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Effects of Running Distance and Performance on Incident Benign Prostatic Hyperplasia

PAUL T. WILLIAMS

Donner Laboratory, Life Sciences Division, Ernest Orlando Lawrence Berkeley National Laboratory, Berkeley, CA

Abstract

Purpose—Benign prostatic hyperplasia (BPH) is generally not considered a preventable condition. Our goal is to assess whether running (a vigorous physical activity) and 10-km race performance (an indicator of cardiorespiratory fitness) reduce BPH risk.

Methods—Prospective cohort study of incident BPH in 28,612 nonsmoking, nonvegetarian, nondiabetic men.

Results—The 1899 men (6.64%) reported physician-diagnosed incident BPH during (mean $\pm$ SD) 7.74 ± 1.84 yr of follow-up. Survival analyses showed significantly lower risk with both longer distance run ($\text{km}\cdot\text{wk}^{-1}$; $P < 0.0001$) and faster 10-km performance ($\text{m}\cdot\text{s}^{-1}$; $P = 0.0004$) independent of age, BMI, and meat, fish, fruit, and alcohol intake. When adjusted for age, the fastest men ($\geq 4.0 \text{ m}\cdot\text{s}^{-1}$) had 32% lower risk than the slowest men ($< 3 \text{ m}\cdot\text{s}^{-1}$; $P = 0.0006$). The decline in incidence extended throughout the performance range, with even the fastest category ($\geq 4.0 \text{ m}\cdot\text{s}^{-1}$) having significantly lower risk than the penultimate fastest category ($3.5\text{--}4.0 \text{ m}\cdot\text{s}^{-1}$; $P = 0.03$). The decline in BPH risk with running distance was independent of performance. BPH incidence was more strongly related to the average of the baseline and the follow-up distance run than to concurrent changes in running distance between baseline and follow-up. Incident BPH was significantly lower in men who ran > 16 than $\leq 16 \text{ km}\cdot\text{wk}^{-1}$ ($P = 0.05$), > 32 than $16\text{--}32 \text{ km}\cdot\text{wk}^{-1}$ ($P = 0.02$), and > 48 than $32\text{--}48 \text{ km}\cdot\text{wk}^{-1}$ ($P = 0.04$).

Conclusions—Greater distances run per week may reduce BPH risk independent of BMI, 10-km performance, and diet. If the relationship is causal, then this health benefit accrues at greater exercise doses and intensities than the minimum guideline levels currently recommended.

Keywords

BPH; LOWER URINARY TRACT SYMPTOMS; PREVENTION; CARDIORESPIRATORY FITNESS

INTRODUCTION

Benign prostatic hyperplasia (BPH) is a common malady among older men that often produces frequent or uncomfortable urination. These urinary symptoms arise from pressure on the urethra due to greater prostate mass and decreased prostatic smooth muscle tone due

Address for correspondence: Paul T. Williams, Ph.D., Donner Laboratory, Life Sciences Division, Ernest Orlando Lawrence Berkeley National Laboratory, Berkeley, CA 94720; ptwilliams@lbl.gov.

Dr. Williams had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

The results of the present study do not constitute endorsement by ACSM.

to reduced sympathetic nervous activity [14]. Bothersome urinary tract symptoms affect over 40% of older men [14]. In 2000, BPH accounted for 105,000 hospitalizations, 117,000 emergency room visits, and 4.4 million office visits [30]. Reported risk factors for BPH include older age, functioning testes, total and central obesity, hypertension, diabetes, low alcohol intake, nonsmoking status, and possibly African American race [9,11–14,18,21,23,25]. Whereas a few studies suggest that physical activity reduces the risk for BPH [10,24] or lower urinary tract symptoms [26,27,33], many report no inverse association [11,18,20,21].

To date, the most compelling evidence relating BPH to physical activity derives from the 30,634 men of the Health Professionals Follow-up Study followed prospectively for 6 yr [24]. Their data showed that the odds for BPH were 24% lower for the highest quintile of physical activity than the lowest quintile, and that men who walked 2 to 3 h·wk⁻¹ had 27% lower risk for BPH than those reporting zero walking hours. They also reported that vigorously intense physical activity was less effective in preventing BPH than low- or moderate-intensity activities. Other studies claim BPH risk increases at higher doses of physical activity [12,18]. These conflicting results present a compelling need to test the relation between BPH and vigorous exercise. Specifically, there seemed to be no *a priori* reason to expect that an inverse relationship between BPH risk and physical activity would not extend throughout higher doses of vigorous exercise, as has been observed for other diseases and their risk factors [34,39]. Moreover, reductions in disease risk due to exercise are often associated with cardiorespiratory fitness [39], which is more effectively increased by vigorous than by moderate exercise [28] and more effectively increased by more-intense than by less intense vigorous exercise [31].

The National Runners' Health Study was specifically designed to assess the dose-response relationship between health and running distance (km·wk⁻¹) and 10-km race performance time (an indicator of cardiorespiratory fitness) in men and women who meet or substantially exceed current minimum physical activity guidelines [34–40]. In addition to providing independent confirmation of a BPH–physical activity relationship, these analyses extend the activity range to higher doses than previously considered. To our knowledge, this is the only article to date to assess the independent effects of a vigorous physical activity and a fitness-related measure on BPH and to relate BPH to changes in a physical activity.

METHODS

The design and the methods of the National Runners' Health Study are described elsewhere [34–40]. Briefly, a two-page questionnaire was distributed nationally to runners identified through a running magazine subscription list and among participants of foot race events. The questionnaire solicited information on demographics, running history, weight history, smoking habits, prior history of heart attack and cancer, and medications for blood pressure, thyroid, cholesterol, and diabetes. Participants were recruited between 1991 and 1994 (primarily 1993), and follow-up questionnaires were obtained between 1999 and 2002. The study protocol was approved by the University of California Berkeley Committee for the Protection of Human Subjects, and all participants signed committee-approved informed consents.

Participants reported whether a physician had told them they had benign prostate enlargement since their baseline questionnaire and reported the year of diagnosis. Diagnoses dated the same year as the baseline survey or earlier were excluded in order that the end points represented only newly diagnosed incidence of disease. Running distances were reported in usual miles run per week. Although other leisure-time physical activities were not recorded for this cohort, data from runners recruited after 1998 (when the question was

added to the survey) show running represents ($\pm$ SD) $91.5 \pm 19.1\%$ of all vigorously intense activity in those men and $73.5 \pm 23.7\%$ of their total leisure-time physical activity. Previously, we reported strong correlations between repeated questionnaires for self-reported running distance ($r = 0.89$) [36], between self-reported and clinically measured height ($r = 0.96$) and weight ($r = 0.96$) [36], and for self-reported running distance versus self-reported BMI and body circumference in cross-sectional analyses [34,40]. Eighty percent of the 54,956 participants of the National Runners' Health Study provided follow-up information or were known deceased. For this report, participant's best 10-km race during the previous 5 yr (reported as finish time in minutes) as speed in meters per second was included as a performance-based measure associated with cardiorespiratory fitness [3,8,17].

Intakes of meat, fish, and fruit were based on the questions "During an average week, how many servings of beef, lamb, or pork do you eat?" "...servings of fish do you eat?" and "...pieces of fruit do you eat?" Alcohol intake was estimated from the corresponding questions for 4-oz. (112 mL) glasses of wine, 12-oz (336 mL) bottles of beer, and mixed drinks and liqueurs. Alcohol was computed as 10.8 g per 4-oz glass of wine, 13.2 g per 12-oz bottle of beer, and 15.1 g per mixed drink. Correlations between these responses and values obtained from 4-d diet records in 110 men were $r = 0.65$ for alcohol, $r = 0.46$ for red meat, $r = 0.38$ for fruit, and $r = 0.19$ for fish intake. These values agree favorably with published correlations between food records and more extensive food frequency questionnaires for red meat ($r = 0.50$), wine ($r = 0.66$), beer ($r = 0.70$), and mixed drinks ($r = 0.72$), somewhat less favorably for fruit intake ($r = 0.50$), and less favorably for fish intake ($r = 0.51$) [19].

Cox proportional hazard analyses (JMP version 5.1, SAS Institute, Cary, NC, USA) were used to estimate the dose-response relationships of incident BPH to distances run and 10-km performance speed. Reported weekly intakes of alcohol, meat, fish, and fruit, along with age and age², were included as covariates. Current smoking, strict vegetarianism, and diabetes were handled by exclusion rather than adjustment because they represented a very small proportion of the sample. Further adjustments for BMI, running distance ($\text{km}\cdot\text{wk}^{-1}$), and 10-km performance ($\text{m}\cdot\text{s}^{-1}$) were included where indicated. In addition, relative risks (i.e., risk ratios of the hazard function) are presented that compare distance and performance intervals to all higher activity and performance levels and to the least active and slowest performing runners. The Cox proportional hazard model was also used to test whether changes in distance run per week were related to the incidence of BPH. Those results are presented adjusted for BMI at baseline and end of follow-up.

RESULTS

At baseline, there were 28,612 men who were nonsmoking, nonvegetarian, and nondiabetic who reported their average weekly running distance, height, body weight, and age and who did not report being diagnosed with BPH before their baseline survey. Their baseline characteristics relative to their reported distance run per week are displayed in Table 1. Reported distance run was associated with significantly younger age, slightly less education, lower BMI and narrower waist circumferences, fewer weekly servings of meat, greater fruit intake, and faster 10-km performance times.

Included among the 28,612 men were 1899 (6.64%) who reported incident BPH during (mean $\pm$ SD) 7.74 ± 1.84 yr of follow-up. The affected men were significantly older than unaffected men (mean $\pm$ SE, 53.6 ± 0.2 vs. 44.0 ± 0.1 yr; $P < 0.0001$). They also ran significantly less per week (34.5 ± 0.5 vs. 38.2 ± 0.1 $\text{km}\cdot\text{wk}^{-1}$; $P < 0.0001$) and ran slower 10-km races (3.71 ± 0.01 vs. 3.96 ± 0.00 $\text{m}\cdot\text{s}^{-1}$; $P < 0.0001$), which could not be ascribed to their difference in age (age-adjusted significance differences of $P = 0.005$ and $P = 0.002$, respectively). The older age of those with BPH accounted for their lower meat consumption

(affected vs. unaffected, 2.65 ± 0.06 vs. 2.85 ± 0.02 servings per week; unadjusted $P = 0.001$ became $P = 0.58$ when age adjusted), higher fish (1.73 ± 0.04 vs. 1.53 ± 0.01 servings per week; $P < 0.0001$ became $P = 0.57$ when age adjusted) and fruit consumption (11.89 ± 0.20 vs. 10.98 ± 0.05 servings per week; $P < 0.0001$ became $P = 0.48$ when adjusted for age), and broader waist circumferences (85.67 ± 0.14 vs. 84.35 ± 0.04 cm; $P < 0.0001$ became $P = 0.55$ when age adjusted). Affected and unaffected men also did not differ in their BMI (23.95 ± 0.06 vs. 23.86 ± 0.02 kg·m⁻²; $P = 0.16$) or weekly alcohol intake (81.32 ± 2.54 vs. 81.02 ± 0.70 mL; $P = 0.91$).

Ten-kilometer performance times were provided on a subset of 24,107 men (84.3%), including 1560 who reported BPH. This fitness subset was younger than men who did not have a 10-km performance time to report (44.48 ± 0.06 vs. 45.63 ± 0.17 yr; $P < 0.0001$), which explains their lower proportion of incident BPH (6.47 ± 0.16 vs. $7.52 \pm 0.39\%$; $P = 0.009$ reduced to $P = 0.76$ when adjusted for age).

Table 2 presents the relative risk from survival analyses for incident BPH versus baseline running distance (km·wk⁻¹) and 10-km performance (m·s⁻¹) both separately and when included simultaneously, and with and without adjustment for BMI. The table shows that both longer running distance and faster 10-km race performance predicted lower BPH risk. The risk reduction attributable to distance was the same for the entire cohort as for the fitness subset (i.e., those reporting 10-km race times). Adjustment for BMI slightly increased the reductions in incidence associated with distance and performance. Restricting the analyses to men 45 yr old and older yielded essentially identical results (not displayed). Figure 1 displays the monotonic risk reductions associated with baseline 10-km performance. Men who ran ≥ 4.5 m·s⁻¹ had 32% lower risk than the slowest performing men ($P = 0.0006$). The dose-response relationship extended across the entire performance range, with men in the fastest performance category having significantly lower risk than men in the penultimate fastest performance category ($P = 0.03$).

There were 25,936 men who provided their running distance on their end of follow-up questionnaire. These data were analyzed in conjunction with their reported baseline level in Table 3. Two models were used differing only in the way the baseline and end of follow-up running distances were parameterized as independent variables. Model 1 included the baseline and the follow-up distances separately as independent variables. In model 1, the significance for the baseline distance represents its effect independent of the end of follow-up distance, and, correspondingly, the significance for the follow-up distance represents its effect independent of baseline distance. These analyses showed that baseline distance was a more significant predictor of BPH than follow-up distance. In model 2, the baseline and the follow-up distances were reparameterized as the average distance run and the change in distance run between baseline and follow-up. The analyses show that the average distance predicted the risk for BPH whereas the change in distance did not. Figure 2 displays the linear decline in risk with average running distance. There was significantly lower risk for men who ran >16 than ≤ 16 km·wk⁻¹ ($P = 0.05$), >32 than $16\text{--}32$ km·wk⁻¹ ($P = 0.02$), and >48 than $32\text{--}48$ km·wk⁻¹ ($P = 0.04$).

DISCUSSION

Dose-response

Our results add to the accumulating evidence that important health benefits accrue at greater exercise doses and greater exercise intensities than currently recommended. Specifically, guidelines from the American College of Sports Medicine and the American Heart Association [16] and the Centers for Disease Control and Prevention [29] emphasize the health benefits of walking 2 miles (3.2 km) briskly on most days of the week (the energy

equivalent of running 8–12 km·wk⁻¹) [1]. This corresponds to the referent activity levels of Figure 2 (<16 km·wk⁻¹) [1]. These guidelines [29], particularly the updated versions [16], acknowledge that additional benefits may accrue for longer, more intense activity. Consistent with this acknowledgement, Figure 2 shows that BPH risk continued to decline with longer running distances through at least 64 km·wk⁻¹. Compared with ≤16 km·wk⁻¹ (guideline levels), running 16–32, 32–48, 48–64, and >64 km·wk⁻¹ reduced BPH risk by 6%, 12%, 24%, and 33%, respectively. In other articles, we have demonstrated that running appears to improve men's health incrementally up to 80 km·wk⁻¹ by lowering BMI [34,40]; through 64 km·wk⁻¹ by raising plasma HDL cholesterol [34], lowering plasma triglycerides and LDL cholesterol concentrations [34], and reducing incident diabetes and hypercholesterolemia [39]; and through 48 km·wk⁻¹ by reducing hypertension [34,39].

The Health Professionals Follow-up Study suggests that physical activity reduces the odds for BPH even if the activity is only of low to moderate intensity (climbing stairs, walking, rowing, or calisthenics) [24]. Our analyses confirm the inverse relationship between physical activity and BPH. This is important because few articles report this benefit compared with other health outcomes such as cardiovascular disease [29]. Small sample size may explain the nonsignificance of the trend between physical activity and BPH in the 9-yr follow-up of the Massachusetts Male Aging study of 1019 men [21] and the lack of a significant total physical activity difference between Chinese men with and without BPH [20].

In addition to providing confirmation, our article extends the findings of the Health Professionals Follow-up Study to higher exercise doses and intensities. The highest quintile of total physical activity they reported was ≥33.8 MET·h per week of activity [24], the energy equivalent of 33 km·wk⁻¹ [1], whereas Figure 2 suggests declining risk through ≥64 km·wk⁻¹. Although the Health Professionals Follow-up Study also demonstrates that running specifically reduces the odds for BPH ($P = 0.02$) [24], their highest category of running was ≥2 h·wk⁻¹. Our Figure 2 shows significant incremental decreases in risk for running over 16 km·wk⁻¹ ($P = 0.05$), 32 km·wk⁻¹ ($P = 0.02$), and 48 km·wk⁻¹ ($P = 0.04$). Whereas the Health Professionals Follow-up Study reported that the decline in BPH risk was less for high intensity (running, jogging, biking, swimming, tennis, racquet ball, or squash) than low- to moderate-intensity physical activity (flights of stairs climbed, walking, rowing, or calisthenics), our data provide ample evidence for dose/dependent, clinically important risk reductions with vigorous exercise.

Mechanisms

Various mechanisms have been proposed to explain how physical activity reduces BPH risk. Androgens are involved in the development and the maintenance of BPH, and men who engage in chronic endurance training have, as a group, significantly lower resting levels of testosterone [15,32]. Whereas this explanation appears apropos to the high-mileage runners reported by us, Platz et al. [24] discounted this explanation for walking and other low- to moderate-intensity physical activities of the Health Professionals Follow-up Study. They attributed their results to reduced sympathetic nervous activity rather than reduced prostate volume because activity was more strongly related to symptomatic BPH than prostatic enlargement.

Our analyses may offer particular clues to the biological process(es) involved: 1) it must have little if any relationship to the leanness of the runners; 2) it must be at least as strongly related to the physical activity as cardiorespiratory fitness; and 3) it must affect BPH chronically rather than acutely. The third clue is derived from the analyses of Table 3, which show that BPH was more strongly related to the baseline than the end of follow-up activity, and more strongly related to average than change in activity during follow-up. These clues distinguish BPH from hypertension, diabetes, and lipoproteins in their relationship to

physical activity. First, in other analyses [34,35,39], we have shown that adjustment for the runners' BMI substantially reduced the decrease in blood pressure, hypertension, diabetes, and triglycerides and the increases in HDL cholesterol associated with running distance (adjustment did not attenuate the exercise–BPH relationship). Second, the risks for hypertension and diabetes appear to be affected more acutely by changes in exercise [37,38]. Third, the risks for hypertension and diabetes show no relationship to running distance when adjusted for 10-km race performance but show a strong relationship to 10-km performance when adjusted for distance [39] (BPH is more strongly related to running distance activity than 10-km performance). Thus, the processes that elevate HDL cholesterol, reduce triglycerides, improve glucose intolerance, and reduce diabetes risk with exercise may be poor candidates for explaining the BPH–exercise relationship. We are of the opinion that if suppression of elevated sympathetic nervous system activity causes the BPH risk reduction due to exercise, then it would be more strongly related to follow-up than baseline running distance, as observed with hypertension.

Cardiorespiratory fitness

The important and influential work by Blair et al. [4,5] shows a variety of health benefits associated with cardiorespiratory fitness, including reduced cardiovascular morbidity and mortality. In formulating public health guidelines for physical activity, there was great utility in interpreting cardiorespiratory fitness as a more objective measure of physical activity than self-reported activity itself [5,29]. However, Table 2 shows that BPH risk has separate, statistically independent associations with cardiorespiratory fitness (as measured by 10-km performance) and vigorous physical activity. Elsewhere, we have demonstrated that declines in the prevalence of antidiabetic, antihypertensive, and LDL-cholesterol-lowering medications with increasing fitness ($\text{m}\cdot\text{s}^{-1}$) were also independent of reported physical activity [39]. Furthermore, we have shown that blood pressure levels were more strongly related to cardiorespiratory fitness than physical activity [35]. These observations suggest the need for a clearer interpretation of fitness than simply as a surrogate for physical activity. For example, cardiorespiratory fitness is known to reflect a substantial hereditary component [6,7], and genetic factors associated with higher fitness levels might also reduce BPH risk. They also suggest that cardiorespiratory fitness *per se* warrants consideration as an important clinically indicated risk factor independent of activity.

Caveats

These analyses are based on self-reported distance run per week, which, although comparable to other self-reported physical activity measures with respect to reproducibility over duplicate questionnaires, is still subject to error and potential bias that could affect the associations described in this article. The prospective cohort design used in our study is less subject to the biases associated with the case–control studies, which rely heavily on the adequacy of matching cases with controls. However, we do not wish to overstate our findings as proof of cause and effect. Although the prospective design ensures that physical activity levels preceded the clinical diagnosis of BPH, the relationship is probably better viewed as cross-sectional between the activity and probability of BPH. There is also the inherent limitation of self-reported physician diagnosis, which, although used by other epidemiological studies of BPH [2,22], is necessarily less accurate than clinically validated conditions. Our survey question on incident BPH has not been verified in relation to medical records or independent physician diagnoses and lacks specificity with respect to symptomatic and asymptomatic disease. In addition, the question does not distinguish prostate enlargement from obstructive and irritative lower urinary tract symptoms. However, the Health Professionals Follow-up Study reported the same inverse association for BPH defined by surgery or by symptoms [24]. This study also found self-reported surgery for prostate enlargement to be fully verified by medical records, and they included self-reported

enlarged prostate detected by digital rectal examination as an end point. Nevertheless, our results are likely to exclude cases where these symptoms were not attributed to BPH. The ability to distinguish benign and malign disease may depend on the experience of the physician. However, it is important to emphasize that misdiagnosis and underreporting of BPH cannot account for the significant association reported here between BPH and running distance and 10-km performance unless the rates of misdiagnosis and underreporting were directly related to the reported distance run or 10-km performance time, which we believe to be unlikely.

We also caution that our analyses of incident BPH in relation to concurrent changes in running distance are not prospective and recall bias may spuriously associate follow-up distance to disease status. Moreover, our analysis of exercise change between baseline and follow-up does not take into account substitution of other activities for running.

Chronologic age is the strongest risk factor for BPH [14,21]. Ten-kilometer performance times decline sharply with age [35], and therefore, the validity of these analyses depend strongly on the adequacy of our age adjustment. Age and age² were included as covariates in all analyses, which appeared sufficient to eliminate the highly significant differences in meat, fish, and fruit consumption between affected and unaffected men and thus presumably would also have eliminated an association between fitness and BPH due solely to age. When the analyses of Tables 2 and 3 were repeated on men 45 yr old and older, the results were essentially indistinguishable from the entire sample of men. (Analyses not displayed.)

Urological complications of BPH could lead to reduced mobility and recreational activities, yet our findings are generally consistent with those for low- to moderately-intense activity, which presumably would be less affected [24]. Dal Maso et al. [10] argue against this explanation because the relationships are consistent in younger and older men. We do not believe that the declining incidence of BPH with increasing running distance is likely due to greater stoicism in more athletic men, for this would presumably be reflected in the relationship of running distance to other maladies including degenerative arthritis ($P = 0.23$), kidney stones ($P = 0.35$), or gastric ulcers ($P = 0.83$), which was not observed (unpublished observations).

CONCLUSIONS

Although BPH has not generally been considered a preventable condition, our findings suggest that exercise may lower BPH risk. They also provide further support for encouraging physical activity at doses and intensities that substantially exceed minimum guideline levels and for recognizing cardiorespiratory fitness as a clinically important risk factor independent of physical activity.

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FIGURE 1.

Relative risk from survival analyses of self-reported physician-diagnosed BPH by 10-km foot race performance ($\text{m}\cdot\text{s}^{-1}$) adjusted for age, age², and weekly intakes of meat, fish, fruit, and alcohol. Additional adjustments for weekly running distance and BMI are indicated by the legend. Significant differences relative to the slowest (presumably least fit) men are coded *P < 0.05, †P < 0.01, ‡P < 0.001, and §P < 0.0001. Numerical probabilities above the bars are the significance of the difference in risk between the range of speeds represented by the bar and all the faster speeds (e.g., men who exceeded $3.5 \text{ m}\cdot\text{s}^{-1}$ had a significantly lower risk for BPH than men who ran between 3.0 and $3.5 \text{ m}\cdot\text{s}^{-1}$, P = 0.01).

FIGURE 2.

Relative risk from survival analyses of self-reported physician-diagnosed BPH versus average kilometer per week running distance between baseline and follow-up adjusted for age, age², and weekly intakes of meat, fish, fruit, and alcohol. Additional adjustment for BMI where indicated by the legend. Significant differences relative to the least active men are coded *P < 0.05, †P < 0.01, ‡P < 0.001, and §P < 0.0001. Numerical probabilities above the bars are the significance of the difference between its risk and all greater distances (e.g., men who ran >48 km·wk⁻¹ had significantly lower risk for BPH than men who ran between 32 and 48 km·wk⁻¹, P = 0.04).

TABLE 1

Baseline characteristics of the sample (mean $\pm$ SD).

	Reported Running Distance (km·wk ⁻¹)					Significance ANOVA (<i>P</i>)
	0–16	16–32	32–48	48–64	>64	
Sample size	3264	7830	8412	4897	4209	<0.0001
Cases	220	630	572	285	192	<0.0001
Age (yr)	45.37 $\pm$ 10.86	45.69 $\pm$ 10.15	45.03 $\pm$ 9.89	44.11 $\pm$ 10.11	42.13 $\pm$ 10.45	<0.0001
Education (yr)	16.52 $\pm$ 2.47	16.66 $\pm$ 2.38	16.54 $\pm$ 2.41	16.44 $\pm$ 2.50	16.35 $\pm$ 2.51	<0.0001
BMI (kg·m ⁻²)	25.16 $\pm$ 3.17	24.45 $\pm$ 2.57	23.86 $\pm$ 2.34	23.24 $\pm$ 2.15	22.55 $\pm$ 2.12	<0.0001
Waist circumference (cm)	87.55 $\pm$ 7.54	85.96 $\pm$ 6.05	84.50 $\pm$ 5.67	82.82 $\pm$ 5.23	80.97 $\pm$ 5.14	<0.0001
Meat servings per week	3.30 $\pm$ 2.82	3.08 $\pm$ 2.72	2.81 $\pm$ 2.61	2.61 $\pm$ 2.62	2.35 $\pm$ 2.56	<0.0001
Fish servings per week	1.53 $\pm$ 1.42	1.55 $\pm$ 1.42	1.55 $\pm$ 1.43	1.55 $\pm$ 1.46	1.49 $\pm$ 1.49	0.20
Fruit pieces per week	9.30 $\pm$ 7.96	10.25 $\pm$ 8.14	11.01 $\pm$ 8.23	11.88 $\pm$ 9.42	12.90 $\pm$ 10.48	<0.0001
Alcohol (mL·wk ⁻¹)	73.99 $\pm$ 106.24	84.53 $\pm$ 115.89	84.54 $\pm$ 116.27	79.38 $\pm$ 112.30	74.93 $\pm$ 111.93	<0.0001
10-km performance (m·s ⁻¹)	3.69 $\pm$ 0.55	3.71 $\pm$ 0.49	3.90 $\pm$ 0.47	4.10 $\pm$ 0.46	4.38 $\pm$ 0.50	<0.0001

TABLE 2

Relative risks (95% confidence interval) from survival analyses of incident BPH versus 10-km race performance and running distance during (mean $\pm$ SD) 7.74 ± 1.84 yr of follow-up.

	Model	Relative Risk	
		10-km race performance (m·s ⁻¹)	Running distance (km·wk ⁻¹)
All			
BMI unadjusted	Distance only		0.996 ^{***} (0.994, 0.998)
BMI adjusted	Distance only		0.995 ^{****} (0.993, 0.998)
Fitness subset			
BMI unadjusted	Performance only	0.840 ^{***} (0.755, 0.934)	
BMI unadjusted	Distance only		0.995 ^{***} (0.993, 0.998)
BMI unadjusted	Performance and distance together	0.892 (0.794, 1.002)	0.997 ^{**} (0.994, 0.999)
BMI adjusted	Performance only	0.812 ^{***} (0.725, 0.911)	
BMI adjusted	Distance only		0.995 ^{****} (0.992, 0.998)
BMI adjusted	Performance and distance together	0.862 [*] (0.763, 0.973)	0.996 ^{**} (0.993, 0.999)

The fitness subset is limited to the 24,107 men of the 28,612 total who had a 10-km performance time to report. All relative risks adjusted for age, age², and weekly intake of meat, fish, fruit, and alcohol. Additional adjustment for physical activity, cardiorespiratory fitness, and BMI were indicated. Relative risks significantly different from one are coded

* $P \leq 0.05$,

** $P \leq 0.01$,

*** $P \leq 0.001$, and

**** $P \leq 0.0001$.

TABLE 3

Relative risks (95% confidence interval) from survival analyses of incident BPH versus baseline and follow-up weekly running distance in 25,936 men during 7.74 ± 1.84 yr of follow-up.

	Model 1		Model 2	
	Baseline km·wk ⁻¹	End of Follow-up km·wk ⁻¹	Average km·wk ⁻¹	km·wk ⁻¹
BMI unadjusted	0.997* (0.994, 1.000)	0.998 (0.995, 1.001)	0.994**** (0.992, 0.997)	1.000 (0.998, 1.003)
BMI adjusted	0.996** (0.993, 0.999)	0.998 (0.995, 1.001)	0.994**** (0.991, 0.997)	1.001 (0.998, 1.003)

All risks adjusted for age, age², and weekly intakes of meat, fish, fruit, and alcohol. Additional adjustment for BMI where indicated. Relative risks significantly different from one are coded

* $P \leq 0.05$,

** $P \leq 0.01$,

*** $P \leq 0.001$, and

**** $P \leq 0.0001$.