研究成果の刊行に関する一覧表(平成 20 年度)

書籍

著者氏名	論文タイトル名	書籍全体の編集者名	書 籍 名	出版社名	出版地	出版年	ページ
宮地元彦	生活習慣病予防に関する保健指導、特定健診・保健指導における運動・身体活動指導	奈良昌治, 山門實監修	人間ドック健診情報管理指導士テキスト―第 2 版―特定健診・特定保健指導	人間ドック学会		2008	176, 361 -179, 368
宮地元彦 , 岡本正一 , 三船智美 , 新倉佳奈江	メタボ解消 ! 10 分エクササイズ on DVD	宮地元彦監修		(株)社会保健研究所		2008	1 -27
宮地元彦	運動の効果 PWV および中心動脈から見た予防医学と生活習慣	家光素行 , 宮地元彦 , 小澤利男編	新しい血圧測定と脈波解析マニュアル	MEDICAL VIEW		2008	186 -189
宮地元彦	婦人科における応用 PWV および中心血圧の臨床応用	林貢一郎 , 宮地元彦 , 小澤利男編	新しい血圧測定と脈波解析マニュアル	MEDICAL VIEW		2008	178 -181
宮地元彦	VI 生活習慣改善指導 【2】運動・身体活動指導	宮地元彦	健診判定基準ガイドライン〔改訂新版〕	文光堂	東京	2008	282 -289
引原有輝 , 宮地元彦	身体活動量の測定・評価	佐藤祐造編	運動療法と運動処方〔第 2 版〕	文光堂	東京	2008	95 -100
宮地元彦	体力・運動能力／身体組成の測定・評価	佐藤祐造編	運動療法と運動処方〔第 2 版〕	文光堂	東京	2008	101 -109
宮地元彦	生活習慣病対策と介護予防における体力の重要性		体力の重要性と測定・評価 ―新体力テスト―	(財)健康・体力づくり事業財団	東京	2008	1 -17
宮地元彦	2章:運動・スポーツの生理学(1), 12章:生活習慣病予防と運動・スポーツ(2)―高血圧症・動脈硬化―, 15章:老化防止・介護予防と運動・スポーツ	宮地元彦 , 樋口満, 福永哲夫(編)	放送大学大学院文化科学研究科テキスト「スポーツ・健康科学」	(財)放送大学教育振興会(東京都港区)		2009	25 -46, 191-207, 242-259

宮地元彦	第1章:特定保健指導における運動・身体活動指導の基本的な考え方,第5章:運動指導におけるリスクマネージメント	宮地元彦(編)	行動変容につなげる保健指導スキルアップBOOK「エビデンスと運動実践から学ぶ運動指導」	中央法規	東京	2009	1 -192
宮地元彦	第2章:運動生理学	宮地元彦 , 青木純一郎, 田畑泉ら(編)	健康運動実践指導者養成用テキスト	(財)健康・体力づくり事業財団	東京	2009	13 -28
宮地元彦	Section 10 トレーニングと循環, 2:心拍出量と血管	宮地元彦 , 宮村実晴(編)	身体トレーニング―運動生理学からみた身体機能の維持・向上	真興交易	東京	2009	288 -294

雑誌

発表者氏名	論文タイトル名	発表誌名	巻号	ページ	出版年
宮地元彦	生活習慣病における運動療法,特集:運動と動脈硬化予防	動脈硬化予防	7(2)	61 -70	2008
宮地元彦	特定健診・保健指導に向けての運動・身体活動指導	日本補完代替医療学会誌	5(2)	115 -122	2008
宮地元彦	特定保健指導における運動指導の方法	体育の科学	58(7)	475 -482	2008
宮地元彦	特定健診と保健指導―メタボリックシンドロームを標的とした動脈計機能評価と対策	Arterial Stiffness 臨床血圧脈波研究会	14	26 -33	2008
宮地元彦	運動・身体活動のエビデンス⑩ 体力レベルが高い場合,低い場合と比較して総死亡リスクを約50%減少させる	健康づくり 健康・体力づくり事業財団(東京)	360; 11		2008
宮地元彦	メタボリックシンドロームに関するサイト,栄養士のためのお役立ちサイト(4)	臨床栄養	112(5)	486 -487	2008
宮地元彦	腹部の筋持久力が脂肪率の予測因子となる,運動・身体活動のエビデンス⑪	健康づくり,健康体力づくり事業財団	(360); 11		2008

宮地元彦	エクササイズガイド 2006 を用いた特定保健指導とは？ 特集 特定健診と特定保健指導ー展望と実際ー	肥満と糖尿病	7(5)	705 -707	2008
宮地元彦	糖尿病診療の最新情報(Ⅲ) 運動療法	ドクターサロン	52(10)	56 -60	2008
H. Kawano, H. Nakagawa, S. Onodera, M. Higuchi, M. Miyachi.	Attenuated increases in blood pressure by dynamic resistance exercise in middle-aged men.	Hypertens Res	31(5)	1045 -1053	2008
M. Miyatani, H. Kawano, K. Masani, Y. Gando, K. Yamamoto, M. Tanimoto, T. Oh, C. Usui, K. Sanada,M. Higuchi, I. Tabata, M. Miyachi.	Required muscle mass for preventing lifestyle-related diseases in Japanese women.	BMC Public Health	18(8)	291	2008
沼田健之, 宮武伸之, 松本純子, 藤井昌史, 宮地元彦.	5 か月間の生活習慣改善教室参加女性における体重変化量と腹囲変化量との関係.	日本予防医学会誌	3(2)	13-16	2008
M. Tanimoto, K. Yamamoto, H. Kawano, Y. Gando, I. Tabata, N. Ishii, M. Miyachi.	Effects of whole-body low-intensity resistance training with slow movement and tonic force generation on muscular size and strength in young men.	J Strength Cond Res	22(6)	1926-1938	2008
山本祥子, 高田和子, 別所京子, 谷本道哉, 宮地元彦, 田中茂穂, 戸谷誠之, 田畑泉.	ボディービルダーの基礎代謝量と身体活動レベルの検討.	栄養学雑誌	66(4)	195 -200	2008
N. Miyatake, S. Matsumoto, M. Fujii, T. Numata.	Reducing waist circumference by at least 3 cm is recommended for improving metabolic syndrome in obese Japanese men.	Diabetes Res Clin Pract	79	191-195	2008
C. Usui, E. Takahashi, Y. Gando, K. Sanada, J. Oka , M. Miyachi, I. Tabata, M. Higuchi.	Resting energy expenditure can be assessed by dual energy X-ray absorptiometry in women regardless of age and fitness.	Eur J Clin Nutr			2008

H. Kawano, M. Tanimoto, K. Yamamoto, K. Sanada, Y. Gando, I. Tabata, M. Higuchi, M. Miyachi.	Resistance training is associated with increased arterial stiffness and blood pressure but does not adversely affect endothelial function as measured by arterial reactivity to the cold pressor test.	Exp Physiol	93	296-302	2008
ZB. Cao, N. Miyatake, M. Higuchi, K. Ishikawa-Takata, M. Miyachi, I. Tabata.	Prediction of VO2max with daily step counts for Japanese adult women.	Eur J Appl Physiol	105(2)	289 -296	2009
M. Miyatani, K. Masani, PI. Oh, M. Miyachi, MR . Popovic, BC. Craven.	Pulse wave velocity for assessment of arterial stiffness among people with spinal cord injury: a pilot study	J Spinal Cord Med	32(1)	72 -78	2009
M. Tanimoto, K. Kawano, Y. Gando, K. Sanada, K Yamamoto, N. Ishii, I. Tabata, M. Miyachi.	Low-intensity resistance training with slow movement and tonic force generation increases basal limb blood flow.	Clin Physiol Funct Imaging	29(2)	128 -135	2009
M. Tanimoto, H. Arakawa, K. Sanada, M. Miyachi, N. Ishii.	Change in muscle activation and force generation patterns during cycling movements due to low-intensity squat training with slow movement and tonic force generation (LST).	Clin Physiol Funct Imaging			2009, In Press

V. 研究成果の刊行物・別刷

以下に主な文献を添付する

- H. Kawano, H. Tanaka, M. Miyachi. Resistance Training and Arterial Compliance: Keeping the Benefits While Minimizing the Stiffening. J. Hypertens. 24(9): 1753-9, 2006
- N. Miyatake, J. Wada, T. Saito, H. Nishikawa, S. Matsumoto, M. Miyachi, H. Makino, T. Numata. Comparison of muscle strength between Japanese men with and without metabolic syndrome. Acta Med Okayama. 61(1): 99-102, 2007
- N. Miyatake, T. Saito, J. Wada, M. Miyachi, I. Tabata, S. Matsumoto, H. Nishikawa, H. Makino, T. Numata. Comparison of ventilatory threshold and exercise habits between Japanese men with and without metabolic syndrome. Diabetes Res Clin Pract, 77(2): 314-319, 2007
- N. Miyatake, S. Matsumoto, M. Miyachi, M. Fujii, T. Numata. Relationship between changes in body weight and waist circumference in Japanes. Eenvironmental Health and Preventive Medicine, 12(5): 220-223, 2007
- H. Kawano, M. Tanimoto, K. Yamamoto, K. Sanada, Y. Gando, I. Tabata, M. Higuchi, M. Miyachi. Resistance training in men is associated with increased arterial stiffness and blood pressure but does not adversely affect endothelial function as measured by arterial reactivity to the cold pressor test. Experimental Physiology, 93(2): 296-302, 2008.
- M. Miyachi, Y. Ohmori, K. Yamamoto, H. Kawano, H. Murakami, A. Morita, S. Watanabe. The Use of a Uniaxial Accelerometer to Assess Physical-activity-related Energy Expenditure in Obese Men and Women: Saku Control Obesity Program (SCOP). Anti-Aging Med, 5(1): 1-5, 2008
- K. Sanada, M. Miyachi, I. Tabata, M. Miyatani, M. Tanimoto, TW. Oh, K. Yamamoto, C. Usui, E. Takahashi, H. Kawano, Y. Gando, M. Higuchi. Muscle mass and bone mineral indices: does the normalized bone mineral content differ with age? Eur J Clin Nutr: In press2008.
- H. Kawano, M. Tanimoto, K. Yamamoto, K. Sanada, Y. Gando, I. Tabata, M. Higuchi, M. Miyachi. Resistance training is associated with increased arterial stiffness and blood pressure but does not adversely affect endothelial function as measured by arterial reactivity to the cold pressor test. Exp. Physiol, 93: 296-302, 2008
- H. Kawano, H. Nakagawa, S. Onodera, M. Higuchi, M. Miyachi. Attenuated increases in blood pressure by dynamic resistance exercise in middle-aged men. Hypertension Research, 31(5): 1045 -1053, 2008

- M. Miyatani, H. Kawano, K. Masani, Y. Gando, K. Yamamoto, M. Tanimono, T. Oh, C. Usui, K. Sanada, M. Higuchi, I. Tabata, M. Miyachi. Required Muscle Mass for Preventing Lifestyle-Related Diseases in Japanese Women. BMC Public Health, 18; 8: 291, 2008
- M. Tanimoto, K. Sanada, K. Yamamoto, H. Kawano, Y. Gando, I. Tabata, N. Ishii, M. Miyachi. Effects of whole-body low-intensity resistance training with slow movement and tonic force generation (LST) on muscular size and strength in young men. JSCR, 22(6): 1926-38, 2008
- M. Miyatani, K. Masani, P. Oh, M. Miyachi, MR. Popovic, BC. Craven. Pulse wave velocity for assessment of arterial stiffness among people with spinal cord injury: a pilot study. J Spinal Cord Med; 32(1); 72 -78, 2009
- M. Tanimoto, H. Kawano, Y. Gando, K. Sanada, K. Yamamoto, N. Ishii, I. Tabata, M. Miyachi. Low-intensity resistance training with slow movement and tonic force generation increases basal limb blood flow. Clin Physiol Funct Imaging. 29(2); 128 -135, 2009

Resistance training and arterial compliance: keeping the benefits while minimizing the stiffening

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Objectives This study aimed to determine the effects of moderate resistance training as well as the combined resistance and aerobic training intervention on carotid arterial compliance.

Background Resistance training has become a popular mode of exercise, but intense weight training is shown to stiffen carotid arteries.

Methods Thirty-nine young healthy men were assigned either to the moderate-intensity resistance training (MODE), the combined resistance training and endurance training (COMBO) or the sedentary control (CONTROL) groups. Participants in the training groups underwent three training sessions per week for 4 months followed by four additional months of detraining.

Results All training groups increased maximal strength in all the muscle groups tested ($P < 0.05$). Carotid arterial compliance (via simultaneous carotid ultrasound and applanation tonometry) decreased approximately 20% after MODE training (from 0.20 ± 0.01 to 0.16 ± 0.01 mm^2/mmHg, $P < 0.01$). No significant changes in carotid arterial compliance were observed in the COMBO (0.20 ± 0.01 to 0.23 ± 0.01 mm^2/mmHg) and CONTROL (0.20 ± 0.01 to 0.20 ± 0.01 mm^2/mmHg) groups. Following the detraining period, carotid arterial compliance returned to the baseline level. Peripheral (femoral) artery compliance did not change in any groups.

Conclusions We concluded that simultaneously performed aerobic exercise training could prevent the stiffening of carotid arteries caused by resistance training in young healthy men. *J Hypertens* 24:1753–1759 © 2006 Lippincott Williams & Wilkins.

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Keywords: arterial structure and compliance, exercise, imaging, cross-training, ultrasonics

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Introduction

The aorta and large arteries play an important role in the cardiovascular system not only as blood conduits to the peripheral tissues, but also as a buffer for pressure changes resulting from intermittent ventricular ejection of blood. By absorbing a proportion of the energy in systole and releasing it in diastole, they maintain coronary blood flow and avoid an increase in left ventricular afterload. Through the impairment of this buffering function, reductions in arterial compliance or increases in arterial stiffness contribute to elevations in systolic blood pressure, left ventricular hypertrophy, and coronary ischemia [1,2]. Indeed, higher arterial stiffness is associated with a greater rate of mortality in patients with end-stage renal failure and essential hypertension [3,4]. Accordingly, any interventions that could act to decrease arterial compliance should be cautiously performed or even avoided.

Resistance training has become a popular modality of exercise performed by most populations, and has become an integral component of exercise recommendations endorsed by a number of national health organizations [5–7]. Resistance training has profound effects on the musculoskeletal system, thereby contributing to the maintenance of functional capacity and the prevention of sarcopenia and osteoporosis [7]. The effects of resistance training on the cardiovascular system, however, are not well understood. We have recently demonstrated that high-intensity resistance training is associated with reduced arterial compliance [8,9]. This finding was initially observed in cross-sectional studies comparing strength-trained individuals and sedentary controls [8] and later confirmed by interventional studies involving several months of resistance training interventions [9]. Considering a number of functional and physiological benefits that resistance training induces, practice of resistance training should not be discouraged. A remaining critical question is whether any type of resistance training could be performed regularly without inducing arterial stiffening. In this context, two strategies appear plausible. First, the intensity and volume of the resistance training used in the previous studies [8,9] were more strenuous and vigorous than those recommended for the comprehensive health programs [5–7]. It is not currently

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known whether moderate resistance training would induce similar arterial stiffening. Second, because regular aerobic exercise has been shown to increase arterial compliance [10,11], simultaneously performed endurance training may negate the effects of resistance training, thereby attenuating or preventing arterial stiffening. Neither of these possibilities has been tested, however.

Accordingly, the primary aim of the present study was to determine the effects of moderate-intensity resistance training as well as the combined strength and endurance training intervention on carotid arterial compliance. We hypothesized that the compliance of carotid arteries would not change following moderate-intensity resistance training as well as combined resistance and aerobic training. At the completion of the exercise intervention period, we implemented a period of detraining. We reasoned that if the observed changes in arterial compliance were induced by the prescribed exercise training, values should return to the baseline levels when the stimuli of exercise training were removed.

Methods

Participants

A total of 39 young healthy men were studied. None of the men had participated in any resistance or endurance training on the regular basis. All subjects were normotensive (< 140/90 mm Hg), non-obese (body mass index < 30 kg/m^2), and free of overt chronic diseases as assessed by medical history, physical examination, and a complete blood chemistry and hematological evaluation. Candidates who smoked in the past 4 years were taking cardiovascular-acting medications or anabolic steroids, or had significant intima–media thickening, plaque formation, and/or other characteristics of atherosclerosis (e.g. ankle–brachial index < 0.9) were excluded. All subjects gave their written informed consent to participate, and all procedures were reviewed and approved by the Institutional Review Board. Subjects were randomly assigned into either the moderate-intensity resistance training group (MODE, $n = 12$), the combined high-intensity resistance training and moderate-intensity aerobic exercise training group (COMBO, $n = 11$), or sedentary control group (CONTROL, $n = 16$). No endurance-training group was included because the primary focus of the present study was on resistance training. Before the intervention period, there were no significant differences in any of the variables between the groups (Table 1).

Table 1 **Selected subject characteristics at baseline**

Variable	CONTROL group	MODE group	COMBO group
N	16	12	11
Age (years)	22 ± 1	20 ± 1	21 ± 1
Height (cm)	172 ± 1	169 ± 2	171 ± 2
Body weight (kg)	68 ± 2	65 ± 2	66 ± 2
Body mass index (kg/m^2)	22 ± 1	23 ± 1	23 ± 1
Body fat (%)	21 ± 1	18 ± 2	21 ± 1
Lean body mass (kg)	55 ± 2	51 ± 1	53 ± 1
Peak oxygen consumption (ml/kg per min)	49 ± 3	52 ± 2	49 ± 2

Data presented as the mean ± SEM. CONTROL, sedentary control group; MODE, moderate-intensity resistance training group; COMBO, combined high-intensity resistance training and moderate-intensity aerobic exercise training group.

Measurements

The exercise intervention groups were studied five times: before training (baseline), at 2 months (midpoint of exercise training), at 4 months (completion of exercise training), at 6 months (midpoint of detraining), and at 8 months (completion of detraining). The non-exercising control group was studied three times: baseline, at 4 months, and at 8 months. In order to avoid potential diurnal variations, subjects were tested at the same time of day throughout the study period [9,10]. Furthermore, prior to each testing, subjects abstained from caffeine and fasted for at least 4 h; most subjects were studied after overnight fast. Subjects in the intervention groups were studied 20–24 h after their last exercise training session to avoid the acute effects of exercise [12], but while they were still considered to be in their normal (i.e. habitually exercising) physiological state.

Incremental exercise

To demonstrate that the participants had been sedentary, we measured the maximal oxygen consumption during an incremental cycle ergometer exercise [13]. The oxygen consumption, heart rate, and ratings of perceived exertion were measured throughout the protocol.

Strength testing

Maximal muscular strength in the intervention groups was assessed before and after resistance training using the following exercises: half squat, bench press, leg extension, leg curls, lat row, and abdominal bend. After 10 warm-up repetitions, one-repetition maximum (1 RM) values were obtained according to established guidelines. The day-to-day coefficient of variation for 1 RM strength in our laboratory is 4 ± 2%. The 1 RM test was not performed in the control group due to the potential risks involved in the testing.

Body composition

The body composition was determined using the bioelectric impedance method (coefficient of variance, 4 ± 2%) [14].

Arterial blood pressure at rest

Chronic levels of arterial blood pressure at rest were measured with a semi-automated oscillometric device (Form PWV/ABI; Colin Medical, Komaki, Aichi, Japan) over the brachial and dorsalis pedis artery. Recordings were made in triplicate with participants in the supine position.

Carotid artery intima–media thickness

The carotid artery intima–media thickness (IMT) was measured from the images derived from an ultrasound machine equipped with a high-resolution linear-array broad-band transducer as previously described [8]. Ultrasound images were analyzed by use of computerized image analysis software. At least 10 measurements of IMT were taken at each segment, and the mean values were used for analysis. This technique has excellent day-to-day reproducibility (coefficient of variance, $3 \pm 1\%$) for the carotid IMT.

Carotid artery stiffness and compliance

A combination of ultrasound imaging of the pulsatile common carotid artery with simultaneous applanation of tonometrically obtained arterial pressure from the contralateral carotid artery permits non-invasive determination of arterial compliance [10,15]. The carotid artery diameter was measured from images derived from an ultrasound machine equipped with a high-resolution linear-array transducer. A longitudinal image of the cephalic portion of the common carotid artery was acquired 1–2 cm distal to the carotid bulb. To assess the effects of peripheral artery compliance, the same procedure was repeated on the common femoral artery. All image analyses were performed by the same investigator who was blinded to the group assignments.

Pressure waveforms and amplitudes were obtained from the common carotid artery with a pencil-type probe incorporating a high-fidelity strain-gauge transducer (SPT-301; Millar Instruments, Houston, Texas, USA) [10,16]. Because baseline levels of blood pressure are subjected to hold-down force, the pressure signal obtained by tonometry was calibrated by equating the carotid mean arterial and diastolic blood pressure to the brachial artery value [9,10]. In addition to arterial compliance [17], we also calculated the β-stiffness index, which provides an index of arterial compliance adjusted for distending pressure [18]. Arterial compliance and the β-stiffness index were calculated using the equations $[(D_1-D_0)/D_0]/[2(P_1-P_0)] \times \pi \times (D_0)^2$ and $[\ln(P_1/P_0)]/[(D_1-D_0)/D_0]$, where D_1 and D_0 are the maximal and minimum diameters, and P_1 and P_0 are the highest and lowest blood pressures. The blood pressure obtained at the ankle (Form PWV/ABI; Colin Medical) was used to calculate the femoral artery compliance. The day-to-day coefficients of variation were 2 ± 1, 7 ± 3, and 5 ± 2 for the carotid artery diameter, pulse pressure, and arterial compliance, respectively. The coefficient of variance for femoral arterial compliance was $7 \pm 4\%$.

Left ventricular dimensions, mass and function

Echocardiography was used to measure the left ventricular dimensions, wall thickness, and stroke volume according to established guidelines [19] as previously described [8]. The left ventricular mass and stroke volume were normalized for the body surface area. The ratio of the average left ventricular wall thickness to the left ventricular internal end-diastolic diameter was used as an index of relative wall thickness [8].

Exercise training intervention

In the first 4 months of study period, participants in all training groups underwent three supervised resistance training sessions per week. During each training session, participants in the COMBO group completed three sets of 8–12 exercises at 80% of 1 RM and subjects in the MODE group completed three sets of 14–16 exercises at 50% of 1 RM, in the following order: leg extension, seated chest press, leg curls, lateral row, squat, and sit-ups. The resistance of each exercise was increased progressively throughout the resistance training period. The recovery time between exercise bouts was controlled at 2-min intervals. Each resistance training session lasted approximately 45 min. Subjects in the COMBO group performed a cycle exercise at 60% of the maximal heart rate for 30 min immediately after each resistance training session. Training assistants verbally encouraged the subjects and ensured proper form and technique at each exercise session. Participants were instructed to refrain from any other regular exercise during the entire study period. Participants in the sedentary control group were instructed not to alter their normal activity levels throughout the study period.

Statistical analyses

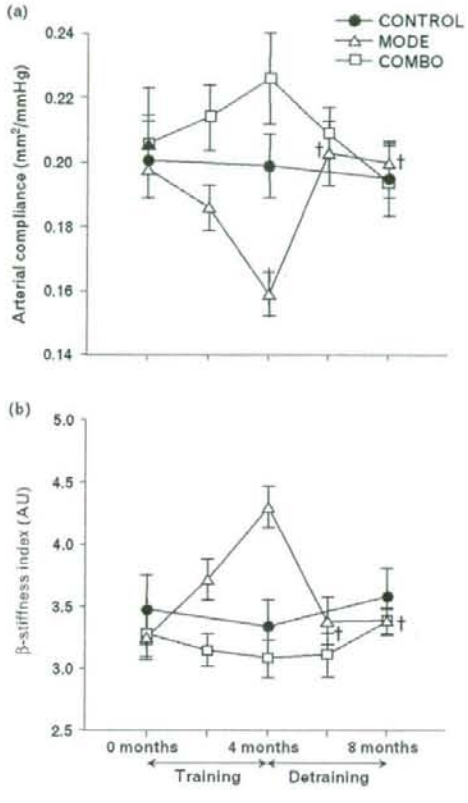
Changes were assessed by two-way analysis of variance (group × time) with repeated measures. In the case of significant F-values, a post-hoc test (Newman–Keuls method) was used to identify significant differences among mean values. To determine whether the changes in arterial compliance and the β-stiffness index were independent of changes in stroke volume, analysis of covariance was performed with stroke volume as the covariate. Pearson's correlation and regression analyses were performed to determine the relation between variables of interest.

Results

Before the intervention period, there were no significant differences in any of the variables between the groups (Table 1). In all groups, there were no changes in height, weight, body mass index, and body surface area throughout the intervention periods.

All the exercise intervention groups increased 1 RM strength significantly in all muscle groups tested ($P < 0.05$ to $P < 0.0001$). Percentage increases in 1 RM strength for the MODE and COMBO groups were 6 and 25% for leg extension, 13 and 14% for leg curl, 10 and 25% for squat, 8 and 17% for lateral row, 6 and 21% for bench press, and 12 and 21% for abdominal bend, respectively.

Fig. 1



Changes in (a) carotid arterial compliance and (b) β-stiffness index for the sedentary control group (CONTROL), the moderate-intensity strength training group (MODE), and the combined aerobic and strength training group (COMBO). Data presented as the mean ± SEM. *$P < 0.05$ versus baseline; †$P < 0.05$ versus 4 months.

Table 2 Hemodynamic and vascular indices

Variable	Baseline	After training	After detraining	Interaction
Brachial systolic blood pressure (mmHg)				
CONTROL group	118 ± 2	119 ± 1	120 ± 2	$F = 2.130$
MODE group	120 ± 3	117 ± 3	115 ± 2	$P = 0.088$
COMBO group	115 ± 2	116 ± 2	115 ± 2	
Brachial diastolic blood pressure (mmHg)				
CONTROL group	68 ± 2	73 ± 2*	73 ± 1	$F = 5.475$
MODE group	71 ± 2	66 ± 2*	68 ± 2	$P > 0.001$
COMBO group	67 ± 1	67 ± 2	67 ± 2	
Brachial pulse pressure (mmHg)				
CONTROL group	49 ± 2	47 ± 1	47 ± 1	$F = 2.407$
MODE group	49 ± 2	52 ± 2	47 ± 2	$P = 0.057$
COMBO group	48 ± 2	49 ± 1	48 ± 1	
Carotid systolic blood pressure (mmHg)				
CONTROL group	101 ± 2	104 ± 2	104 ± 1	$F = 1.653$
MODE group	105 ± 3	105 ± 4	104 ± 3	$P = 0.170$
COMBO group	99 ± 2	97 ± 2	98 ± 2	
Carotid pulse pressure (mmHg)				
CONTROL group	33 ± 2	32 ± 1	32 ± 1	$F = 2.383$
MODE group	36 ± 2	39 ± 3	36 ± 2†	$P = 0.059$
COMBO group	31 ± 1	30 ± 1	32 ± 1	
Carotid lumen diameter (mm)				
CONTROL group	5.91 ± 0.11	5.94 ± 0.14	6.06 ± 0.11	$F = 1.839$
MODE group	6.03 ± 0.13	6.02 ± 0.10	6.02 ± 0.11	$P = 0.131$
COMBO group	5.79 ± 0.09	5.91 ± 0.07	5.81 ± 0.09	
Δ Carotid lumen diameter (mm)				
CONTROL group	0.66 ± 0.03	0.66 ± 0.04	0.63 ± 0.03	$F = 3.460$
MODE group	0.74 ± 0.02	0.66 ± 0.04*	0.76 ± 0.04†	$P = 0.012$
COMBO group	0.71 ± 0.04	0.72 ± 0.03	0.69 ± 0.04	
Carotid intima–media thickness (mm)				
CONTROL group	0.50 ± 0.01	0.52 ± 0.02	0.50 ± 0.02	$F = 1.803$
MODE group	0.46 ± 0.01	0.45 ± 0.02	0.46 ± 0.01	$P = 0.138$
COMBO group	0.47 ± 0.01	0.52 ± 0.01	0.51 ± 0.02	
Femoral compliance (mm²/mmHg)				
CONTROL group	0.10 ± 0.01	0.09 ± 0.01	0.08 ± 0.01	$F = 0.950$
MODE group	0.08 ± 0.01	0.09 ± 0.01	0.08 ± 0.01	$P = 0.441$
COMBO group	0.09 ± 0.01	0.07 ± 0.01	0.07 ± 0.01	

Data presented as the mean ± SEM. CONTROL, sedentary control group; MODE, moderate-intensity resistance training group; COMBO, combined high-intensity resistance training and moderate-intensity aerobic exercise training group. *$P < 0.05$ versus baseline. †$P < 0.05$ versus after the training period.

The magnitude of increases was larger ($P < 0.05$) in the COMBO group than in the MODE group in all exercises except for the leg curl.

There were no significant differences in baseline arterial compliance and β-stiffness index between all four groups (Fig. 1). Carotid arterial compliance decreased after 4 months of MODE interventions ($P < 0.01$). In contrast, arterial compliance did not decrease, but rather tended to increase ($P = 0.06$), after 4 months of the COMBO intervention. Following the detraining period, arterial compliance values returned to the baseline level. Alterations in arterial compliance were primarily due to changes in arterial distension as the carotid pulse pressure remained unchanged (Table 2). In general, qualitatively similar results (although inverse in direction) were obtained by use of the β-stiffness index ($P < 0.01$). The femoral arterial compliance, an index of the compliance of peripheral (muscular) artery, did not change in any groups. In all groups, there were no significant changes in brachial and carotid systolic blood pressures, carotid IMT, and carotid lumen diameter (Table 2).

In all groups, there were no significant changes in heart rate at rest throughout the study period (Table 3). All the resistance training interventions increased the left ventricular mass index and the relative wall thickness ($P < 0.001$). In the COMBO group, the stroke volume index tended to increase during the training period ($P = 0.07$). There were no significant changes in the stroke volume index in any other groups. Following the detraining period, left ventricular structural and functional indices returned to baseline and were no longer significantly different from baseline. There were no such changes in the sedentary control group throughout the study period. To determine whether changes in stroke volume, a determinant of arterial compliance, could confound the interpretation of the present results, we performed several different analyses. When we performed a

Table 3 **Cardiac indices**

Variable	Baseline	4 months	8 months	Interaction
Heart rate at rest (beats/min)				
CONTROL group	58 ± 3	56 ± 2	57 ± 2	$F = 0.254$
MODE group	55 ± 3	54 ± 2	53 ± 2	$P = 0.908$
COMBO group	52 ± 3	48 ± 1	50 ± 1	
Left ventricular mass index (g/m^2)				
CONTROL group	131 ± 7	132 ± 7	131 ± 7	$F = 11.940$
MODE group	139 ± 4	151 ± 4*	137 ± 4†	$P < 0.001$
COMBO group	125 ± 5	143 ± 6*	127 ± 6†	
Relative wall thickness (%)				
CONTROL group	19.5 ± 0.4	19.7 ± 0.4	19.8 ± 0.4	$F = 15.793$
MODE group	19.0 ± 0.5	20.7 ± 0.5*	19.3 ± 0.5†	$P < 0.001$
COMBO group	19.0 ± 1.0	20.2 ± 0.9*	18.9 ± 0.9†	
Stroke volume index (ml/m^2)				
CONTROL group	47 ± 2	47 ± 2	46 ± 2	$F = 1.861$
MODE group	51 ± 1	50 ± 1	50 ± 1	$P = 0.130$
COMBO group	48 ± 2	50 ± 2	48 ± 2	

Data presented as the mean ± SEM. CONTROL, sedentary control group; MODE, moderate-intensity resistance training group; COMBO, combined high-intensity resistance training and moderate-intensity aerobic exercise training group. *$P < 0.05$ versus baseline. †$P < 0.05$ versus 4 months.

univariate correlation analysis between the stroke volume index and carotid arterial compliance in a pooled population, these two functions were not correlated ($r = 0.05$, $P = 0.93$). Additionally, changes in carotid arterial compliance were not associated with changes in stroke volume index in the combined exercise group ($r = 0.19$, $P = 0.26$). Moreover, when analysis of covariance was performed with the stroke volume as the covariate, the overall results on carotid arterial compliance were essentially the same.

Discussion

The major findings of the present study are as follows. First, resistance training performed at a moderate intensity produced a magnitude of arterial stiffening similar to high-intensity resistance training previously reported [9]. Second, concurrently performed endurance training minimized arterial stiffening that was accompanied by high-intensity resistance training. These results suggest that a simultaneously performed aerobic training could negate and prevent the stiffening of carotid arteries caused by resistance training.

Historically, resistance training had been regarded as unsafe for individuals at high risk for future cardiac events because of the abrupt increases in blood pressure and myocardial oxygen demand during high-intensity resistance training [20]. These marked increases in arterial blood pressure during resistance exercise were thought to be initiating factors for arterial stiffening [8]. The majority of recent studies, however, have documented that low to moderate resistance training is a safe and viable form of exercise training as blood pressure increases are within the clinically acceptable range during moderate-intensity resistance training [21]. For these reasons, we hypothesized that resistance training performed at a moderate intensity would not result in a decrease in arterial compliance. In contrast to our working hypothesis, moderate resistance training significantly decreased arterial compliance (from 0.20 ± 0.01 to 0.16 ± 0.01 mm^2/mmHg), and the magnitude of the reduction in arterial compliance was similar to that we previously observed in high-intensity resistance training (from 0.20 ± 0.02 to 0.16 ± 0.01 mm^2/mmHg) [9]. Moreover, these changes in arterial compliance returned to the baseline levels a few months after the cessation of training, confirming that the change in carotid arterial compliance was indeed due to the effect of the moderate resistance training intervention. Furthermore, reductions in arterial compliance were accompanied by significant increases in left ventricular mass index and relative wall thickness, important clinical correlates of arterial stiffening. Even moderate-intensity resistance training therefore appears to stiffen or harden the large elastic arteries. Our present study provides a warning that even moderate resistance training, which is typically recommended to the general public, should be prescribed cautiously, especially for high-risk populations. However, one important consideration that should be emphasized is that the volume (i.e. three sets) of moderate-intensity resistance training used in the present study was still greater than that typically recommended for comprehensive health programs, where only one set of resistance exercises is recommended [6,7]. We therefore cannot exclude the possibility that moderate-intensity resistance training performed with fewer sets may not result in a reduction in arterial compliance.

In contrast to resistance training, regular aerobic exercise is shown to be efficacious in preventing and reversing arterial stiffening in healthy adults [10,11]. We hypothesized that by combining the stiffening effects of resistance training and the destiffening effects of endurance training, both interventions would negate each other and would cause no changes in arterial compliance. In the present study, we demonstrated that simultaneously performed endurance training prevented the reduction in arterial compliance that was accompanied by high-intensity resistance training. Additionally, there was a tendency for arterial compliance to increase with combined endurance and resistance training. From the standpoint of exercise adherence and compliance, this type of 'cross-training' is highly beneficial as it is more enjoyable and breaks the boredom that often results from long-term participation in a single exercise mode [22,23]. Taken together, these findings suggest that combined resistance and aerobic training may be an effective countermeasure for the unfavorable effects of strenuous resistance training.

It is not clear what physiological mechanisms explain the effects of combined training on arterial compliance. Chronic or repeated increases in flow exert their effects on endothelial vasodilatation by modulating the expression of nitric oxide synthase [24]. Carotid arteries

experience increases in blood flow and shear stress during aerobic exercise bouts [25,26], whereas carotid blood flow does not appear to change during resistance exercises [27,28]. Consistent with this, endothelial function is improved with regular aerobic exercise [29,30] as well as with combined resistance and aerobic training [31,32]. Resistance training alone, however, appears to have no effects on flow-mediated vasodilation [33]. One possibility is therefore that the combined aerobic and resistance training may have increased nitric oxide bioavailability, which in turn may have negated the opposing effects of resistance training on the arterial wall. Future studies will be needed to determine the physiological mechanisms underlying the influence of resistance and aerobic training on carotid arterial compliance.

Although endurance training performed concurrently with resistance training prevented the stiffening of carotid arteries, the magnitude of increases was larger in the combined training group than in the moderate-intensity training group in all exercises except for the leg curl. The strength gains were consistently smaller in the combined training group compared with the previously studied high-intensity resistance training alone [9], especially in the lower limbs. This occurred despite the fact that the same training intensity and volume were prescribed to both groups. These results are consistent with a number of previous studies demonstrating that subjects who perform a combination of endurance and strength training achieve lower strength gains than subjects performing weight training alone [34–36]. It should therefore be noted that simultaneous endurance and resistance training may prevent arterial stiffening, but could attenuate optimum gains in muscular strength. In order to minimize the antagonistic effects of endurance training on strength gains, it is recommended that strength and endurance training be performed on alternate days [36]. A smaller strength gain in the combined training group might confound the interpretation of our findings. The moderate-intensity resistance training that achieved much smaller strength gains, however, experienced a similar magnitude of arterial stiffening to the high-intensity training group. The effect of resistance training on arterial compliance therefore does not appear to be dependent upon the training intensity or strength gains.

There are several limitations of the present study that should be emphasized. First, the combined training group that performed moderate-intensity resistance training was not included in the present study. Because simultaneously performed endurance training negated the effects of 'high-intensity' resistance training, however, it is fairly reasonable to assume that it would negate the effects of 'moderate-intensity' (i.e. lesser stimuli) resistance training as well. Second, although arterial compliance and blood pressure often change simultaneously with interventions, changes in arterial compliance were not associated with the corresponding changes in blood pressure in the present study. Because changes in the elastic property of arteries appear to precede changes in blood pressure [37], it is possible that a longer duration of resistance training may have increased blood pressure. Third, we studied relatively small numbers of subjects in each group ($n = 11–16$), and included only young healthy men. Future studies targeting high-risk populations (e.g. the elderly) are needed.

In light of the current recommendation that resistance training should be incorporated into exercise prescription [5–7], the effects of resistance training to stiffen large elastic arteries are of particular concern. We examined two strategies that potentially prevent arterial stiffening associated with resistance training. We demonstrated that moderate-intensity resistance training produced significant reductions in arterial compliance. In contrast, combined resistance and aerobic training did not result in decreases in carotid arterial compliance. These results suggest that in order to negate and prevent the stiffening of carotid arteries caused by resistance training, aerobic training should be performed simultaneously with resistance training.

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References

1 Rajkumar C, Cameron JD, Christophidis N, Jennings GL, Dart AM. Reduced systemic arterial compliance is associated with left ventricular hypertrophy and diastolic dysfunction in older people. *J Am Geriatr Soc* 1997; **45**:803–808.

2 Pak PH, Maughan L, Baughman KL, Kass DA. Marked discordance between dynamic and passive diastolic pressure-volume relations in idiopathic hypertrophic cardiomyopathy. *Circulation* 1996; **94**:52–60.

3 Blacher J, Guerin AP, Pannier B, Marchais SJ, Safar ME, London GM. Impact of aortic stiffness on survival in end-stage renal disease. *Circulation* 1999; **99**:2434–2439.

4 Laurent S, Boutouyrie P, Asmar R, Gautier I, Laloux B, Guize L, *et al.* Aortic stiffness is an independent predictor of all-cause and cardiovascular mortality in hypertensive patients. *Hypertension* 2001; **37**:1236–1241.

5 Diabetes mellitus and exercise. *Diabetes Care* 2000; **23 (Suppl 1)**: S50–S54.

6 American College of Sports Medicine Position Stand. Exercise and physical activity for older adults. *Med Sci Sports Exerc* 1998; **30**: 992–1008.

7 Pollock ML, Franklin BA, Balady GJ, Chaitman BL, Fleg JL, Fletcher B, *et al.* AHA Science Advisory. Resistance exercise in individuals with and without cardiovascular disease: benefits, rationale, safety, and prescription; an advisory from the Committee on Exercise, Rehabilitation, and Prevention, Council on Clinical Cardiology, American Heart Association; position paper endorsed by the American College of Sports Medicine. *Circulation* 2000; **101**:828–833.

8 Miyachi M, Donato AJ, Yamamoto K, Takahashi K, Gates PE, Moreau KL, Tanaka H. Greater age-related reductions in central arterial compliance in resistance-trained men. *Hypertension* 2003; **41**:130–135.

9 Miyachi M, Kawano H, Sugawara J, Takahashi K, Hayashi K, Yamazaki K, *et al.* Unfavorable effects of resistance training on central arterial compliance: a randomized intervention study. *Circulation* 2004; **110**:2858–2863.

10 Tanaka H, Dinenno FA, Monahan KD, Clevenger CM, DeSouza CA, Seals DR. Aging, habitual exercise, and dynamic arterial compliance. *Circulation* 2000; **102**:1270–1275.

11 Tanaka H, DeSouza CA, Seals DR. Absence of age-related increase in central arterial stiffness in physically active women. *Arterioscler Thromb Vasc Biol* 1998; **18**:127–132.

12 DeVan AE, Anton MM, Cook JN, Neidre DB, Cortez-Cooper MY, Tanaka H. Acute effects of resistance exercise on arterial compliance. *J Appl Physiol* 2005; **98**:2287–2291.

13 Miyachi M, Tanaka H, Yamamoto K, Yoshioka A, Takahashi K, Onodera S. Effects of one-legged endurance training on femoral arterial and venous size in healthy humans. *J Appl Physiol* 2001; **90**:2439–2444.

14 Bolanowski M, Nilsson BE. Assessment of human body composition using dual-energy x-ray absorptiometry and bioelectrical impedance analysis. *Med Sci Monit* 2001; **7**:1029–1033.

15 Lage SG, Polak JF, O'Leary DH, Creager MA. Relationship of arterial compliance to baroreflex function in hypertensive patients. *Am J Physiol* 1993; **265**:H232–H237.

16 Kelly R, Hayward C, Avolio A, O'Rourke M. Noninvasive determination of age-related changes in the human arterial pulse. *Circulation* 1989; **80**:1652–1659.

17 Van Merode T, Hick PJ, Hoeks AP, Rahn KH, Reneman RS. Carotid artery wall properties in normotensive and borderline hypertensive subjects of various ages. *Ultrasound Med Biol* 1988; **14**:563–569.

18 Hirai T, Sasayama S, Kawasaki T, Yagi S. Stiffness of systemic arteries in patients with myocardial infarction. A noninvasive method to predict severity of coronary atherosclerosis. *Circulation* 1989; **80**:78–86.

19 Cheitlin MD, Alpert JS, Armstrong WF, Aurigemma GP, Beller GA, Bierman FZ, *et al.* ACC/AHA Guidelines for the Clinical Application of Echocardiography. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Clinical Application of Echocardiography). Developed in colloboration with the American Society of Echocardiography. *Circulation* 1997; **95**:1686–1744.

20 MacDougall J. Morphological changes in human skeletal muscle following strength training and immobilization. In: Jones NLM, McComas AJ, editors. *Human muscle power*. Champaign, IL: Human Kinetics; 1986. pp. 269–285.

21 Haslam DRS, McCartney N, McKelvie RS, MacDougall JD. Direct measurements of arterial blood pressure during formal weightlifting in cardiac patients. *J Cardiopulm Rehab* 1988; **8**:213–225.

22 Tanaka H. Effects of cross-training. Transfer of training effects on VO2 max between cycling, running and swimming. *Sports Med* 1994; **18**:330–339.

23 Tanaka H, Swensen T. Impact of resistance training on endurance performance. A new form of cross-training? *Sports Med* 1998; **25**:191–200.

24 Delp MD, McAllister RM, Laughlin MH. Exercise training alters endothelium-dependent vasoreactivity of rat abdominal aorta. *J Appl Physiol* 1993; **75**:1354–1363.

25 Jiang ZL, Yamaguchi H, Tanaka H, Takahashi A, Tanabe S, Utsuyama N, *et al.* Blood flow velocity in the common carotid artery in humans during graded exercise on a treadmill. *Eur J Appl Physiol Occup Physiol* 1995; **70**:234–239.

26 Ogoh S, Fadel PJ, Zhang R, Selmer C, Jans O, Secher NH, Raven PB. Middle cerebral artery flow velocity and pulse pressure during dynamic exercise in humans. *Am J Physiol Heart Circ Physiol* 2005; **288**:H1526–H1531.

27 Dickerman RD, McConathy WJ, Smith GH, East JW, Rudder L. Middle cerebral artery blood flow velocity in elite power athletes during maximal weight-lifting. *Neurol Res* 2000; **22**:337–340.

28 Edwards MR, Martin DH, Hughson RL. Cerebral hemodynamics and resistance exercise. *Med Sci Sports Exerc* 2002; **34**:1207–1211.

29 Clarkson P, Montgomery HE, Mullen MJ, Donald AE, Powe AJ, Bull T, *et al.* Exercise training enhances endothelial function in young men. *J Am Coll Cardiol* 1999; **33**:1379–1385.

30 Fuchsjäger-Mayrl G, Pleiner J, Wiesinger GF, Sieder AE, Quittan M, Nuhr MJ, *et al.* Exercise training improves vascular endothelial function in patients with type 1 diabetes. *Diabetes Care* 2002; **25**:1795–1801.

31 Walsh JH, Bilsborough W, Maiorana A, Best M, O'Driscoll GJ, Taylor RR, Green DJ. Exercise training improves conduit vessel function in patients with coronary artery disease. *J Appl Physiol* 2003; **95**:20–25.

32 Maiorana A, O'Driscoll G, Cheetham C, Dembo L, Stanton K, Goodman C, *et al.* The effect of combined aerobic and resistance exercise training on vascular function in type 2 diabetes. *J Am Coll Cardiol* 2001; **38**:860–866.

33 Rakobowchuk M, McGowan CL, de Groot PC, Hartman JW, Phillips SM, MacDonald MJ. Endothelial function of young healthy males following whole body resistance training. *J Appl Physiol* 2005; **98**:2185–2190.

34 Dudley GA, Djamil R. Incompatibility of endurance- and strength-training modes of exercise. *J Appl Physiol* 1985; **59**:1446–1451.

35 Hickson RC. Interference of strength development by simultaneously training for strength and endurance. *Eur J Appl Physiol Occup Physiol* 1980; **45**:255–263.

36 Sale DG, Jacobs I, MacDougall JD, Garner S. Comparison of two regimens of concurrent strength and endurance training. *Med Sci Sports Exerc* 1990; **22**:348–356.

37 Liao D, Arnett DK, Tyroler HA, Riley WA, Chambless LE, Szklo M, Heiss G. Arterial stiffness and the development of hypertension. The ARIC study. *Hypertension* 1999; **34**:201–206.

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Short Communication

Comparison of Muscle Strength between Japanese Men with and without Metabolic Syndrome

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We compared muscle strength between Japanese men with and without metabolic syndrome. We used data for 323 Japanese men with metabolic syndrome and 893 Japanese men without the syndrome. Metabolic syndrome was defined by a new criterion in Japan, and the parameters for muscle strength, *i.e.* grip strength, leg strength were measured. Leg strength was found to be significantly higher in subjects with metabolic syndrome than in those without, while muscle strength per body weight was significantly lower in subjects with the syndrome. Lower muscle strength per body weight may be one of the characteristic features in subjects with metabolic syndrome.

Key words: metabolic syndrome, grip strength, leg strength

Metabolic syndrome is a common disorder and has become a public challenge in Japan. For example, 30.7% of men and 3.6% of women have been diagnosed as having metabolic syndrome using the new criterion in Japan [1]. The metabolic syndrome has been associated with an increased risk of cardiovascular disease [2], proteinuria [3], and elevation of hepatic enzymes [4]. Lifestyle modifications, especially exercise, are important for preventing and improving metabolic syndrome. However, the link between metabolic syndrome using the new criterion in Japan and muscle strength remains to be investigated. In this study, we compared muscle strength between Japanese men with and without metabolic syndrome.

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Subjects and Methods

Subjects. We used the data for 1,216 Japanese men, aged 20-79 years, who met the following criteria, 1) received annual health checkups from June 1997 to May 2005 at Okayama Southern Institute of Health, 2) received fasting blood examination and muscle strength measurements, and 3) obtained written informed consent.

Anthropometric measurements. Anthropometric parameters *i.e.* height, weight, and waist circumference were measured. The waist circumference was measured at the umbilical level.

Definition of metabolic syndrome. Meta-

bolic syndrome was defined, among men with a waist circumference in excess of 85 cm, as having 2 or more components from the following: 1) Dyslipidemia: triglycerides ≧ 150 mg/dl and/or HDL cholesterol < 40 mg/dl, 2) High blood pressure: blood pressure ≧ 130/85 mmHg, 3) Impaired glucose tolerance: fasting plasma glucose ≧ 110 mg/dl [5].

Measurements of muscle strength. To assess muscle strength, grip and leg strength were measured. Grip strength was measured by using THP-10 (SAKAI, Tokyo, Japan), while leg strength was measured by COMBIT CB-1 (MINATO, Osaka, Japan). Isometric leg strength was measured as follows: the subject sat in a chair, grasping the armrest in order to fix the body position. The dynamometer was then attached to the subject's ankle joint by a strap. They next extended the leg to 60 degrees [6]. In addition, to standardize the influence of the total body weight, we calculated the muscle strength (kg) per body weight (kg) [7].

Statistical analysis. Data are expressed as mean ± standard deviation (SD) values. A comparison of parameters between the 2 groups was made using the unpaired t-test and covariance analysis: $p < 0.05$ was considered to be statistically significant.

Results

A total of 323 men (26.6%) were diagnosed as having metabolic syndrome, and the measurements of muscle strength in subjects with and without metabolic syndrome (n = 893) are indicated in the Table. The age was significantly higher in subjects with metabolic syndrome and thus, to avoid the influence of age on muscle strength, we used the age as a covariate and compared the muscle strength using covariance analysis. In subjects with metabolic syndrome, leg strength was significantly higher compared with subjects without metabolic syndrome. However, muscle strength per body weight was significantly lower in subjects with metabolic syndrome.

We then analyzed the groups with and without each component of metabolic syndrome. The age was significantly higher in subjects with abdominal obesity, impaired glucose tolerance, dyslipidemia, and high blood pressure compared with the subjects without each component. Based on the comparison of muscle strength adjusting for age, leg strength was significantly higher in subjects with abdominal obesity, dyslipidemia, and high blood pressure. In subjects with impaired glucose tolerance, leg strength was significantly lower than in subjects without impaired glucose tolerance. In subjects with abdominal obesity, the left grip strength was significantly higher compared with subjects without abdominal obesity. However, the leg strength per body weight in subjects with abdominal obesity was significantly lower. In addition, the grip strength per body weight in subjects with abdominal obesity, dyslipidemia, and high blood pressure was also significantly lower.

Discussion

We compared muscle strength in metabolic syndrome men with that in non-metabolic syndrome men using the criterion in Japan.

In some literature, cardiorespiratory fitness is closely associated with metabolic syndrome [8, 9]. However, the relationship between muscle strength and metabolic syndrome, especially using the new criterion in Japan, has not been clearly investigated. Jurca R *et al.* have reported examining the associations for muscle strength and cardiorespiratory fitness with the prevalence of metabolic syndrome by cross sectional [10] and longitudinal study [11]. They concluded that muscle strength has an inverse association with metabolic syndrome prevalence using the National Cholesterol Education Program (NCEP) definition. In this study, by using the new criterion in Japan, leg strength was found to be significantly higher in subjects with metabolic syndrome than in those without the syndrome. However, muscle strength per body weight was significantly lower in subjects with metabolic syndrome than that in those without the syndrome. Leg strength per body weight in subjects with abdominal obesity was significantly lower, and grip strength per body weight in subjects with abdominal obesity, dyslipidemia, and high blood pressure was also significantly lower. These findings may stress the clinical significance of such components on muscle strength per body weight in subjects with metabolic syndrome. Although aerobic exercise has been advocated as the most suitable exercise for metabolic syndrome, it is difficult for subjects with

Table 1 Comparison of parameters between subjects with and without metabolic syndrome

	Mean ± SD		p Unpaired t test	p Adjusting for age
	Metabolic syndrome (+)	Metabolic syndrome (−)		
Number of subjects	323	893		
Age	49.4 ± 11.0	45.4 ± 12.5	< 0.0001	
Right grip strength (kg)	44.3 ± 8.2	43.7 ± 8.4		0.3232
Left grip strength (kg)	42.3 ± 8.0	41.9 ± 7.6		0.1779
Leg strength (kg)	67.8 ± 17.7	65.2 ± 17.0		0.0016
Right grip strength(kg)/body weight(kg)	0.56 ± 0.10	0.65 ± 0.12		< 0.0001
Left grip strength(kg)/body weight(kg)	0.54 ± 0.10	0.62 ± 0.11		< 0.0001
Leg strength(kg)/body weight(kg)	0.86 ± 0.20	0.96 ± 0.22		0.0058
	Waist circumference (+)	Waist circumference (−)		
Number of subjects	600	616		
Age	47.6 ± 11.1	45.3 ± 13.1	0.0013	
Right grip strength (kg)	45.0 ± 8.3	42.7 ± 8.3		0.2376
Left grip strength (kg)	43.1 ± 7.8	40.9 ± 7.5		0.0130
Leg strength (kg)	69.0 ± 17.2	62.9 ± 16.6		0.0002
Right grip strength(kg)/body weight(kg)	0.58 ± 0.10	0.67 ± 0.12		< 0.0001
Left grip strength(kg)/body weight(kg)	0.55 ± 0.11	0.64 ± 0.11		< 0.0001
Leg strength(kg)/body weight(kg)	0.88 ± 0.20	0.99 ± 0.23		< 0.0001
	Impaired glucose tolerance (+)	Impaired glucose tolerance (−)		
Number of subjects	282	934		
Age	51.5 ± 10.7	44.9 ± 12.2	< 0.0001	
Right grip strength (kg)	41.7 ± 8.0	44.5 ± 8.3		0.0553
Left grip strength (kg)	39.9 ± 7.9	42.6 ± 7.6		0.1340
Leg strength (kg)	62.7 ± 17.7	66.9 ± 16.9		0.0221
Right grip strength(kg)/body weight(kg)	0.58 ± 0.11	0.64 ± 0.12		0.1935
Left grip strength(kg)/body weight(kg)	0.56 ± 0.11	0.61 ± 0.11		0.0565
Leg strength(kg)/body weight(kg)	0.87 ± 0.21	0.95 ± 0.22		0.4832
	Dyslipidemia (+)	Dyslipidemia (−)		
Number of subjects	577	639		
Age	47.6 ± 11.7	45.4 ± 12.6	0.0014	
Right grip strength (kg)	43.2 ± 8.5	44.4 ± 8.1		0.3572
Left grip strength (kg)	41.5 ± 8.0	42.4 ± 7.5		0.2205
Leg strength (kg)	65.3 ± 17.8	66.4 ± 16.6		0.0155
Right grip strength(kg)/body weight(kg)	0.60 ± 0.11	0.65 ± 0.11		0.0034
Left grip strength(kg)/body weight(kg)	0.57 ± 0.11	0.62 ± 0.11		0.0055
Leg strength(kg)/body weight(kg)	0.90 ± 0.22	0.97 ± 0.22		0.3452
	High blood pressure (+)	High blood pressure (−)		
Number of subjects	703	513		
Age	48.9 ± 11.8	43.1 ± 11.9	< 0.0001	
Right grip strength (kg)	43.7 ± 8.4	44.0 ± 8.2		0.2065
Left grip strength (kg)	42.0 ± 8.0	42.0 ± 7.4		0.0843
Leg strength (kg)	65.9 ± 17.7	65.9 ± 16.5		0.0001
Right grip strength(kg)/body weight(kg)	0.61 ± 0.11	0.65 ± 0.12		0.0006
Left grip strength(kg)/body weight(kg)	0.58 ± 0.11	0.62 ± 0.11		0.0020
Leg strength(kg)/body weight(kg)	0.91 ± 0.22	0.97 ± 0.22		0.8945

lower leg strength per body weight to support their entire body weight, and it is also difficult to carry out aerobic exercise *i.e.* walking and jogging. In addition, resistance training increases muscle quantity and insulin action [12, 13] and reduces visceral adipose tissue [14]. These findings suggest that resistance exercise training should be considered in primary prevention of metabolic syndrome.

Potential limitations remain in our study. First, the cross-sectional study design in our study makes it difficult to infer causality between metabolic syndrome and muscle strength. Second, although reductions in basal leg blood flow [15] and resting metabolic rate [16] have been implicated in the pathogenesis of metabolic syndrome, we could not prove the mechanism of the link between metabolic syndrome and muscle strength. Therefore, our findings are applicable to clinical and public health practice settings. In conclusion, lower muscle strength per body weight is characteristic in Japanese men with metabolic syndrome. Further intervention studies are necessary to test the effects of the prevention and treatment of metabolic syndrome.

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References

1. Miyatake N, Kawasaki Y, Nishikawa H, Takenami S and Numata T: Prevalence of metabolic syndrome in Okayama prefecture, Japan. Intern Med (2006) 45: 107–108.
2. Wilson PW, Kannel WB, Silbershatz H and D'Agostino RB: Clustering of metabolic factors and coronary heart disease. Arch Intern Med (1999) 159: 1104–1109.
3. Miyatake N, Wada J, Kawasaki Y, Matsumoto S, Makino H and Numata T: Relationship between metabolic syndrome and proteinuria in the Japanese population. Intern Med (2006) 45: 599–603.
4. Miyatake N, Matsumoto S, Makino H and Numata T: Comparison of hepatic enzymes between Japanese men with and without metabolic syndrome. Acta Med Okayama (2007) 61: 31–34.
5. Definition and the diagnostic standard for metabolic syndrome-Committee to evaluate diagnostic standards for metabolic syndrome. Nippon Naika Gakkai Zasshi (2005) 94: 794–809 (in Japanese).
6. Kigawa A, Yamamoto T, Koyama Y, Kageyama S and Arima K: Evaluation of knee extensor strength for prevention of sports injury. Japan Ortho Soc Sports Med (1987) 6: 141–145 (in Japanese).
7. Miyatake N, Takahashi K, Wada J, Nishikawa H, Morishita A, Suzuki H, Kunitomi M, Makino H, Kira S and Fujii M: Changes in serum leptin concentrations in overweight men after exercise. Diabetes Obes Metab (2004) 6: 332–337.
8. Laaksonen DE, Lakka HM, Salonen JT, Niskanen LK, Rauramaa R and Lakka T: Low levels of leisure-time physical activity and cardiorespiratory fitness predict development of the metabolic syndrome. Diabetes Care (2002) 25: 1612–1618.
9. Katzmarzyk PT, Lenon AS, Wilmore JH, Skinner JS, Rao DC, Rankinen T and Bouchard C: Targeting the metabolic syndrome with exercise: evidence from the HERITAGE Family Study. Med Sci Sports Exerc (2003) 35: 1703–1709.
10. Jurca R, Lamonte MJ, Church TS, Earnest CP, Fitzgerald SJ, Barlow CE, Jordan AN, Kampert JB and Blair SN: Associations of muscle strength and fitness with metabolic syndrome in men. Med Sci Sports Exerc (2004) 36: 1301–1307.
11. Jurca R, lamonte MJ, Barlow CE, Kampert JB, Church TS and Blair SN: Association of muscular strength with incidence of metabolic syndrome. Med Sci Sports Exerc (2005): 37: 1849–1855.
12. Andersen JL, Schjerling P, Andersen LL and Dela F: Resistance training and insulin action in humans: effects of de-training. J Physiol (2003) 551: 1049–1058.
13. Holten MK, Zacho M, Gaster M, Juel C, Wojtaszewski JF and Dela F: Strength training increases insulin-mediated glucose uptake, GLUT4 content, and insulin signaling in skeletal muscle in patients with type 2 diabetes. Diabetes (2004) 294–305.
14. Park SK, Park JH, Kwon YC, Kim HS, Yoon MS and Park HT: The effect of combined aerobic and resistance exercise training on abdominal fat in obese middle-aged women. J Physiol Anthoropol Appl Human Sci (2003) 22: 129–135.
15. Miyachi M, Tanaka H, Kawano H, Okajima M and Tabata I: Lack of age-related decreases in basal whole leg blood flow in resitance-trained men. J Appl Physiol (2005) 99: 1384–1390.
16. Toth MJ and Poehlman ET: Resting metabolic rate and cardiovascular disease risk in resistance- and aerobic-trained middle-aged women. Int J Obes Relat Metab Disord (1985) 19: 691–698.

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Comparison of ventilatory threshold and exercise habits between Japanese men with and without metabolic syndrome

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Abstract

Objective: We compared the levels of ventilatory threshold (VT) and exercise habits in subjects with metabolic syndrome with those in age, sex-matched subjects without metabolic syndrome.
Methods: We used data of 155 Japanese men (47.1 ± 9.2 years) with metabolic syndrome; the diagnosis was given by the definition and the diagnostic standard for metabolic syndrome in Japan. The influence of metabolic syndrome on oxygen uptake, work rate and heart rate at VT, and exercise habits were evaluated.
Results: Oxygen uptake and work rate at VT in subjects with metabolic syndrome were significantly lower than those in subjects without metabolic syndrome even after adjusting for body mass index (BMI). The number of subjects with exercise habits was significantly lower in metabolic syndrome. The subjects with exercise habits were significantly older than that in subjects without exercise habits. Furthermore, oxygen uptake and work rate at VT were significantly higher in subjects with exercise habits than those in subjects without exercise habits.
Conclusion: Lower level of VT was characteristic in subjects with metabolic syndrome. Promotion of exercise habits is necessary for preventing and improving metabolic syndrome in Japanese men.

Keywords: Metabolic syndrome; Ventilatory threshold; Exercise habits

1. Introduction

Metabolic syndrome is characterized by abdominal obesity, high blood pressure, dyslipidemia and impaired glucose tolerance [1]. New criterion in Japan has been defined in April 2005 and 30.7% in men and 3.6% in women are diagnosed as having metabolic syndrome using the new criterion in Japan [1,2]. Exercise is considered as a useful method for preventing metabolic syndrome and improving each component of metabolic syndrome. The ventilatory threshold (VT) is defined as the upper limit of the aerobic exercise and is thought to serve as an accurate and reliable standard for exercise prescription [3]. Since the exercise intensity at VT is not harmful to cardiovascular function, it can be safely applied to patients with myocardial infarction as exercise prescription [4]. However, the relationship

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between exercise habits and metabolic syndrome, also between physical fitness such as aerobic exercise level defined by VT and metabolic syndrome using the new criterion in Japan are not fully discussed.

In this study, we evaluated the parameters at VT and exercise habits between Japanese men with and without metabolic syndrome.

2. Subjects and methods

2.1. Subjects

The total number of Japanese men with metabolic syndrome, aged 24–68 years, was 155 and they were enrolled into annual health check-ups at Okayama Southern Institute of Health with written informed consent. They were compared with 115 men who were age and sex matched and without metabolic syndrome.

2.2. Anthropometric measurements

The anthropometric measurements were performed by using the following parameters such as height, body weight, body mass index (BMI) and waist circumference. BMI was calculated by weight/[height]^2 (kg/m^2). The waist circumference was measured at the umbilical level [5].

2.3. Blood pressure measurements

Blood pressure of each participant was measured after resting at least 15 min in the sitting position.

2.4. Blood sampling and assays

We measured overnight fasting serum levels of high density lipoprotein (HDL) cholesterol, triglycerides (L Type Wako Triglyceride H, Wako Chemical, Osaka) and plasma glucose.

2.5. Definition of metabolic syndrome

The syndrome was defined [1], among men with a waist circumference in excess of 85 cm and women with a waist circumference in excess of 90 cm [6], as having 2 or more components from among the following: (1) dyslipidemia: triglyceride $\geqq$150 mg/dl and/or HDL cholesterol $\geqq$40 mg/dl, (2) high blood pressure: blood pressure $\geqq$130/85 mmHg, (3) impaired glucose tolerance: fasting plasma glucose $\geqq$110 mg/dl.

2.6. Exercise testing

A graded ergometer exercise protocol [7] was performed. Two hours after breakfast, a resting ECG was recorded and blood pressure was measured. Then, all participants were given graded exercise after 3 min of pedaling on an unloaded bicycle ergometer (Excalibur V2.0, Lode BV, Groningen, Netherlands). The profile of incremental workloads was automatically defined by the methods of Jones [7], in which the workloads reach the predicted $\dot{V}O_2$ max in 10 min. A pedaling cycle of 60 rpm was maintained. Loading was terminated when the appearance of symptoms forced the subject to stop. During the test, ECG was monitored continuously together with the recording of heart rate (HR). Expired gas was collected and rates of oxygen consumption ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$) were measured breath-by-breath using a cardiopulmonary gas exchange system (Oxycon Alpha, Mijnhrdt b.v., Netherlands). VT was determined by the standard of Wasserman et al. [3], Davis et al. [8], and the V-slope method of Beaver [9] from $\dot{V}O_2$, $\dot{V}CO_2$ and minute ventilation ($\dot{V}E$). At VT, $\dot{V}O_2$ (ml/(kg min)), work rate (W), and heart rate (beats/min) were measured and recorded.

2.7. Exercise habits

The data on exercise habits were obtained at interviews by well-trained staff in a structured way according to the National Nutrition Survey in Japan [10]. The subjects were asked if they currently exercise (over the level of 30 min per session, two times per week and prolonged duration for 3 months). When the answer was "yes", they were classified as subjects with exercise habits. When the answer was "no", they were classified as subjects without exercise habits.

2.8. Statistical analysis

Data are expressed as mean ± standard deviation (S.D.) values. Relationship between metabolic syndrome and exercise habits was tested using χ^2-test and comparison of parameters between two groups was used by unpaired t-test: $P < 0.05$ was considered to be statistically significant.

3. Results

Table 1 shows the comparison of age, body weight, BMI and parameters at VT between subjects with metabolic ($n = 155$) and without metabolic syndrome ($n = 155$). There was no significant difference of age between the subjects with and without metabolic syndrome. Oxygen uptake at VT and work rate at VT in subjects with metabolic syndrome was significantly lower than those in subjects without metabolic syndrome. Heart rate at VT in subjects with metabolic syndrome was similar to that in subjects without metabolic syndrome.

We also compared the levels of parameters at VT between the groups with and without each component of definition of metabolic syndrome in Japan (Table 2). Of 310 subjects, 56 subjects were diagnosed as having type 2 diabetes mellitus (fasting plasma glucose

Table 1
Comparison of parameters at VT between metabolic and non-metabolic subjects in men

	Metabolic syndrome (+)	Metabolic syndrome (−)
Number of subjects	155	155
Age	47.1 ± 9.2	47.1 ± 9.2
Body weight (kg)	80.6 ± 12.4	72.9 ± 11.1[a]
BMI (kg/m^2)	28.0 ± 3.6	25.6 ± 3.4[a]
Oxygen uptake at VT (ml/(kg min))	14.2 ± 2.7	16.1 ± 3.6[a]
Work rate at VT (W)	75.6 ± 17.1	80.7 ± 22.5[b]
Heart rate at VT (beat/min)	105.1 ± 12.3	106.5 ± 11.3

VT: ventilatory threshold, BMI: body mass index.
[a] $P < 0.01$ vs. metabolic syndrome (+).
[b] $P < 0.05$ vs. metabolic syndrome (+).

≦126 mg/dl). There was no significant difference of age between subjects with or without abdominal obesity or dyslipidemia and high blood pressure. However, there was a significant difference of age between subgroups with or without impaired glucose tolerance. Oxygen uptake at VT in subjects with abdominal obesity, dyslipidemia, impaired glucose tolerance and high blood pressure were significantly lower than those in subjects without such components of metabolic syndrome. Work rate at VT and heart rate at VT in subjects with impaired glucose tolerance were also significantly lower than those in subjects without impaired glucose tolerance. In addition, we compared the levels of parameters at VT between the groups with and without various combinations of each component (Table 2). Oxygen uptake at VT in subjects with two or

Table 2
Comparison of parameters at VT with and without subcriterion of metabolic syndrome in men

	Waist circum ference (+)	Waist circum ference (−)
Number of subjects	243	67
Age	47.0 ± 8.9	47.5 ± 10.0
Oxygen uptake at VT (ml/(kg min))	14.4 ± 2.7	17.8 ± 3.8[a]
Work rate at VT (W)	78.1 ± 19.3	78.4 ± 23.0
Heart rate at VT (beat/min)	105.2 ± 11.9	108.0 ± 11.3
	Dyslipidemia (+)	Dyslipidemia (−)
Number of subjects	175	135
Age	46.4 ± 8.8	48.0 ± 9.6
Oxygen uptake at VT (ml/(kg min))	14.7 ± 3.1	15.7 ± 3.6 a
Work rate at VT (W)	76.5 ± 18.5	80.3 ± 21.9
Heart rate at VT (beat/min)	106.0 ± 12.0	105.4 ± 11.6
	Impaired glucose tolerance (+)	Impaired glucose tolerance (−)
Number of subjects	112	198
Age	49.5 ± 9.0	45.8 ± 9.0[a]
Oxygen uptake at VT (ml/(kg min))	14.1 ± 2.7	15.7 ± 3.5[a]
Work rate at VT (W)	73.8 ± 16.6	80.7 ± 21.5[a]
Heart rate at VT (beat/min)	104.0 ± 11.4	106.8 ± 12.0[b]
	High blood pressure (+)	High blood pressure (−)
Number of subjects	231	79
Age	47.6 ± 8.9	45.7 ± 9.8
Oxygen uptake at VT (ml/(kg min))	14.8 ± 3.0	16.2 ± 3.9[a]
Work rate at VT (W)	77.8 ± 19.6	79.2 ± 21.5
Heart rate at VT (beat/min)	105.1 ± 11.8	107.7 ± 11.6
	Dyslipidemia (+) and impaired glucose tolerance (+)	Dyslipidemia (−) and/or impaired glucose tolerance (−)
Number of subjects	67	243
Age	47.8 ± 8.8	46.9 ± 9.3
Oxygen uptake at VT (ml/(kg min))	14.0 ± 2.8	15.4 ± 3.4[a]
Work rate at VT (W)	74.9 ± 18.6	79.1 ± 20.4
Heart rate at VT (beat/min)	105.8 ± 11.3	105.8 ± 12.0