strength (tertiles)								
High	1,252/195,299	1.00	2,371/202,217	1.08 (1.01, 1.16); *P*=0.028	3,747/110,714	1.90 (1.77, 2.03); *P*<0.001	1.08 (1.01, 1.16); *P*=0.028	1.90 (1.77, 2.03); *P*<0.001
Medium	1,268/173,644	1.19 (1.10, 1.29); *P*<0.001	2,893/162,087	1.60 (1.49, 1.71); *P*<0.001	6,590/176,171	2.25 (2.11, 2.39); *P*<0.001	1.35 (1.25, 1.44); *P*<0.001	1.89 (1.77, 2.01); *P*<0.001
Low	1,006/145,789	1.58 (1.45, 1.72); *P*<0.001	2,340/142,972	1.92 (1.79, 2.06); *P*<0.001	12,541/225,532	3.07 (2.88, 3.27); *P*<0.001	1.22 (1.13, 1.31); *P*<0.001	1.94 (1.81, 2.08); *P*<0.001
HRs (95% CI) for medium muscular strength within strata of aerobic capacity		1.19 (1.10, 1.29); *P*<0.001		1.48 (1.39, 1.56); *P*<0.001		1.19 (1.14, 1.23); *P*<0.001		
HRs (95% CI) for low muscular strength within strata of aerobic capacity		1.58 (1.45, 1.72); *P*<0.001		1.78 (1.67, 1.88); *P*<0.001		1.62 (1.55, 1.68); *P*<0.001		
Interaction on additive scale, lowest vs. highest tertiles: RERI (95% CI)					0.59 (0.45, 0.73); *P*<0.001			
Interaction on multiplicative scale, lowest vs. highest tertiles: Ratio of HRs (95% CI)					1.02 (0.93, 1.12); *P*=0.62			

HR = hazard ratio, RERI = relative excess risk due to interaction

[a]HRs are adjusted for year of the military conscription exam, body mass index, family history of diabetes, education, and neighborhood socioeconomic status.

Ann Intern Med. Author manuscript; available in PMC 2016 November 03.