Discussion

Our prospective data show that the duration of the walk to work was associated with a decreased risk for incident hypertension, even after adjustment for age, body mass index, alcohol consumption, leisure-time physical activity (regular physical exercise at least once weekly or less than once weekly), smoking status (current smoker, past smoker, or nonsmoker), systolic and diastolic blood pressure at baseline, and fasting plasma glucose level. Multivariate analysis also showed that regular physical exercise at least once weekly was inversely related to the risk for incident hypertension.

Previous studies have shown an inverse association between physical activity and the incidence of hypertension. Vigorous exercise was inversely associated with the risk for hypertension in a cohort of Harvard University alumni followed for 16 to 50 years and in a cohort of University of Pennsylvania alumni followed for 15 years, but no significant relation between walking and the incidence of hypertension was seen in either study (10, 11). In those studies, the analysis was adjusted only for age and body mass index and questions on walking were not limited to walking to work. In our study, the duration of the walk to work was associated with a decreased risk for hypertension. In Japan, an employee often stays at the same job until retirement, and there are few opportunities for transfer in the company from which our study participants were recruited. Thus, the duration of a man's to walk to work may be a consistent and reliable indicator of mild physical activity status throughout his adult life.

We did not identify the reason why the duration of the walk to work reduced the risk for hypertension. Controlled studies have showed that even mild exercise, especially walking, reduces blood pressure in hypertensive (21, 22) and normotensive (23) persons. In a population-based study of elderly persons, light-intensity physical activity, including walking, significantly decreased systolic and diastolic blood pressures (24). The intensity of exercise does not seem to be related to the magnitude of the decrease in blood pressure (25). In our study, walking seemed to have a positive effect in reducing the risk for hypertension. The explanatory mechanisms are probably multifactorial.

We considered the possibility that participants with a longer walk to work might have had more active lifestyles. However, only 33.3% of participants who belonged to the group with the longest walk to work engaged in vigorous activity at least once a week. Therefore, there did not appear to be any association between the duration of the walk to work and a more active lifestyle. When we examined the relation between the duration of the walk to work and the risk for hypertension, we adjusted the data for leisure-time physical activity in addition to other cofounders. Therefore, the relation between the duration of the walk to work and the risk for hypertension was independent of leisure-time physical activity.

Our study has some potential limitations. First, all participants were registered employees of the same company. Thus, our results may not be representative of the general population; however, these relations are thought to apply to many men who work outside the home. The relative homogeneity of the cohort may actually enhance the study's internal validity. Because of the relatively uniform education background and socioeconomic status of the men in our cohort, these variables were unlikely to represent confounding factors. Second, all our study participants were men. It remains to be determined whether our results also apply to women. Finally, we could not include several confounding variables, such as diet and family history. It is well known that family history and consumption of salt are associated with hypertension (26, 27). A recent study showed that a diet rich in fruits, vegetables, and low-fat dairy foods that includes reduced saturated and total fats significantly lowers blood pressure (28). Therefore, the history of food intake and fam-

Table 4. Number Needed To Walk To Avoid One Case of Hypertension during 10 Years of Follow-up*

Walk to Work	All Participants	Cases of Hypertension	Absolute Risk Reduction (95% CI)	Number Needed To Walk (95% CI)
	n	*n (%)*		
Model 1				
0–10 minutes	2240	207 (9.2)		
11–20 minutes	1766	146 (8.3)	0.009 (0.0088–0.0092)†	111.1 (108.7–113.6)‡
Model 2				
0–10 minutes	2240	207 (9.2)		
≥21 minutes	404	22 (5.4)	0.038 (0.0377–0.0383)§	26.3 (26.1–26.5)‡

* The number needed to walk is the number of men needed to walk to work in order to prevent one incident case of hypertension.
† Absolute risk reduction = case rates of hypertension (0 to 10 minutes of walking) − case rates of hypertension (11 to 20 minutes of walking).
‡ Number needed to walk = 1/absolute risk reduction.
§ Absolute risk reduction = case rates of hypertension (0 to 10 minutes of walking) − case rates of hypertension (≥21 minutes of walking).

ily history of hypertension should be included in future studies.

In conclusion, our results provide evidence that the duration of the walk to work has an independent effect on the risk for hypertension. Even persons who drive to work or use public transportation may benefit from parking or leaving their transportation more than a 20-minute walk from the office. We believe that physicians should recommend walking to work as an adjunct to proper weight control, reduction of alcohol consumption, and leisure-time physical activity.

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References

1. **Leon AS.** Physical activity levels and coronary heart disease. Analysis of epidemiologic and supporting studies. Med Clin North Am. 1985;69:3-20.
2. **Leon AS, Connett J, Jacobs DR Jr, Rauramaa R.** Leisure-time physical activity levels and risk of coronary heart disease and death. The Multiple Risk Factor Intervention Trial. JAMA. 1987;258:2388-95.
3. **Paffenbarger RS Jr, Wing AL, Hyde RT.** Physical activity as an index of heart attack risk in college alumni. Am J Epidemiol. 1978;108:161-75.
4. **Paffenbarger RS Jr, Wing AL, Hyde RT.** Physical activity as an index of heart attack risk in college alumni. 1978. Am J Epidemiol. 1995;142:889-903.
5. **Slattery ML, Jacobs DR Jr, Nichaman MZ.** Leisure time physical activity and coronary heart disease death. The US Railroad Study. Circulation. 1989;79: 304-11.
6. **Blair SN, Kohl HW 3d, Paffenbarger RS Jr, Clark DG, Cooper KH, Gibbons LW.** Physical fitness and all-cause mortality. A prospective study of healthy men and women. JAMA. 1989;262:2395-401.
7. **Arroll B, Beaglehole R.** Does physical activity lower blood pressure: a critical review of the clinical trials. J Clin Epidemiol. 1992;45:439-47.
8. The fifth report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure (JNC V). Arch Intern Med. 1993;153: 154-83.
9. 1993 guidelines for the management of mild hypertension. Memorandum from a World Health Organization/International Society of Hypertension meeting. Guidelines Subcommittee of the WHO/ISH Mild Hypertension Liaison Committee. Hypertension. 1993;22:392-403.
10. **Paffenbarger RS Jr, Wing AL, Hyde RT, Jung DL.** Physical activity and incidence of hypertension in college alumni. Am J Epidemiol. 1983;117:245-57.
11. **Paffenbarger RS Jr, Jung DL, Leung RW, Hyde RT.** Physical activity and hypertension: an epidemiological view. Ann Med. 1991;23:319-27.
12. **Siconolfi SF, Lasater TM, Snow RC, Carleton RA.** Self-reported physical activity compared with maximal oxygen uptake. Am J Epidemiol. 1985;122: 101-5.
13. **LaPorte RE, Black-Sandler R, Cauley JA, Link M, Bayles C, Marks B.** The assessment of physical activity in older women: analysis of the interrelationship and reliability of activity monitoring, activity surveys, and caloric intake. J Gerontol. 1983;38:394-7.
14. **Washburn RA, Adams LL, Haile GT.** Physical activity assessment for epidemiologic research: the utility of two simplified approaches. Prev Med. 1987; 16:636-46.
15. **Washburn RA, Goldfield SR, Smith KW, McKinlay JB.** The validity of self-reported exercise-induced sweating as a measure of physical activity. Am J Epidemiol. 1990;132:107-13.
16. Arterial hypertension. Report of a WHO expert committee. World Health Organ Tech Rep Ser. 1978;(628):7-56.
17. **Miettinen O.** Estimability and estimation in case-referent studies. Am J Epidemiol. 1976;103:226-35.
18. **Cook RJ, Sackett DL.** The number needed to treat: a clinical useful measure of treatment effect. BMJ. 1995;310:452-4.
19. **Altman DG.** Confidence intervals for the number needed to treat. BMJ. 1998;317:1309-12.
20. **McQuay HJ, Moore RA.** Using numerical results from systematic reviews in clinical practice. Ann Intern Med. 1997;126:712-20.
21. **Seals DR, Reiling MJ.** Effect of regular exercise on 24-hour arterial pressure in older hypertensive humans. Hypertension. 1991;18:583-92.
22. **Hagberg JM, Montain SJ, Martin WH 3d, Ehsani AA.** Effect of exercise training in 60- to 69-year-old persons with essential hypertension. Am J Cardiol. 1989;64:348-53.
23. **Kingwell BA, Jennings GL.** Effect of walking and other exercise programs upon blood pressure in normal subjects. Med J Aust. 1993;158:234-8.
24. **Reaven PD, Barrett-Connor E, Edelstein S.** Relation between leisure-time physical activity and blood pressure in older women. Circulation. 1991;83: 559-65.
25. **Fagard R, Bielen E, Hespel P.** Physical exercise in hypertension. In: Laragh JH, Brenner BM, eds. Hypertension: Pathophysiology, Diagnosis, and Management. 2d ed. New York: Raven Pr; 1990:1985-98.
26. **Williams RR, Hunt SC, Hasstedt SJ, Hopkins PN, Wu LL, Berry TD, et al.** Are there interactions and relations between genetic and environmental factors predisposing to high blood pressure? Hypertension. 1991;18(3 Suppl): I29-37.
27. **Stamler J, Rose G, Stamler R, Elliott P, Dyer A, Marmot M, et al.** INTERSALT study findings. Public health and medical care implications. Hypertension. 1989;14:570-7.
28. **Appel LJ, Moore TJ, Obarzanek E, Vollmer WM, Svetkey LP, Sacks FM, et al.** A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. N Engl J Med. 1997;336:1117-24.

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対象の内訳		ヒト	動物	地　域	国内	研究の種類	縦断研究
	対象	一般健常者	空白		(　　)		コホート研究
	性別	男性	(　　)		(　　)		(　　)
	年齢	35-60歳			(　　)		前向き研究
	対象数	5000～10000			(　　)		(　　)
調査の方法	質問紙	(　　)					
アウトカム	予　防	高血圧症予防	な し	な し	な し		(　　)
	維持・改善	な し	な し	な し	な し	(　　)	(　　)

図　表

Table 2. Relative Risk for Hypertension According to Duration of the Walk to Work

Variable	Person-Years of Follow-up	Cases of Hypertension, *n*	Multivariate Relative Risk (95% CI)*	Further Multivariate Relative Risk (95% CI)†
Walk to work‡				
0–10 minutes	30 796	337	1.00 (reference)	1.00 (reference)
11–20 minutes	23 266	242	0.91 (0.77–1.08)	0.88 (0.75–1.04)
≥21 minutes	5722	47	0.70 (0.59–0.95)	0.71 (0.52–0.97)
Walk to work as a continuous variable (per 10 minutes)			0.88 (0.78–0.98)	0.88 (0.79–0.98)

* Adjusted for age, body mass index, alcohol consumption, leisure-time physical activity (regular physical exercise at least once weekly or less than once weekly), smoking status (current smoker, past smoker, or nonsmoker), and fasting plasma glucose level.
† Adjusted for age, body mass index, alcohol consumption, leisure-time physical activity (regular physical exercise at least once weekly or less than once weekly), smoking status (current smoker, past smoker, or nonsmoker), fasting plasma glucose level, systolic blood pressure, and diastolic blood pressure.
‡ *P* for trend = 0.02 for multivariate relative risk and further multivariate relative risk.

図表掲載箇所	P23, Table2
概　要 (800字まで)	The Osaka Health Survey(日本)において、6017名の男性(35－60歳)を対象に、職場までの歩行時間や余暇時間における身体活動と、高血圧症発症との関係について検討をした研究である。仕事への歩行については、「あなたはオフィスまで歩いてくるのにどのくらいの時間かかりますか？」を0-10分、11-20分、21分以上に分類している。余暇時間での運動は、「あなたはジョギングやサイクリング、水泳、テニス、汗をかくような十分な長さ(30分以上続く)の活動を定期的に行っていますか？もしそうであれば、週に何回行っていますか？それはどのような運動ですか？」と尋ねている。これをもとに、週1回より少ないもの、週1回、週2回以上にも分類した。職場までの歩行時間が0－10分の者に比較して、11－20分、21分以上の者では、高血圧発症リスクが、0.88(0.75-1.04)、0.71(0.52-0.97)で有意な低下を示した。また週当たりの運動が週1回未満の者と比較して、1回、2回以上の者では、リスクが0.65(0.47-0.90)、0.72(0.59-0.88)と有意な低下を示した。
結　論 (200字まで)	余暇時間における運動や通勤での歩行時間は、高血圧発症と関連していることが示された。
エキスパートによるコメント (200字まで)	高血圧は、日本のもっとも多い罹患率を示す疾患の一つであり、これらが通勤での歩行時間を多く確保することで予防できる可能性が示されたことは、非常に意義深い。忙しい年代の人にとっても、比較的ハードル低く行える通勤での歩行が広く推奨され、疾病予防につながることが期待される。

担当者:村上晴香

Body Mass Index, Physical Activity, and Bladder Cancer in a Large Prospective Study

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Abstract

Increased body size and lack of physical activity are associated with increased risk of several cancers, but the relations of body mass index (BMI) and physical activity to bladder cancer are poorly understood. We investigated the associations between BMI, physical activity, and bladder cancer in the NIH-AARP Diet and Health Study, a prospective cohort of 471,760 U.S. men and women, followed from 1995 to 2003. During 3,404,642 person-years of follow-up, we documented 1,719 incident cases of bladder cancer. Compared with normal weight, obesity was associated with an up to 28% increased risk for bladder cancer. The multivariate relative risks of bladder cancer for BMI values of 18.5 to 24.9 (reference), 25.0 to 29.9, 30.0 to 34.9, and ≥35 kg/m^2 were 1.0, 1.15, 1.22, and 1.28 (95% confidence interval, 1.02-1.61; P_{trend} = 0.028). The association between BMI and bladder cancer was consistent among subgroups defined by gender, education, smoking status, and other potential effect modifiers. In contrast, physical activity showed no statistically significant relation with bladder cancer. After multivariate adjustment, including BMI, the relative risks of bladder cancer for increasing frequency of physical activity [0 (reference), <1, 1-2, 3-4, and ≥5 times a week] were 1.0, 0.85, 0.89, 0.91, and 0.87 (95% confidence interval, 0.74-1.02; P_{trend} = 0.358), respectively. In conclusion, these findings provide support for a modest adverse effect of adiposity on risk for bladder cancer. In contrast, our results do not suggest a relation between physical activity and bladder cancer. (Cancer Epidemiol Biomarkers Prev 2008;17(5):1214–21)

Introduction

Bladder cancer is the fourth most common malignancy in men and the ninth most common in women in the United States (1). In the United States in 2007, an estimated 67,000 new bladder cancer cases will occur and 13,750 people will die from this disease (1). Established risk factors for bladder cancer include tobacco use, occupational exposure to specific carcinogens such as aromatic amines, schistosomiasis, drinking tap water with arsenic, certain drugs such as phenacetin-containing analgesics, and familial history of bladder cancer (2).

Nearly two thirds of U.S. adults are currently overweight [body mass index (BMI) between 25.0 and 29.9 kg/m^2] or obese (BMI ≥ 30.0 kg/m^2; ref. 3) and more than half are insufficiently physically active (4). Substantial epidemiologic evidence suggests that adiposity is related to increased risk and physical activity to decreased risk of cancer (5), but bladder cancer has not been consistently linked to either body size or physical activity. In epidemiologic studies examining adiposity and bladder cancer (6-20), no statistically significant association has been seen in most (7-9, 11, 13-16, 18-20), but the majority of those studies has been limited by small numbers of cases, especially cases with a BMI ≥ 30.0 kg/m^2. Although three prospective studies (6, 10, 12) and one large case-control study (17) found a positive relation of adiposity to risk of bladder cancer, only one (12) of those studies explored this association in detail. Likewise, few studies have investigated physical activity in relation to bladder cancer (12, 15, 21-28). An apparent protective effect of physical activity on risk for bladder cancer is limited to results from one study (27).

Given the high prevalence of adiposity and physical inactivity in the United States (3, 4), we conducted a detailed, prospective investigation in the NIH-AARP Diet and Health Study. With over 1,700 bladder cancer cases, this is the largest study to date to examine BMI and physical activity in relation to this important malignancy.

Materials and Methods

Study Population. In 1995 to 1996, 566,402 members of AARP ages 50 to 71 years and residing in one of six U.S. states (California, Florida, Louisiana, New Jersey, North Carolina, and Pennsylvania) or two metropolitan areas (Atlanta, GA, and Detroit, MI) satisfactorily completed and returned a mailed questionnaire on medical history, diet, and physical activity to initiate the NIH-AARP Diet and Health Study (29). The study was approved by the

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Table 1. Baseline characteristics according to BMI and physical activity

Characteristics	BMI (kg/m^2)				Physical activity (times a week)*				
	18.5-24.9	25.0-29.9	30.0-34.9	≥35.0	0	<1	1-2	3-4	≥5
Participants (n)	162,353	204,189	75,350	29,886	83,558	64,730	102,945	128,003	92,524
Age (y)	62.1	62.1	61.6	60.8	62.0	61.1	61.5	62.2	62.4
Gender (%)									
Men	51.9	71.2	64.6	46.0	52.2	58.9	61.9	63.4	67.2
Women	49.1	29.8	36.4	54.0	48.8	41.1	38.1	36.6	32.8
Race									
White	92.4	91.9	90.9	88.7	89.5	91.8	92.5	91.8	92.6
Non-White	7.6	8.1	9.1	11.3	10.5	8.2	7.5	8.2	7.4
Smoking status (%)									
Current smoker	17.0	12.5	10.8	9.2	20.4	16.9	14.1	10.0	9.3
Former smoker	43.8	53.3	54.9	52.9	45.3	48.3	49.2	52.8	53.9
Never smoker	39.2	34.2	34.3	37.9	34.4	34.8	36.7	37.2	36.8
BMI (kg/m^2)	22.8	27.2	31.9	39.5	28.7	27.9	27.3	26.6	26.1
Physical activity (times a week)	2.7	2.5	2.0	1.5	0	0.5	1.5	3.5	5.5
College education (%)	44.3	40.4	34.4	29.3	28.3	37.6	41.2	44.8	44.6
Married or living as married (%)	66.1	75.2	70.6	58.5	62.4	68.6	71.1	72.8	74.1
Red meat intake (servings/1,000 kcal/d)	29.6	35.8	39.4	41.0	37.9	37.3	36.2	32.5	30.8
Fruit and vegetable intakes (servings/1,000 kcal/d)	3.7	3.4	3.4	3.4	3.1	3.2	3.4	3.7	3.9
Nonalcoholic beverage intake (mL/d)	1,865	2,020	2,068	2,023	2,004	1,969	1,972	1,937	2,012
Alcohol intake (servings/wk)	6.7	7.5	6.6	4.5	6.8	6.9	6.8	6.6	7.4
Menopausal hormone therapy (%)†									
Current users	50.9	54.2	37.1	29.3	38.1	43.8	45.4	48.9	47.2
Former users	8.5	9.4	9.8	8.9	8.7	9.5	9.0	9.3	8.6
Never users	40.5	46.4	53.1	61.7	53.2	46.6	45.6	41.8	44.2
Ever used oral contraceptive use (%)†	42.2	39.5	36.8	34.3	37.1	39.9	40.4	41.5	39.7
Parity (no. live-born children)†	2.1	2.2	2.3	2.3	2.2	2.2	2.2	2.2	2.2
NSAID use (%)	49.1	51.3	54.4	57.2	49.6	52.5	52.1	52.4	49.6

NOTE: All values (except age) were directly standardized to the age distribution of the cohort.
*Physical activity was defined as activity that lasted ≥20 min and caused either increases in breathing or heart rate or working up a sweat.
† Among women only.

Special Studies Institutional Review Board of the U.S. National Cancer Institute.

At baseline, we excluded individuals with diagnosed cancer other than nonmelanoma skin cancer (n = 52,561), participants with emphysema (n = 13,764), those with missing data on body weight or height (n = 12,118), those who were underweight (defined as BMI < 18.5 kg/m^2; n = 4,825), and subjects with missing information on physical activity (n = 5,106) or smoking (n = 6,268). The remaining analytical cohort comprised 287,941 men and 183,819 women.

Identification of Incident Bladder Cancer Cases. We identified incident cases of bladder cancer by probabilistic linkage to the state cancer registries serving our cohort. Beyond the eight original states of our cohort, our cancer registry ascertainment area was recently expanded by three additional states (Texas, Arizona, and Nevada) to capture cancer cases occurring among participants who moved to those states during follow-up. The North American Association of Central Cancer Registries certifies all 11 cancer registries (30). We conducted a validation study comparing registry findings with self-reports and medical records and found that ~90% of all cancer cases in our cohort were validly identified using linkage to cancer registries (31). Vital status was ascertained by linkage of the cohort to the Social Security Administration Death Master file. Additional cases of fatal bladder cancer were identified through linkage to the National Death Index Plus, which provided verification of death and information on cause of death. For matching purposes, we have virtually complete data on first and last name, address history, gender, and date of birth. Social Security number is available for 85% of the NIH-AARP cohort.

Cancer sites were identified by anatomic site and histologic code of the *International Classification of Disease for Oncology, Second and Third Editions* (32). The primary endpoint for the present analysis was total bladder cancer (*International Classification of Disease for Oncology, Third Edition* code C67.0-C67.9), which included cancers with the following morphology: transitional cell carcinoma (8050, 8120-8122, 8130), squamous cell carcinoma (8051-8076), adenocarcinoma (8140-8145, 8190-8231, 8260-8263, 8310, 8480-8490, 8560, 8570), and not otherwise specified carcinomas (8010-8034). A total of 87.4% were transitional cell, 1.3% were squamous cell, 1.4% were adenocarcinoma, and 9.9% were other or not specified bladder cancers.

Assessment of BMI and Physical Activity. Information on weight and height was collected using the baseline questionnaire and was used to calculate BMI and to divide participants into four BMI categories (18.5-24.9, 25.0-29.9, 30.0-34.9, and ≥35.0 kg/m^2) hat incorporated the definitions of normal weight (18.5-24.9 kg/m^2), overweight (25.0-29.9 kg/m^2), and obesity (≥30.0 kg/m^2), respectively, proposed by the WHO (33).

In correspondence with the American College of Sports Medicine physical activity guidelines that recommend at least 20 min of continuous vigorous exercise three times a week for improving cardiorespiratory fitness (34), we asked participants to report the average frequency (never, rarely, 1-3 times a month, 1-2 times a week, 3-4 times a week, and ≥5 times a week) during the past year that they engaged in activities of any type that lasted ≥20 min and caused either increases in breathing or heart rate or working up a sweat. We collapsed the bottom two categories to ensure sufficient numbers of cases in the reference category. A questionnaire similar to the one used in this cohort showed good interrater reliability (percentage agreement = 0.76; κ = 0.53) and reasonable validity (percentage agreement = 0.71; κ = 0.40) as assessed by a computer science and applications activity monitor (35). Additional evidence of the validity of our physical activity instrument is shown by the capability of our assessment of physical activity to predict lower risk of mortality from coronary heart disease (36).

In a supplementary questionnaire mailed in 1996 to 1997, we requested information on sedentary behavior by asking about the average number of hours a day currently spent watching television or videos (0, <1, 1-2, 3-4, 5-6, 7-8, and ≥9 h). We collapsed the bottom three categories to provide adequate statistical power. We also requested information on weight and height at age 18 and physical activity at age 15 to 18 years. The supplementary questionnaire was mailed 6 months after baseline and was returned by 60% of the baseline questionnaire respondents. Thus, in analyses involving sedentary behavior, we began follow-up in 1996 and excluded participants who had a cancer diagnosis before return of the supplementary questionnaire.

Statistical Analysis. Each participant accrued follow-up time beginning at the scan date of the baseline questionnaire and ending at the date of diagnosis of bladder cancer, move out of the registry ascertainment area, death, or the end of follow-up in December 31, 2003, whichever came first. Cox proportional hazards regression (37) with age as the underlying time metric was used to estimate the relations of BMI and physical activity to bladder cancer. We examined potential violation of the proportional hazards assumption and found no discrepancies from the assumption of proportional hazards.

The relation of BMI to bladder cancer was estimated in three models. One model was adjusted for age and gender. A second model was additionally adjusted for race/ethnicity; education; a combination of smoking status, time since quitting for former smokers, and smoking intensity for former and current smokers; family history of any cancer, marital status; intakes of red meat; the combination of fruit and vegetables; total beverages, except alcohol; alcohol; menopausal hormone therapy; use of oral contraceptives; and parity (the latter three variables for women only). A third model was additionally adjusted for physical activity. The association between physical activity and bladder cancer was estimated in three similar models, with the exception that physical activity was replaced by BMI in the third model.

In a subset of study participants, we collected information on use of nonsteroidal anti-inflammatory drugs (NSAID). We used those data to assess whether the relations of BMI or physical activity to bladder cancer were confounded by NSAID use. Tests of linear trend across categories were conducted by modeling the mean values of exposures as a single continuous variable in the multivariate model, the coefficient for which was evaluated using a Wald test.

To examine whether the associations of BMI or physical activity to bladder cancer risk were modified by other potential risk factors for bladder cancer, such as gender, age at baseline, race/ethnicity, education, smoking status, intakes of fruits and vegetables, red meat, beverages, and alcohol, and NSAID use, we conducted tests for multiplicative interaction using likelihood ratio tests. We also evaluated the relations of BMI and physical activity to bladder cancer within strata of potential effect modifying variables. All relative risks (RR) are presented with 95% confidence intervals (95% CI), and reported P values are based on two-sided tests. All analyses were conducted using SAS release 9.1 (SAS Institute).

Results

During 3,404,642 person-years of follow-up, we documented 1,719 newly incident cases of bladder cancer, of which 1,470 were diagnosed in men and 249 in women. Participants with high BMI were less physically active, were more likely to be NSAID users, had a lower education level, and consumed less fruit and vegetables than those with normal BMI. In contrast, subjects with high physical activity were leaner, were more educated, and consumed more fruit and vegetables than less active subjects. At baseline, participants with high BMI or those

Table 2. RR of bladder cancer according to BMI

Variable	BMI (kg/m^2)				P_{trend}
	18.5-24.9	25.0-29.9	30.0-34.9	≥35.0	
Person-years	1,174,879	1,474,882	541,963	212,917	
No. cases	479	845	301	94	
Age- and sex-adjusted RR (95% CI)	1.0	1.18 (1.05-1.32)	1.29 (1.11-1.49)	1.34 (1.08-1.68)	0.002
Multivariate RR without physical activity (95% CI)*	1.0	1.16 (1.03-1.29)	1.23 (1.06-1.43)	1.30 (1.04-1.63)	0.016
Multivariate RR with physical activity (95% CI) †	1.0	1.15 (1.03-1.29)	1.22 (1.05-1.42)	1.28 (1.02-1.61)	0.028

*The multivariate model used age as the underlying time metric and included the following covariates: gender (women, men), a combination of smoking status (never, former, current), time since quitting for former smokers (≥10, 5-9, 1-4, <1 y) and smoking intensity for former and current smokers (1-10, 11-20, 21-30, 31-40, 41-60, ≥61 cigarettes per day), race/ethnicity (White, Black, Hispanic, other race/ethnicity), education (less than high school, high school, vocational school or some college, college graduate, postgraduate), marital status (married or living as married, other), family history of cancer (yes, no), intakes of red meat (quintiles), fruit and vegetables combined (quintiles), nonalcoholic beverages (quintiles), and alcohol (0, <1, 1-3, >3 servings/d), menopausal hormone therapy (current, past, never), oral contraceptive use (ever, never), and parity (0, 1-2, ≥3).
† Adjustment for physical activity included the following categories: 0, <1, 1-2, 3-4, ≥5 times a week.

Table 3. Multivariate relative risk of bladder cancer to according BMI in participants defined by selected variables

Variable	No. cases	BMI (kg/m²)				P_{trend}	$P_{interaction}$
		18.5-24.9	25.0-29.9	30.0-34.9	≥35.0		
Gender							
Men	1,470	1.0	1.21 (1.07-1.37)	1.21 (1.03-1.43)	1.25 (0.96-1.63)	0.079	0.061
Women	249	1.0	0.84 (0.62-1.15)	1.38 (0.96-1.96)	1.37 (0.87-2.18)	0.146	
Age at baseline (y)							
<65	798	1.0	1.14 (0.96-1.36)	1.27 (1.02-1.57)	1.26 (0.92-1.72)	0.026	0.919
≥65	921	1.0	1.16 (0.99-1.35)	1.17 (0.95-1.45)	1.32 (0.94-1.84)	0.376	
Race/ethnicity							
White	1,629	1.0	1.16 (1.03-1.31)	1.22 (1.05-1.43)	1.29 (1.02-1.64)	0.024	0.849
Non-White	90	1.0	0.99 (0.61-1.64)	1.19 (0.63-2.24)	0.93 (0.24-2.50)	0.942	
Education							
Some college or less	1,097	1.0	1.13 (0.98-1.31)	1.25 (1.04-1.49)	1.19 (0.89-1.57)	0.137	0.662
College graduate or postgraduate	622	1.0	1.19 (0.99-1.43)	1.15 (0.89-1.49)	1.47 (0.99-1.19)	0.104	
Smoking status							
Current smoker	389	1.0	1.13 (0.89-1.41)	1.17 (0.88-1.61)	1.07 (0.62-1.87)	0.502	0.862
Former smoker	1,047	1.0	1.20 (1.03-1.40)	1.24 (1.02-1.51)	1.32 (0.99-1.76)	0.179	
Never smoker	283	1.0	1.05 (0.79-1.38)	1.29 (0.89-1.86)	1.42 (0.82-2.46)	0.028	
Physical activity*							
Inactive	914	1.0	1.09 (0.92-1.28)	1.06 (0.86-1.29)	1.33 (1.01-1.74)	0.352	0.087
Active	805	1.0	1.22 (1.03-1.43)	1.44 (1.16-1.79)	0.98 (0.61-1.57)	0.020	
Fruit and vegetable intakes †							
Low	998	1.0	1.07 (0.92-1.25)	1.17 (0.96-1.42)	1.41 (1.07-1.87)	0.102	0.107
High	721	1.0	1.26 (1.06-1.50)	1.29 (1.02-1.64)	1.05 (0.70-1.55)	0.171	
Red meat intake ‡							
Low	750	1.0	1.21 (1.02-1.43)	1.34 (1.06-1.69)	1.19 (0.79-1.79)	0.023	0.606
High	969	1.0	1.11 (0.95-1.30)	1.15 (0.94-1.39)	1.30 (0.98-1.72)	0.289	
Nonalcoholic beverage intake§							
Low	751	1.0	1.11 (0.93-1.31)	1.08 (0.85-1.36)	0.95 (0.65-1.39)	0.474	0.179
High	968	1.0	1.21 (1.03-1.41)	1.36 (1.12-1.66)	1.56 (1.17-2.07)	0.017	
Alcohol use							
No	360	1.0	1.15 (0.89-1.49)	1.21 (0.88-1.66)	1.32 (0.85-2.40)	0.175	0.989
Yes	1,359	1.0	1.16 (1.02-1.32)	1.23 (1.04-1.46)	1.27 (0.97-1.66)	0.071	
NSAID use∥							
No	480	1.0	1.23 (0.99-1.52)	1.22 (0.91-1.64)	1.20 (0.74-1.96)	0.183	0.458
Yes	406	1.0	1.02 (0.80-1.29)	1.24 (0.92-1.66)	0.80 (0.47-1.37)	0.989	

NOTE: The multivariate models were adjusted for covariates listed in Table 2 footnote. In each case, the stratification variable was excluded from the model. Within each stratum, the category of subjects with a BMI of 18.5 to 24.9 served as the reference group.
* Inactive was defined as engaging in <20 min of continuous vigorous exercise three times a week. Active was defined as engaging in at least 20 min of continuous vigorous exercise three times a week.
† The strata of low and high fruit and vegetable intakes were defined based on the cut point representing the median value of 3.2 servings/1,000 kcal/d.
‡ The strata of low and high red meat intakes were defined based on the cut point representing the median value of 31.4 g/1,000 kcal/d.
§ The strata of low and high nonalcoholic beverage intakes were defined based on the cut point representing the median value of 1,782 mL/d.
∥ The analysis that was stratified by NSAID use was conducted using data from a subcohort of study participants for whom we had collected information regarding NSAID use.

with high physical activity levels were less likely to currently smoke but were more likely to have formerly smoked than their lean or less active counterparts (Table 1).

Participants who were overweight or obese had a greater risk of bladder cancer than normal weight subjects. After adjustment for age and gender, individuals with BMI levels of 18.5 to 24.9 (reference), 25.0 to 29.9, 30.0 to 34.9, and ≥ 35 kg/m² had RRs of 1.0, 1.18, 1.29, and 1.34 (95% CI, 1.08-1.68; P_{trend} = 0.002; Table 2). After additional adjustment for multiple variables, the inverse association was slightly attenuated (RR comparing extreme BMI categories, 1.30; 95% CI, 1.04-1.63; P_{trend} = 0.016). Further adjustment for physical activity had only minor effect (RR comparing extreme BMI categories, 1.28; 95% CI, 1.02-1.61; P_{trend} = 0.028). When we limited the analyses to transitional cell carcinomas (*n* = 1,503), statistical power was slightly reduced, but results were essentially unaltered (multivariate RR comparing extreme BMI categories, 1.23; 95% CI, 0.96-1.58; P_{trend} = 0.044).

In a secondary analysis, we repeated our main analysis after excluding all cases of bladder cancer that occurred during the first 2 years of follow-up (*n* = 423 cases excluded). Results were somewhat attenuated, showing a multivariate RR for the highest versus lowest category of BMI of 1.19 (95% CI, 0.91-1.56; P_{trend} = 0.165).

We examined BMI at age 18 and physical activity at age 15 to 18 in relation to bladder cancer risk. The multivariate RRs for the highest versus lowest categories of BMI at age 18 and physical activity at age 15 to 18 years were 1.76 (95% CI, 1.13-2.76) and 0.92 (95% CI, 0.77-1.09), respectively.

To investigate whether the association between BMI and bladder cancer varied across strata defined by gender, age, race/ethnicity, education, smoking, physical activity, intakes of fruit and vegetables, beverages, and alcohol, and NSAID use, stratified models were fit according to levels of those variables (Table 3). A positive association between BMI and bladder cancer was observed in virtually all subgroups. The relation of BMI to bladder cancer appeared to be strong among never smokers, modest among former smokers, and weak among current smokers, but tests of interaction indicated no differences across strata.

In age- and gender-adjusted analyses, participants who reported engaging in increasing levels of physical activity [0 (reference), <1, 1-2, 3-4, and ≥5 more times a week] had RRs of 1.0, 0.80, 0.79, 0.75, and 0.71 (95% CI, 0.61-0.82; P_{trend} < 0.001; Table 4). After adjustment for multiple potential confounders, except BMI, the inverse association was substantially attenuated, but the point estimate for the highest physical activity level remained statistically significant, with a RR comparing the highest with the lowest level of physical activity of 0.85 (95% CI, 0.73-0.99; P_{trend} = 0.188). After further adjustment for BMI, the relation between physical activity and bladder cancer was no longer statistically significant (RR, 0.87; 95% CI, 0.74-1.02; P_{trend} = 0.358). When we limited the analyses to transitional cell cancers (n = 1,503), results were essentially unchanged (multivariate RR comparing extreme physical activity categories, 0.85; 95% CI, 0.72-1.01; P_{trend} = 0.131).

Statistically nonsignificant inverse associations between physical activity and bladder cancer were noted for virtually all subgroup analyses defined by gender, age, race/ethnicity, education, smoking, BMI, intakes of fruit and vegetables, beverages, and alcohol, and NSAID use. Formal tests of interaction were not statistically significant (Table 5). The relations of BMI or physical activity with bladder cancer risk were not confounded by NSAID use (data not shown).

We investigated the association between sedentary behavior and bladder cancer. In an analysis that was adjusted for multiple variables, including physical activity, the RRs of bladder cancer for watching television or videos for <3 (reference), 3 to 4, 5 to 6, 7 to 8, and ≥9 h/d were 1.0, 1.01, 0.94, 1.05, and 0.99 (95% CI, 0.66-1.51). Additional control for BMI yielded risk estimates of 1.0, 1.00, 0.93, 1.03, and 0.98 (95% CI, 0.65-1.49).

Discussion

In this prospective study of nearly 500,000 men and women followed for up to 8 years, we found a modest but graded positive association between BMI and risk of bladder cancer. Compared with normal weight, overweight was associated with 15% increase in risk, and obesity was related to an up to 28% increased risk. The positive association between BMI and bladder cancer was independent of other known risk factors for bladder cancer, including age, race, and smoking, suggesting that avoidance of adiposity may play an important role in the prevention of bladder cancer.

In addition, initial analyses suggested that physical activity was associated with reduced risk of bladder cancer. However, part of the effect of physical activity may be related to its influence on weight control because the inverse association between physical activity and bladder cancer did not persist after adjustment for BMI. This suggests that the apparent protective influence of physical activity on bladder cancer operates through a mechanism involving reduced body mass. Findings for BMI and physical activity from earlier in life were consistent with those based on current BMI and physical activity.

Our finding of a modest positive relation between BMI and bladder cancer is consistent with results from 8 (6-12, 16) of a total of 11 (6-16) prospective studies on this topic. Three record linkage–based cohort studies (6-8) compared high with low BMI levels and reported RRs of bladder cancer of 1.13 (95% CI, 1.06-1.20), 1.2 (95% CI, 1.0-1.5), and 1.2 (95% CI, 1.0-1.6), respectively. Those studies (6-8) did not control for smoking; failure to adjust for smoking may have resulted in an overestimation of the positive association between BMI and bladder cancer in those studies because most adult ever smokers are former smokers and BMI is positively related both to former smoking and to bladder cancer risk. In contrast, failure to adjust for current smoking may have resulted in an underestimation of the relation. One cohort study (16) noted a bladder cancer risk of 1.28 (95% CI, 0.73-2.25) for obese versus normal weight women, whereas a cohort study of men (9) found a RR of 2.3 (95% CI, 0.9-5.7) for high versus low body weight. A cohort study (11) that used mortality from bladder cancer as an endpoint compared obese with normal weight subjects and published RRs of 1.14 (95% CI, 0.88-1.46) for men and 1.34 (95% CI, 0.91-1.95) for women. In another cohort study of bladder cancer mortality (10), a statistically significant positive association with adiposity emerged only after excluding the first 20 years of follow-up (RR comparing overweight with normal weight men,

Table 4. RR of bladder cancer according to physical activity

Variable	Physical activity (times a week)*					P_{trend}
	0	<1	1-2	3-4	≥5	
Person-years	591,296	467,197	745,4094	929,663	671,077	
No. cases	334	217	363	469	336	
Age- and sex-adjusted RR (95% CI)	1.0	0.80 (0.68-0.95)	0.79 (0.68-0.91)	0.75 (0.65-0.86)	0.71 (0.61-0.82)	<0.001
Multivariate RR without BMI (95% CI) †	1.0	0.85 (0.72-1.01)	0.88 (0.75-1.02)	0.89 (0.77-1.03)	0.85 (0.73-0.99)	0.188
Multivariate RR with BMI (95% CI) ‡	1.0	0.85 (0.72-1.02)	0.89 (0.76-1.03)	0.91 (0.78-1.05)	0.87 (0.74-1.02)	0.358

* Physical activity was defined as activities that lasted ≥20 min and caused either increases in breathing or heart rate or working up a sweat.
† The multivariate model used age as the underlying time metric and included the following covariates: gender (women, men), a combination of smoking status (never, former, current), time since quitting for former smokers (≥10, 5-9, 1-4, <1 y) and smoking intensity for former and current smokers (1-10, 11-20, 21-30, 31-40, 41-60, ≥61 cigarettes per day), race/ethnicity (White, Black, Hispanic, other race/ethnicity), education (less than high school, high school, vocational school or some college, college graduate, postgraduate), marital status (married or living as married, other), family history of cancer (yes, no), intakes of red meat (quintiles), fruit and vegetables combined (quintiles), nonalcoholic beverages (quintiles) and alcohol (0, <1, 1-3, >3 servings/d), menopausal hormone therapy (current, past, never), oral contraceptive use (ever, never), and parity (0, 1-2, ≥3).
‡ Adjustment for BMI included the following categories: 18.5-24.9, 25.0-29.9, 30.0-34.9, ≥35.0 kg/m^2.

Table 5. Multivariate RR of bladder cancer according to physical activity in participants defined by selected variables

Variable	No. cases	Physical activity (times a week)					P_{trend}	$P_{interaction}$
		0	<1	1-2	3-4	≥5		
Gender								
Men	1,470	1.0	0.84 (0.69-1.02)	0.86 (0.73-1.02)	0.86 (0.73-1.01)	0.87 (0.73-1.03)	0.345	0.317
Women	249	1.0	0.88 (0.58-1.34)	0.98 (0.68-1.42)	1.19 (0.84-1.69)	0.78 (0.49-1.23)	0.879	
Age at baseline (y)								
<65	798	1.0	0.78 (0.62-0.99)	0.83 (0.67-1.03)	0.81 (0.66-1.01)	0.89 (0.71-1.12)	0.685	0.652
≥65	921	1.0	0.93 (0.73-1.28)	0.94 (0.67-1.16)	0.98 (0.81-1.19)	0.86 (0.69-1.07)	0.343	
Race/ethnicity								
White	1,629	1.0	0.86 (0.72-1.02)	0.88 (0.76-1.03)	0.89 (0.77-1.04)	0.87 (0.74-1.03)	0.336	0.892
Non-White	90	1.0	0.75 (0.35-1.60)	0.89 (0.47-1.72)	1.12 (0.62-2.01)	0.82 (0.41-1.66)	0.952	
Education								
Some college or less	1,097	1.0	0.80 (0.65-0.99)	0.92 (0.76-1.10)	0.98 (0.83-1.17)	0.93 (0.76-1.12)	0.796	0.131
College graduate or postgraduate	622	1.0	0.93 (0.69-1.24)	0.81 (0.62-1.06)	0.77 (0.59-0.99)	0.78 (0.59-1.02)	0.064	
Smoking status								
Current smoker	389	1.0	0.93 (0.68-1.27)	0.93 (0.69-1.24)	0.91 (0.67-1.23)	1.14 (0.83-1.58)	0.509	0.489
Former smoker	1,047	1.0	0.83 (0.66-1.04)	0.87 (0.72-1.06)	0.86 (0.72-1.04)	0.78 (0.64-0.96)	0.064	
Never smoker	283	1.0	0.80 (0.50-1.29)	0.84 (0.56-1.27)	1.01 (0.69-1.48)	0.97 (0.65-1.45)	0.509	
BMI (kg/m^2)								
<25.0	479	1.0	0.80(0.57-1.03)	0. 83 (0.62-1.11)	0.79 (0.60-1.06)	0.79 (0.59-1.06)	0.224	0.928
25.0-29.9	845	1.0	0.85 (0.66-1.09)	0.83 (0.66-1.03)	0.87 (0.70-1.07)	0.85 (0.68-1.07)	0.429	
≥30.0	395	1.0	0.89 (0.65-1.25)	1.04 (0.78-1.39)	1.11 (0.83-1.48)	1.02 (0.73-1.44)	0.498	
Fruit and vegetable intakes*								
Low	998	1.0	0.88 (0.72-1.09)	0.93 (0.77-1.12)	0.95 (0.79-1.14)	0.88 (0.72-1.09)	0.512	0.963
High	721	1.0	0.80 (0.59-1.08)	0.82 (0.63-1.05)	0.84 (0.66-1.06)	0.84 (0.65-1.07)	0.521	
Red meat intake†								
Low	750	1.0	0.83 (0.63-1.09)	0.82 (0.64-1.04)	0.87 (0.69-1.08)	0.87 (0.67-1.09)	0.642	0.937
High	969	1.0	0.87 (0.69-1.08)	0.93 (0.77-1.13)	0.93 (0.77-1.13)	0.87 (0.69-1.07)	0.396	
Nonalcoholic beverage intake‡								
Low	751	1.0	0.75 (0.57-0.98)	0.85 (0.67-1.07)	0.90 (0.73-1.12)	0.84 (0.66-1.06)	0.658	0.717
High	968	1.0	0.94 (0.75-1.17)	0.91 (0.65-1.12)	0.91 (0.75-1.11)	0.91 (0.74-1.12)	0.432	
Alcohol use								
No	360	1.0	0.86 (0.58-1.27)	1.10 (0.79-1.53)	1.04 (0.76-1.42)	1.18 (0.86-1.63)	0.219	0.221
Yes	1,359	1.0	0.84 (0.69-1.02)	0.83 (0.69-0.98)	0.86 (0.73-1.02)	0.79 (0.66-0.95)	0.087	
NSAID use§								
No	480	1.0	0.87 (0.62-1.22)	0.96 (0.72-1.28)	0.84 (0.63-1.12)	0.93 (0.69-1.26)	0.652	0.234
Yes	406	1.0	0.77 (0.54-1.11)	0.77 (0.56-1.06)	0.93 (0.69-1.25)	0.70 (0.49-0.98)	0.297	

NOTE: The multivariate models were adjusted for covariates listed in Table 2 footnote. In each case, the stratification variable was excluded from the model. Within each stratum, the category of subjects with a BMI of 18.5 to 24.9 served as the reference group.
*The strata of low and high fruit and vegetable intakes were defined based on the cut point representing the median value of 3.2 servings/1,000 kcal/d.
† The strata of low and high red meat intakes were defined based on the cut point representing the median value of 31.4 g/1,000 kcal/d.
‡ The strata of low and high nonalcoholic beverage intakes were defined based on the cut point representing the median value of 1,782 mL/d.
§The analysis that was stratified by NSAID use was conducted using data from a subcohort of study participants for whom we had collected information regarding NSAID use.

1.68; 95% CI, 1.06-2.65). A third cohort study on bladder cancer mortality found no relation with excess weight (13).

In contrast, two cohort studies (14, 15) documented a statistically nonsignificant inverse association between BMI and bladder cancer, with RRs comparing high with low BMI of 0.74 (95% CI, 0.45-1.22) and 0.63 (95% CI, 0.33-1.19), respectively. As noted by the authors of one of those studies (15), one plausible reason for the inverse relation of BMI to bladder cancer is residual confounding by smoking that may have persisted even after control for smoking intensity and duration.

In a large cohort study (12), the initial modest association between BMI and bladder cancer (RR comparing high versus low BMI, 1.16; 95% CI, 0.89-1.52) was strengthened and became statistically significant (RR, 1.33; 95% CI, 1.01-1.76) after excluding 423 cases diagnosed in the initial years of follow-up. Other than chance, we have no explanation for the attenuation of the BMI and bladder cancer relation after excluding cases diagnosed in the initial years of follow-up in our study.

As opposed to the majority of cohort studies that showed a positive albeit not always statistically significant association between BMI and bladder cancer (6-12, 16), the four available case-control investigations of BMI and bladder cancer (17-20) produced largely divergent findings. Only one case-control study (17) suggested a statistically significant increased RR of bladder cancer comparing high with low BMI of 1.27 (95% CI, 1.01-1.58). One case-control study (18) found a statistically nonsignificant inverse association between BMI and bladder cancer (odds ratio, 0.61; 95% CI, 0.33-1.14), and two additional case-control studies (19, 20) each reported a null association but did not present actual risk estimates.

Our results showed a lack of an independent association between physical activity and bladder cancer, which is compatible with most (12, 15, 21-26) but not all (27, 28) previous studies in this area. One occupational cohort study (27) reported an increased risk of bladder cancer for sedentary occupations with standardized incidence ratios ranging from 1.10 (95% CI, 1.02-1.18) to 1.33 (95% CI, 1.07-1.61). Those positive associations with sedentary behavior may have been overestimated as a result of confounding by BMI. We found no relation between our measure of sedentary behavior and bladder cancer in the AARP cohort.

In contrast, one cohort study (28) found an increased risk of bladder cancer for high versus low levels of recreational activity (RR, 2.06; 95% CI, 1.08-3.95), although the test for trend did not reach statistical significance (P_{trend} = 0.09). That study (28) evaluated physical activity in relation to a large number of individual cancer sites; multiple comparisons and a small number of bladder cancer cases (n = 92) could have yielded a positive finding for bladder cancer by chance.

Important strengths of our study include its prospective design, a large number of cases, a high follow-up rate, and reasonably detailed information on potential bladder cancer risk factors. In particular, we were able to adjust for cigarette smoking habits, including smoking status, intensity, and time since quitting. BMI was positively associated with former smoking but was inversely related to current smoking in our data, and in analyses that were stratified by smoking status, the relation of BMI to bladder cancer was stronger among former than current smokers. An analysis including only participants who never smoked (n = 283 bladder cancer cases) yielded a stepwise positive association between increasing BMI level and bladder cancer. This observation suggests that smoking did not substantially confound our findings.

Despite numerous advantageous features of our study, one limitation is that weight and height were assessed by self-report, a method that is subject to error; however, self-reported weight and height have been found to be highly accurate (38). Measurement error in assessing physical activity is a more likely issue (39), but the physical activity assessment in the NIH-AARP study is very similar to an instrument with documented reasonable validity and reproducibility (35). Moreover, our physical activity tool predicts decreased cardiovascular mortality in this cohort (36).

Although this study is the largest known prospective investigation to date to examine the relations of BMI and physical activity to bladder cancer risk, our participants are predominantly Caucasians. Thus, we had limited statistical power to evaluate relations among non-White individuals, for whom bladder cancer incidence rates are known to differ from rates among Caucasians (40).

Biological mechanisms underlying the positive association between BMI and bladder cancer are speculative. Excess body fat is associated with elevated production of insulin, and insulin is a mitogenic factor that may also enhance tumor growth by increasing free insulin-like growth factor-I (41), which in turn stimulates cell proliferation and suppresses apoptosis (42) and has been linked to bladder cancer (43). Although hyperinsulinemia per se has not been implicated in bladder carcinogenesis, type 2 diabetes is directly associated with bladder cancer (44). Adiposity is also accompanied by low-grade, systemic inflammation (45), which may play a role in bladder carcinogenesis as suggested by positive relations of circulating levels of inflammatory markers, such as C-reactive protein and interleukin-6, to bladder cancer mortality (46, 47).

We conclude that overweight and obesity are associated with a modest increase in bladder cancer risk. Thus, bladder cancer may be added to the list of cancers potentially related to adiposity. In contrast, physical activity is not independently related to bladder cancer risk.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

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References

1. American Cancer Society. Cancer facts and figures 2007. Atlanta (GA): American Cancer Society; 2007.
2. Pelucchi C, Bosetti C, Negri E, Malvezzi M, La Vecchia C. Mechanisms of disease: the epidemiology of bladder cancer. Nat Clin Pract Urol 2006;3:327–40.
3. Flegal KM, Graubard BI, Williamson DF, Gail MH. Excess deaths associated with underweight, overweight, and obesity. JAMA 2005; 293:1861–7.
4. Center of Disease Control. Adult participation in recommended levels of physical activity—United States, 2001 and 2003. MMWR Morb Mortal Wkly Rep 2005;54:1208–12.
5. IARC. Handbooks of cancer prevention. Volume 6: weight control and physical activity. Lyon (France): IARC Press; 2002.
6. Samanic C, Gridley G, Chow WH, Lubin J, Hoover RN, Fraumeni JF, Jr. Obesity and cancer risk among White and Black United States veterans. Cancer Causes Control 2004;15:35–43.
7. Moller H, Mellemgaard A, Lindvig K, Olsen JH. Obesity and cancer risk: a Danish record-linkage study. Eur J Cancer 1994;30A:344–50.
8. Wolk A, Gridley G, Svensson M, et al. A prospective study of obesity and cancer risk (Sweden). Cancer Causes Control 2001;12:13–21.
9. Whittemore AS, Paffenbarger RS, Jr., Anderson K, Lee JE. Early precursors of urogenital cancers in former college men. J Urol 1984; 132:1256–61.
10. Batty GD, Shipley MJ, Jarrett RJ, Breeze E, Marmot MG, Smith GD. Obesity and overweight in relation to organ-specific cancer mortality in London (UK): findings from the original Whitehall study. Int J Obes (Lond) 2005;29:1267–74.
11. Calle EE, Rodriguez C, Walker-Thurmond K, Thun MJ. Overweight, obesity, and mortality from cancer in a prospectively studied cohort of U.S. adults. N Engl J Med 2003;348:1625–38.
12. Holick CN, Giovannucci EL, Stampfer MJ, Michaud DS. Prospective study of body mass index, height, physical activity and incidence of bladder cancer in US men and women. Int J Cancer 2007;120:140–6.
13. Lew EA, Garfinkel L. Variations in mortality by weight among 750,000 men and women. J Chronic Dis 1979;32:563–76.
14. Rapp K, Schroeder J, Klenk J, et al. Obesity and incidence of cancer: a large cohort study of over 145,000 adults in Austria. Br J Cancer 2005; 93:1062–7.
15. Tripathi A, Folsom AR, Anderson KE. Risk factors for urinary bladder carcinoma in postmenopausal women. The Iowa Women's Health Study. Cancer 2002;95:2316–23.
16. Cantwell MM, Lacey JV, Jr., Schairer C, Schatzkin A, Michaud DS. Reproductive factors, exogenous hormone use and bladder cancer risk in a prospective study. Int J Cancer 2006;119:2398–401.

17. Pan SY, Johnson KC, Ugnat AM, Wen SW, Mao Y. Association of obesity and cancer risk in Canada. Am J Epidemiol 2004;159:259–68.
18. Pelucchi C, La Vecchia C, Negri E, Dal Maso L, Franceschi S. Smoking and other risk factors for bladder cancer in women. Prev Med 2002;35:114–20.
19. Harris RE, Chen-Backlund JY, Wynder EL. Cancer of the urinary bladder in Blacks and Whites. A case-control study. Cancer 1990;66: 2673–80.
20. Vena JE, Graham S, Freudenheim J, et al. Diet in the epidemiology of bladder cancer in western New York. Nutr Cancer 1992;18:255–64.
21. Dosemeci M, Hayes RB, Vetter R, et al. Occupational physical activity, socioeconomic status, and risks of 15 cancer sites in Turkey. Cancer Causes Control 1993;4:313–21.
22. Brownson RC, Chang JC, Davis JR, Smith CA. Physical activity on the job and cancer in Missouri. Am J Public Health 1991;81:639–42.
23. Paffenbarger RS, Jr., Hyde RT, Wing AL. Physical activity and incidence of cancer in diverse populations: a preliminary report. Am J Clin Nutr 1987;45:312–7.
24. Soll-Johanning H, Bach E. Occupational exposure to air pollution and cancer risk among Danish urban mail carriers. Int Arch Occup Environ Health 2004;77:351–6.
25. Severson RK, Nomura AM, Grove JS, Stemmermann GN. A prospective analysis of physical activity and cancer. Am J Epidemiol 1989;130:522–9.
26. Schnohr P, Gronbaek M, Petersen L, Hein HO, Sorensen TI. Physical activity in leisure-time and risk of cancer: 14-year follow-up of 28,000 Danish men and women. Scand J Public Health 2005;33:244–9.
27. Ji J, Granstrom C, Hemminki K. Occupation and bladder cancer: a cohort study in Sweden. Br J Cancer 2005;92:1276–8.
28. Wannamethee SG, Shaper AG, Walker M. Physical activity and risk of cancer in middle-aged men. Br J Cancer 2001;85:1311–6.
29. Schatzkin A, Subar AF, Thompson FE, et al. Design and serendipity in establishing a large cohort with wide dietary intake distributions: the National Institutes of Health-American Association of Retired Persons Diet and Health Study. Am J Epidemiol 2001;154:1119–25.
30. North American Association of Central Cancer Registries (NAACCR). Standards for completeness, quality, analysis, and management of data. In: North American Association of Central Disease Registries, editor. NAACCR; 2004.
31. Michaud DS, Midthune D, Hermansen S, et al. Comparison of cancer registry case ascertainment with SEER estimates and self-reporting in a subset of the NIH-AARP Diet and Health Study. J Reg Manag 2005; 32:70–5.
32. Fritz AG, Percy C, Jack A, et al. editors. International classification of diseases for oncology: ICD-O. Geneva (Switzerland): WHO; 2000.
33. WHO. Physical status: the use and interpretation of anthropometry. Report No. 854. WHO Technical Report Series. Geneva (Switzerland): WHO; 1995.
34. American College of Sports Medicine. The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness in healthy adults. Med Sci Sports Exerc 1990;22:265–74..
35. Marshall AL, Smith BJ, Bauman AE, Kaur S. Reliability and validity of a brief physical activity assessment for use by family doctors. Br J Sports Med 2005;39:294–7.
36. Leitzmann MF, Park Y, Blair A, et al. Physical activity recommendations and decreased risk of mortality. Arch Intern Med 2007;167: 2453–60.
37. Cox DR. Regression models and lifetables. J R Stat Soc B 1972;34: 187–220.
38. Willett WC. Nutritional epidemiology. New York: Oxford University Press; 2006.
39. Sallis JF, Saelens BE. Assessment of physical activity by self-report: status, limitations, and future directions. Res Q Exerc Sport 2000;71: S1–14.
40. Madeb R, Messing EM. Gender, racial and age differences in bladder cancer incidence and mortality. Urol Oncol 2004;22:86–92.
41. Suikkari AM, Koivisto VA, Rutanen EM, Yki-Jarvinen H, Karonen SL, Seppala M. Insulin regulates the serum levels of low molecular weight insulin-like growth factor-binding protein. J Clin Endocrinol Metab 1988;66:266–72.
42. Iwamura M, Ishibe M, Sluss PM, Cockett AT. Characterization of insulin-like growth factor I binding sites in human bladder cancer cell lines. Urol Res 1993;21:27–32.
43. Zhao H, Grossman HB, Spitz MR, Lerner SP, Zhang K, Wu X. Plasma levels of insulin-like growth factor-1 and binding protein-3, and their association with bladder cancer risk. J Urol 2003;169:714–7.
44. Larsson SC, Orsini N, Brismar K, Wolk A. Diabetes mellitus and risk of bladder cancer: a meta-analysis. Diabetologia 2006;49:2819–23.
45. Festa A, D'Agostino R, Jr., Williams K, et al. The relation of body fat mass and distribution to markers of chronic inflammation. Int J Obes Relat Metab Disord 2001;25:1407–15.
46. Hilmy M, Bartlett JM, Underwood MA, McMillan DC. The relationship between the systemic inflammatory response and survival in patients with transitional cell carcinoma of the urinary bladder. Br J Cancer 2005;92:625–7.
47. Andrews B, Shariat SF, Kim JH, Wheeler TM, Slawin KM, Lerner SP. Preoperative plasma levels of interleukin-6 and its soluble receptor predict disease recurrence and survival of patients with bladder cancer. J Urol 2002;167:1475–81.

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対象の内訳		ヒト	動物	地 域	欧米	研究の種類	縦断研究	
	対象	一般健常者	空白		( )		コホート研究	
	性別	男女混合	( )		( )		( )	
	年齢	50～71歳(62歳)	[redacted]		( )		前向きコホート	
	対象数	10000以上			( )		( )	
調査の方法	質問紙	( )						
アウトカム	予 防	なし	なし	ガン予防	なし		( )	
	維持・改善	な し	な し	な し	なし	( )	( )	

図 表

Table 4. RR of bladder cancer according to physical activity

Variable	Physical activity (times a week)*					P_{trend}
	0	<1	1-2	3-4	≥5	
Person-years	591,296	467,197	745,4094	929,663	671,077	
No. cases	334	217	363	469	336	
Age- and sex-adjusted RR (95% CI)	1.0	0.80 (0.68-0.95)	0.79 (0.68-0.91)	0.75 (0.65-0.86)	0.71 (0.61-0.82)	<0.001
Multivariate RR without BMI (95% CI)†	1.0	0.85 (0.72-1.01)	0.88 (0.75-1.02)	0.89 (0.77-1.03)	0.85 (0.73-0.99)	0.188
Multivariate RR with BMI (95% CI)‡	1.0	0.85 (0.72-1.02)	0.89 (0.76-1.03)	0.91 (0.78-1.05)	0.87 (0.74-1.02)	0.358

*Physical activity was defined as activities that lasted ≥20 min and caused either increases in breathing or heart rate or working up a sweat.
† The multivariate model used age as the underlying time metric and included the following covariates: gender (women, men), a combination of smoking status (never, former, current), time since quitting for former smokers (≥10, 5-9, 1-4, <1 y) and smoking intensity for former and current smokers (1-10, 11-20, 21-30, 31-40, 41-60, ≥61 cigarettes per day), race/ethnicity (White, Black, Hispanic, other race/ethnicity), education (less than high school, high school, vocational school or some college, college graduate, postgraduate), marital status (married or living as married, other), family history of cancer (yes, no), intakes of red meat (quintiles), fruit and vegetables combined (quintiles), nonalcoholic beverages (quintiles) and alcohol (0, <1, 1-3, >3 servings/d), menopausal hormone therapy (current, past, never), oral contraceptive use (ever, never), and parity (0, 1-2, ≥3).
‡ Adjustment for BMI included the following categories: 18.5-24.9, 25.0-29.9, 30.0-34.9, ≥35.0 kg/m².

図表掲載箇所	
概 要 (800字まで)	＜目的＞BMIと身体活動と膀胱がん発症との関連を明らかにすること。＜方法＞コホート名：NIH-AARP Diet and Health Study、対象者数：471760人、追跡期間：8年、身体活動評価方法詳細：少なくとも20分以上で汗をかくような運動を週何回か。運動の単位：回／週。分位毎の運動は、分位1：なし、分位2：1回／週未満、分位3：1-2回／週、分位4：3-4回／週、分位5：5-7回／週であった。＜結果＞分位1：1、分位2：0.85(0.72-1.02)、分位3：0.89(0.76-1.03)、分位4：0.91(0.78-1.05)、分位5：0.87(0.74-1.02)であった。非肥満者と比べて、肥満者は膀胱がん発症リスクは28%有意に高かった。
結 論 (200字まで)	アメリカの男女を対象に、運動の膀胱ガン予防効果がないことが示唆された。
エキスパートによるコメント (200字まで)	希少な膀胱がんの発症に運動の予防効果が大きくないことを、50万人近い参加者を用いて検討した価値のある研究。

担当者　　宮地元彦

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Original Contribution

Physical Activity and Television Watching in Relation to Risk of Type 2 Diabetes

The Black Women's Health Study

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Few modifiable risk factors for type 2 diabetes have been documented in the high-risk population of US black women. The authors used data from 45,668 black women aged 21–69 years, followed biennially from 1995 to 2005 in the Black Women's Health Study, to estimate incidence rate ratios for type 2 diabetes comparing various levels of physical activity and television watching. Cox proportional hazards models were used to control confounding factors. During 10 years of follow-up, 2,928 incident cases of type 2 diabetes were identified. Vigorous activity was inversely associated with type 2 diabetes risk ($P_{trend} < 0.0001$); the incidence rate ratio for ≥7 hours per week was 0.43 (95% confidence interval (CI): 0.31, 0.59) relative to no activity. Brisk walking for ≥5 hours per week was associated with reduced type 2 diabetes risk (incidence rate ratio = 0.67, 95% CI: 0.49, 0.92) relative to no walking. Television watching was associated with an increased type 2 diabetes risk: The incidence rate ratio was 1.86 (95% CI: 1.54, 2.24) for ≥5 hours relative to <1 hour of television per day, independent of physical activity. These observational data suggest that black women might reduce their risk of developing type 2 diabetes by increasing their time spent walking or engaged in vigorous physical activity and by limiting television watching.

African continental ancestry group; diabetes mellitus, type 2; exercise; incidence; motor activity; television; walking; women's health

Abbreviations: BWHS, Black Women's Health Study; CI, confidence interval; MET, metabolic equivalent.

Type 2 diabetes has reached epidemic proportions in the United States and affects an estimated 20.6 million people (1, 2). The burden of this disease is particularly large among African-American women, among whom the prevalence is almost twice that of non-Hispanic whites (2). Although epidemiologic studies support a role for physical activity in preventing type 2 diabetes (3–15), there has been little attention focused on the impact of this factor in the high-risk population of African-American women. A recent randomized clinical trial (Diabetes Prevention Program) showed that an intensive lifestyle modification (healthy diet and moderate physical activity of 30 minutes a day for 5 days a week) reduced the incidence of type 2 diabetes by 50% as compared with placebo (10). We undertook to examine how the actual levels of physical activity in a large population of African-American women were related to type 2 diabetes incidence. With detailed questionnaire data collected every 2 years from over 40,000 women in the Black Women's Health Study (BWHS), we were able to assess the influence of vigorous activity, walking, and television watching on the incidence of type 2 diabetes in black women.

MATERIALS AND METHODS

The BWHS, a collaborative project of Boston University and Howard University, is an ongoing prospective follow-up study of African-American women from across the United States (16). The study began in 1995 when women aged 21–69 years were enrolled through postal questionnaires mailed to subscribers of *Essence* magazine, members of several professional organizations, and friends and relatives of early respondents. The baseline questionnaire collected

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information on demographics, medical and reproductive history, height, weight, physical activity, and cigarette and alcohol use, among other factors. Dietary intake was assessed by using a modified version of the NCI [National Cancer Institute]-Block food frequency questionnaire (17, 18).

After exclusion of 5,500 women who had completed the questionnaire but were outside the age range 21–69 years, who had not completed the questionnaire satisfactorily, or whose addresses were judged to be invalid, 59,052 women comprised the cohort that has been followed. Biennial follow-up questionnaires collect updated information on incident disease, weight, smoking, physical activity, and other factors. Follow-up has averaged over 80% of the baseline cohort over 5 completed questionnaire cycles.

Analyses are based on follow-up from 1995 through 2005, with follow-up beginning at age 30 years. We excluded women who did not reach the age of 30 years by the end of follow-up (n = 1,330); who reported a history of diabetes (n = 2,930) or gestational diabetes (n = 636) at baseline; who reported stroke (n = 359), myocardial infarction (n = 447), or cancer (n = 1,144) at baseline (whose questionnaire assessment of physical activity and diet may not reflect long-term patterns because these conditions may have caused modifications of physical activity level and diet); who were pregnant at baseline (n = 957); or who had missing data on weight, height, or physical activity at baseline (n = 5,581). The final analysis cohort consisted of 45,668 women.

Case definition

Each follow-up questionnaire asked about new diagnoses of diabetes during the previous 2-year period. Incident diabetes was reported by 2,928 women during follow-up.

We assessed the accuracy of self-reported diabetes among a random sample of 227 women whose physicians provided data from their medical records. The diagnosis of type 2 diabetes was confirmed for 218 (96%) of the women. Of the remaining 9 women, 3 did not have diabetes, 2 had type 1 diabetes, 2 had gestational diabetes, 1 had steroid-induced diabetes, and 1 was classified as having metabolic syndrome.

Exposure measurement

Physical activity data were obtained on each follow-up questionnaire. Subjects were asked separate questions on the number of hours per week spent on vigorous physical activity (e.g., running, swimming), walking for exercise, and walking to and from work. Response options were 0, <1, 1–2, 3–4, 5–6, 7–9, and ≥10 hours per week. Participants were asked on the 2003 questionnaire to classify their usual walking pace as casual/strolling (<2 mph), average/normal (2–<3 mph), fairly brisk (3–<4 mph), and brisk/striding (≥4 mph) (1 mile = 1.6 km). In our analysis of walking and diabetes risk, we used data on walking pace from the 2003 questionnaire to reflect their usual walking pace at all time periods. We created a variable that grouped participants into categories based on both walking pace (casual, average, or brisk (fairly brisk and brisk)) and amount of walking, taking into account both walking for exercise and walking to and from work. We created cumulative average variables for each individual based on activity data from each questionnaire that preceded the occurrence of diabetes or end of follow-up; when data are available for a number of different time periods, cumulative averages give a better representation of long-term exposure, as compared with the standard follow-up approach of updating the exposure variable for each time period (19). The number of hours per day spent watching television was also assessed in each questionnaire with possible response categories of 0, <1, 1–2, 3–4, and ≥5 hours per day.

Physical activity assessment in the BWHS was validated in a study conducted at the Howard University Cancer Center (20). Actigraphs (activity monitors) were worn by 101 BWHS participants during their waking hours for 7 days. They also completed 7-day physical activity diaries and completed the BWHS questions on physical activity. Significant positive correlations were observed between BWHS questionnaire data and actigraph measurements for total activity (metabolic equivalent (MET)-hours/week), r = 0.28, $P < 0.01$; walking, r = 0.26, $P < 0.01$; and vigorous activity, r = 0.40, $P < 0.01$. Correlations between the diary data and the BWHS questionnaire data were significant for total activity (MET-hours/week), r = 0.32, $P < 0.01$, as well as vigorous activity, r = 0.41, $P < 0.01$.

Data analysis

Age- and time-stratified Cox proportional hazards models were used to calculate incidence rate ratios, also known as hazard ratios, and 95% confidence intervals (21). Person-years were calculated from baseline to the year of diagnosis of type 2 diabetes, loss to follow-up, death, or the end of follow-up in 2005, whichever came first. Incidence rate ratios for diabetes were calculated for the levels of each physical activity measure relative to the lowest level. Time-varying covariates were reassigned every 2 years by using the Anderson-Gill data structure (22). The Anderson-Gill data structure creates a new record for every follow-up cycle at which the participant is at risk and assigns covariate values reported for that specific questionnaire cycle. The covariates included in the multivariable models were age (continuous), questionnaire cycle, family history of diabetes (biologic parents, siblings, or children), years of education (≤12, 13–15, 16, ≥17), household income in dollars (≤15,000, 15,001–25,000, 25,001–35,000, 35,001–50,000, 50,001–100,000, and >100,000), marital status (single, married, divorced/separated/widowed), cigarettes smoked per day (0, <15, 15–24, and ≥25), alcoholic drinks per week (0, 1–6, 7–13, ≥14), energy intake in kilocalories (quintiles), and coffee consumption in cups/week (<1, 1, 2–3, ≥4) (1 cup = 236.6 ml) . We carried out analyses with and without terms for body mass index (weight (kg)/height $(m)^2$) that we hypothesized to be in the causal pathway between physical activity and type 2 diabetes risk. Tests for linear trend across categories of physical activity variables were carried out by including in the regression model an ordinal term for increasing levels of exposure. Departure from the proportional hazards assumption was tested by using a likelihood ratio test comparing models with and without cross-product terms between exposure and age.

Table 1. Baseline Characteristics by Hours of Vigorous Physical Activity and Television Watching, the Black Women's Health Study, 1995–2005[a]

	Vigorous Physical Activity						Television Watching					
	0 hours/week			≥7 hours/week			0 or <1 hour/day			≥5 hours/day		
	No.	%	Mean (SD)	No.	%	Mean (SD)	No.	%	Mean (SD)	No.	%	Mean (SD)
Study participants	14,449	31.6		3,310	7.2		5,285	11.6		6,674	14.6	
Age, years			41.4 (10.5)			36.2 (9.1)			38.4 (9.5)			37.9 (10.6)
Body mass index, kg/m^2			29.2 (7.3)			25.9 (5.1)			26.5 (5.8)			29.1 (7.5)
Energy intake, kcal/day			1,729 (1,038)			1,645 (952)			1,536 (845)			1,970 (1,227)
Carbohydrates, % of energy			49.7 (9.5)			53.2 (9.7)			52.3 (9.6)			49.5 (9.7)
Fat, % of energy			34.5 (7.9)			30.3 (8.4)			31.5 (8.2)			34.6 (8.1)
Family history of diabetes		27.6			24.9			26.3			26.4	
Education, ≥17 years		18.9			22.0			33.5			13.4	
Income, >$100,000		9.3			13.7			19.5			6.2	
Married/living as married		41.3			33.6			41.5			34.3	
Cigarette use, ≥15/day		14.8			9.6			10.0			14.6	
Alcohol use, ≥7 drinks/day		6.6			7.1			3.5			10.8	
No coffee consumption		48.4			52.2			49.6			52.7	
No vigorous activity								25.2			41.7	
No walking for exercise		32.5			9.8			16.9			22.5	
Watching television, ≥5 hours/day		19.3			18.2							

Abbreviation: SD, standard deviation.
[a] The data shown are for extreme categories of vigorous activity and television watching.

RESULTS

Table 1 shows the baseline characteristics of the study population by vigorous physical activity and television watching. At baseline, 31.6% of the cohort reported no vigorous physical activity. Compared with women who exercised ≥7 hours per week, women who reported no vigorous activity were older, had a higher mean body mass index, smoked more, had a higher percentage of energy from fat, and were less likely to walk for exercise. At baseline, 14.6% of the population watched ≥5 hours of television per day. Compared with women who watched <1 hour per day of television, those who watched ≥5 hours per day were less educated; had a higher mean body mass index, higher energy intake, higher percentage of energy from fat, and greater use of cigarettes and alcohol; and were less likely to participate in vigorous physical activity or to walk for exercise. The correlation coefficient for the relation between vigorous activity and television watching was −0.035 ($P < 0.0001$).

There were 2,928 incident cases of type 2 diabetes ascertained during 182,994 person-years of follow-up. In the multivariable model, vigorous physical activity was inversely associated with the risk of type 2 diabetes (Table 2). The incidence rate ratio for ≥7 hours per week of vigorous activity relative to 0 hours was 0.43 (95% confidence interval (CI): 0.31, 0.59) ($P_{\text{trend}} < 0.0001$). When body mass index was added to the model, the incidence rate ratio was 0.57 (95% CI: 0.41, 0.79) (data not shown). Brisk walking was also associated with a reduced risk: The incidence rate ratio for walking at a brisk pace for ≥5 hours per week relative to no walking was 0.67 (95% CI: 0.49, 0.92). With inclusion of terms for body mass index in the regression model, the incidence rate ratio was 0.87 (95% CI: 0.64, 1.19) (data not shown).

Television watching was positively associated with the risk of type 2 diabetes (Table 2). The incidence rate ratio was 1.86 (95% CI: 1.54, 2.24) for watching ≥5 hours per day relative to <1 hour per day ($P_{\text{trend}} < 0.0001$). In a multivariable model that included body mass index, the incidence rate ratio was 1.56 (95% CI: 1.29, 1.89) (data not shown).

The inverse association between vigorous physical activity and type 2 diabetes risk was present at all levels of body mass index (Table 3), even among those with a body mass index of ≥30, the World Health Organization definition of obesity (23). The positive association of television watching and risk of type 2 diabetes was also present at all levels of body mass index. The association was strongest in those with a body mass index of <25, among whom the incidence rate ratio for watching ≥5 hours per day of television was 2.49 (95% CI: 1.24, 5.02) relative to <1 hour per day ($P_{\text{trend}} = 0.01$). The associations for vigorous activity and television watching were similar among premenopausal and postmenopausal women (data not shown).

Figure 1 shows the combined effect of vigorous physical activity and television watching. The reference group was those women who reported ≥3 hours per week of vigorous activity and <1 hour per day of television watching. Type 2 diabetes risk increased with decreasing physical activity in each category of television watching and increased with increased television watching in each category of physical

Table 2. Incidence Rate Ratios for the Association of Physical Activity, Television Watching, and Risk of Type 2 Diabetes, the Black Women's Health Study, 1995–2005

	Cases, no.	Person-Years, no.	Age-adjusted Incidence Rate Ratio	Multivariable[a]	
				Incidence Rate Ratio	95% Confidence Interval
Vigorous physical activity, hours/week					
0	994	42,665	1.0	1.0	
<1	994	51,646	0.79	0.90	0.82, 0.99
1–2	668	51,057	0.62	0.77	0.69, 0.85
3–4	170	21,368	0.41	0.53	0.45, 0.63
5–6	63	9,101	0.38	0.49	0.38, 0.64
≥7	39	7,157	0.33	0.43	0.31, 0.59
			$P_{trend} < 0.0001$	$P_{trend} < 0.0001$	
Walking for exercise or transport, pace[b]					
No walking	69	5,375	1.0	1.0	
Casual, hours/week					
<1	182	8,598	1.06	1.04	0.78, 1.37
1–2	310	14,097	1.06	1.09	0.84, 1.42
3–4	113	4,726	1.15	1.21	0.89, 1.65
≥5	106	4,459	1.17	1.21	0.89, 1.65
Average, hours/week					
<1	176	10,386	0.92	1.02	0.77, 1.35
1–2	404	25,439	0.81	0.96	0.74, 1.24
3–4	198	11,242	0.84	1.04	0.78, 1.37
≥5	178	11,347	0.78	0.95	0.71, 1.26
Brisk, hours/week					
<1	67	5,432	0.73	0.91	0.64, 1.27
1–2	192	17,582	0.59	0.78	0.59, 1.03
3–4	107	10,735	0.49	0.69	0.51, 0.95
≥5	115	12,417	0.46	0.67	0.49, 0.92
Television watching, hours/day					
0–<1	135	16,390	1.0	1.0	
1–2	935	68,284	1.51	1.43	1.19, 1.71
3–4	1,146	67,901	1.77	1.53	1.28, 1.83
≥5	712	30,419	2.38	1.86	1.54, 2.24
			$P_{trend} < 0.0001$	$P_{trend} < 0.0001$	

[a] The multivariable incidence rate ratio included terms for age, time period, family history of diabetes, years of education, family income, marital status, cigarette use, alcohol use, energy intake, coffee consumption, vigorous activity, television watching, and walking.

[b] The pace of walking was reported on only 1 follow-up questionnaire, and values are missing for those who did not fill out that questionnaire.

activity. The incidence rate ratio for no vigorous physical activity and watching ≥5 hours per day of television was 3.64 (95% CI: 2.23, 5.92).

DISCUSSION

In this large prospective study of black women, vigorous physical activity was associated with a reduced risk of type 2 diabetes. Walking at a brisk pace, but not slow walking, was also associated with a reduction in risk. The risk of type 2 diabetes was increased among women who spent an appreciable amount of time watching television, and this increase was apparent at every level of physical activity.

The present findings on vigorous physical activity in black women are consistent with those reported for white women in previous studies (4, 5, 7, 9, 12–14). Results from

Table 3. Incidence Rate Ratios for the Association of Vigorous Physical Activity and Television Watching With Risk of Type 2 Diabetes, Stratified by Body Mass Index, the Black Women's Health Study, 1995–2005

Body Mass Index, kg/m^2	Vigorous Physical Activity				Television Watching			
	Hours/ Week	No. of Cases (N = 2,928)	Incidence Rate Ratio[a]	95% Confidence Interval	Hours/ Day	No. of Cases (N = 2,928)	Incidence Rate Ratio[a]	95% Confidence Interval
<25	0	57	1.0		0–<1	10	1.0	
	<1	58	0.97	0.65, 1.43	1–2	67	1.64	0.84, 3.19
	1–2	50	0.89	0.59, 1.36	3–4	69	1.71	0.87, 3.34
	≥3	32	0.74	0.46, 1.19	≥5	51	2.49	1.24, 5.02
			P_{trend} = 0.08				P_{trend} = 0.01	
25–29	0	236	1.0		0–<1	38	1.0	
	<1	236	0.92	0.76, 1.12	1–2	270	1.42	1.01, 2.00
	1–2	186	0.84	0.68, 1.04	3–4	297	1.41	1.00, 1.98
	≥3	90	0.65	0.50, 0.85	≥5	143	1.57	1.09, 2.25
			P_{trend} = 0.001				P_{trend} = 0.07	
30–34	0	250	1.0		0–<1	44	1.0	
	<1	268	0.95	0.79, 1.14	1–2	249	1.08	0.78, 1.49
	1–2	173	0.81	0.65, 0.99	3–4	300	1.10	0.80, 1.52
	≥3	83	0.75	0.57, 0.98	≥5	181	1.29	0.92, 1.81
			P_{trend} = 0.01				P_{trend} = 0.07	
≥35	0	451	1.0		0–<1	43	1.0	
	<1	432	0.93	0.81, 1.07	1–2	349	1.37	1.00, 1.89
	1–2	259	0.93	0.79, 1.11	3–4	480	1.35	0.99, 1.86
	≥3	67	0.57	0.43, 0.74	≥5	337	1.59	1.15, 2.19
			P_{trend} = 0.002				P_{trend} = 0.01	

[a] Adjusted for age, time period, family history of diabetes, years of education, family income, marital status, cigarette use, alcohol use, energy intake, coffee consumption, vigorous activity, television watching, and walking.

the Diabetes Prevention Program trial showed that a lifestyle intervention consisting of diet and physical activity was effective in reducing the incidence of diabetes among all racial subgroups, including African Americans (10). In the Women's Health Initiative observational cohort, there was no significant association among African-American women, but statistical power was limited because there were only 395 diabetic cases among the black women (13). The Women's Health Initiative cohort included only postmenopausal women, but this does not explain the difference between their results and ours. We found an inverse association of physical activity with diabetes in both pre- and postmenopausal women. The other epidemiologic studies that have included an appreciable number of black women have been cross-sectional in design (5, 6).

We had enough statistical power to assess the association of physical activity and type 2 diabetes within strata of body mass index, and we found that physical activity is beneficial regardless of body mass index. Previous studies of physical activity and diabetes that have assessed physical activity within strata of body mass index have provided inconsistent results (3, 4, 9, 12).

Our results showing that walking at a brisk pace is associated with a reduction in risk of type 2 diabetes are consistent with findings on white women from the Nurses' Health Study (7). The Women's Health Initiative did not find a significant association of walking with diabetes risk in black women, but statistical power was limited (13).

The positive association between television watching and type 2 diabetes risk in our study of black women is similar to that found in 2 previous studies of white women (24) and men (25). Importantly, this association was independent of physical activity and other known risk factors for type 2 diabetes.

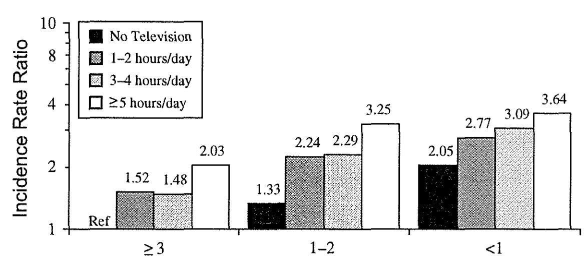

Figure 1. Incidence rate ratios for type 2 diabetes by levels of vigorous physical activity and television watching, the Black Women's Health Study, 1995–2005. The reference category for all other strata is no television watching and ≥3 hours/week of physical activity (Ref).

Body mass index, which is influenced in part by levels of physical activity and is a strong independent risk factor for type 2 diabetes, may be an intermediate in the association of physical activity and diabetes or a confounder of the association. When we controlled for body mass index in multivariable analyses, the association between physical activity and type 2 diabetes was reduced slightly, but a strong inverse association still remained. In addition, the inverse association of physical activity with diabetes risk was present at all levels of body mass index, including the <25 category of body mass index where confounding from body mass index would be smallest. The latter 2 observations suggest that confounding, if present, played a minor role in the present study and that mechanisms other than body mass index may also play a role in the reduction in risk.

The relation between type 2 diabetes and physical activity may be mediated through increased insulin sensitivity due to increases in levels of the glucose transporter protein GLUT-4 and muscle glycogen synthase activity, a decrease in serum triglyceride concentration, and an increase in muscle capillary network (15, 26). Physical activity can also lead to weight loss or maintenance of a healthy weight (27), which in turn can lead to a lower risk of type 2 diabetes.

The positive association between television watching and type 2 diabetes might be explained by 2 mechanisms (28). First, television watching is related to a lower expenditure of energy, which in turn can lead to obesity, weight gain, and increased risk of diabetes. Adjustment for body mass index attenuated the incidence rate ratios in our study; this is compatible with the association of television watching with type 2 diabetes being at least partially mediated through obesity. Another possible mechanism is that television watching leads to a higher caloric intake and a relatively unhealthy dietary pattern. Participants in our study who watched more television had a higher energy intake and a higher fat and carbohydrate intake as compared with those who watched no television.

One of the main strengths of our study is the prospective study design, which reduces the potential of recall bias. In addition, the BWHS has high follow-up rates that reduce the possibility of bias resulting from selective losses. The sample size was large, providing excellent statistical power to assess effects overall and in subgroups. We were able to adjust for a large number of possible confounding variables.

Physical activity was self-reported. Because we had repeated measures of physical activity, we were able to get a better representation of long-term physical activity. Our validation study showed significant correlations of questionnaire responses with diary and actigraph measurement of physical activity. Nonetheless, measurement errors are likely but should be nondifferential, which would move estimates for the highest exposure categories toward the null. To the extent that there was misclassification of exposure, the true association of activity with diabetes risk may be even stronger than shown here.

Identification of type 2 diabetes cases was based on self-report. A validation study indicated that type 2 diabetes was reported with a high degree of accuracy. Some women with undiagnosed type 2 diabetes were undoubtedly misclassified as noncases, but the prevalence of undiagnosed disease was likely to be low (29). Physicians are well aware of the high risk of diabetes among African-American women, and it seems likely that BWHS participants were screened for the disease during the course of regular check-ups. Access to health care is quite good among BWHS participants: 93% reported that they had health insurance in 1997, and 98% reported that they had visited a physician/hospital in the past 2 years.

The BWHS participants were from 17 states across the United States with approximately equal numbers living in the Northeast, South, West, and Midwest. In addition, 97% of the participants have completed high school or a higher level of education. Among the US black female population of the same ages, 83% have at least a high school education (30). In this respect, our results should be applicable to most US black women, except possibly the approximately 17% who have not completed high school.

The present observational findings suggest that physical activity may reduce the risk of type 2 diabetes in African-American women, a population at high risk of the disease. Recent recommendations for physical activity for adults include "moderate-intensity physical activities for at least 30 minutes on 5 or more days of the week" (Centers for Disease Control and Prevention) and "vigorous-intensity physical activity 3 or more days per week for 20 or more minutes per occasion" (Healthy People 2010) (31). Like the majority of adults in the United States, most African-American women do not meet recommended levels of physical activity. Our results for vigorous activity and brisk walking suggest that levels approximating those recommended might indeed be protective against type 2 diabetes. Regular brisk walking may be easier to implement than vigorous physical activity. Reducing sedentary behaviors, such as television watching, or at least reducing the excess eating that often accompanies it, might also be effective. A necessary first step for the translation of scientific findings into behavioral change is dissemination of the health information to those affected, and the current results provide the basis for educating African-American women about the benefits of physical activity in preventing type 2 diabetes.

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REFERENCES

1. Engelgau MM, Geiss LS, Saaddine JB, et al. The evolving diabetes burden in the United States. *Ann Intern Med*. 2004; 140(11):945–950.

2. Centers for Disease Control and Prevention. National diabetes fact sheet: general information and national estimates on diabetes in the United States, 2005. Atlanta, GA: Centers for Disease Control and Prevention, US Department of Health and Human Services, 2005. (NIH publication 06-3892). (http://www.cdc.gov/diabetes/pubs/pdf/ndfs_2005.pdf).
3. Manson JE, Rimm EB, Stampfer MJ, et al. Physical activity and incidence of non-insulin-dependent diabetes mellitus in women. *Lancet*. 1991;338(8770):774–778.
4. Manson JE, Nathan DM, Krolewski AS, et al. A prospective study of exercise and incidence of diabetes among US male physicians. *JAMA*. 1992;268(1):63–67.
5. Lipton RB, Liao Y, Cao G, et al. Determinants of incident non-insulin dependent diabetes mellitus among blacks and whites in a national sample: the NHANES I Epidemiologic Follow-up Study. *Am J Epidemiol*. 1993;138(10):826–839.
6. Sherman JA, Lama J, Raghunatham TE, et al. Physical activity and NIDDM in African-Americans: the Pitt County Study. *Diabetes Care*. 1998;21(4):555–562.
7. Hu FB, Sigal RJ, Rich-Edwards JW, et al. Walking compared with vigorous physical activity and risk of type 2 diabetes in women. *JAMA*. 1999;282(15):1433–1439.
8. Wannamethee SG, Shaper GA, Alberti KG. Physical activity, metabolic factors, and the incidence of coronary heart disease and type 2 diabetes. *Arch Intern Med*. 2000;160(14):2108–2116.
9. Folsom AR, Kushi LH, Hong CP. Physical activity and incident diabetes mellitus in postmenopausal women. *Am J Public Health*. 2000;90(1):134–138.
10. Knowler WC, Barrett-Connor E, Fowler SC, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med*. 2002;346(6):393–403.
11. Hu G, Qiao Q, Silventoinen K, et al. Occupational, commuting, and leisure-time physical activity in relation to risk for type 2 diabetes in middle-aged Finnish men and women. *Diabetoloigia*. 2003;46(3):322–329.
12. Weinstein AR, Sesso HD, Lee IM, et al. Relationship of physical activity vs body mass index with type 2 diabetes in women. *JAMA*. 2004;292(10):1188–1194.
13. Hsia J, LieLing W, Allen C, et al. Physical activity and diabetes risk in postmenopausal women. *Am J Prev Med*. 2005;28(1):19–25.
14. Rana JS, Li TY, Manson J, et al. Adiposity compared with physical inactivity and risk of type 2 diabetes in women. *Diabetes Care*. 2007;30(1):53–58.
15. Jeon CY, Lokken RP, Hu FB, et al. Physical activity of moderate intensity and risk of type 2 diabetes: a systematic review. *Diabetes Care*. 2007;30(3):744–752.
16. Rosenberg L, Adams-Campbell L, Palmer JR. The Black Women's Health Study: a follow-up study for causes and preventions of illness. *J Am Med Womens Assoc*. 1995;50(2):56–58.
17. Block G, Hartman AM, Naughton D. A reduced dietary questionnaire. Development and validation. *Epidemiology*. 1990;1(1):58–64.
18. Kumanyika SK, Mauger D, Mitchell DC, et al. Relative validity of food frequency questionnaire nutrient estimation in the Black Women's Health Study. *Ann Epidemiol*. 2003;13(2):111–118.
19. Hu FB, Stampfer MJ, Rimm E, et al. Dietary fat and coronary heart disease: a comparison of approaches for adjusting for total energy intake and modeling repeated dietary measurements. *Am J Epidemiol*. 1999;149(6):531–540.
20. Carter-Nolan PL, Adams-Campbell LL, Makambi K, et al. Validation of physical activity instruments: Black Women's Health Study. *Ethn Dis*. 2006;16(4):943–947.
21. SAS Institute, Inc. *SAS/STAT User's Guide. Version 8.02*. Cary, NC: SAS Institute, Inc; 2002.
22. Therneau TM. Extending the Cox model. In: Lin DY, Fleming TR, eds. *Proceedings of the First Seattle Symposium in Biostatistics: Survival Analysis*. New York, NY: Springer Verlag; 1997.
23. Obesity and overweight. Geneva, Switzerland: Office of Health Communications and Public Relations, World Health Organization; 2006. (http://whqlibdoc.who.int/fact_sheet/2006/FS_311.pdf).
24. Hu FB, Li TY, Colditz GA, et al. Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women. *JAMA*. 2003;289(14):1785–1791.
25. Hu FB, Leitzzman MF, Stampfer MJ, et al. Physical activity and television watching in relation to risk of type 2 diabetes mellitus in men. *Arch Intern Med*. 2001;161(12):1542–1548.
26. Henriksson J. Influence of exercise on insulin sensitivity. *J Cardiovasc Risk*. 1995;2(4):303–309.
27. Catenacci VA, Wyatt HR. The role of physical activity in producing and maintaining weight loss. *Nat Clin Pract Endocrinol Metab*. 2007;3(7):518–529.
28. Hu FB. Sedentary lifestyle and risk of obesity and type 2 diabetes. *Lipids*. 2003;38(2):103–108.
29. Harris MI, Flegal KM, Cowie CC, et al. Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in U.S. adults. The Third National Health and Nutrition Examination Survey, 1988–1994. *Diabetes Care*. 1998;21(4):518–524.
30. Day J, Curry A. Educational attainment in the United States: March 1995. Washington, DC: Bureau of the Census, US Department of Commerce; 2006. (http://www.census.gov/prod/2/pop/p20/p20-489.pdf).
31. Physical activity for everyone. Atlanta, GA: Centers for Disease Control and Prevention, US Department of Health and Human Services; 2007. (http://www.cdc.gov/nccdphp/dnpa/physical/recommendations/index.htm).

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対象の内訳		ヒト	動物	地　域	欧米	研究の種類	縦断研究
	対象	一般健常者	空白		(　　)		コホート研究
	性別	女性	(　　)		(　　)		(　　)
	年齢	21-69歳	[redacted]		(　　)		前向き研究
	対象数	10000以上			(　　)		(　　)
調査の方法	質問紙	(　　)					
アウトカム	予　防	な し	糖尿病予防	な し	な し	(　　)	(　　)
	維持・改善	な し	な し	な し	な し	(　　)	(　　)

図　表

Table 2. Incidence Rate Ratios for the Association of Physical Activity, Television Watching, and Risk of Type 2 Diabetes, the Black Women's Health Study, 1995–2005

	Cases, no.	Person-Years, no.	Age-adjusted Incidence Rate Ratio	Multivariable[a] Incidence Rate Ratio	Multivariable[a] 95% Confidence Interval
Vigorous physical activity, hours/week					
0	994	42,665	1.0	1.0	
<1	994	51,646	0.79	0.90	0.82, 0.99
1–2	668	51,057	0.62	0.77	0.69, 0.85
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≥7	39	7,157	0.33	0.43	0.31, 0.59
			P_{trend} < 0.0001	P_{trend} < 0.0001	
Walking for exercise or transport, pace[b]					
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≥5	712	30,419	2.38	1.86	1.54, 2.24
			P_{trend} < 0.0001	P_{trend} < 0.0001	

[a] The multivariable incidence rate ratio included terms for age, time period, family history of diabetes, years of education, family income, marital status, cigarette use, alcohol use, energy intake, coffee consumption, vigorous activity, television watching, and walking.
[b] The pace of walking was reported on only 1 follow-up questionnaire, and values are missing for those who did not fill out that questionnaire.

図表掲載箇所	P431, Table2
概　要 (800字まで)	本研究は、アメリカのThe Black Women's Health Studyに参加した黒人女性45,668名を対象に10年間の追跡調査を行い、身体活動やテレビ視聴時間が2型糖尿病発症に及ぼす影響について検討したものである。質問紙によって、週当たりの高強度身体活動を実施した時間、歩行による運動または移動手段としての歩行を実施した時間および速度、1日当たりのテレビ視聴時間を尋ねた。高強度身体活動の実施時間を0時間/週、1時間/週未満、1-2、3-4、5-6、7時間/週以上の6群に、歩行時間を、のんびり(2mph未満)、普通(2-3mph)、やや速い(3-4mph)、早歩き(4mph以上)のペースでそれぞれ、1時間/週未満、1-2、3-4、5時間/週の4群に、テレビ視聴時間を、1時間/日未満、1-2、3-4、5時間/日以上の4群に分類した。高強度身体活動の実施が0時間/週の集団と比較すると、時間の延長につれて全ての群で糖尿病発症リスクがそれぞれ0.90(95%信頼区間:0.82-0.99)、0.77(0.69-0.85)、0.53(0.45-0.63)、0.49(0.38-0.64)、0.43(0.31-0.59)と量反応的に減少した(Ptrend<0.0001)。歩行時間0時間の集団と比較すると、のんびり歩行、普通歩行では糖尿病発症リスクの減少はみられなかったが、早歩きを週当たり3-4時間、または5時間以上実施している集団では、糖尿病発症リスクが0.69(0.51-0.95)、0.67(0.49-0.92)と有意に減少した。また、テレビ視聴時間1時間/日未満の集団と比較すると、1-2、3-4、5時間/日以上の全ての集団で、糖尿病発症リスクが1.43(1.19-1.71)、1.53(1.28-1.83)、1.86(1.54-2.24)と量反応的に上昇した(Ptrend<0.0001)。また、高強度身体活動とテレビ視聴時間の延長の複合効果として、高強度活動3時間/週以上かつ視聴1時間/日未満の集団と比較すると高強度活動1時間/未満かつ視聴5時間/日以上の集団で糖尿病発症リスクは3.64(2.23-5.92)と跳ね上がった。
結　論 (200字まで)	黒人女性コホートにおいて、高強度身体活動や歩行時間の延長は2型糖尿病発症の抑制に効果を示すことが明らかとなった。また、不活動時間(テレビ視聴)の延長が糖尿病発症リスクを高めることが明らかとなった。
エキスパートによるコメント (200字まで)	身体活動基準の策定に用いられた研究の1つである。糖尿病の発症は我が国においても年々増加しており、糖尿病発症を予防することは非常に重要な課題である。本研究では、高強度身体活動とテレビ視聴時間の相互作用が認められており、高強度身体活動を実施しておらず、テレビの視聴時間が多い人においては、特に重点的な支援を行うことが必要であることが示されている。

担当者　久保絵里子・村上晴香・宮地元彦