Original Article

Relationship between aerobic capacity and cardiovascular disease risk factors in Thai men and women with normolipidemia and dyslipidemia

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Abstract. [Purpose] This research aimed to investigate the relationship between aerobic capacity ($VO_{2,peak}$) and cardiovascular risk factors in normolipidemic and dyslipidemic Thai men and women. [Subjects and Methods] We recruited 104 dyslipidemic and 100 healthy participants. Fasting blood samples were analyzed for lipid and blood glucose levels. Anthropometry, blood pressure, and body composition were measured before exercise. Each subject underwent exercise testing to determine $VO_{2,\ peak}$. Heart rate (HR) was recorded throughout the exercise test. [Results] Dyslipidemic participants had a lower $VO_{2,\ peak}$ than normolipidemic participants (p<0.01). In normolipidemic male participants, $VO_{2,\ peak}$ was positively correlated with high density lipoprotein cholesterol (HDL-C) levels and negatively correlated with low density lipoprotein cholesterol (LDL-C) levels and triglycerides to HDL-cholesterol (TG/HDL-C) ratios; in females, $VO_{2,\ peak}$ was negatively correlated with age, total cholesterol, and LDL-C. In dyslipidemic males, $VO_{2,\ peak}$ was positively correlated with HDL-C levels and negatively correlated with age, LDL-C and TG levels, and percent body fat; in females, $VO_{2,\ peak}$ was positively correlated with resting HR and heart rate recovery and negatively correlated with age, TG/HDL-C, and waist circumference. [Conclusion] There was a relationship between aerobic capacity and cardiovascular disease risk factors in both normolipidemic and dyslipidemic participants. This relationship was affected by gender.
Key words: Peak oxygen consumption, Lipid profiles, Heart rate

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INTRODUCTION

Dyslipidemia is a noncommunicable disease characterized by abnormal blood lipid levels[1], in addition to elevated levels of total cholesterol (TC), triglycerides (TG), low density lipoprotein cholesterol (LDL-C), or decreased levels of high density lipoprotein cholesterol (HDL-C). The disease is associated with a high risk of cardiovascular disease (CVD)[2], which is a leading cause of death as a result of atherosclerosis[3–5]. Dyslipidemia also contributes to a loss of endothelial derived nitric oxide, resulting in reduced exercise-induced nitric oxide production[6]. This decreases endothelial vasodilation, which in turn decreases blood flow, leading to increased CVD risk[7, 8]. In addition, graded maximal exercise reduces vasodilation via exercise-induced oxidative stress[9]. Both mechanisms decrease oxygen transport capacity of the vasculature, either at the peripheral vasculature or by centrally affecting the heart[10]. Thus, aerobic capacity or maximal oxygen consumption ($VO_{2,\ max}$) is normally used to determine cardiorespiratory fitness[11–15]. A low $VO_{2,\ max}$ is associated with CVD risk factors (i.e., lipid profiles[16]) such as obesity[17, 18] and hypertension[19]. Sex additionally affects CVD risk[20, 21]. A previous study reported that waist circumference (WC) had better discriminative potential than waist to hip circumference ratio (WHR) in women but did not perform significantly better than body mass index (BMI) in either gender[21]. Moreover, the WC of Malaysian men and women is a better discriminator of metabolic syndrome than WHR. However, no studies have investigated the correlation between $VO_{2,\ peak}$ and CVD risk factors, nor the effect of gender on this correlation, in Thai patients with dyslipidemia, despite there being differences

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between ethnicities in heart health behaviors, risk factors for CVD, and cardiopulmonary fitness[20]. Therefore, this study aimed to investigate the correlation between $VO_{2, peak}$ and CVD risk factors, including anthropometry, body composition, and hemodynamic factors, in both healthy, sedentary and dyslipidemic Thai participants.

SUBJECTS AND METHODS

We enrolled 104 dyslipidemic Thai men and women (aged 47 ± 6.3 years) and 100 healthy, sedentary, normolipidemic Thai men and women (aged 47 ± 7.1 years) for this study. Individuals with dyslipidemia were diagnosed according to the following criteria: total cholesterol (TC) levels ≥ 240 mg/dL, TG levels ≥ 200 mg/dL, LDL-C levels ≥ 160 mg/dL, and HDL-C levels < 35 mg/dL. No participants had underlying CVD, hypertension, diabetes mellitus, orthopedic problems, neuromuscular disorder, liver disease, kidney disease, or infections, nor had anyone received any medicine for dyslipidemia. This study was approved by the Ethical Committee of Khon Kaen University in accordance with the 1964 Declaration of Helsinki (revised in 1983), and written informed consent was obtained from each subject.

All participants were asked to refrain from consumption of alcohol or tobacco during the 12 hours prior to testing. Drinking tea or coffee was not allowed for at least 2 hours before the trial. To ensure consistent baseline activity levels, participants were also asked to refrain from strenuous exercise beginning 48 hours prior to the trial. Participants then underwent a routine medical examination, including completion of a health-risk questionnaire, to obtain medical history, an examination of cardiac function using 12-lead electrocardiography, and blood pressure (BP) measurement. Blood samples were obtained from an antecubital vein and glucose levels, urea nitrogen, creatinine, uric acid, albumin, aspartate aminotransferase, alanine aminotransferase, and alkaline phosphatase measured to determine kidney and liver function. TC, TG, and HDL-C were measured using a chemistry analyzer (Beckman Synchron CX4; Beckman Coulter, Inc., Brea, CA, USA), and LDL-C was calculated using the Friedewald equation (LDL-C = TC − HDL-C − $0.20 \times$ TG). All blood chemistry parameters were analyzed in the chemistry laboratory of Srinagarind Hospital, Faculty of Medicine, Khon Kaen University, Thailand. Participants were asked to record their food intake and physical activity for 3 days (2 weekdays and 1 weekend day), which was used to calculate their daily food intake and amount of physical activity 1 week before the start of the experiment. Dietary records were analyzed using INMUCAL software (Mahidol University, Thailand).

$VO_{2, peak}$ test: In this test, each subject performed a continuous, 3-minute graded exercise protocol (increased by 15 Watts for women and 20 Watts for men) on an electrically braked cycle ergometer (Corival, Lode B.V., Groningen, the Netherlands). The initial work rates, determined according to participants' fitness status, were 30 Watts for women and 50 Watts for men. The tests were performed using a Vmax 22 system (SensorMedics Co., Yorba Linda, CA, USA) configured to the breath-by-breath mode. The gas analysis system was calibrated before each trial using commercially available precision gases (15% oxygen, 5% carbon dioxide, and balance nitrogen), and flow calibration was carried out using a 3-liter syringe. Expired gases were collected during the last 10 seconds of the last minute of each workload, to determine the following: oxygen consumption (VO_2), carbon dioxide production (VCO_2), respiratory exchange ratio (RER), and minute ventilation (VE). Borg scores determining dyspnea and leg muscle fatigue were recorded in the last 15 seconds of each workload. The $VO_{2, peak}$ of each participant was determined when any of the following criteria were achieved: 1) participant's VO_2 reached a plateau despite an increase in workload; 2) HR reached 85% of age-predicted maximum HR (220 − age); 3) participant's RER ≥ 1.15; or 4) participant was unable to sustain pedal frequency at 50 rotations per minute. Immediately following the exercise test, participants were placed in the sitting position. Heart rate recovery (HRR) was defined as the difference between heart rate at the termination of strenuous exercise and after the first minute of the recovery or cool-down period.

All data are expressed as mean ± SD. Descriptive statistics were used to express the baseline characteristics of participants. An independent t-test and Mann-Whitney U test were used to determine mean differences of the variables under study in healthy, sedentary, normolipidemic and dyslipidemic participants, and a Pearson correlation test was used to determine correlation between $VO_{2,}$peak and CVD risk factors in both categories of participant. If the statistical probability (p-value) was less than 0.05, the difference was considered to be of statistical significance.

RESULTS

Participants in both groups had high percent body fat (%BF; men > 20%, women > 30%). Combined data showed that dyslipidemic participants had higher levels of TG (p<0.01), TC (p<0.01), and LDL-C (p<0.01) and higher triglycerides to HDL-cholesterol (TG/HDL-C) ratios (p<0.01) than normolipidemic participants but dyslipidemic participants had lower $VO_{2, peak}$ (p<0.05) (Table 1). However, there were no significant differences between normolipidemic and dyslipidemic participants in age, height, body mass (BM), BMI, %BF, fat mass (FM), fat-free mass (FFM), WC, hip circumference (HC), WHR, HDL-C levels, fasting blood glucose levels, systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), resting HR, or HRR (Table 1).

In normolipidemic participants, women had lower TG, TG/HDL-C ratios (p<0.01), and lower levels of SBP (p<0.01), DBP (p<0.05), and MAP (p<0.01) than men. However, there were no significant differences between sexes in age, height, BM, BMI, %BF, FM, FFM, WC, HC, weight/height ratio, TC, LDL-C, glucose levels, resting HR, HRR, or $VO_{2, peak}$ (Table 2).

In dyslipidemic participants, women had lower height (p<0.01), WC (p<0.05), TG levels (p<0.05), and TG/HDL-C ratios (p<0.05) than men but higher LDL-C levels (p<0.05). However, there were no significant differences between women and men in age, BM, BMI, %BF, FM, FFM, HC, WHR, TC, HDL-C, fasting blood glucose levels, SBP, DBP, MAP, resting HR, HRR, or $VO_{2, peak}$ (Table 3). There were

Table 1. Characteristics of normolipidemic and dyslipidemic participants

	Normolipidemia (n = 100)	Dyslipidemia (n = 104)	95% CI Mean difference
Age (years)	47.4 ± 7.1	47.5 ± 6.3	0.1 (−1.7 to 2.0)
Height (cm)	160.4 ± 8.2	159.5 ± 8.0	−0.9 (−3.1 to 1.4)
BM (kg)	60.5 ± 9.8	61.9 ± 11.1	1.4 (−1.5 to 4.3)
BMI (kg/m^2)	23.5 ± 3.0	24.2 ± 3.1	0.7 (−0.2 to 1.5)
BF (%)	30.4 ± 5.2	30.4 ± 5.6	0.0 (−1.5 to 1.5)
FM (kg)	18.6 ± 5.0	18.9 ± 5.6	0.4 (−1.2 to 1.9)
FFM (kg)	41.9 ± 6.4	42.9 ± 7.4	1.0 (−0.9 to 2.9)
WC (cm)	79.4 ± 9.5	81.1 ± 8.6	1.7 (−0.8 to 4.2)
HC (cm)	93.9 ± 6.9	95.0 ± 6.9	1.1 (−0.6 to 2.8)
WHR	0.84 ± 0.01	0.85 ± 0.01	0.01 (−0.01 to 0.03)
TG (mg/dL)	102.5 ± 37.5	174.6 ± 104.2*	72.2 (50.4 to 93.9)
TC (mg/dL)	199.2 ± 22.8	241.1 ± 41.8*	41.9 (32.5 to 51.2)
LDL-C (mg/dL)	126.5 ± 21.4	152.0 ± 49.6*	25.5 (14.9 to 36.2)
HDL-C (mg/dL)	51.9 ± 12.5	52.4 ± 17.5	0.5 (−3.7 to 4.7)
TG/HDL-C	2.1 ± 1.0	3.8 ± 3.3*	1.7 (1.0 to 2.4)
FBG (mg/dL)	78.9 ± 8.8	79.4 ± 8.3	0.5 (−1.9 to 2.8)
SBP (mmHg)	117.7 ± 13.3	119.1 ± 15.0	1.4 (−2.5 to 5.3)
DBP (mmHg)	74.7 ± 9.4	76.4 ± 10.1	1.7 (−1.0 to 4.4)
MAP (mmHg)	89.0 ± 9.9	90.6 ± 10.6	1.6 (−1.2 to 4.4)
Resting HR (/min)	74.5 ± 9.4	77.8 ± 10.0	3.2 (0.6 to 5.9)
HRR (/min)	29.3 ± 8.2	27.2 ± 8.1	−1.5 (−3.8 to 0.7)
VO$_{2,\text{peak}}$ (mL/kg/min)	24.2 ± 7.9	21.0 ± 8.1*	−3.2 (−5.4 to −1.0)

Values are expressed as mean ± SD; n = 204 participants (100 normolipidemic, 104 dyslipidemic). 95% CI: 95% confidence interval; BM: body mass; BMI: body mass index; %BF: percentage of body fat; FM: fat mass; FFM: fat-free mass; WC: waist circumference; HC: hip circumference; WHR: waist to hip circumference ratio; TG: triglycerides; TC: total cholesterol; LDL-C: low density lipoprotein cholesterol; HDL-C: high density lipoprotein cholesterol; FBG: fasting blood glucose; SBP: systolic blood pressure; DBP: diastolic blood pressure; MAP: mean arterial pressure; HR: heart rate; HRR: heart rate recovery; VO$_{2,\text{peak}}$: peak oxygen consumption
*Significantly different between groups ($p < 0.01$)

no significant differences between normolipidemic and dyslipidemic participants, or between sexes in either group, for total caloric intake, dietary composition, or total energy expenditure.

In normolipidemic men, VO$_{2,\text{peak}}$ was positively correlated to HDL-C levels ($p<0.01$) and peak exercise HR ($p<0.01$) and negatively correlated to LDL-C levels ($p<0.05$) and TG/HDL-C ratios ($p<0.01$). However, VO$_{2,\text{peak}}$ was not correlated to TG, TC, glucose levels, BM, BMI, %BF, FM, FFM, WC, HC, WHR, SBP, DBP, MAP, resting HR, or HRR (Table 4). In women, VO$_{2,\text{peak}}$ was positively correlated to peak exercise HR ($p<0.01$) and negatively correlated to age ($p<0.05$), TC levels ($p<0.05$), and LDL-C levels ($p<0.01$). However, there were no correlations between VO$_{2,\text{peak}}$ and TG, HDL-C levels, TG/HDL-C ratios, glucose levels, BM, BMI, %BF, FM, FFM, WC, HC, WHR, SBP, DBP, MAP, resting HR, or HRR (Table 4).

In dyslipidemic men, VO$_{2,\text{peak}}$ was positively correlated to HDL-C levels ($p<0.01$) and peak exercise HR ($p<0.01$) and negatively correlated to age ($p<0.05$), TG levels ($p<0.05$), LDL-C levels ($p<0.05$), and %BF ($p<0.05$). However, there were no relationships in men between VO$_{2,\text{peak}}$ and TC

levels, TG/HDL-C ratios, glucose levels, BM, BMI, FM, FFM, WC, HC, WHR, SBP, DBP, MAP, resting HR, or HRR (Table 4). In women, VO$_{2,\text{peak}}$ was positively correlated to resting HR ($p<0.05$), HRR ($p<0.01$), and peak exercise HR ($p<0.01$) and negatively correlated to age ($p<0.05$), TG/HDL-C ($p<0.05$), and WC ($p<0.05$). However, VO$_{2,\text{peak}}$ was not related to TG, TC, LDL-C, HDL-C, glucose levels, BM, BMI, %BF, FM, FFM, WC, HC, WHR, SBP, DBP, or MAP (Table 4).

DISCUSSION

This is the first study demonstrating a significant relationship between aerobic capacity and many CVD risk factors in normolipidemic and dyslipidemic men and women. Gender also plays a role in the relationship between aerobic capacity and a variety of CVD risk factors. In addition, Thai men and women with dyslipidemia have an impaired aerobic capacity, as indicated by the VO$_{2,\text{peak}}$ values measured in this study.

To the best of our knowledge, this is the first study to explore the relationship between aerobic capacity and CVD risk factors in both normolipidemic and dyslipidemic

Table 2. Characteristics of normolipidemic participants

	Males (n = 49)	Females (n = 51)	95% CI Mean difference
Age (years)	48.3 ± 8.2	47.4 ± 5.8	−0.8 (−3.6 to 0.9)
Height (cm)	167.1 ± 4.9	153.9 ± 4.8	−13.2 (−15.1 to −11.3)
BM (kg)	66.4 ± 8.1	54.7 ± 7.8	−11.7 (−14.9 to −8.6)
BMI (kg/m^2)	23.8 ± 2.6	23.2 ± 3.2	−0.6 (−1.7 to 0.6)
BF (%)	28.8 ± 4.8	31.9± 5.2	3.2 (1.2 to 5.1)
FM (kg)	19.4 ± 5.0	17.8 ± 5.0	−1.6 (−3.6 to 0.4)
FFM (kg)	47.1 ± 4.3	36.9 ± 3.5	−10.1 (−11.7 to −8.5)
WC (cm)	83.8 ± 7.5	75.2 ± 9.3	−8.6 (−12.0 to −5.3)
HC (cm)	94.5 ± 3.9	93.3 ± 6.3	−1.3 (−3.4 to 0.8)
WHR	0.89 ± 0.06	0.80 ± 0.06	−0.08 (−0.1 to −0.06)
TG (mg/dL)	117.2 ± 40.3	88.3 ± 28.6[**]	−28.8 (−42.7 to –15.0)
TC (mg/dL)	199.6 ± 23.9	198.8 ± 21.9	−0.7 (−9.8 to 8.4)
LDL-C (mg/dL)	123.6 ± 24.5	129.3 ± 17.7	5.6 (−2.8 to 14.1)
HDL-C (mg/dL)	52.2 ± 15.0	51.6 ± 9.7	−0.6 (−5.6 to 4.4)
TG/HDL-C	2.4 ± 1.2	1.8 ± 0.7[**]	−0.7 (−1.0 to −0.3)
FBG (mg/dL)	80.2 ± 7.8	77.7 ± 10.0	−2.5 (−6.0 to 0.9)
SBP (mmHg)	120.7 ± 15.1	114.8 ± 10.7[**]	−5.9 (−11.0 to −0.7)
DBP (mmHg)	76.9 ± 10.8	72.5 ± 7.4[*]	−5.1 (−8.5 to −1.7)
MAP (mmHg)	91.5 ± 11.5	86.6 ± 7.4[**]	−4.9 (−8.7 to −1.0)
Resting HR (/min)	73.8 ± 9.5	75.3 ± 9.4	1.5 (−2.2 to 5.3)
HRR (/min)	27.6 ± 8.0	30.8 ± 8.1	3.2 (0.0 to 6.4)
VO$_{2,\,peak}$ (mL/kg/min)	24.2 ± 7.3	24.1 ± 8.5	−0.2 (−3.3 to 3.0)

Values are expressed as mean ± SD; n = 100 participants (49 males, 51 females).
95% CI: 95% confidence interval; BM: body mass; BMI: body mass index; %BF: percentage of body fat; FM: fat mass; FFM: fat-free mass; WC: waist circumference; HC: hip circumference; WHR: waist to hip circumference ratio; TG: triglycerides; TC: total cholesterol; LDL-C: low density lipoprotein cholesterol; HDL-C: high density lipoprotein cholesterol; FBG: fasting blood glucose; SBP: systolic blood pressure; DBP: diastolic blood pressure; MAP: mean arterial pressure; HR: heart rate; HRR: heart rate recovery; VO$_{2,\,peak}$: peak oxygen consumption
[*]Significantly different from men (p < 0.05)
[**]Significantly different from men (p < 0.01)

participants in their 40s. Previous studies have shown this relationship in younger people[22, 23], patients with diabetes[23], overweight and obese[24], and African and Western participants[24, 25], and hypercholesterolemic mice[26]. However, dyslipidemia can be found in older people; therefore, correlation among normolipidemic and dyslipidemic participants who are older than 40 years of age should be further explored to provide useful data for the promotion of health in a broader age range.

The results of this study show that in female normolipidemic and both sexes of dyslipidemic participants, aerobic capacity decreases with age. This is consistent with previous studies that have found this relationship in healthy middle-aged (mean age: 39 years) and older (mean age: 68 years) male and female participants[27] and healthy males ages 22 to 65 years[28]. In addition, no subject in this study had a relationship between aerobic capacity and FFM and BMI. This is supported by the study of Yagura et al[15]. This may indicate that aerobic capacity was not influenced by skeletal muscle mass but rather by lipid profiles among the middle-aged normolipidemic and dyslipidemic Thai participants in

this study. However, a previous study reported the opposite results, showing that obesity indicated by BMI was independently associated with coronary endothelial dysfunction in patients with normal or mildly diseased coronary arteries[29]. This implies that BMI is also independently related to aerobic capacity.

Interestingly, these relationships are not only rooted in lipid abnormality but also hemodynamics and obesity in our dyslipidemic study population. The mechanism behind these relationships may be the effect of loss of endothelial derived nitric oxide (EDNO) owing to abnormal lipid profiles, resulting in reduced exercise-induced nitric oxide production[6]. This decreased endothelial vasodilation decreases blood flow and increases blood pressure. In addition, maximal exercise reduces vasodilation via exercise-induced oxidative stress[8]. Capillary volume and surface area are not matched to maximal oxygen demand[29]. Thus, if both of these mechanisms decrease oxygen transport capacity of the vasculature, either at the peripheral vasculature or by centrally affecting the heart, the effect could be a decrease in aerobic capacity. Another possible explanation could be the effect

Table 3. Characteristics of dyslipidemic participants

	Males (n = 52)	Females (n = 52)	95% CI Mean difference
Age (years)	48.0 ± 6.3	47.0 ± 6.4	−1.0 (−3.4 to 1.5)
Height (cm)	165.5 ± 6.1	153.5 ± 4.5[#]	−12.0 (−14.1 to −9.9)
BM (kg)	67.0 ± 9.3	56.7 ± 10.4	−10.3 (−14.2 to −6.5)
BMI (kg/m^2)	24.4 ± 2.5	23.9 ± 3.6	−0.5 (−1.7 to 0.7)
%BF	28.3 ± 6.1	32.5 ± 4.1	4.2 (2.2 to 6.3)
FM (kg)	19.2 ± 5.7	18.7 ± 5.6	−0.5 (−2.7 to 1.7)
FFM (kg)	47.8 ± 5.7	38.0 ± 5.5	1.1 (−12.0 to −7.6)
WC (cm)	84.8 ± 6.5	77.4 ± 8.8[*]	−7.4 (−10.4 to −4.4)
HC (cm)	94.7 ± 5.8	95.3 ± 7.9	0.6 (−2.1 to 3.3)
WHR	0.89 ± 0.01	0.81 ± 0.01	−0.09 (−0.1 to −0.07)
TG (mg/dL)	214.5 ± 113.0	134.8 ± 76.8[*]	−79.7 (−117.3 to −42.1)
TC (mg/dL)	236.4 ± 42.7	245.7 ± 40.7	9.3 (−6.9 to 25.5)
LDL-C (mg/dL)	138.1 ± 54.8	165.9 ± 39.6[*]	27.8 (9.2 to 46.4)
HDL-C (mg/dL)	52.0 ± 19.6	52.8 ± 15.2	0.8 (−6.1 to 7.6)
TG/HDL-C	4.8 ± 4.0	2.7 ± 1.7[*]	−2.1 (−3.3 to −0.9)
FBG (mg/dL)	80.3 ± 7.0	78.5 ± 9.4	−1.7 (−4.9 to 1.5)
SBP (mmHg)	123.7 ± 14.0	114.6 ± 14.7	−9.1 (−14.7 to −3.5)
DBP (mmHg)	80.2 ± 10.0	72.5 ± 8.9	−7.7 (−11.3 to −4.0)
MAP (mmHg)	94.7 ± 10.2	86.5 ± 9.5	−8.2 (−12.0 to −4.4)
Resting HR (/min)	77.3 ± 10.1	78.2 ± 9.9	0.9 (−3.0 to 4.8)
HRR (/min)	26.9 ± 8.2	28.5 ± 8.1	1.6 (−1.5 to 4.8)
VO$_{2, peak}$ (mL/kg/min)	22.2 ± 8.8	19.8 ± 7.2	−2.5 (−5.6 to 0.7)

Values are expressed as mean ± SD; n = 104 participants (52 males, 52 females)
95% CI: 95% confidence interval; BM: body mass; BMI: body mass index; %BF: percentage of body fat; FM: fat mass; FFM: fat-free mass; WC: waist circumference; HC: hip circumference; WHR: waist to hip ratio; TG: triglycerides; TC: total cholesterol; LDL-C: low density lipoprotein cholesterol; HDL-C: high density lipoprotein cholesterol; FBG: fasting blood glucose; SBP: systolic blood pressure; DBP: diastolic blood pressure; MAP: mean arterial pressure; HR: heart rate; HRR: heart rate recovery; VO$_{2, peak}$: peak oxygen consumption
[*]Significantly different from men (p<0.05)

of high plasma cholesterol, which decreases oxyhemoglob in content via decreasing erythrocyte membrane fluidity and its permeability to O$_2$. Decreased oxyhemoglobin content would contribute to a reduction of O$_2$ tissue supply resulting in reduced aerobic capacity, as seen in this study.

The lipid profiles, hemodynamic and obesity measurements shown in this study appear to be useful tools to indirectly evaluate the performance of cardiopulmonary systems[30, 31], which could additionally contribute to predicting prognosis and all-cause mortality in patients with CVD. In addition, although the atherosclerotic process was not monitored in this study, the significant correlations found suggest the usefulness of lipid profiles to determine cardiopulmonary function. This may assist medical staff in prescribing exercise regimens that improve cardiopulmonary function, effectively preventing CVD in individuals with dyslipidemia.

Surprisingly, though there was no difference in anthropometry between normolipidemic and dyslipidemic participants, the relationship between aerobic capacity and anthropometry was found to be different between these participants. There was no relationship to anthropometry in normolipidemic participants, but this relationship was found in dyslipidemic participants, which may be owing to normal cardiorespiratory and anthropometry values in normolipidemic participants. Moreover, this may imply that lipid parameters play more of a role than anthropometry in cardiorespiratory fitness. Additionally, because obesity is an independent risk factor for CVD[30, 31], the relationship may be absent in a normolipidemic state but present in a dyslipidemic state.

Dyslipidemic participants had a lower VO$_{2, peak}$ than normolipidemic participants (p<0.01). In normolipidemic male participants, VO$_{2, peak}$ was positively correlated with levels of HDL-C and negatively correlated with levels of LDL-C and TG/HDL-C ratios, whereas in female participants, VO$_{2, peak}$ was negatively correlated with age, TC, and LDL-C. In dyslipidemic male participants, VO$_{2, peak}$ was positively correlated with HDL-C levels and negatively correlated with age, LDL-C and TG levels, and %BF; in female participants, VO$_{2, peak}$ was positively correlated with resting HR and HRR and negatively correlated with age, TG/HDL-C, and WC. Although there was a low correlation coefficient in some lipid profiles, such as for LDL-C and TG, these

Table 4. Correlations between VO$_{2, peak}$ and cardiovascular risk factors in participants with normolipidemia and dyslipidemia

	Normolipidemia		Dyslipidemia	
	Males (n = 49)	Females (n = 51)	Males (n = 52)	Females (n = 52)
Age (years)	−0.15	−0.34[+]	−0.30[+]	−0.33[+]
TG (mg/dL)	−0.16	−0.02	−0.29[+]	−0.23
TC (mg/dL)	0.06	−0.31[+]	0.05	0.11
LDL-C (mg/dL)	−0.29[+]	−0.49[++]	−0.36[+]	−0.01
HDL-C (mg/dL)	0.53[++]	0.15	0.48[++]	0.24
TG/HDL-C	−0.45[++]	−0.14	−0.09	−0.32[+]
FBG (mg/dL)	−0.08	−0.04	−0.13	−0.09
BM (kg)	−0.03	−0.17	−0.04	−0.06
BMI (kg/m^2)	−0.04	−0.12	−0.08	−0.20
%BF	−0.02	−0.19	−0.30[+]	−0.03
FM (kg)	0.02	−0.18	0.16	−0.08
FFM (kg)	0.08	−0.11	−0.22	−0.11
WC (cm)	−0.12	−0.07	−0.07	−0.31[+]
HC (cm)	0.06	−0.07	−0.03	−0.27
WHR	−0.19	−0.08	−0.07	−0.14
SBP (mmHg)	0.15	0.03	0.06	0.01
DBP (mmHg)	0.19	−0.13	0.17	0
MAP (mmHg)	0.19	−0.07	0.12	0.02
Resting HR (/min)	−0.02	0.19	0.06	0.32[+]
Peak HR (/min)	0.67[++]	0.52[++]	0.76[++]	0.64[++]
HRR (/min)	0.24	0.13	0.19	0.37[++]

Values are expressed as correlation coefficient (r)
BM: body mass; BMI: body mass index; %BF: percentage of body fat; FM: fat mass; FFM: fat-free mass; WC: waist circumference; HC: hip circumference; WHR: waist to hip circumference ratio; TG: triglycerides; TC: total cholesterol; LDL-C: low density lipoprotein cholesterol; HDL-C: high density lipoprotein cholesterol; FBG: fasting blood glucose; SBP: systolic blood pressure; DBP: diastolic blood pressure; MAP: mean arterial pressure; HR: heart rate; HRR: heart rate recovery; VO$_{2, peak}$: peak oxygen consumption
[+]Correlation is significant at the 0.05 level (p<0.05)
[++]Correlation is significant at the 0.01 level (p<0.01)

parameters are still useful. These lipid profiles themselves can be used to indicate CVD risk because they have been shown to be related to CVD risk[2].

A strength of this study was that VO$_{2, peak}$ was directly measured by gas analysis using graded maximal exercise. The test was performed by strictly following exercise termination criteria. Therefore, the aerobic capacity determined by VO$_{2, peak}$ in this study is valid.

A limitation of this study was the assessment of body composition, such as %BF, which was assessed by measuring skin fold thickness and then calculating fat mass. However, the reliability of measurements was controlled by keeping coefficient of variation to less than 3%.

A further study investigating mechanisms of the gender effect on relationships between aerobic capacity and CVD risk factors in a dyslipidemic Thai population may be worthwhile. The results of this study suggest that there are relationships between aerobic capacity and many CVD risk factors in normolipidemic and dyslipidemic Thai men and women, and that sex plays a role in defining the relationships in both groups.

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