Regular physical activity attenuates the blood pressure response to public speaking and delays the development of hypertension

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Objectives **The objective of this study was to investigate the effect of regular physical activity on the haemodynamic response to public speaking and to evaluate the long-term effect of exercise on development of hypertension.**

Participants **We assessed 75 sedentary and 44 active participants screened for stage 1 hypertension with consistent activity habits and 63 normotensive individuals as control.**

Methods **The blood pressure (BP) response to public speaking was assessed with beat-to-beat noninvasive recording. Definition of incident hypertension was based either on clinic or 24-h BP measurement.**

Results **The BP response to public speaking was greater in the hypertensive than the normotensive participants ($P = 0.018/0.009$). Among the former, sedentary participants showed increased BP reactivity to the speech test ($45.2 \pm 22.6/22.2 \pm 11.5$ mmHg, $P < 0.01/<0.001$ versus controls), whereas physically active participants had a response similar to that of controls ($35.4 \pm 18.5/18.5 \pm 11.5$ mmHg, $P =$ not significant). During a median follow-up of 71 months, ambulatory BP did not virtually change in the active participants ($-0.9 \pm 7.8/-0.0 \pm 4.7$ mmHg) and increased in their sedentary peers ($2.8 \pm 9.8/3.2 \pm 7.4$ mmHg, $P = 0.08/0.003$ versus active). Active participants were less likely to develop incident hypertension than sedentary ones. After controlling for several confounders including baseline heart rate, the hazard ratio was 0.53 [95% confidence interval (CI) 0.31–0.94] for clinic hypertension and 0.60 (95% CI 0.37–0.99) for ambulatory hypertension. Inclusion of BP response to public speaking into the Cox model influenced the strength of the association only marginally [hazard ratio = 0.55 (95% CI 0.30–0.97) and hazard ratio = 0.59 (95% CI 0.36–0.99), respectively].**

Conclusion **Regular physical activity attenuates the BP reaction to psychosocial stressors. However, this mechanism seems to be only partially responsible for the long-term effect of exercise on BP. *J Hypertens* 28:1186–1193 © 2010 Wolters Kluwer Health | Lippincott Williams & Wilkins.**

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Abbreviations: BP, blood pressure; CIs, two-sided 95% confidence intervals; HARVEST, Hypertension and Ambulatory Recording Venetia Study

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Introduction

A number of studies [1–4] have shown that cardiovascular reactivity to tasks such as mental arithmetic, stroop colour test, or psychosocial challenges predicts future values of blood pressure (BP) and risk of hypertension, and that exaggerated stress reactivity may contribute to the development of cardiovascular disease. It is thus important to understand how to effectively reduce stress reactivity before cardiovascular complications develop. Aerobic fitness has been found to be associated with reduction in cardiovascular reactivity to psychophysiological stressors [5], but a recent review of 73 studies failed to demonstrate a significant effect of physical fitness on reactivity [6]. The conflicting results of previous studies may be due to the different stressors employed in the various laboratories. It is known that the nature of the stressor may be of critical importance in determining the cardiovascular response to stress because the amount of 'stress' mainly depends on the participant's degree of involvement in the particular task [7,8]. The tests currently used to assess BP reactivity in the laboratory, such as mental arithmetic, stroop colour test, or mirror tracing, can hardly reflect stressful situations of daily life [6]. Recent studies performed with the public speaking test, a socially salient stressor that involves interpersonal components and elicits a strong β-adrenergic response, have shown that regular physical activity can reduce the heart rate (HR) [9,10] and BP [10,11] responses to stressful situations both in children [10] and adults [9,11]. The stress-buffering effect of exercise may be more important

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in patients potentially hyperreactive to stress, such as young individuals in the early stage of hypertension.

Because of the unresolved controversy and potential public health importance of the topic, we investigated the BP and HR responses to public speaking in a group of young hypertensive patients screened for stage 1 hypertension stratified according to whether they were sedentary or physically active and compared them with those in a group of normotensive controls. To establish whether there was an association of decreased haemodynamic response to stress and physical conditioning with future BP, in the hypertensive patients, we examined the long-term risk of developing hypertension.

Methods

Study population

The present study involved 119 patients screened for stage 1 hypertension aged 18–45 years from the Hypertension and Ambulatory Recording Venetia Study (HARVEST) [12,13]. The individuals taking part in this subproject were all those recruited at the Padova Center whose physical activity habits were consistent throughout the period of observation. Patients with diabetes mellitus, nephropathy, and cardiovascular disease were excluded. Patients' clinical characteristics are reported in Table 1. The higher prevalence of men among our study participants confirms previous observations of a much higher prevalence of men in the young-to-middle-age segment of the hypertensive population [14]. Patients' recruitment was obtained with the collaboration of the local general practitioners who were instructed during local meetings. Consecutive patients with the above-mentioned clinical characteristics seen in the offices of the general practitioners and willing to participate in the study were eligible for recruitment and were sent to the referral Center. Sixty-three normotensive individuals of similar age and sex distribution served as controls for the speech test study. All were asymptomatic, had no history of cardiovascular disease, and were normal at physical examination. Standard ECG, blood chemistry, and urinalysis were normal in all of the individuals. Their BP measured six times over a 2-week period was always lower than 140/90 mmHg.

Procedures

The procedures followed were in accordance with the institutional guidelines. All individuals underwent physical examination, anthropometry, blood chemistry, and urine analysis. Participants completed questionnaires about their medical history, family history of hypertension, physical activity, and dietary habits including coffee intake, alcohol use, and cigarette smoking. Categorization of lifestyle factors in the present study was based on previously published data, showing the effects of various types of lifestyle factor categorization on BP in a similar population of North-east Italy [12,15]. Physical activity was assessed using a standardized questionnaire [12,15]. Briefly, activities were classified on the basis of relative intensity, adapting the activity intensity codes established and validated by the Minnesota Heart Survey [16]. Participants were categorized as sedentary if they did not regularly perform sports activity and active if they performed sports, such as running, jogging, cycling, swimming, soccer, tennis, and so on, at least once a week during the previous 2 months [17]. Smoking habits were categorized as nonsmokers (class 0) and smokers (one or more cigarettes per day, class 1). Coffee consumption was categorized according to the number of caffeine-containing cups of coffee drunk per day: nondrinkers (0 cups/day), moderate drinkers (one to three cups/day), and heavy drinkers (four or more cups/day). Alcohol intake was calculated by summing the total number of millilitres

Table 1 Clinical characteristics of the study participants by physical activity status

		Physical activity		*P*	
Variable	Total ($n=119$)	Sedentary ($n=75$)	Active ($n=44$)	Unadjusted	Adjusted for age and sex
Female sex, *n* (%)	21 (17.6)	17 (23%)	4 (9%)	0.06	–
Age (years)	29.1 ± 8.9	30.2 ± 8.6	27.3 ± 9.2	NS	–
BMI (kg/m^2)	24.4 ± 3.3	24.4 ± 3.6	24.4 ± 2.8	NS	NS
Baseline SBP (mmHg)	143.4 ± 9.8	142.4 ± 9.5	144.9 ± 10.3	NS	NS
Baseline DBP (mmHg)	90.8 ± 6.0	91.1 ± 5.2	90.3 ± 7.3	NS	NS
Baseline heart rate (bpm)	73.4 ± 9.4	75.8 ± 10.1	69.5 ± 6.7	0.0001	0.0001
24-h SBP (mmHg)	130.8 ± 10.1	129.4 ± 10.5	133.1 ± 9.1	0.051	NS
24-h DBP (mmHg)	79.3 ± 7.4	79.9 ± 7.5	78.2 ± 7.3	NS	NS
24-h heart rate (bpm)	71.8 ± 8.7	73.4 ± 8.0	68.9 ± 9.1	0.007	0.02
White-coat effect, SBP (mmHg)	12.5 ± 11.0	12.9 ± 11.3	11.9 ± 10.7	NS	NS
White-coat effect, DBP (mmHg)	11.5 ± 7.3	11.1 ± 7.2	12.1 ± 7.4	NS	NS
Parental hypertension, *n* (%)	75 (63.6)	47 (63.5)	28 (63.6)	NS	–
Cigarette smokers, *n* (%)	23 (19.3)	16 (21.3)	7 (15.9)	NS	–
Alcohol drinkers, *n* (%)	41 (34.4)	25 (33.3)	16 (36.4)	NS	–
Coffee drinkers, *n* (%)	84 (70.6)	57 (76.0)	27 (61.4)	NS	–
Follow-up change in clinic SBP (mmHg)[a]	−4.5 ± 14.1	−2.2 ± 14.5	−8.3 ± 12.6	0.018	0.052
Follow-up change in clinic DBP (mmHg)[a]	−1.1 ± 9.2	0.5 ± 8.9	−3.9 ± 9.1	0.013	0.006
Follow-up change in 24-h SBP (mmHg)[a]	1.4 ± 9.2	2.8 ± 9.8	−0.9 ± 7.8	0.024	0.082
Follow-up change in 24-h DBP (mmHg)[a]	2.0 ± 6.7	3.2 ± 7.4	−0.0 ± 4.7	0.005	0.003

Values are given as mean ± SD unless otherwise specified. BP, blood pressure; NS, not significant. [a]Adjusted also for baseline BP and time.

of daily alcohol consumption of wine, beer, and liqueurs according to the formula of Criqui *et al.* [18]. Participants were then categorized as nondrinkers (class 0), light drinkers (less than 50 g/day, class 1), and moderate-to-heavy drinkers (50 g or more of alcohol/day, class 2). Details about the interview, lifestyle assessment, and criteria used for participants' classification according to lifestyle were reported elsewhere [12,15]. Baseline BP was the mean of six readings obtained during two visits performed 2 weeks apart. BMI was considered as an index of adiposity (weight divided by the height squared). The study was approved by the Ethics Committee of the University of Padova, and a written informed consent was given by the participants.

Blood pressure measurement

At each visit, office BP was measured in triplicate in the supine position according to the recommendations of the British Society of Hypertension. In the hypertensive patients, 24-h ambulatory BP monitoring was performed with the A&D TM-2430 (A&D Co., Ltd., Tokyo, Japan) or the ICR SpaceLabs 90207 (SpaceLabs Inc., Redmond, Washington, USA). The procedures used for the validation and the application of the devices have been described elsewhere [12]. During the recordings, patients were asked to engage in their ordinary daily activities, to keep a diary of them, and to go to bed not later than 2300 h. BP was measured every 15 min during the waking hours and every 30 min at night. The ambulatory measurement was repeated at the end of the study.

To assess the BP response to public speaking, BP was monitored with a finger device (Finometer; Finapres Medical Systems, Amsterdam, The Netherlands), which provides beat-to-beat BP measurements. This technique correlates closely with direct intra-arterial measurements [19]. The finger BP recording was performed with the patient in the semi-recumbent position on an arm chair with the head elevated about 30° from horizontal. The cuffed hand was positioned at the heart level. The equipped arm of the patient was held in the same position, and BP was recorded continuously throughout the laboratory examination.

Speech test

The assessment of the BP reaction to public speaking was assessed in an out-patient clinic and was embedded in the larger clinical evaluation [20]. All patients were naive to the applied stress procedure, and none had an experience with speaking in public. Participants were asked to refrain from drinking alcohol or beverages containing caffeine and from smoking for at least 24 h before the study. The test was performed in the afternoon in a quiet room with a temperature of approximately 22°C. The test procedure was explained to the patients in the laboratory room. Participants were given instructions to prepare a speech on an assigned topic covering specific points. They were instructed to talk as if actually involved in the situations and to express their feelings. Participants had 3 min to mentally prepare their speech, after which they delivered their speech for 3 min in front of a group of observers unknown to them who had to evaluate their performance. The speech test was followed by a 3-min recovery period. Although the participants were in a supine position, the Finometer cuff was applied to their finger and the device was calibrated. Participants rested quietly for the first 10 min of the testing to establish an average of resting baseline BP. To calculate the BP and HR reaction to public speaking, the beat-to-beat readings obtained with the device were averaged. The BP response to speech test was calculated from the difference between the BP recorded during the test and the initial resting period. To compute this difference, we used the last 5 min of the initial baseline recording and the whole period of the test, which in a previous study showed better correlations with other clinical variables than the maximum BP changes during the test [20]. A similar procedure was used to calculate the BP change during recovery.

Follow-up

Office BP was assessed monthly during the first 3 months of follow-up, then after 6 months, and every 6 months thereafter. Participants were followed until they developed hypertension requiring antihypertensive treatment according to the guidelines available at the time. To identify the participants with hypertension needing antihypertensive treatment, we followed the guidelines of the British Hypertension Society until 1999 [21,22], the 1999 World Health Organization/International Society of Hypertension (WHO/ISH) guidelines until 2003 [23], and the 2003 and 2007 European Society of Cardiology/European Society of Hypertension (ESC/ESH) guidelines thereafter [24,25]. Participants who did not meet the criteria for treatment were checked at 6-month intervals unless they dropped-out. If the BP level was above the operational threshold level at follow-up visits, the patient was rescheduled for a visit within 2–4 weeks. If BP was still above the limit, the patient was given antihypertensive drug treatment, otherwise the patient was checked at monthly intervals. All data used for the present analysis were collected before starting the antihypertensive therapy.

Definitions

In the present study, clinic hypertension was defined as an average SBP of at least 140 mmHg and/or a DBP of at least 90 mmHg from two consecutive visits occurring after at least 6 months of observation [26]. Ambulatory hypertension was defined as an average 24-h SBP of at least 130 mmHg and/or 24-h DBP of at least 80 mmHg [25]. The white-coat effect was calculated from the difference between clinic and average 24-h BP at baseline assessment.

Data analysis

Differences between groups (sedentary versus active) were assessed with the *t*-test for the variables normally

distributed. Data were adjusted for age and sex by use of linear regression analysis. The significance of differences in categorical variables was assessed with the χ^2 test and the Fisher exact test. Follow-up changes in BP were evaluated using a general linear model, with the value for the measurement of interest serving as the dependent variable and physical activity group, age, sex, baseline BP, and follow-up length serving as independent variables. To compare the normotensive individuals with the two groups of hypertensive patients, the analysis of covariance (ANCOVA) test and the Tukey–Kramer multiple comparisons post-hoc test were used. The risk of hypertension related to physical activity status was assessed in multivariable Cox proportional hazards models adjusting for sex, age, BMI, family history for hypertension, smoking status, alcohol consumption, coffee use, and baseline BP. Other explanatory variables included in the models were BP and HR changes during public speaking. No violations to the proportional hazards assumption were detected by inspection of survival curves. Estimates of hazard ratio and corresponding two-sided 95% confidence intervals (CIs) relating physical activity status to risk of hypertension were computed from the Cox models. A two-tailed probability value of less than 0.05 was considered significant. Data are presented as mean ± SD, unless specified, or as percentages. Analyses were performed using Systat versions 10 and 11 (SPSS Inc., Evanston, Illinois, USA).

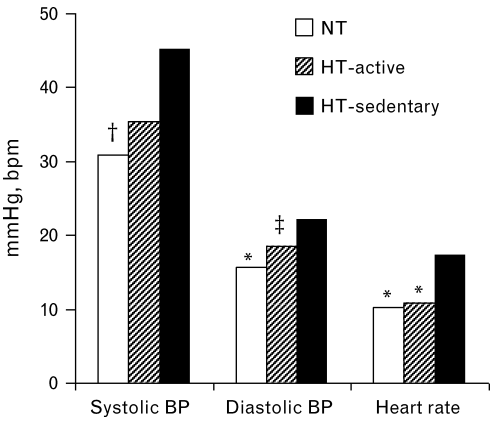
Results

The hypertensive patients were slightly younger than the normotensive individuals of control (29.1 ± 8.8 versus 31.6 ± 6.8 years, $P = 0.06$), while no difference was observed for sex distribution (men, 82.3 versus 76.7%, $P = 0.36$) or BMI (24.6 ± 3.3 versus 24.3 ± 2.6 kg/m^2, $P = 0.76$). Clinic SBP and DBP both at baseline (143.4 ± 9.8/90.8 ± 6.0 versus 124.1 ± 9.5/70.9 ± 9.0 mmHg, $P < 0.001$/<0.001) and before the speech test (140.3 ± 21.2/80.9 ± 10.3 versus 125.8 ± 13.2/71.5 ± 8.7 mmHg, $P < 0.001$/<0.001) were higher in the former. Among the hypertensive patients, the number of patients categorized as sedentary was 75 and of those categorized as active was 44 (22 amateurs and 22 competitive athletes). Clinical characteristics of the sedentary versus active individuals are shown in Table 1. Active individuals were slightly younger than sedentary ones, were more frequently male, and had lower clinic and 24-h HR. After adjustment for age and sex, baseline BP, either measured in the clinic or with ambulatory monitoring, did not differ significantly between the groups. Also, no between-group difference was found for the systolic and diastolic white-coat effects. When individuals were re-evaluated before the speech test, the clinic BP difference between the two groups increased, though not to a significant level, as SBP was 141.4 ± 22.7 mmHg in the sedentary and 138.4 ± 18.4 mmHg in the active individuals (age and-sex-adjusted $P = 0.21$), and DBP was 81.5 ± 11.2 and 79.9 ± 8.6 mmHg, respectively (age and-sex-adjusted $P = 0.20$).

Blood pressure and heart rate response to public speaking

During the speech test, BP and HR rose in all of the individuals, and the BP increase was greater in the hypertensive (42.0 ± 21.7/21.0 ± 11.9 mmHg) than the normotensive (31.2 ± 17.3/16.0 ± 7.8 mmHg) individuals (age and-sex-adjusted $P = 0.018$/0.009). Also, the increase in HR was greater in the hypertensive patients [15.4 ± 12.4 versus 10.5 ± 7.8 beats/min (bpm), $P = 0.016$]. The BP and HR responses to the laboratory test in the normotensive individuals and the hypertensive patients divided according to whether they were sedentary or physically active are shown in Fig. 1. The sedentary hypertensive patients exhibited a larger reaction to the speech test than the normotensive individuals of control or the active hypertensive individuals (Fig. 1 and Table 2). Differences were highly significant especially for HR. Similar results were observed during the recovery period. The BP increase from baseline in SBP (23.4 ± 19.2 versus 12.9 ± 10.2 mmHg, $P = 0.0005$), DBP (11.5 ± 10.5 versus 6.7 ± 6.6 mmHg, $P = 0.005$), and HR (3.7 ± 10.1 versus 0.5 ± 4.3 bpm, $P = 0.07$) was greater in the hypertensive than in the normotensive individuals. Among the hypertensive patients, all changes were greater in the sedentary than in the active individuals (Fig. 2, and Table 2). The differences in the BP reaction to the speech test and the recovery period between the sedentary and active individuals remained significant also after adjustment for baseline HR (Table 2).

Fig. 1



Blood pressure and heart rate reaction to public speaking (test: baseline) in the hypertensive patients divided according to physical activity status and a group of normotensive individuals of control. Data are adjusted for age, sex, and baseline blood pressure or heart rate., BP, blood pressure; HT-active, physically active hypertensive patients; HT-sedentary, sedentary hypertensive patients; NT, normotensive individuals of control. *$P < 0.001$, †$P < 0.01$, ‡$P < 0.05$ versus HT-sedentary.

Table 2 Blood pressure and heart rate changes during the speech test and the recovery period in the hypertensive patients grouped by physical activity status

Variable	Sedentary	Active	Adjusted *P*	*P* adjusted also for baseline heart rate
n	75	44		
Speech test				
SBP increase (mmHg)	45.2 ± 22.6	35.4 ± 18.5	0.004[a]	0.004
DBP increase (mmHg)	22.2 ± 11.5	18.5 ± 11.5	0.039[a]	0.039
Heart rate increase (bpm)	17.3 ± 13.4	10.9 ± 10.4	0.0007[b]	NA
Recovery				
SBP increase (mmHg)	26.1 ± 20.3	18.9 ± 16.6	0.006[a]	0.005
DBP increase (mmHg)	12.2 ± 10.6	10.3 ± 10.5	NS[a]	NS
Heart rate increase (bpm)	5.0 ± 10.8	1.5 ± 8.6	0.003[b]	NA

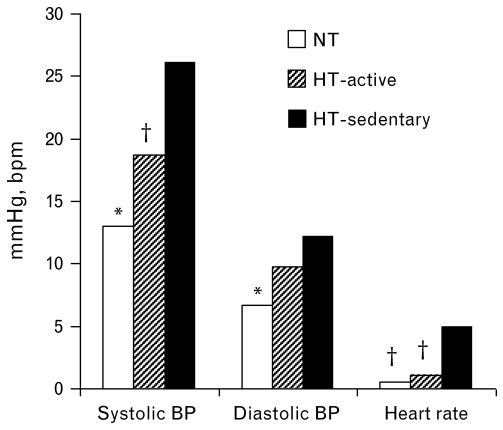
BP, blood pressure; NA, not applicable; NS, non-significant. [a]Adjusted for age, sex, BMI, parental hypertension, smoking, alcohol, coffee use, and for baseline BP. [b]Adjusted for age, sex, BMI, parental hypertension, smoking, alcohol, coffee use, and for baseline heart rate.

Follow-up

In the present study, median follow-up for the whole hypertensive cohort was 71 (interquartile range 33–140) months. Within the first 3 months of observation, clinic BP declined by 3.1 ± 20.1/9.9 ± 11.9 mmHg in the whole hypertensive group. A SBP of less than 140 mmHg and a DBP of less than 90 mmHg were found in 53.6% of the individuals before the accomplishment of the speech test. At follow-up end, both clinic HR (70.9 ± 10.7 versus 75.3 ± 10.3 bpm, $P = 0.03$ adjusted for age and sex) and 24-h HR (68.6 ± 9.6 versus 73.6 ± 7.9 bpm, $P = 0.007$) were still lower in the active than in the sedentary hypertensive patients. During the follow-up, there was a decline in clinic BP among the active individuals and little or no change among the sedentary ones (Table 1). In the active group, there was a slight decline in ambulatory SBP and no change in ambulatory DBP, whereas among the sedentary individuals, both systolic and diastolic BPs increased during the period of observation (Table 1). Body weight increased in both active and sedentary individuals (2.6 ± 5.2 and 2.5 ± 8.4 kg, respectively) without significant difference between the two groups ($P = 0.95$).

The frequency of incident hypertension during the follow-up was 58.8% when it was defined according to clinic BP measurement and 76.5% when it was diagnosed with the ambulatory measurement. The risk of clinic hypertension was statistically significantly smaller in the active individuals compared with the sedentary ones in fully adjusted Cox models (hazard ratio = 0.49, 95% CI 0.27–0.89, $P = 0.019$). Inclusion of baseline HR in the model slightly reduced the strength of the association (hazard ratio = 0.53, 95% CI 0.31–0.94, $P = 0.028$). Also, ambulatory hypertension was developed less frequently by the active individuals (hazard ratio = 0.56, 95% CI 0.33–0.95, $P = 0.029$, and HR = 0.60, 95% CI 0.37–0.99, $P = 0.044$, respectively). Inclusion of changes in BP and HR during public speaking in the models affected the strength of the association between physical activity status and risk of hypertension only marginally (Tables 3 and 4). We evaluated the prediction of the mental preparation, of the speech test, and of the mean of the two periods. The

Fig. 2



Blood pressure and heart rate changes during the recovery period (recovery: baseline) in the hypertensive patients divided according to physical activity status and a group of normotensive individuals of control. Data are adjusted for age, sex, and baseline blood pressure or heart rate. BP, blood pressure; HT-active, physically active hypertensive patients; HT-sedentary, sedentary hypertensive patients; NT, normotensive individuals of control. *$P < 0.001$, †$P < 0.01$ versus HT-sedentary.

Table 3 Hazard ratios and 95% confidence interval for development of clinic hypertension according to a multivariable Cox model

Variable	Hazard ratio (95% CI)	*P*
Physical activity (yes/no)	0.55 (0.30–0.97)	0.039
SBP change during the speech test (mmHg)[a]	1.03 (1.00–1.06)	0.050
BMI (kg/m^2)	1.10 (1.02–1.20)	0.019
Sex (female)	0.91 (0.40–2.03)	0.82
Age (years)	1.03 (0.99–1.06)	0.10
Parental hypertension (yes/no)	1.04 (0.71–1.51)	0.84
Smoking (yes/no)	1.07 (0.76–1.49)	0.70
Alcohol drinking (yes/no)	1.33 (0.79–1.63)	0.18
Coffee use (0, 1–3, >3 cups)	1.19 (0.70–2.02)	0.67
Baseline SBP (mmHg)	1.01(0.98–1.03)	0.50
Baseline DBP (mmHg)	0.99 (0.95–1.03)	0.68
Baseline Heart rate (bpm)	1.02 (0.99–1.04)	0.16
DBP change during the speech test (mmHg)	0.97(0.92–1.02)	0.25
Heart rate change during the speech test (bpm)	0.98 (0.95–1.01)	0.18

CI, confidence interval. [a]Mean of the whole test period (mental preparation and speech).

Table 4 Hazard ratios and 95% confidence interval for development of ambulatory hypertension according to a multivariable Cox model

Variable	Hazard ratio (95% CI)	*P*
Physical activity (yes/no)	0.59 (0.36–0.99)	0.045
SBP change during the speech test (mmHg)[a]	1.02 (1.00–1.05)	0.069
BMI (kg/m^2)	1.08 (1.00–1.16)	0.057
Sex (female)	0.92 (0.45–1.90)	0.83
Age (years)	1.02 (0.98–1.05)	0.32
Parental hypertension (yes/no)	1.04 (0.71–1.51)	0.84
Smoking (yes/no)	1.14 (0.83–1.56)	0.41
Alcohol drinking (yes/no)	1.00 (0.61–1.64)	0.98
Coffee use (0, 1–3, >3 cups)	0.77 (0.48–1.25)	0.29
Baseline SBP (mmHg)	1.02 (0.85–1.22)	0.80
Baseline DBP (mmHg)	0.99 (0.95–1.03)	0.53
Baseline heart rate (bpm)	1.01(0.99–1.03)	0.23
DBP change during the speech test (mmHg)	0.98 (0.94–1.02)	0.35
Heart rate change during the speech test (bpm)	0.99 (0.96–1.01)	0.23

CI, confidence interval. [a]Mean of the whole test period (mental preparation and speech).

SBP response to the laboratory stressor showed a weak association with outcome in all models. For preparation, the univariate *P* value was 0.09 for clinic hypertension and 0.057 for ambulatory hypertension. The multivariable *P* values were 0.046 and 0.071 for clinic hypertension and for ambulatory hypertension, respectively. For speech, the univariate *P* value was 0.09 for clinic hypertension and 0.057 for ambulatory hypertension. The multivariable *P* values were 0.067 and 0.069 for clinic hypertension and for ambulatory hypertension, respectively. For the whole test period (preparation and speech), the univariate *P* value was 0.10 for clinic hypertension and 0.054 for ambulatory hypertension. The multivariable *P* values were 0.050 and 0.069 for clinic hypertension and for ambulatory hypertension, respectively (Tables 3 and 4).

Discussion

In this homogeneous cohort of participants screened for stage 1 hypertension, those who performed regular physical activity had a smaller BP reaction to public speaking and a lower risk of developing hypertension than the sedentary ones. After a median follow-up of 71 months, ambulatory BP remained virtually unchanged among the participants who engaged in sports activities, whereas it increased among the sedentary ones. The favourable effect of exercise was independent of several confounding factors, including BP, HR, and BMI at baseline and follow-up changes in body weight.

Physical activity and cardiovascular response to stress exposure

There are a number of important factors related to lifestyle that are independently associated with a greater risk of hypertension including psychosocial factors [2–4,27,28], which are also known to be affected by exercise [6,9–11]. A reduction of BP responsiveness to stressors has been described in physically active individuals [5,11], which may be involved with the BP-lowering effect of regular physical activity. However, previous studies of the relationship between physical conditioning and stress reactivity provided conflicting results. Recent reviews have shown that there was no effect of physical activity on reactivity in studies that used the stroop colour-word conflict test, and that fitness was even associated with small increases in reactivity for mental arithmetic [6]. In patients with hypertension or parental hypertension, cardiorespiratory fitness was found to be positively related to cardiovascular reactivity to active and passive stressor tasks [29–31]. Inconsistencies among previous studies are likely to be due to different experimental conditions and, in particular, to the lack of standardization of the type of stressor employed by the investigators. The tests more often used to assess BP reactivity in the laboratory were mental arithmetic, stroop colour test, or mirror tracing. These laboratory tasks elicit weaker haemodynamic and sympatho-adrenal medullary responses than can be elicited by psychosocial stressors. Al'Absi *et al.* [32] found a much greater and consistent cardiovascular and neuroendocrine activation during three public speaking presentations than during three sessions of mental arithmetic. Similar results were obtained by other authors [33]. Simulated public speaking incorporates several challenging components, including the emotional load of the topics discussed, fear of a poor performance, and the demand of maintaining poise and control in front of the audience [32,34]. Only a few recent studies did evaluate the effects of regular aerobic exercise on the BP and HR responses to public speaking, showing blunted reactivity in trained individuals [9–11].

In the present study, the favourable effect of exercise on BP reactivity to stress was also documented during the recovery period. Little attention has been placed by previous investigators on studying recovery from stress. Delayed recovery after stress has been reported in individuals with a parental history of hypertension [35], and low levels of fitness have been associated with delayed recovery after stress in hypertensive patients [36]. The greater BP increase during recovery in our sedentary individuals is in agreement with those previous findings.

Physical activity and risk of hypertension

A large number of studies have shown a significant effect of regular aerobic exercise on BP, with net reductions of resting and ambulatory daytime systolic and diastolic BPs of 3.0/2.4 and 3.3/3.5 mmHg, respectively [37]. However, the above results were obtained in short-term intervention studies performed in small groups of individuals who were sedentary at the time of baseline investigation. Little is known about the long-term effects of exercise on BP in physically active individuals with mildly elevated BP at baseline assessment. In our study, we had the opportunity of tracking the natural history of BP over the long term in the absence of the confounding

effect of antihypertensive treatment. At baseline, active participants had similar clinic BP and ambulatory BP to those of their sedentary counterpart because they had been selected on the basis of their initial BP level. However, during the follow-up, ambulatory BP increased only in the sedentary individuals, and sedentary lifestyle was associated with a doubled risk of hypertension. The greater follow-up decrease in clinic BP observed in the active group was not the consequence of a more pronounced alarm reaction at initial examination because the white-coat effect was similar in the active and the sedentary groups at baseline. According to the international guidelines, individuals with low cardiovascular risk profile and BP within the stage 1 hypertensive range, such as those enrolled in the present study, should be monitored for extended periods with only nonpharmacological treatment [21–25]. The current data provide important confirmation of this recommendation because in many of our participants, clinic BP decreased to below 140/90 mmHg during the first months of follow-up and remained below that level for several years.

Possible mechanisms that link the effect of exercise on stress reactivity to risk of hypertension

Similarities between central and peripheral cardiovascular responses to exercise and psychosocial stressors have lead to the theory of 'cross-stressor adaptation'. According to this view, adaptations to repeated bouts of exercise can also lead to adaptations to daily life stressors [38–40]. The increase in BP during stressful situations is primarily due to elevation in cardiac output with little changes in peripheral resistance, suggesting that the BP response to mental stress is mediated by central neural modulation of the β-adrenergic system [41,42]. Physical fitness has been shown to modulate autonomic [41,43] responses to stressors. Although not all studies have been positive [44], chronic aerobic exercise can result in decreased cardiac–sympathetic drive, in part, by enhancing baroreceptor sensitivity [45,46]. Central autonomic adaptations in response to regular physical activity would thus be responsible for the reduced BP responsiveness to psychological stress [41,42] and might also represent a mechanistic pathway for the decreased tendency to develop hypertension in physically active individuals [1–3]. However, in the present study, inclusion of baseline HR in the ANCOVA models caused only negligible changes in the level of statistical significance, suggesting that autonomic adaptation may not be the only mechanism for the effect of regular exercise training on BP reactivity to stressors.

In the present study, the BP response to the psychological challenge was a weak predictor of future hypertension, irrespective of whether we considered the mental preparation period, the speech period, or the average of the two periods as a predictor of outcome (P values between 0.045 and 0.071 in the multivariable regressions). When the BP responses to public speaking were incorporated in the Cox models, the association of physical activity status with incident hypertension remained significant. This again suggests that the favourable effect of regular aerobic exercise on future BP was only partially due to central autonomic modulation, and that also other mechanisms were operative. Among these, enhanced endothelial vasodilator function, suppression of the activity of the renin–angiotensin–aldosterone system, and reduction of insulin resistance are well known mechanisms by which exercise training may prevent or delay the onset of hypertension [37]. An effect of exercise-related weight reduction on BP has been demonstrated in many studies [37]. However, in the present study, baseline BMI did not differ according to physical activity status, and the slight increase in body weight during the follow-up was of similar magnitude in the active and the sedentary individuals.

Study limitations

A limitation of our investigation is that our definition of physical activity status was based on patients' interview. Therefore, misclassification must be considered. However, the significant between-group difference in clinic and ambulatory HRs at baseline and follow-up ends corroborates the validity of our classification and suggests that activity status was consistent during the follow-up. In addition, previous data have shown that reactivity effects related to training were similar when fitness was measured directly or indirectly [6].

Another possible limitation is that the stress reactivity study was cross-sectional in nature. Cross-sectional comparisons of physical activity groups do not permit causal inferences, but they typically yield greater differences in fitness than the changes in fitness feasibly obtained during short-term exercise training [47].

In conclusion, our study demonstrates that exercise has favourable effects on BP reactivity to psychological stressors and on the risk of developing hypertension. Whether and to what extent the long-term effect of exercise on BP was mediated by the attenuated response to stress is difficult to say. However, the present findings support a strategy of exercise training as an initial approach in the management of young sedentary patients in the early stage of hypertension, chiefly in those hyperreactive to psychosocial tasks. Physically active people should be encouraged to continue their exercise programmes, irrespective of whether they perform leisure-time or competitive athletics.

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