



## Effects of Moderate and Vigorous Physical Activity on Heart Rate Variability in a British Study of Civil Servants

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Physical inactivity and low resting heart rate variability (HRV) are associated with increased coronary heart disease incidence. In the Whitehall II study of civil servants aged 45–68 years (London, United Kingdom, 1997–1999), the strength of the association of moderate and vigorous activity with higher HRV was examined. Five-minute recordings of heart rate and HRV measures were obtained from 3,328 participants. Calculated were time domain (standard deviation of NN intervals) and high-frequency-power measures as indicators of cardiac parasympathetic activity and low-frequency power of parasympathetic-sympathetic balance. Leisure-time physical activity (metabolic equivalent-hours per week) was categorized as moderate ( $\geq 3$ – $< 5$ ) and vigorous ( $\geq 5$ ). Moderate and vigorous physical activity were associated with higher HRV and lower heart rate. For men, linear trends of higher low-frequency power with increasing quartile of vigorous activity (304.6 (low), 329.0, 342.4, 362.5 (high);  $p < 0.01$ ) and lower heart rate with increasing quartile of moderate activity (69.6 (low), 69.2, 68.9, 67.8 (high);  $p < 0.05$ ) were found. These associations remained significant after adjustment for smoking and high alcohol intake. For men whose body mass index was  $> 25$  kg/m<sup>2</sup>, vigorous activity was associated with HRV levels similar to those for normal-weight men who engaged in no vigorous activity. Vigorous activity was associated with higher HRV, representing a possible mechanism by which physical activity reduces coronary heart disease risk.

coronary disease; exercise; heart rate; population

Abbreviations: BMI, body mass index; HRV, heart rate variability; MET, metabolic equivalent; SDNN, standard deviation of all NN intervals.

Because the prevalence of physical inactivity has risen over the last decade (1–3), its association with coronary heart disease incidence (4–7) is of growing importance. Studies of coronary heart disease risk and physical activity have predominantly focused on vigorous activity (8, 9). Physical inactivity may lead to coronary heart disease via increased adiposity, reduced lean body mass, reduced cardiovascular fitness (6), raised blood pressure (10), reduced glucose tolerance, lowered insulin sensitivity (11), and adverse lipid profile (12). However, the relative importance of each of these mechanisms is unclear, and they do not totally explain the reduction in coronary heart disease observed in studies

that include increased physical activity. Disturbances in autonomic function are associated with each of these potential mechanisms linking physical inactivity to coronary disease and may be an additional way in which physical activity reduces coronary heart disease morbidity.

Low resting heart rate variability (HRV), a marker of autonomic function, has been related to an increased risk of all-cause mortality (13) and to the incidence of ventricular arrhythmias and coronary heart disease events (14–17), after adjustment for risk factors. HRV is an indicator of the interaction between cardiac sympathetic and parasympathetic activity, which causes changes in the beat-to-beat intervals

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and changes in the frequency components of the heart rate. Short-term variations in the beat-to-beat interval are reduced by decreased parasympathetic activity or sympathetic overstimulation. High-frequency power is a marker of parasympathetic activity; low levels of high-frequency power indicate lower responsiveness to parasympathetic activity. Low-frequency power reflects a combination of both parasympathetic and sympathetic modulations. Low levels of high-frequency power and low levels of low-frequency power are associated with risk of coronary heart disease (17).

Higher HRV has been reported in athletes (18–20). In addition, increases in HRV may follow aerobic training in previously sedentary people (21–24).

However, in general populations, the relation between physical activity and heart rate and its variability remains uncertain. Those population-based studies that have assessed the association of physical activity with HRV in healthy men and women were relatively small (Fagard et al.,  $n = 614$  (25); Horsten et al.,  $n = 300$  (26); and Molgaard et al.,  $n = 104$  (27)). These studies found high levels of physical activity to be associated with higher levels of low-frequency power and high-frequency power (25, 27). In these, as with the intervention studies, the focus has been on the association between HRV and aerobic exercise. Because high body weight, measured in terms of body mass index (BMI), is related to both low levels of physical activity and depression of parasympathetic activity (28–32), it may be a biologic mediator in the association between physical activity and increased HRV. It is unclear whether total activity, affecting energy expenditure and body weight, or the intensity of activity is more important in increasing HRV (33).

The aims of this investigation were to determine the extent to which moderate and vigorous physical activity were associated with higher HRV in a large population-based study and whether any associations observed differed with body weight. The homogeneity of occupational activity among participants in the Whitehall II study of civil servants, with more than 92 percent employed in nonmanual occupations, makes this cohort a good group to examine regarding the relation between leisure-time activity and risk of disease.

## MATERIALS AND METHODS

### Participants

All nonindustrial civil servants aged 35–55 years working in the London, United Kingdom, offices of 20 departments were invited to participate in this study. Recruitment (phase 1) took place during 1985–1988. The final cohort consisted of 10,308 (3,414 women) participants, with an overall response rate of 73 percent. However, the actual rate was probably higher because 4 percent of those on the employee list had moved before the study began and thus were not eligible for inclusion. Data presented here are from the fifth phase of data collection (phase 5). At phase 5 (1997–1999), all study participants known to be alive and in the country were invited to attend a screening clinic. Although 6,554 (1,909 women) participants aged 45–68 years attended the clinic, heart rate was recorded for only 3,349 because of staff availability. Participants who did not undergo HRV record-

ings did not differ significantly from those who did with respect to age, sex, employment grade, smoking, and alcohol intake. A total of 3,328 participants (994 women) completed HRV recordings and the physical activity questionnaire. The University College of London ethics committee approved the study.

### Physical activity questionnaire

The physical activity questionnaire included 20 items on the amount of time participants spent walking and cycling and involved in sports, gardening activities, housework, and house maintenance (such as home improvements) and was derived from the Minnesota leisure-time activity questionnaire (34). Open items enabled participants to report additional activities. To indicate usual activity, participants were asked about their total hours spent on these activities during the last 4 weeks. These numbers were averaged to calculate total hours per week on each activity item, and a metabolic equivalent (MET) value was assigned by using a compendium of activity energy costs (35). One MET is the metabolic energy expended lying quietly and is equivalent to approximately 1 kcal per kilogram of body weight per hour. The MET value reflects the intensity of the activity as a multiple of one MET. Therefore, a 70-kg person walking at a moderate pace (MET value of 3.5) for 1 hour would expend 3.5 METs or 245 kcal. Time spent per week on each item was multiplied by the MET value of the activity to give MET-hours per week. Physical activity was expressed as MET-hours per week rather than kcal per week because the latter calculation includes the participant's body weight, which prevents direct comparison between participants without adjustment for body weight and moreover may confound the relation between physical activity and HRV. Total physical activity was calculated by summing the MET-hours for all items. MET values were used to categorize activities as light (<3 METs), moderate ( $\geq 3$ –<5 METs), and vigorous ( $\geq 5$  METs), and the number of MET-hours per week spent on each activity was obtained.

### Measurement of HRV

HRV measurements were designed, conducted, and analyzed in accordance with the standards proposed by the European Society of Cardiology/North American Society of Pacing and Electrophysiology (ESC/NASPE) Task Force (36). Limb and chest leads were placed in standard positions, and a 12-lead electrocardiogram (Mingorec; Siemens, Munich, Germany) was recorded. After this procedure was completed, participants rested supine for at least 5 minutes in a quiet room. Five minutes of beat-to-beat heart rate data were sampled at a frequency of 500 Hz by using a dedicated personal computer and software (Kardiosis; Tapa, Inc., Ankara, Turkey) to obtain digitized recording of R waves. A heartbeat is caused by ventricular contraction, which in turn is caused by electrical depolarization detected on the surface of the skin as the QRS complex. The interval between successive R waves reflects successive heartbeats. HRV recordings were excluded from further analysis if they had frequent atrial or ventricular ectopics (defined as more than

10 percent of all heartbeats). Manual methods of removing electrocardiogram artifacts and ectopics used in previous studies have the potential for observer error and were not considered feasible in a study of this size. Therefore, a computerized algorithm was developed for QRS detection and ectopic beat identification that incorporates both timing and morphologic information from the electrocardiogram (37). When validated against manual review by a cardiologist on 69 healthy volunteers, the algorithm had a sensitivity of 92 percent and a specificity of 95 percent for detecting supraventricular ectopics with normal QRS complexes (37). Singular ectopics were eliminated, and R-R sequence was linearly interpolated.

HRV was analyzed in the time domain (standard deviation of all NN intervals (SDNN)) to assess changes in beat-to-beat intervals that occur between consecutive heartbeats. The frequency domain measures were analyzed by using the autoregressive method (38).

Frequency domain measures were computed by integration over their frequency intervals in two frequency bands: 0.04–0.15 Hz (low-frequency power, in milliseconds squared) and 0.15–0.4 Hz (high-frequency power, in milliseconds squared). Low-frequency power reflects both parasympathetic and sympathetic modulations; the high-frequency component is a function of the variation in parasympathetic tone (36).

### Screening examination

At the screening examination, height, weight, and waist and hip circumferences were measured. Waist circumference was taken as the smallest circumference at or below the costal margin and hip circumference at the level of the greater trochanter by using a fiberglass tape measure at 600 g tension. BMI was calculated from weight and height (kg/m<sup>2</sup>) measurements. Blood pressure (in mmHg) was measured twice with the Hawksley random zero sphygmomanometer (Hawksley & Sons Ltd., West Sussex, United Kingdom) after a 5-minute rest. High density lipoprotein cholesterol concentration was determined from fasting serum samples.

Participants completed a questionnaire that included items on smoking and on alcohol intake (units per week) as well as a rating of their general health as excellent, very good, good, fair, or poor. Employment grades were grouped into three categories: unified grades 1–7 (administrators in Whitehall II), executive officers, and clerical and support staff (low grade). Details of these methods have been described previously (39).

### Statistical analysis

Student's *t* test was used to compare total activity between men and women. Total physical activity and moderate activity expressed in MET-hours per week were split into sex-specific quartiles. For vigorous activity, we categorized participants into either a "none" group or into sex-specific tertiles of MET-hours per week for those who reported some vigorous activity. Tests for linear trend were used to determine differences in mean age and the proportions of subjects

with a low employment grade and who were current smokers between the total activity quartiles. Age-adjusted means of risk factors between physical activity quartiles were also assessed by using tests for linear trend with age as a covariate.

The HRV measures—SDNN, low-frequency power, and high-frequency power—were transformed by natural logarithm because their distributions were skewed. Because the activity quartiles and HRV variables were related to age and sex, the linear trend analyses were carried out separately for men and women, and means of the HRV measures were adjusted for age as a covariate. The activity quartiles were treated as a continuous variable to assess the significance of that linear term. These linear regressions were used to quantify the percentage difference in the HRV variables from none to the highest category of vigorous activity, with adjustment for age and light and moderate activity quartiles as covariates. No adjustment for BMI was made in the main analyses. Since sick people may have lower activity levels and, independently, have lower HRV, we further adjusted for self-reported general health. These trend analyses were then stratified by BMI, <25 and ≥25, with adjustment for the risk factors smoking and high alcohol intake. An interaction term for BMI and vigorous activity was fitted into the model to test whether the effect of vigorous activity differed between the two BMI categories. In this paper, the HRV values are expressed as geometric means; however, resting heart rate, which was normally distributed, is expressed as a mean with standard error.

### RESULTS

Compared with women, men reported higher levels of total activity (39.4 (standard deviation, 21.5) vs. 36.0 (standard deviation, 19.8) MET-hours per week,  $p < 0.001$ ) and vigorous activity (48 percent vs. 31 percent reported some vigorous activity,  $p < 0.001$ ). Men and women accumulated MET-hours per week via different activities. The three moderate activities reported most frequently by men were making home improvements, cleaning the car, and mowing the grass; those for women were doing laundry, vacuuming, and dusting. Men and women reported similar levels of walking (median: men, 4.5 hours; women, 4.33 hours per week) and gardening activities (median, 30 minutes per week for both men and women), although men also recorded more strenuous gardening tasks such as digging. The vigorous activities reported most frequently were jogging, gym workouts, and tennis for men and floor exercises and aerobics for women.

Table 1 shows participant characteristics by total physical activity quartile. For men, total physical activity was higher at older ages ( $p < 0.001$ ). Participants reporting high total physical activity were likely to have a higher employment grade, be nonsmokers, have a lower mean BMI and waist-hip ratio, and have higher high density lipoprotein cholesterol levels.

Table 2 shows linear trends of lower resting heart rate ( $p < 0.001$ ), higher SDNN ( $p = 0.09$ ), and higher low-frequency power ( $p < 0.01$ ) and high-frequency power ( $p < 0.05$ ) with

**TABLE 1. Age and age-adjusted risk factors,\* by total physical activity quartile, for men and women in the Whitehall II study of civil servants ( $n = 3,328$ ), London, United Kingdom, 1997–1999**

Factor and sex	Total physical activity quartile†				<i>p</i> for trend
	1 (low)	2	3	4 (high)	
No. of participants					
Men	583	584	583	584	
Women	248	250	248	248	
Age (years)					
Men	54.7	55.1	55.5	57.5	<0.001
Women	56.9	56.4	55.6	57.3	0.76
Low employment grade (%)					
Men	23.7	14.4	11.8	9.8	<0.001
Women	70.6	52.4	55.6	56.0	<0.01
Current smoker (%)					
Men	12.2	8.1	8.8	7.9	0.15
Women	14.2	14.3	10.0	8.2	<0.01
Alcohol intake (%)					
Men: $\geq 31$ units/week	13.2	16.1	13.4	15.2	0.42
Women: $\geq 21$ units/week	4.8	9.2	6.9	6.5	0.28
Systolic blood pressure (mmHg)					
Men	123.0	123.9	122.9	122.8	0.51
Women	122.4	122.1	122.0	122.2	0.74
Diastolic blood pressure (mmHg)					
Men	79.9	79.8	79.0	78.0	0.01
Women	76.4	75.6	75.2	75.4	0.36
Body mass index (kg/m <sup>2</sup> )					
Men	26.6	25.9	26.1	25.7	<0.001
Women	27.2	26.3	26.1	26.6	<0.01
Waist-hip ratio					
Men	0.941	0.928	0.920	0.915	<0.001
Women	0.820	0.799	0.798	0.793	<0.001
HDL‡ cholesterol (mmol/liter)					
Men	1.32	1.34	1.37	1.41	<0.001
Women	1.59	1.65	1.72	1.63	<0.01

\* Values are expressed as means unless stated otherwise.

† Sex-specific quartiles in metabolic equivalent-hours/week (men: <24.2, 24.2–36.09, 36.1–51.8, >51.8; women: <22.3, 22.3–34.69, 34.7–47.1, >47.1).

‡ HDL, high density lipoprotein.

increasing activity levels for men. These trends were not observed for women.

As illustrated in table 3, moderate physical activity showed a dose-response relation with lower resting heart rate and high low-frequency power, with higher levels of moderate activity reported by men, after adjustment for age and for light and vigorous activity. No associations were seen with resting heart rate or low-frequency power for women or with high-frequency power for either sex. Men and women who participated in vigorous activity had signif-

icantly lower resting heart rates, after adjustment for age and for light and moderate activity, than those who did not participate in vigorous activity. For men, a clear dose-response relation was evident between greater reported participation in vigorous activity and higher SDNN, low-frequency power, and high-frequency power ( $p < 0.05$ ,  $p < 0.01$ , and  $p < 0.01$ , respectively). This finding was not observed for women. Adjustment for smoking and high alcohol intake did not alter the results. Those men in the top category of vigorous activity had an 8 percent higher SDNN,

**TABLE 2. Age-adjusted means of heart rate variability measures, by total physical activity quartile, for men and women in the Whitehall II study of civil servants, London, United Kingdom, 1997–1999**

Measure and sex	Total physical activity quartile*				p for trend
	1 (low)	2	3	4 (high)	
No. of participants					
Men	583	584	583	584	
Women	248	250	248	248	
Resting heart rate† (beats per minute)					
Men	70.7	70.1	68.4	66.4	<0.001
Women	71.7	70.2	71.0	70.7	0.49
Standard deviation of NN intervals‡					
Men	32.9	34.7	34.5	34.8	0.09
Women	32.5	33.0	31.7	32.3	0.76
Low-frequency power‡ (milliseconds squared)					
Men	284.6	332.0	337.0	342.4	<0.01
Women	233.5	246.9	233.5	243.2	0.88
High-frequency power‡ (milliseconds squared)					
Men	104.8	118.3	116.3	125.2	<0.05
Women	133.8	146.9	129.8	141.0	0.61

\* Sex-specific quartiles in metabolic equivalent-hours/week (men: <24.2, 24.2–36.09, 36.1–51.8, >51.8; women: <22.3, 22.3–34.69, 34.7–47.1, >47.1).

† Arithmetic mean.

‡ Geometric mean.

**TABLE 3. Age-adjusted means of heart rate variability measures, by moderate physical activity quartile and vigorous activity category, for men and women in the Whitehall II study of civil servants, London, United Kingdom, 1997–1999**

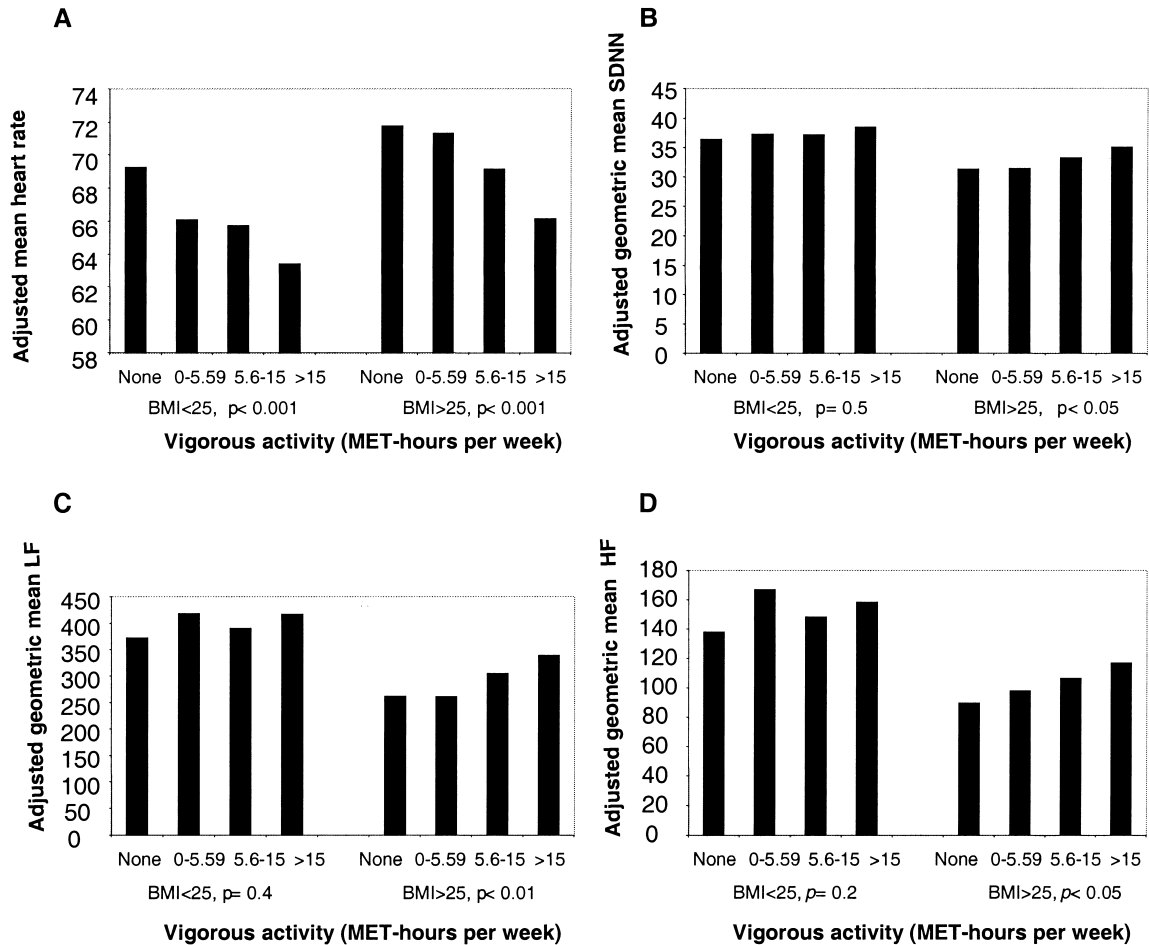
Measure and sex	Moderate activity quartile*				Test for trend p	Vigorous activity category†				Test for trend p
	1 (low)	2	3	4 (high)		None	Low	Medium	High	
Resting heart rate (beats per minute)‡										
Men	69.6	69.2	68.9	67.8	<0.05	70.6	70.0	67.0	65.0	<0.001
Women	70.6	71.4	71.3	70.2	0.53	71.4	70.1	68.5	70.5	<0.05
Standard deviation of NN interval§										
Men	33.1	34.9	34.5	34.4	0.19	33.4	34.2	35.1	36.1	<0.05
Women	33.4	32.4	31.2	32.5	0.45	32.2	31.2	32.2	34.8	0.31
Low-frequency power (milliseconds squared)§										
Men	292.7	333.0	334.0	334.6	<0.05	304.6	329.0	342.4	362.5	<0.01
Women	253.7	240.8	222.7	241.5	0.50	234.2	224.3	260.3	268.8	0.35
High-frequency power (milliseconds squared)§										
Men	107.7	119.2	117.7	119.5	0.32	107.1	123.6	124.2	131.0	<0.01
Women	142.3	141.2	127.2	141.5	0.64	134.8	141.2	131.9	163.7	0.42

\* Means were adjusted for age and for light and vigorous physical activity. Sex-specific cutpoints (men: <16.8, 16.8–25.69, 25.7–37.3, >37.3; women: <12.8, 12.8–22.19, 22.2–32.6, >32.6).

† Means were adjusted for age and for light and moderate physical activity. Sex-specific cutpoints (men: none, 0–5.59, 5.6–15, >15; women: none, 0–4.9, 5–11.7, >11.7).

‡ Arithmetic mean.

§ Geometric mean.



**FIGURE 1.** Mean heart rate variability measures, by vigorous physical activity, for men in the Whitehall II study of civil servants ( $n = 2,334$ ) stratified by body mass index (BMI; weight (kg)/height (m)<sup>2</sup>) as <25 and  $\geq 25$  and adjusted for age, light and moderate activity, smoking, and high alcohol intake, London, United Kingdom, 1997–1999. A, resting heart rate; B, standard deviation of NN intervals (SDNN); C, low-frequency (LF) power; D, high-frequency (HF) power. MET, metabolic equivalent.

19 percent higher low-frequency power, and 23 percent higher high-frequency power than those men who reported no vigorous activity, after adjustment for age and for light and moderate activity. Because physical activity and HRV may be seasonal, we adjusted for month of screening. To investigate whether illness might confound the association, we also adjusted for self-reported general health. Neither adjustment changed the results (data not shown).

As illustrated in figure 1, resting heart rate was significantly lower in those men in both the low and high BMI groups who participated in vigorous activity, after adjustment for age, light and moderate physical activity, smoking, and high alcohol intake. For men whose BMI was  $>25$ , high levels of vigorous activity were associated with HRV levels similar to those of men of low BMI who did not participate in vigorous activity. For men, the dose-response relations found between vigorous activity and the HRV variables appeared to be confined to those whose BMI was  $>25$ . However, tests for interaction between the trend in vigorous

activity and the two BMI categories indicated that the difference between these trends was nonsignificant ( $p = 0.25$  for SDNN,  $p = 0.17$  for low-frequency power, and  $p = 0.24$  for high-frequency power). Similar trends were also seen for women but reached significance with only resting heart rate ( $p < 0.05$ ) and SDNN ( $p = 0.04$ ). When stratified by BMI, the relations of moderate physical activity with resting heart rate and low-frequency power for men were no longer significant.

## DISCUSSION

For men, total leisure-time physical activity and both moderate- and vigorous-intensity activity were associated with higher HRV independent of age. Furthermore, when participants were stratified into normal-weight and overweight/obese categories, a clear dose-response relation between vigorous activity and HRV was confined to those men who were overweight or obese. To our knowledge, this

is the first large, population-based study to investigate the relation between both moderate and vigorous activity and cardiac autonomic function.

### Resting heart rate and HRV measures

Men had significantly lower resting heart rates with increasing levels of total, moderate, and vigorous activity; for women, lower heart rates were associated with increasing vigorous activity. Lower resting heart rates have been reported in athletes (20, 25, 40) and in those in the general population who participate in leisure-time sports (25). It is agreed that lower heart rates are at least partially the result of increased parasympathetic tone (19, 20, 23, 24, 33). The frequency-domain measures of HRV indicate parasympathetic and sympathetic modulations. It is unclear whether either or both an increase in parasympathetic activity and a decrease in sympathetic activity are responsible for an increase in low-frequency power. We found that low-frequency power increased with greater participation in vigorous activity, in agreement with other observational studies (26, 27), intervention studies (21, 41), and studies of athletes (19, 33, 40). Low-frequency power was also higher with increasing levels of moderate activity. One of the possible adaptations to activity is an increase in parasympathetic tone and thus an increase in the high-frequency component (23, 42). We found that vigorous activity, but not moderate activity, was associated with higher high-frequency power in men. This finding suggests that parasympathetic tone may be increased by high-intensity activity such as jogging but not more moderate activity such as walking, in common with some studies of vigorous activity (20, 27, 33, 40, 43–45), but not all (21, 26, 41). The higher parasympathetic tone, and thus higher HRV, may be as a result of improved cardiovagal baroreflex sensitivity, which is known to increase with aerobic exercise (46), or via improvement in central regulation of the autonomic outflow.

### Effect of body weight

For men who were overweight or obese, a dose-response relation was found between vigorous activity and measures of HRV. Higher levels of vigorous activity in this group of men were associated with low resting heart rates and high levels of HRV that were similar to those in men of normal weight who did not participate in vigorous activity. Some of the beneficial effects of physical activity on coronary heart disease morbidity and mortality, in particular increased physical fitness, have been reported to be independent of body weight (47). This report supports our finding that higher levels of vigorous activity, which would be reflected in higher fitness levels, are associated with higher levels of HRV in overweight men independently of the risk factors smoking and high alcohol intake. However, these men did not have a lower resting heart rate or higher HRV compared with leaner men who performed no vigorous activity. This finding suggests that, although high levels of vigorous activity may be of benefit in those who are overweight, vigorous activity may not surpass the advantages of having a low BMI. When the cohort was stratified by BMI, associa-

tions between moderate activity and HRV were no longer significant, suggesting that the effect of moderate activity on HRV could be mediated by body weight. The relation between vigorous activity and HRV for women and men whose BMI was low also seemed to be mediated by the effect of activity on body weight. However, possible influences of diet and energy intake were not examined in this study. Longitudinal research is required to determine whether vigorous activity can increase HRV in overweight people independently of the effect of body weight and diet.

### Women, physical activity, and cardiac autonomic function

For women, physical activity was associated with resting heart rate but not other measures of HRV, a finding consistent with other studies (25). Compared with men, women had lower measures of SDNN and of low-frequency power and higher heart rates and high-frequency power. These gender differences have been observed in other studies (25, 44, 48). This lack of association for women may reflect gender differences in autonomic function, with a predominance of the parasympathetic activity in women (44), so that the effect of physical activity regarding these measures is less pronounced than that in men. It could also be in part because women report lower levels of total physical activity and less vigorous activity (31 percent of women compared with 48 percent of men participated in vigorous activity) or that men and women attained MET-hours in different ways (49). The questionnaire may have been unable to assess women's activity behavior as well as men's, which might explain the lack of association found for women.

### Limitations of the study

Measuring physical activity in large, population-based studies usually relies on questionnaires, in which moderate activities such as walking and home-related tasks may be reported less accurately than vigorous activities such as jogging or swimming that are planned and structured. Such measurement error may bias results toward the null (50). Similarly, it was only practicable in this large study to obtain measures of HRV by administering a 5-minute supine electrocardiogram, but this procedure is considered representative of 24-hour ambulatory recordings (51). Direct comparison with other studies is difficult, because there is no standardized method for the frequency domain measures and therefore no universal unit (25). In a population-based study in Belgium, sports activity was significantly inversely associated with heart rate, and occupational activity was positively related to high-frequency power and inversely related to low-frequency:high-frequency ratio in 202 men (25).

### Public health importance

It is important that public health recommendations distinguish whether cardioprotective effects might extend beyond vigorous activity to include moderate activity. Increasing levels of moderate activity may be a more attainable goal than taking up vigorous exercise. With jobs becoming

increasingly sedentary, physical activities outside of work assume greater importance. Forty percent of participants who had retired were categorized in the top quartile compared with 20 percent of those who were employed ( $p < 0.001$ ); for women, the proportions were 29 percent and 23 percent, respectively ( $p = 0.09$ ). This finding suggests that retirement may be a critical time for people to increase their activity levels. Our study indicates that moderate leisure-time activities, such as walking and gardening, are associated with higher HRV, which in other studies has been associated with lower coronary heart disease risk (14, 17). Participants who reported high levels of moderate and vigorous activity were more likely to be from a high socioeconomic group, as measured by employment grade, suggesting that promotion of physical activity should be focused on lower socioeconomic groups.

Intervention studies in healthy people have concentrated on increasing fitness through programs of aerobic training, with most (21, 24) but not all (52, 53) finding an increase in HRV and a decrease in resting heart rate after substantial periods of aerobic exercise. One intervention study investigated moderate-intensity activity and, after 5 months, found no dose-response result between the moderate- and vigorous-activity intervention arms (41).

In conclusion, this cross-sectional study has shown associations between moderate and vigorous activity and HRV. Increased HRV may be an important mechanism by which physical activity reduces the risk of coronary heart disease, which may operate independently or in concert with effects on other risk factors. To determine the effects of different intensities of activity per se on HRV, longitudinal studies, together with smaller intervention studies, are needed.

## REFERENCES

1. Prentice AM, Jebb SA. Obesity in Britain: gluttony or sloth? *BMJ* 1995;311:437-9.
2. Powell KE, Blair SN. The public health burdens of sedentary living habits: theoretical but realistic estimates. *Med Sci Sports Exerc* 1994;26:851-6.
3. US Department of Health and Human Services. Physical activity and health: a report of the Surgeon General. Atlanta, GA: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, 1996.
4. Paffenbarger RS Jr, Hyde RT, Jung DL, et al. Epidemiology of exercise and coronary heart disease. *Clin Sports Med* 1984;3:297-318.
5. Powell KE, Thompson PD, Caspersen CJ, et al. Physical activity and the incidence of coronary heart disease. *Annu Rev Public Health* 1987;8:253-87.
6. Blair SN, Brodney S. Effects of physical inactivity and obesity on morbidity and mortality: current evidence and research issues. *Med Sci Sports Exerc* 1999;31(11 suppl):S646-S662.
7. Haapanen N, Miilunpalo S, Vuori I, et al. Association of leisure time physical activity with the risk of coronary heart disease, hypertension and diabetes in middle-aged men and women. *Int J Epidemiol* 1997;26:739-47.
8. Morris JN, Everitt MG, Pollard R, et al. Vigorous exercise in leisure-time: protection against coronary heart disease. *Lancet* 1980;2:1207-10.
9. Paffenbarger RS Jr, Blair SN, Lee IM, et al. Measurement of physical activity to assess health effects in free-living populations. *Med Sci Sports Exerc* 1993;25:60-70.
10. Duncan JJ, Farr JE, Upton SJ, et al. The effects of aerobic exercise on plasma catecholamines and blood pressure in patients with mild essential hypertension. *JAMA* 1985;254:2609-13.
11. Koivisto VA, Yki-Jarvinen H, DeFronzo RA. Physical training and insulin sensitivity. *Diabetes Metab Rev* 1986;1:445-81.
12. Wood PD, Stefanick ML, Williams PT, et al. The effects on plasma lipoproteins of a prudent weight-reducing diet, with or without exercise, in overweight men and women. *N Engl J Med* 1991;325:461-6.
13. Tsuji H, Venditti FJ Jr, Manders ES, et al. Reduced heart rate variability and mortality risk in an elderly cohort. The Framingham Heart Study. *Circulation* 1994;90:878-83.
14. Tsuji H, Larson MG, Venditti FJ Jr, et al. Impact of reduced heart rate variability on risk for cardiac events. The Framingham Heart Study. *Circulation* 1996;94:2850-5.
15. Algra A, Tijssen JG, Roelandt JR, et al. Heart rate variability from 24-hour electrocardiography and the 2-year risk for sudden death. *Circulation* 1993;88:180-5.
16. Dekker JM, Schouten EG, Klootwijk P, et al. Heart rate variability from short electrocardiographic recordings predicts mortality from all causes in middle-aged and elderly men. The Zutphen Study. *Am J Epidemiol* 1997;145:899-908.
17. Liao D, Cai J, Rosamond WD, et al. Cardiac autonomic function and incident coronary heart disease: a population-based case-cohort study. The ARIC Study. *Am J Epidemiol* 1997;145:696-706.
18. Boutcher SH, Nugent FW, McLaren PF, et al. Heart period variability of trained and untrained men at rest and during mental challenge. *Psychophysiology* 1998;35:16-22.
19. Yataco AR, Fleisher LA, Katzell LI. Heart rate variability and cardiovascular fitness in senior athletes. *Am J Cardiol* 1997;80:1389-91.
20. Shin K, Minamitani H, Onishi S, et al. Autonomic differences between athletes and nonathletes: spectral analysis approach. *Med Sci Sports Exerc* 1997;29:1482-90.
21. Schuit AJ, van Amelsvoort LG, Verheij TC, et al. Exercise training and heart rate variability in older people. *Med Sci Sports Exerc* 1999;31:816-21.
22. Stein PK, Ehsani AA, Domitrovich PP, et al. Effect of exercise training on heart rate variability in healthy older adults. *Am Heart J* 1999;138(3 pt 1):567-76.
23. Seals DR, Chase PB. Influence of physical training on heart rate variability and baroreflex circulatory control. *J Appl Physiol* 1989;66:1886-95.
24. Levy WC, Cerqueira MD, Harp GD, et al. Effect of endurance exercise training on heart rate variability at rest in healthy young and older men. *Am J Cardiol* 1998;82:1236-41.
25. Fagard RH, Pardaens K, Staessen JA. Influence of demographic, anthropometric and lifestyle characteristics on heart rate and its variability in the population. *J Hypertens* 1999;17:1589-99.
26. Horsten M, Ericson M, Perski A, et al. Psychosocial factors and heart rate variability in healthy women. *Psychosom Med* 1999;61:49-57.
27. Molgaard H, Hermansen K, Bjerregaard P. Spectral components of short-term RR interval variability in healthy subjects and effects of risk factors. *Eur Heart J* 1994;15:1174-83.
28. Laederach-Hofmann K, Mussgay L, Ruddel H. Autonomic cardiovascular regulation in obesity. *J Endocrinol* 2000;164:59-66.
29. Rossi M, Marti G, Ricordi L, et al. Cardiac autonomic dysfunction in obese subjects. *Clin Sci (Lond)* 1989;76:567-72.
30. Aronne L, Machintosh R, Rosenbaum M, et al. Cardiac auto-



- onomic nervous system activity in obese and never-obese young men. *Obes Res* 1997;5:354–9.
31. Hirsch J, Leibel RL, Mackintosh R, et al. Heart rate variability as a measure of autonomic function during weight change in humans. *Am J Physiol* 1991;261(6 pt 2):R1418–R1423.
  32. Zahorska-Markiewicz B, Kuagowska E, Kucio C, et al. Heart rate variability in obesity. *Int J Obes Relat Metab Disord* 1993;17:21–3.
  33. Davy KP, Miniclier NL, Taylor JA, et al. Elevated heart rate variability in physically active postmenopausal women: a cardioprotective effect? *Am J Physiol* 1996;271(2 pt 2):H455–H460.
  34. Taylor HL, Jacobs DR Jr, Schucker B, et al. A questionnaire for the assessment of leisure time physical activities. *J Chronic Dis* 1978;31:741–55.
  35. Ainsworth BE, Haskell WL, Leon AS, et al. Compendium of physical activities: classification of energy costs of human physical activities. *Med Sci Sports Exerc* 1993;25:71–80.
  36. Task force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Heart rate variability: standards of measurement, physiological interpretation and clinical use. *Circulation* 1996;93:1043–65.
  37. Acar B, Savlieva I, Hemingway H, et al. Automated ectopic beat elimination in short-term heart rate variability measurement. *Comput Methods Programs Biomed* 2000;63:123–31.
  38. Kay SM, Marple SL. Spectrum analysis—a modern perspective. *Proc IEEE* 1981;69:1380–419.
  39. Marmot MG, Bosma H, Hemingway H, et al. Contribution of job control and other risk factors to social variations in coronary heart disease. *Lancet* 1997;350:235–40.
  40. Jensen-Urstad K, Bouvier F, Saltin B, et al. High prevalence of arrhythmias in elderly male athletes with a lifelong history of regular strenuous exercise. *Heart* 1998;79:161–4.
  41. Loimaala A, Huikuri H, Oja P, et al. Controlled 5-mo aerobic training improves heart rate but not heart rate variability or baroreflex sensitivity. *J Appl Physiol* 2000;89:1825–9.
  42. Goldsmith RL, Bigger JT Jr, Steinman RC, et al. Comparison of 24-hour parasympathetic activity in endurance-trained and untrained young men. *J Am Coll Cardiol* 1992;20:552–8.
  43. Goldsmith RL, Bigger JT Jr, Bloomfield DM, et al. Physical fitness as a determinant of vagal modulation. *Med Sci Sports Exerc* 1997;29:812–17.
  44. Rossy LA, Thayer JF. Fitness and gender-related differences in heart period variability. *Psychosom Med* 1998;60:773–81.
  45. Dixon EM, Kamath MV, McCartney N, et al. Neural regulation of heart rate variability in endurance athletes and sedentary controls. *Cardiovasc Res* 1992;26:713–19.
  46. Monahan KD, Dinunno FA, Tanaka H, et al. Regular aerobic exercise modulates age-associated declines in cardiovagal baroreflex sensitivity in healthy men. *J Physiol* 2000;529:263–71.
  47. Blair SN, Brodney S. Effects of physical inactivity and obesity on morbidity and mortality: current evidence and research issues. *Med Sci Sports Exerc* 1999;31:S646–662.
  48. Huikuri HV, Pikkujamsa SM, Airaksinen KE, et al. Sex-related differences in autonomic modulation of heart rate in middle-aged subjects. *Circulation* 1996;94:122–5.
  49. Ainsworth BE. Issues in the assessment of physical activity in women. *Res Q Exerc Sport* 2000;71(2 suppl):S37–S42.
  50. Rennie KL, Wareham NJ. The validation of physical activity instruments for measuring energy expenditure: problems and pitfalls. *Public Health Nutr* 1998;1:265–71.
  51. Bigger JT, Fleiss JL, Rolnitzky LM, et al. The ability of several short-term measures of RR variability to predict mortality after myocardial infarction. *Circulation* 1993;88:927–34.
  52. Boutcher SH, Stein P. Association between heart rate variability and training response in sedentary middle-aged men. *Eur J Appl Physiol Occup Physiol* 1995;70:75–80.
  53. Davy KP, Willis WL, Seals DR. Influence of exercise training on heart rate variability in post-menopausal women with elevated arterial blood pressure. *Clin Physiol* 1997;17:31–40.