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Original Article

Effect of Physical Activity on the Prevalence of Metabolic Syndrome and Left Ventricular Hypertrophy in Apparently Healthy Adults

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Background/Purpose: Metabolic syndrome and left ventricular hypertrophy (LVH) carry high cardiovascular risks. We performed a cross-sectional study to evaluate the effect of different amounts of physical activity (PA) on the prevalence of metabolic syndrome and LVH in our study population.

Methods: This study was a cross-sectional survey of 1494 apparently healthy subjects: 776 men with a mean age of 57.6 ± 12.3 years, and 718 women with a mean age of 56.4 ± 11.0 years. The metabolic syndrome was defined according to modified criteria of the National Cholesterol Education Program Adult Treatment Panel III. LVH was diagnosed by electrocardiography voltage criteria. The amount of PA was determined with a questionnaire and stratified into low, moderate or high levels.

Results: The prevalence of metabolic syndrome and its components was as follows: metabolic syndrome, 15.5%; obesity, 29.7%; high triglyceride level, 21.7%; low high-density lipoprotein–cholesterol level, 35.9%; high blood pressure, 56.9%; and impaired fasting glucose, 13.1%. A high amount of PA (> 14 km per week walking distance) was significantly associated with lower prevalence of metabolic syndrome [odds ratio (OR) = 0.53, $p = 0.001$], lower prevalence of obesity (OR = 0.56, $p = 0.001$), triglyceridemia (OR = 0.58, $p = 0.007$) and LVH (OR = 0.37, $p = 0.006$).

Conclusion: This study suggests that high amounts of PA are inversely correlated with the prevalence of metabolic syndrome and LVH in men and women.

Key Words: left ventricular hypertrophy, metabolic syndrome, physical activity

It is well known that metabolic syndrome increases the risk of cardiovascular disease, which is the leading cause of mortality and morbidity in developed and developing countries. Metabolic syndrome is characterized by obesity, glucose intolerance, elevated blood pressure and dyslipidemia. Each component of metabolic syndrome predisposes people to atherosclerosis, and when clustered together, these components promote atherosclerosis even more prominently. Lifestyle

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modification, such as physical activity (PA) and dietary habits, have been shown to reduce the risk of cardiovascular diseases[1–4] and the prevalence of metabolic syndrome.[5–7] A sedentary lifestyle predicts the development of metabolic syndrome.[8,9] However, the effect of different amounts of moderate-intensity PA is unclear.

Left ventricular hypertrophy (LVH) is related to the increased risk of cardiovascular disease and mortality,[10] as well as stroke and transient ischemic attack.[11] It has been demonstrated to be an independent predictor of cardiovascular events.[12] The effect of PA on LVH is not completely understood. Strenuous PA is known to cause LVH and dilatation as normal physiologic adaptations in athletes.[13,14] Conversely, low-to-moderate-intensity exercise has been shown to decrease blood pressure and regression of LVH in subjects with severe hypertension.[15] However, the effect of different amounts of PA on LVH in the general population remains unknown.

Metabolic syndrome is known to be a risk factor for cardiovascular disease and it has been extensively studied in the western hemisphere. However, the data for the Taiwanese population are limited. Therefore, we carried out a cross-sectional population study to elucidate the prevalence of metabolic syndrome in Taiwan. Furthermore, we wanted to investigate the association of various amounts of PA on the prevalence of metabolic syndrome and LVH in our middle-aged population.

Materials and Methods

Recruitment of study participants

We recruited 1494 apparently healthy subjects aged > 40 years in 1992–1993. These subjects comprised 776 men with a mean age of 57.6 ± 12.3 years, and 718 women with a mean age of 56.4 ± 11.0 years from Taipei city (1992) and San-Tze village (1993), a suburban area of Taipei (1042 and 452 subjects, respectively). All subjects were randomly selected from the general population in those areas. We randomly selected one out of every 100 administrative units in Taipei and San-Tze village. All participants provided informed consent before enrollment. A detailed medical record was obtained, which included basic personal and demographical data, complete medical history including current medication use and smoking status, educational level, and menopausal status for women. Physical examination, including measurement of body weight and height, was performed for the individual participants. Sitting blood pressure was measured three times with a conventional sphygmomanometer. Study subjects were instructed to fast for 12 hours overnight, and blood samples were collected for biochemistry tests, including fasting blood glucose, lipid profiles and uric acid, using standard laboratory methods in the hospital.

Definition of metabolic syndrome and left ventricular hypertrophy

Metabolic syndrome was defined according to the criteria of the National Cholesterol Education Program Adult Treatment Panel III with slight modification. Subjects with metabolic syndrome had three or more of the following criteria: obesity [body mass index (BMI) ≥ 27 kg/m^2]; serum triglycerides ≥ 150 mg/dL (1.7 mmol/L); serum high-density lipoprotein (HDL)-cholesterol < 40 mg/dL (1.04 mmol/L) in men or < 50 mg/dL (1.29 mmol/L) in women; blood pressure ≥ 130/85 mmHg or hypertensive patients taking regular medication; and fasting plasma glucose ≥ 110 mg/dL (6.1 mmol/L) or diabetic patients taking regular hypoglycemic agents.

LVH was diagnosed by 12-lead electrocardiography according to Framingham voltage criteria[12] as follows: R aVL > 11 mm and R V4–6 > 25 mm, or S V1–3 > 25 mm, or S V1 or V2 + R V5 or V6 > 35 mm, or R I + S III > 25 mm. The diagnosis of LVH was made by two independent cardiologists with an inter-rater agreement of 99.7%.

Assessment of daily PA

Daily PA was evaluated with a semi-quantitative questionnaire to assess the amount of daily PA in the previous month. The questionnaire asked: "How far do you walk every day on average?

(a) < 0.5 km (500 m); (b) between 500 m and 2 km; or (c) > 2 km." We applied the reported walking distance as a rough measurement of the amount of daily PA.[16] Based on their weekly walking distance, all participants were classified into three categories: sedentary life (low amount of PA); walking < 3.5 km per week; moderate amount of PA, walking 3.5–14 km per week; and high amount of PA, walking > 14 km per week.

Statistical analysis

To analyze the prevalence of metabolic syndrome, we divided our study population into four age categories: 40–49, 50–59, 60–69, and ≥ 70 years. The prevalence of metabolic syndrome in each age category was calculated separately for men and women. Continuous variables were presented as mean ± standard deviation and categorical variables were reported as percentages. Differences between mean values were assessed by Student's *t* test and differences between proportions were calculated by the χ^2 test. One-way analysis of variance was used to compare the mean values of each metabolic factor as continuous variables across different PA categories. To determine the effects of different amounts of moderate-intensity PA on the prevalence of metabolic syndrome and its components, as well as LVH, we applied multiple logistic regression analyses with adjustments for sex, age and smoking habits. The odds ratio (OR) was calculated using the low amount of moderate-intensity PA (sedentary lifestyle) group as a reference. Further stratified analysis for men and women was performed with logistic regression with adjustments for age, smoking and menopausal status (for women). A two-tailed *p* value of < 0.05 was considered significant. STATA Intercooled version 7.0 (StataCorp, College Station, TX, USA) was used for all statistical calculations.

Results

Epidemiology of metabolic syndrome

The overall prevalence of metabolic syndrome according to the modified criteria of the National Cholesterol Education Program Adult Treatment Panel III was 15.5% and the age-standardized prevalence was 14.7% (World Health Organization World Standard Population 2000). Figure 1 shows that the prevalence of metabolic syndrome significantly increased with age for both sexes. The prevalence of metabolic syndrome in men for the different age groups was as follows: 10.7% in 40–49 years, 16.6% in 50–59 years, 21.0% in 60–69 years, and 19.6% in > 70 years. In women, the prevalence of metabolic syndrome in the different age groups was: 7.2% in 40–49 years, 11.9% in 50–59 years, 22.2% in 60–69 years, and 22.7% in > 70 years. These data demonstrated that men had higher prevalence of metabolic syndrome than women in the age groups 40–49 years and 50–59 years ($p = 0.19$ and 0.01, respectively). Conversely, women had slightly higher prevalence of metabolic syndrome in the age group 60–69 years or older, although the differences were not statistically significant.

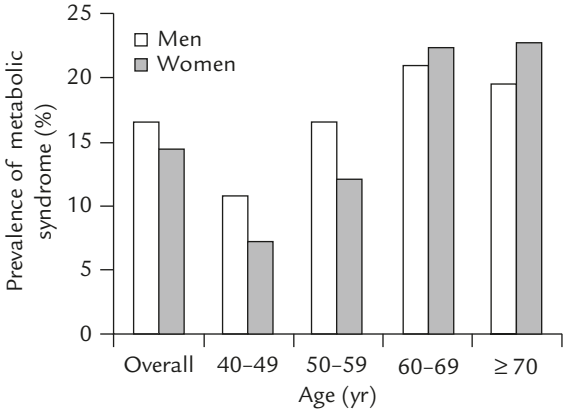

Figure 1. Prevalence of metabolic syndrome in Taiwanese population in 1993.

High blood pressure (> 130/85 mmHg) was the most common metabolic abnormality (56.9%), followed by low HDL-cholesterol (35.9%), obesity (29.7%), high triglyceride level (21.7%), and impaired fasting glucose (13.1%). Table 1 shows the prevalence of each component of metabolic syndrome stratified by age group and sex. In women, all components of metabolic syndrome increased significantly after postmenopausal age, whereas in men, only the prevalence of high blood pressure and impaired fasting glucose increased

Table 1. The prevalence of each component of the metabolic syndrome stratified by age and sex*

Metabolic factors	Sex	*n*	Age (yr)				*p*
			40–49	50–59	60–69	≥70	
Obesity, BMI ≥ 27 kg/m^2	M	199	22.3	26.3	25.6	30.1	0.42
	F	245	22.1	37.1*	38.6*	46.4*	<0.01
Triglycerides ≥ 150 mg/dL	M	194	23.7	32.6	21.5	23.1	0.07
	F	130	9.9*	15.2*	27.0	25.8	<0.01
HDL-cholesterol < 40 mg/dL	M	254	33.3	30.1	34.8	33.8	0.78
HDL-cholesterol < 50 mg/dL	F	283	33.0	39.7	42.8	50.5*	0.03
Blood pressure ≥ 130/85 mmHg	M	459	45.2	58.4	67.1	71.3	<0.01
	F	391	36.4*	56.0	64.2	76.3	<0.01
Fasting blood glucose ≥ 110 mg/dL	M	115	8.6	18.0	16.3	19.0	0.01
	F	82	5.0	12.3	17.1	13.4	<0.01

**p < 0.05 between men and women. BMI = body mass index; HDL = high-density lipoprotein.*

with older age. In the 40–49-year-old age group, the prevalence of obesity, low HDL-cholesterol and impaired fasting glucose was similar for men and women; hypertriglyceridemia and high blood pressure were even more common in men (23.7% *vs*. 9.9%, $p < 0.01$ and 45.2% *vs*. 36.4%, $p = 0.05$, respectively). However, the reverse was noted after postmenopausal age, when women had significantly higher prevalence of obesity (37.1% *vs*. 26.3%, $p = 0.02$). The prevalence of all other components of metabolic syndrome was similar between men and women.

Association of different amounts of PA with prevalence of metabolic syndrome and LVH

Baseline characteristics of the participants are shown in Table 2. Compared with controls, subjects with metabolic syndrome were significantly older (59.7 ± 11.7 *vs*. 56.6 ± 11.6 years, $p < 0.001$). These subjects also had higher serum total cholesterol (200.6 ± 40.5 mg/dL *vs*. 188.0 ± 46.0 mg/dL, $p < 0.001$), were more likely to be smokers (30.7% *vs*. 22.4%, $p = 0.05$), and were less physically active (24.7% *vs.* 17.8%, $p = 0.04$). Sex distribution was similar for metabolic syndrome and controls.

Table 3 displays the effect of different amounts of moderate-intensity PA on the profiles of several metabolic parameters. High amounts of PA were significantly associated with lower BMI ($p = 0.04$), lower triglyceride level ($p = 0.02$) and higher HDL-cholesterol in women ($p < 0.01$). No obvious difference was noted for the remaining metabolic factors.

As shown in Figure 2, subjects with high amounts of PA had lower prevalence of metabolic syndrome. These phenomena were valid for men and women. After adjustment for age, sex and smoking habit, multiple logistic regression analyses showed that, when compared with sedentary lifestyle, high amounts of PA were associated with decreased risk of metabolic syndrome [OR = 0.61, 95% confidence interval (CI) = 0.36–0.86, $p = 0.001$], but moderate amounts of PA had no significant risk reduction (OR = 0.83, 95% CI = 0.56–1.23, $p = 0.40$) (Table 4). For each component of metabolic syndrome, a high amount PA was significantly associated with lower prevalence of obesity (OR = 0.56, $p = 0.001$), hypertriglyceridemia (OR = 0.58, $p = 0.007$) and low HDL-cholesterol (OR = 0.73, $p = 0.05$). These effects were not observed in the moderate PA group. The level of PA had no significant association with high blood pressure or impaired fasting glucose.

Multiple logistic regression analysis revealed that LVH was correlated with systolic blood pressure (OR = 1.3, $p < 0.01$) (data not shown), and inversely associated with high amounts of PA (OR = 0.40, 95% CI = 0.18–0.83, $p = 0.01$) (Table 4) after controlling for sex, age, BMI, blood pressure,

and smoking habits. Moderate PA did not have a significant correlation with prevalence of LVH. Separate analyses for men and women revealed a similar trend for the inverse correlation between high PA and prevalence of metabolic syndrome and its components, except that high PA was significantly associated with lower prevalence of high BP in men (OR = 0.61, $p = 0.05$) but not in women (Table 5). The benefit of high PA on the prevalence of LVH seemed to be greater in women than in men (OR = 0.20 in women and 0.43 in men) (Table 5).

Table 2. Baseline characteristics of study population, with and without the metabolic syndrome*

	Overall ($n = 1494$)	Without MS ($n = 1263$)	With MS ($n = 231$)	p
Age (yr)	57.1 ± 11.7	56.6 ± 11.6	59.7 ± 11.7	< 0.001
Sex				
Male	776	648 (83.5)	128 (16.5)	
Female	718	615 (85.7)	103 (14.3)	0.250
Systolic BP (mmHg)	128.8 ± 20.0	126.5 ± 19.5	141.9 ± 19.1	< 0.001
Diastolic BP (mmHg)	81.6 ± 11.0	80.4 ± 10.5	88.1 ± 11.3	< 0.001
BMI (kg/m^2)	24.2 ± 3.5	23.7 ± 3.1	26.6 ± 4.1	< 0.001
Serum HDL cholesterol (mg/dL)	50.0 ± 15.2	52.4 ± 14.9	36.8 ± 8.8	< 0.001
Serum triglycerides (mg/dL)	116.4 ± 80.6	100.0 ± 56.2	216.9 ± 113.4	< 0.001
Fasting blood glucose (mg/dL)	95.5 ± 35.0	92.3 ± 34.5	113.3 ± 42.2	< 0.001
Serum total cholesterol (mg/dL)	190.0 ± 45.4	188.0 ± 46.0	200.6 ± 40.5	< 0.001
Serum uric acid (mg/dL)	6.4 ± 13.2	6.4 ± 1.4	6.9 ± 1.8	0.570
Prevalence of hypertension	266 (17.8)	182 (14.4)	84 (36.4)	< 0.001
Prevalence of diabetes	87 (5.8)	45 (3.6)	42 (18.2)	< 0.001
Prevalence of smoking	354 (23.7)	283 (22.4)	71 (30.7)	0.050
Amount of PA				
High	502 (33.6)	434 (34.4)	68 (29.4)	
Moderate	710 (47.5)	589 (46.6)	121 (52.4)	
Low (sedentary lifestyle)	282 (18.9)	225 (17.8)	57 (24.7)	0.070

**Data presented as n (%) or mean ± standard deviation. MS = metabolic syndrome; BP = blood pressure; PA = physical activity.*

Table 3. Effects of the amount of physical activity on various metabolic factors*

	Sedentary	Moderate PA	High PA	p
BMI (kg/m^2)	25.4 ± 3.5	24.1 ± 3.4	23.1 ± 3.4	0.04
HDL-cholesterol (mg/dL)				
Male	44.4 ± 13.4	46.0 ± 14.8	47.5 ± 14.1	0.11
Female	51.3 ± 13.7	55.0 ± 15.3	55.8 ± 18.5	< 0.01
Triglycerides (mg/dL)	127.0 ± 93.4	120.2 ± 78.0	110.3 ± 71.0	0.02
Fasting glucose (mg/dL)	93.2 ± 25.4	96.7 ± 30.0	94.6 ± 33.2	0.51
Systolic BP	131.0 ± 22.0	129.2 ± 20.0	130.2 ± 19.8	0.18
Diastolic BP	82.1 ± 11.0	82.3 ± 11.0	81.2 ± 10.0	0.27
Uric acid (mg/dL)	6.0 ± 1.8	6.2 ± 1.7	6.0 ± 1.6	0.17

**Data presented as mean ± standard deviation. BMI = body mass index; BP = blood pressure; PA = physical activity.*

Discussion

The age-adjusted prevalence of metabolic syndrome in our population was 14.65%, which was clearly lower than that reported in the American (24.0%),[17] Asian Indian (41.1%),[18] Arab American (23.0%)[19] and Iranian (33.7%) populations.[20] It was obvious that the prevalence of metabolic syndrome increased with age for both sexes. In younger age groups, men had higher prevalence of metabolic syndrome than women had, whereas women after postmenopausal age had a higher prevalence of metabolic syndrome than men. This phenomenon could be attributed to the protective effect of estrogen during the reproductive age of women, in parallel with the well-known lower cardiovascular risks in premenopausal women. Among the metabolic syndrome traits, high normal blood pressure was the most common in our middle age population (56.9%), followed by low HDL-cholesterol (35.9%), obesity (29.7%), high triglyceride level (21.7%), and impaired fasting glucose (13.1%). Likewise, high blood pressure was also the most common among the metabolic syndrome traits in middle-aged Asian Indian[19] and Iranian[20] populations. These findings are in contrast with the prevalence of metabolic abnormalities in the United States population reported in the National Health and Nutrition Examination Survey III,[18] in which abdominal obesity (38.6%) and low HDL-cholesterol (37.1%) were more prevalent than high normal blood pressure (34.0%). With regard to the racial differences, the frequencies of metabolic abnormalities in Taiwanese resemble those in African Americans, rather than those in Caucasians and Mexican Americans.

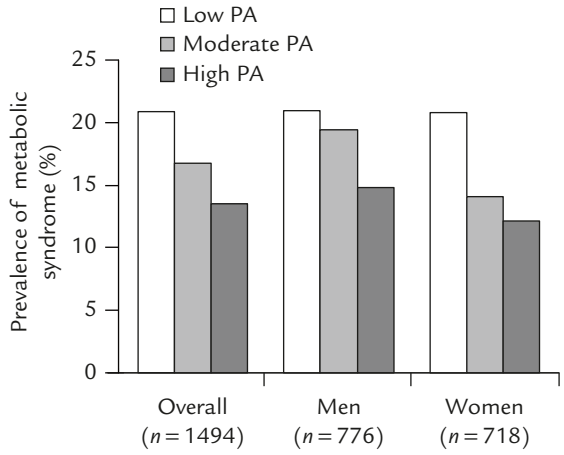

Figure 2. Prevalence of metabolic syndrome stratified by amount of physical activity. PA = physical activity.

Table 4. Effect of moderate and high amount of physical activity on the prevalence of the metabolic syndrome, its components and the prevalence of left ventricular hypertrophy, after adjustments for sex, age and smoking habit

	PA	OR (95% CI)	*p*
Metabolic syndrome	Moderate	0.83 (0.56–1.23)	0.400
	High	0.61 (0.36–0.86)	0.010
Obesity, BMI > 27 kg/m^2	Moderate	0.73 (0.53–1.00)	0.050
	High	0.56 (0.40–0.80)	0.001
Low HDL-cholesterol	Moderate	0.81 (0.58–1.14)	0.210
	High	0.73 (0.53–1.00)	0.050
High triglycerides	Moderate	0.87 (0.61–1.23)	0.460
	High	0.58 (0.39–0.86)	0.007
High BP	Moderate	0.91 (0.65–1.27)	0.610
	High	0.78 (0.55–1.11)	0.190
Impaired fasting glucose	Moderate	1.33 (0.84–2.11)	0.210
	High	0.88 (0.54–1.45)	0.630
LVH*	Moderate	0.77 (0.43–1.40)	0.390
	High	0.40 (0.18–0.83)	0.010

**Also adjusted for systolic and diastolic BP and BMI. BMI = body mass index; BP = blood pressure; OR = odds ratio; CI = confidence interval; LVH = left ventricular hypertrophy.*

Table 5. Effect of high amount of PA on the prevalence of the metabolic syndrome, its components and the prevalence of left ventricular hypertrophy stratified for men and women, after adjustments for age, smoking habit and menopausal status (for women)*

	Male	*p*	Female	*p*
Metabolic syndrome	0.63 (0.35–1.01)	0.05	0.52 (0.27–0.98)	0.05
Obesity, BMI > 27 kg/m^2	0.54 (0.33–0.88)	0.01	0.61 (0.37–0.99)	0.05
Low HDL-cholesterol	0.72 (0.51–1.01)	0.05	0.54 (0.34–0.86)	0.01
High triglycerides	0.61 (0.36–0.98)	0.04	0.60 (0.32–1.00)	0.05
High BP	0.61 (0.37–1.00)	0.05	0.84 (0.52–1.37)	0.50
Impaired fasting glucose	1.04 (0.53–2.05)	0.14	0.75 (0.35–1.58)	0.45
LVH	0.43 (0.20–0.99)	0.05	0.20 (0.05–0.81)	0.02

**Data presented as odds ratio (95% confidence interval). BMI = body mass index; HDL = high-density lipoprotein; BP = blood pressure; LVH = left ventricular hypertrophy.*

In the present cross-sectional study, we compared the association of three different amounts of PA with overall prevalence of metabolic syndrome and its components. We found that the prevalence of metabolic syndrome was lower in the high PA group (walking at least 14 km per week). However, the association was not observed in subjects with moderate or low PA (walking < 14 km per week). This result indicated that a high amount of PA was inversely correlated with risk of metabolic syndrome if the amount of daily PA exceeded 14 km per week walking distance. Moderate amounts of PA were not associated with reduced prevalence of metabolic syndrome. After stratification for each component of metabolic syndrome, we found that prevalence of obesity and hypertriglyceridemia, but not other components, was significantly reduced in the high-PA group. This result was consistent with the finding of previous study by Kraus et al,[21] which showed that high amounts of PA (average 17.4 km per week) had greater benefit than low-to-moderate amounts of PA (average 11 km per week) in terms of lipid profiles, and both high and low amounts of PA were beneficial when compared with the sedentary group. The low amount of PA in the study of Kraus et al is equivalent to high PA in our study. The amount of PA in our study ranged from < 3.5 km per week to > 14 km per week, which could be regarded as being the lower extension of the PA spectrum used by Kraus et al. The beneficial effect of walking was also reported in a substudy of the Women's Health Initiative Observational Study, which demonstrated that walking was associated with a similar reduction in risk of cardiovascular events as vigorous exercise among postmenopausal women. Moreover, a brisker walking pace and fewer hours spent sitting daily also predicted lower risks.[22]

We applied BMI instead of waist circumference as a criterion for metabolic syndrome in this study. Although obesity (defined by BMI) and abdominal obesity (defined by waist circumference) are not interchangeable, several studies have confirmed their close correlation.[23] Furthermore, BMI and waist circumference are independent predictors of cardiovascular risk profiles.[24] In addition, our study also revealed that high-to-moderate-intensity PA was associated with less LVH, after control for confounding factors, such as blood pressure, BMI, sex, age and menopausal status. Likewise, this benefit was absent in the group with a moderate amount of moderate-intensity PA.

A previous study has shown that athletes who undertake regular vigorous PA have LVH and dilatation.[14] In contrast, hypertensive subjects who take regular low-to-moderate-intensity PA have been shown to have regression of LVH.[8] This discrepancy suggests that the intensity of PA, in addition to the amount, is another important factor that affects LVH. We demonstrated that high amounts of PA were significantly associated with decreased prevalence of LVH, even though it did not seem to be associated with decreased blood

pressure. Although it is well known that high blood pressure induces LVH, we propose three explanations for the benefit of high PA in relation to reduced LVH prevalence, independent of blood pressure. First, it is well known that persons who regularly undertake high amounts of PA have slower resting heart rate. The oversimplified physiological explanation is that slower heart rate reduces the overall cardiac workload and subsequently prevents LVH. Conversely, high-intensity PA in athletes imposes greater stress on the heart, which might subsequently induce LVH, denoted as physiological cardiac hypertrophy. Second, Braith et al[25] reported that subjects with regular training programs have lower plasma levels of catecholamines, angiotensin II, arginine vasopressin, and aldosterone. This suppression of neurohormonal stimulation might attenuate the progression of LVH. Third, PA has been reported to increase endogenous antioxidant defense capacity, which plays an important role in the development of LVH.[26] However, further studies are needed to elucidate the exact mechanism that underlies the correlation of PA and LVH.

Although our study was based on an epidemiological survey carried out more than 15 years ago, we did demonstrate the beneficial effect of high amounts of PA on the prevalence of metabolic syndrome and LVH, and this was not likely to have changed with time. Furthermore, comparison of our data with a recent report by Chen et al revealed that the prevalence of metabolic syndrome in Taiwan has increased dramatically during the past decade.[27] This underlines an urgent need to develop a better strategy to prevent metabolic syndrome and cardiovascular diseases.

There are several limitations to our study. First, we used a questionnaire to document the average daily walking distance in the previous month, which could have induced a recall bias. Furthermore, the recalled daily walking distance might only have provided a rough estimate and not the overall PA of an individual. Objective measurement with exercise prescription in a prospective study[21] would be more reliable than a questionnaire-based method in a cross-sectional study. Second, we did not include some potential confounders, such as diet and socioeconomic status. However, in a large-scale epidemiological study, these data were also based on self-reported questionnaires, which again, could have introduced bias. Third, our retrospective study design could not address the causal effect relationship between PA and metabolic syndrome or LVH.

In summary, metabolic syndrome is prevalent in Taiwan, although the prevalence in our middle-aged population seems to be lower than that reported in western and other Asian populations. Higher amounts of PA are inversely correlated with prevalence of metabolic syndrome and LVH. However, this beneficial effect is seen only if the amount of PA exceeds the walking distance of 14 km per week. Future studies are required to elucidate the mechanisms that underlie the correlation between LVH and PA.

References

1. Paffenbarger RS, Jr., Hyde RT, Wing AL, et al. The association of changes in physical-activity level and other lifestyle characteristics with mortality among men. *N Engl J Med* 1993;328:538–45.
2. Leon AS, Sanchez OA. Response of blood lipids to exercise training alone or combined with dietary intervention. *Med Sci Sports Exerc* 2001;33:S502–15.
3. Leon AS, Connett J, Jacobs DR, Jr., et al. Leisure-time physical activity levels and risk of coronary heart disease and death. The Multiple Risk Factor Intervention Trial. *JAMA* 1987;258:2388–95.
4. de Lorgeril M, Salen P, Martin JL, et al. Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction: final report of the Lyon Diet Heart Study. *Circulation* 1999;99:779–85.
5. Panagiotakos DB, Pitsavos C, Chrysohoou C, et al. Impact of lifestyle habits on the prevalence of the metabolic syndrome among Greek adults from the ATTICA study. *Am Heart J* 2004;147:106–12.
6. Yao M, Lichtenstein AH, Roberts SB, et al. Relative influence of diet and physical activity on cardiovascular risk factors in urban Chinese adults. *Int J Obes Relat Metab Disord* 2003;27:920–32.
7. Katzmarzyk PT, Leon AS, Wilmore JH, et al. Targeting the metabolic syndrome with exercise: evidence from the HERITAGE Family Study. *Med Sci Sports Exerc* 2003;35:1703–9.

8. Hagberg JM, Brown MD. Does exercise training play a role in the treatment of essential hypertension? *J Cardiovasc Risk* 1995;2:296–302.
9. Laaksonen DE, Lakka HM, Salonen JT, et al. Low levels of leisure-time physical activity and cardiorespiratory fitness predict development of the metabolic syndrome. *Diabetes Care* 2002;25:1612–8.
10. Levy D, Garrison RJ, Savage DD, et al. Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. *N Engl J Med* 1990; 322:1561–6.
11. Bikkina M, Levy D, Evans JC, et al. Left ventricular mass and risk of stroke in an elderly cohort. The Framingham Heart Study. *JAMA* 1994;272:33–6.
12. Levy D, Salomon M, D'Agostino RB, et al. Prognostic implications of baseline electrocardiographic features and their serial changes in subjects with left ventricular hypertrophy. *Circulation* 1994;90:1786–93.
13. Maron BJ, Epstein SE, Roberts WC. Causes of sudden death in competitive athletes. *J Am Coll Cardiol* 1986;7:204–14.
14. Pelliccia A, Culasso F, Di Paolo FM, et al. Physiologic left ventricular cavity dilatation in elite athletes. *Ann Intern Med* 1999;130:23–31.
15. Kokkinos PF, Narayan P, Colleran JA, et al. Effects of regular exercise on blood pressure and left ventricular hypertrophy in African–American men with severe hypertension. *N Engl J Med* 1995;333:1462–7.
16. Ainsworth BE, Haskell WL, Leon AS, et al. Compendium of physical activities: classification of energy costs of human physical activities. *Med Sci Sports Exerc* 1992; 24:71–80.
17. Ford ES, Giles WH, Dietz WH. Prevalence of the metabolic syndrome among US adults: findings from the third National Health and Nutrition Examination Survey. *JAMA* 2002;287:356–9.
18. Ramachandran A, Snehalatha C, Satyavani K, et al. Metabolic syndrome in urban Asian Indian adults—a population study using modified ATP III criteria. *Diabetes Res Clin Pract* 2003;60:199–204.
19. Jaber LA, Brown MB, Hammad A, et al. The prevalence of the metabolic syndrome among arab americans. *Diabetes Care* 2004;27:234–8.
20. Azizi F, Salehi P, Etemadi A, et al. Prevalence of metabolic syndrome in an urban population: Tehran Lipid and Glucose Study. *Diabetes Res Clin Pract* 2003;61:29–37.
21. Kraus WE, Houmard JA, Duscha BD, et al. Effects of the amount and intensity of exercise on plasma lipoproteins. *N Engl J Med* 2002;347:1483–92.
22. Manson JE, Greenland P, LaCroix AZ, et al. Walking compared with vigorous exercise for the prevention of cardiovascular events in women. *N Engl J Med* 2002;347: 716–25.
23. Anuurad E, Shiwaku K, Nogi A, et al. The new BMI criteria for asians by the regional office for the western pacific region of WHO are suitable for screening of overweight to prevent metabolic syndrome in elder Japanese workers. *J Occup Health* 2003;45:335–43.
24. Janssen I, Katzmarzyk PT, Ross R. Body mass index, waist circumference, and health risk: evidence in support of current National Institutes of Health guidelines. *Arch Intern Med* 2002;162:2074–9.
25. Braith RW, Edwards DG. Neurohormonal abnormalities in heart failure: impact of exercise training. *Congest Heart Fail* 2003;9:70–6.
26. Bloomer RJ. Effect of exercise on oxidative stress biomarkers. *Adv Clin Chem* 2008;46:1–50.
27. Chen PC, Chien KL, Hsu HC, et al. C-reactive protein and the metabolic syndrome correlate differently with carotid atherosclerosis between men and women in a Taiwanese community. *Metabolism* 2008;57:1023–8.