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Effects of home-based breathing exercise on heart rate variability, cardio-ankle vascular index, respiratory muscle strength and maximum oxygen consumption in female participants with normal weight obesity

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ABSTRACT

Background: Obesity causes autonomic imbalance and decreases exercise capacity, which limit exercise and physical activity. Normal-weight obesity (NWO) is defined as normal body mass index (BMI), but increased body fat percentage (BF%) associated with cardiorespiratory impairment in early adulthood. The benefit of slow breathing has shown to enhance cardiorespiratory functions in overweight and generally obese people. Moreover, the slow breathing affects to the heart rate variability (HRV), cardio ankle vascular index (CAVI), respiratory muscle strength and indirect maximum oxygen consumption (indirect VO_2max) has not been studied in female individuals with NWO.

Objectives: To determine the effects of slow breathing exercise on HRV, CAVI, respiratory muscle strength and VO_2max in female participants aged 20-30 years, who have normal BMI (18.5-22.9 kg/m^2, n=19 each group) and high body fat (BF) (BF ≥30%), compared with a control group, which carried out routine physical activity.

Materials and methods: Participants with NWO were divided randomly into two groups: control and intervention group, which performed breathing exercise at home. All of the participants were measured for body composition, blood pressure, HRV and CAVI. An electronic pressure transducer and cycle ergometer were applied to assess respiratory muscle strength and indirect VO_2max. All measurements were recorded before and after 4 weeks when completely intervention. The demographic data between the groups was analyzed the significant difference by using the independent t test. The data at baseline and four weeks after intervention was determine by two way ANOVA. *P*-value less than 0.05 was considered statistically significant.

Results: Participants with NWO, who received breathing exercise at home, showed lower autonomic imbalance, and systolic blood pressure, whereas improved maximum inspiratory pressure (MIP), but no statistical change in maximum expiratory pressure (MEP), indirect VO_2max, CAVI or ankle brachial index (ABI) (p>0.05).

Conclusion: Slow breathing exercise can improve autonomic balance and inspiratory muscle strength in physically inactive female NWO participants.

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Introduction

Obesity is a major contributing factor in public health problems worldwide. A previous study indicated accumulated excessively regarding body fat percentage (BF%) associated with the risk of unhealthy and illness.[1] At present, the number of individuals with normal body mass index (BMI) and high body fat (BF), known as normal weight obesity (NWO), has increased in developing and undeveloped countries.[2] NWO may be prevalent in up to one-third of Asian individuals and has higher risk of causing cardiovascular diseases (CVD).[2,3]

Heart rate variability (HRV) which represents the balance of autonomic function can be used to predict the risk of disease or likelihood of death due to CVD.[4] The cardio ankle vascular index (CAVI) is a noninvasive parameter that indicates abnormality of the artery in the preclinical stages of diseases.[5,6] The adverse effect of overweight and obesity on autonomic function and arterial stiffness has been observed in numerous studies.[7-9] Individuals with normal BMI, with high BF%, tend to have greater risk of CVD due to unaware that underlying health risk behavior.[10] A normal body shape without any signs and symptoms of disease has not been diagnosed, could lead to a misleading perception of exercise. Thus, it is important to recognize and promote exercise before diseases progression.[3]

Maximal oxygen consumption (VO_2 max) is an indicator reflect to the physical fitness, which presents maximum capacity of an individual's body to carry oxygen through the vascular system to active muscles during exercise or performing physical activity.[11] The circulatory and respiratory systems are controlled by the autonomic nervous system (ANS) and important for the maintenance of physical daily life. High BF% is associated with lower lung volumes and respiratory function in young adults.[12] Demand for ventilation, increased work of breathing, respiratory muscle inefficiency and low respiratory compliance are respiratory complications in obesity.[13] Overweight and obese subjects often experience increased breathlessness and decreased efficiency of respiratory muscle function during exercise, which contribute to dyspnea, and consequently avoidance of exercise.[13,14]

The beneficial effects of slow breathing exercise at 6 breaths per min have demonstrated improvement in respiratory efficiency, arterial oxygen saturation and decreased respiratory effort.[15] A study by Rachatakarn *et al*. showed that slow deep breathing exercise improved maximum inspiratory muscle strength in younger individuals, but the study recruited a small number of participants (i.e. n=5-6 per group) and conducted in a laboratory setting. Fifty percent of all the participants dropped out because the experimental center was located inconveniently, and transportation, travel time and cost were an obstacle to regular participation in physical activity and exercise.[16,17] Additionally, previous studies showed the improvement of HRV, blood pressure and breathing pattern.[18,19] Thus, this study investigated the effects of slow breathing on HRV, CAVI, respiratory muscle strength and VO_2max in inactive NWO female volunteers, who were aged 20-30 years. It also promoted exercise at home.

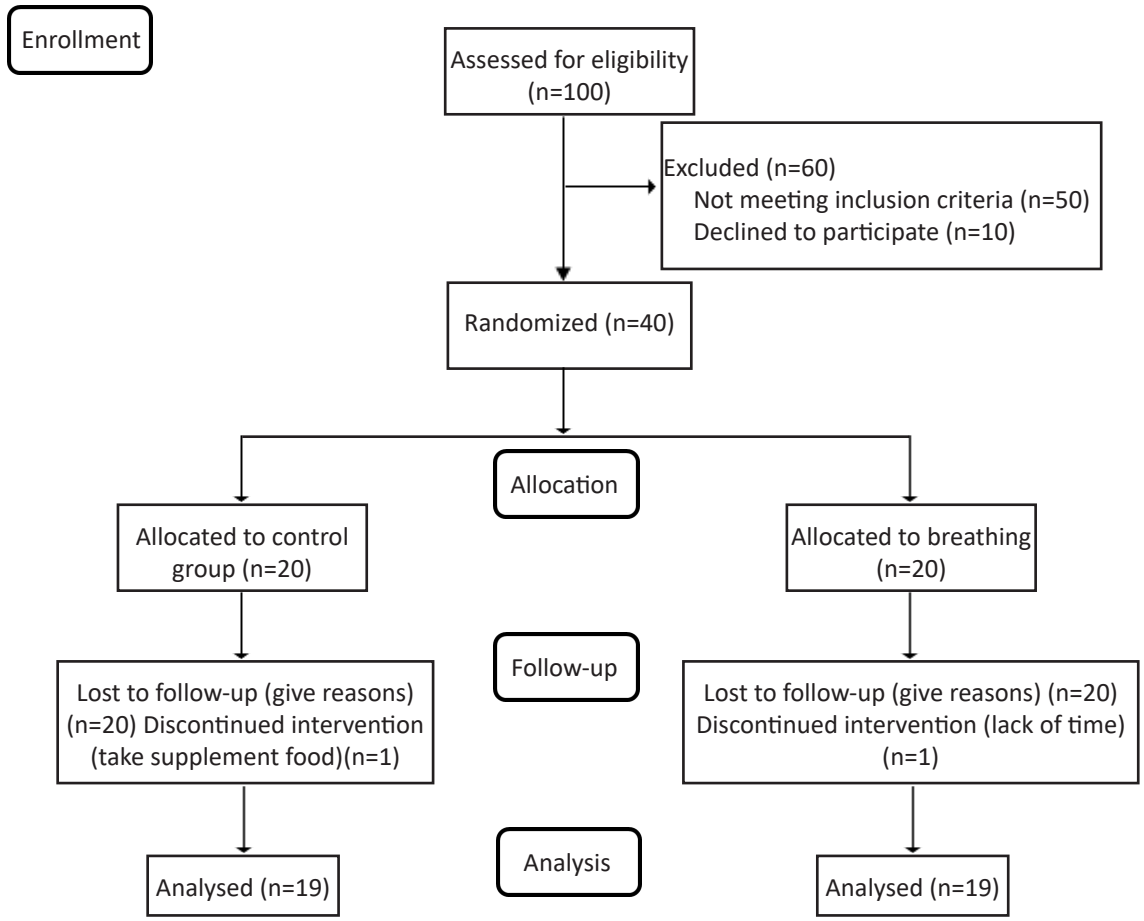
Materials and methods

Participant recruitment

Eligible participants from Naresuan University and nearby Thapho subdistrict, Muang district, Phisanulok, Thailand had enrolled for this study. The inclusion criteria included female participants, aged 20-30 years, with normal BMI (18.5-22.9 mL/kg^2)[20] and high BF (≥30%),[20,21] who performed moderate intensity exercise less than 30 minutes for 5 days per week (150 minutes per week) or vigorous intensity exercise less than 25 minutes for 3 days per week (75 minutes per week).[22,23] The exclusion criteria were as follows: people who smoke or had underlying cardiopulmonary, neurological and musculoskeletal disease, and general obesity, characterized by 25 kg/m^2. Subjects who supplemented stimulating beverages, such as alcohol, caffeine or drugs that stimulate the central nervous system (CNS) within 48 hours before the experiment, and including participants who could not completely follow the protocol were excluded.[23,24] In addition, participants who had mind–body training such as yoga, Qigong and meditation were also excluded from this study. This study was performed at the Research Laboratory, Department of Physical Therapy, Faculty of Allied Health Sciences, Naresuan University, according to the tenets of the Declaration of Helsinki and approved by the Naresuan University Institutional Review Board (IRB No.219/62). The calculation of the sample size was based on the previous mean maximum inspiratory pressure (MIP) results from the study of Rachatakarn et al. (2016).[16] The sample size was set the confidence level at 0.50, the power of the test at 0.80. The result was 16 participants per group. With the 20 percent dropout rate considered, the calculation indicated that the study required at 20 per group. All of the participants received a full explanation of the protocol and possible consequences of the experiment before signing an informed consent form and participating in the study.

Procedures

The participants were divided randomly into two groups: intervention and control (CON) (n=19 per group). The study design was randomized, experimental study, single blind intervention in physically inactive female participants with NWO. The effects of slow deep breathing on HRV, CAVI, respiratory muscle strength and indirect VO_2max were investigated. All of the participants practiced slow deep breathing at home, with a 1:1 allocation rate, according to the block plan generated by the website (www.randomization.com). Randomization was concealed by using a block randomization technique (4 participants per block) by a researcher. Allocation was performed before commencing the study program.



Experimental design

The participants were interviewed using a questionnaire and the demographic data included age, gender, occupation, personal medical history, smoking status and physical activity level. The vital signs, anthropometric composition, HRV, respiratory muscle strength and indirect VO_2max of the participants were recorded before and after 4 weeks of intervention by cardio-thoracic technologists, physiotherapist, research assistants. The participants were given written instructions, necessary information and a diary for recording exercise during the intervention period. All participants were followed to confirm completion of the protocol and ensure all the necessary information was recorded through telephone communication.

Home-based breathing exercise

The participants were instructed to put their dominant hand on their abdomen and non-dominant hand on their upper chest. All of them followed the same instruction for the breathing exercise, using a metronome application on their smart phone. Then they were instructed to slow deep breath with four seconds of inspiration and six seconds of expiration periods, and then one minute of rest interval period by a physical therapist who has more than four years of cardiopulmonary clinical experience and being the same person throughout the program. They were instructed to breathe deeply and slowly at six cycles per minute, for at least 30 minutes, continually 5 days per week for 4 weeks.[18] Those who followed the breathing exercise in accordance with the same instruction according to the protocol were included in this study. The control group performed their normal routine of activity in daily life, and recorded necessary information in their physical activity diary and food consumption. After completing the post-measurement, all participants received health and physical education.

Measuring anthropometric composition

Body composition was measured by using bioelectrical impedance analysis (BIA) (HBF 375, Omron Healthcare Co. Ltd., Kyoto, Japan). All of the participants wore light clothing in adequate privacy of the laboratory. After completing the pre criteria instructions, following the standard guideline.[25] The participants were measured BMI, visceral fat, fat free mass and body fat.[26] They were then asked to stand with their feet on electrodes in the main unit and their weight equally distributed. They kept their back and knees straight and looked straight in front of them, while gripping electrodes with dry palms and arms held straight out horizontally at a 90° angle to the body until the measuring was completed. The BMI, visceral fat, fat free mass and body fat of the participants were recorded.[27]

Heart rate variability measurement

The skin of all participants was cleaned with electrocardiogram (ECG) electrolyte gel. HRV was measured in a room with stable temperature of 25°C , and the participants laid in a resting supine position for 20 min, while the breathing rate was being controlling at 12 breaths/min by a metronome.28 Lead II was recorded the ECG activity by placing electrode on the right arm (RA) and another on the left leg (LL) by chest physical therapist. An ADinstruments lab chart version 8 with HRV module version 2.0.3 (Power Lab 4 SP, ADInstruments, Australia) was used as data acquisition software for recording ECG readings. Beat to beat intervals (R-R intervals) were calculated after correction of artifacts and entropic beats. The average from each R-R interval was measured and considered for the time and frequency domain of HRV,[29] by following the guidelines.[30] The changes in heart rate over time or beat-to-beat alterations between successive normal cardiac cycles were measured. The criteria of successive cardiac waveforms in the ECG signal included P, QRS and T waves without amount of ectropic beats (premature atrial or ventricular, supraventricular contractions). The peak of the R wave without artifacts from normal QRS complex, and the duration between two adjacent R wave peaks is measured. Time and frequency domain of HRV were analyzed though analysis of R-R intervals (the time between the two successive R waves in ECG). The time-domain indices of HRV used in this analysis were the standard deviation (SD) of the N-N intervals (SDNN) and root mean square difference of successive normal R-R intervals (RMSSD). Frequency-domain indices of HRV, including total power, Low Frequency (LF: 0.04-0.15 Hz), High Frequency (HF: 0.15-0.40 Hz), and LF/HF – ratio of absolute LF to HF power were analyzed.[30]

Ankle-brachial index and cardio-ankle vascular index measurement

After rest, all participants were assessed for arterial stiffness in the supine position using a vascular screening device (VS-1500N, Fukuda Denshi Co. Ltd., Tokyo, Japan). The ankle-brachial index (ABI) and CAVI were assessed by ECG, arm and leg blood pressure and phonograph.[5] The participants were measured for leg and arm blood pressure simultaneously, while using ECG and phonocardiography for calculating pulse wave velocity (PWV), which was then used as a factor in calculating ABI and CAVI.[31]

Respiratory muscle strength testing

The participants were familiarized with the instrument and procedure before the actual test. Maximal inspiratory pressure (MIP) and maximum expiratory pressure (MEP) measurements were conducted in accordance with the guideline of American Thoracic Society/European Respiratory Society statement on respiratory muscle testing.[32] An electronic pressure transducer (MicroRPM, Micromedical Ltd., Kent, United Kingdom) was used to measure respiratory muscle strength before and after treatment by a physical therapist. MIP testing were measured in deep inspiration from residual volume after maximum exhalation.[33] For the measurement of the MEP, the participants were instructed to forced expiration form total lung capacity.[33] Participants were allowed to rest for 1-2 minutes in between tests. The test was performed at least 3 times, and accomplished for a maximum of 5 maneuvers. The highest value of 3 measurements was chosen for analysis and compared with the reference value.[34]

Measurement of indirect maximal oxygen consumption (indirect VO_2max)

The indirect VO_2max was assessed by submaximal Astrand-Ryhming protocol by a cycle ergometer (Monark 828E, Monark Exercise AB, Vansbro, Sweden). Prior to performing the actual test, all of the participants were familiarized with the protocol and 2-3 minutes warm up period, which set a rhythm at 50 repetitions per minutes (RPM). A Kilo Pond intensity was performed initially to maintain the heart rate (HR) at between 120-170 beats/minute, which conducted for given level of submaximal work load level, using a heart rate monitor to keep a continuous record for six minutes. Constant HR at the fifth and sixth minute was determined in order to correct VO_2max as follows: 35 absolute $\dot{V}O_2$max (L/min) and relative VO_2max (mL/kg/min) were calculated from the age factor and body weight of the participants, respectively.[35] During the test, if participants with shortness of breath or wheezing, claudication or angina onset were followed according to the American College of Sports Medicine (ACSM) guidelines for exercise testing, and the prescription, the participants were stopped from the test.[36]

Statistical data analysis

Statistical difference of all data were analysis by a SPSS for Windows program, version 17.0 (IBM Corp. New York, NY, USA), and mean and standard deviation were presented when the normal distribution of data was demonstrated. The independent t test was performed for statistical analysis of the demographic data between groups, in addition, the two way ANOVA was used for comparing within and between groups. Regarding all of the parameters, $p<0.05$ was considered a statistically significant difference.

Results

Baseline characteristics of the participants

The statistical results of this study found that the demographic data, age, height, weight, BMI of all participants were not significant between groups ($p>0.05$). Body composition include fat free mass, body fat and visceral fat, which is a type of body fat that stored within the abdominal cavity and around internal organs. There was no significant difference in body fat, visceral fat and fat free mass between the two groups ($p>0.05$) (Table 1). Participants in control group excluded from the study due to take a supplement food during the study, while participants in the intervention group who depart from the study due to lack of time. Beside those, all of the other participants (control group n=19, intervention group n=19) completed the study with the percentage exercise adherence was more than 90%.

Table 1 General characteristics of the participants.

Parameters	Control (n=19)	Intervention (n=19)	*p* value
Age (year)	22.95±2.37 (20-28)	22.58±2.83 (20-29)	0.507
Height (cm)	160.67±5.43 (150-165)	160.47±6.23 (153-168)	0.851
Weight (Kg)	56.92±4.58 (46.5-61.3)	57.16±5.16 (48.5-64.8)	0.747
BMI (Kg/m^2)	21.53±0.69 (20.67-22.52)	22.15±0.77 (20.72-22.96)	0.562
Body fat(%)	32.38±2.05 (30.1-35.2)	33.06±2.56 (30.4-36.8)	0.358
Visceral fat (%)	6.26±2.09 (3.8-9.2)	5.56±2.06 (3.5-8.4)	0.868
Fat free mass (%)	28.42±3.74 (23.6-33.8)	26.55±3.43 (22.4-31.2)	0.211

Body mass index; BMI, Kg/m^2; kilogram per square meter, %; percentage. p value was statistically analyzed with the independent t test.

Comparison of cardiovascular parameters, respiratory strength and indirect VO_2max

There was a significant difference of systolic blood pressure (SBP) and MIP between the two groups (p<0.05). The mean MIP in the intervention group was 87.84±10.01 cmH_2O, which showed a significant difference when compared to the control group (76.84 ± 9.74 cmH_2O). The same was observed for SBP in the intervention group (101.53±5.70 mmHg), compared to the control group (109.78±7.10 mmHg) (p<0.05). Diastolic blood pressure (DBP), MEP and VO_2max were not significantly different when compared to the control group (p>0.05) (Table 2).

Table 2 Cardiovascular parameters, respiratory strength and VO2max between groups at pre and post intervention.

Parameters	Control (n=19)		Intervention (n=19)	
	Pre	Post	Pre	Post
HR (BPM)	82.63±8.94 (70-93)	85.58±9.51 (74-98)	83.10±8.84 (70-96)	80.94±6.61 (70-90)
SBP (mmHg)	108.89±8.08 (98-120)	109.78±7.10 (100-120)	108.94±7.31 (100-120)	101.53±5.70*† (90-110)
DBP (mmHg)	68.16±6.34 (56-80)	69.95±7.12 (60-80)	67.68±5.77 (58-76)	66.11±6.03 (56-80)
MIP (cmH_2O)	78.84±10.44 (67-96)	76.84±9.74 (65-90)	79.89±8.05 (68-94)	87.84±10.01*† (75-104)
MEP (cmH_2O)	95.21±10.67 (83-115)	93.78±8.95 (82-104)	92.94±10.81 (80-110)	94.57±10.34 (80-117)
VO2max (ml/kg/min)	38.01±6.48 (30.6-45.4)	36.19±5.91 (27.8-43.2)	35.97±7.02 (27.4-45.6)	38.85±5.52 (30.3-46.8)

*HR, heart rate; BPM, beats per min; SBP, systolic blood pressure; DBP, diastolic blood pressure; mmHg, millimeter of mercury; MIP, maximal inspiratory pressure; MEP, maximal expiratory pressure; VO_{2max}, maximal oxygen consumption; ml/kg/min, milliliter per kilogram per minute. *p<0.05 for significant differences from pre-intervention, †p<0.05 for significant differences between the groups.*

Comparison of heart rate variability analysis

The mean of SDNN and RMSSD in the intervention group, which was significantly higher than that pre-intervention (p<0.05). There was a significant difference in HF, LFnu and LF/HF between the groups (p<0.05). The mean HFnu and LF/HF, which was significantly higher than that pre intervention and between the groups (p<0.05). Furthermore, the mean of LFnu at 4 weeks of intervention in the control group was higher than pre-test. While, LFnu in the intervention group was lower than that pre-test and after 4 week of intervention, compared to control group (Table 3).

Table 3 Heart rate variability analysis (HRV) between group at pre- and post-slow deep breathing intervention.

Parameters	Control (n=19)		Intervention (n=19)	
	Pre	Post	Pre	Post
SDNN (ms)	60.03±10.52	63.70±13.74	58.36±10.71	67.74±11.22*
	(39.75-74.62)	(42.14-78.54)	(40.11-74.98)	(46.21-83.67)
RMSSD (ms)	42.23±10.14	45.04±14.36	40.91±11.05	52.69±13.73*
	(24.12-60.34)	(21.16-65.07)	(28.63-58.84)	(32.41-67.96)
LF (n.u.)	55.73±10.95	64.92±11.14*	56.44±11.58	45.43±12.30*†
	(36.69-70.33)	(47.40-79.83)	(35.46-74.27)	(20.77-65.39)
HF (n.u.)	38.77±11.27	40.07±12.89	34.32±10.24	56.06±10.62*†
	(24.93-58.64)	(23.38-64.28)	(20.08-55.17)	(36.43-74.95)
LF/HF	1.52±0.42	1.76±0.65	1.67±0.59	0.83±0.37*†
	(0.78-2.37)	(0.97-2.66)	(1.16-2.41)	(0.31-1.55)

*SDNN (standard deviation of N to N interval), RMSSD (root mean square of N to N interval), LF (low frequency), HF (high frequency), LF/HF (ratio of low and high frequency), ms (millisecond), *p<0.05 for significant differences from pre-intervention, †p<0.05 for significant differences between the groups.*

Comparison of CAVI and ABI

The mean CAVI and ABI were not significantly different between groups (p>0.05). The mean CAVI and ABI in the control group were 6.53±0.52 and 1.02±0.05, respectively, while that in the intervention group were 6.56±0.50 and 1.01±0.04, respectively (Table 4).

Table 4 Cardio ankle vascular index (CAVI) and Ankle brachial index (ABI) between groups at pre- and post-intervention.

Parameters		Control (n=19)		Intervention (n=19)	
		Pre	Post	Pre	Post
CAVI	**Right**	6.51±0.47	6.51±0.52	6.51±0.60	6.53±0.53
		(5.60-7.30)	(5.40-7.40)	(5.70-7.80)	(5.70-7.30)
	Left	6.54±0.45	6.56±0.53	6.61±0.60	6.59±0.48
		(5.80-7.30)	(5.30-7.40)	(5.80-7.90)	(5.70-7.40)
	Mean	6.52±0.45	6.53±0.52	6.56±0.60	6.56±0.50
		(5.75-7.25)	(5.35-7.40)	(5.75-7.85)	(5.70-7.35)
ABI	**Right**	1.02±0.05	1.02±0.05	1.02±0.05	1.01±0.04
		(0.94-1.14)	(0.94-1.14)	(0.98-1.16)	(0.96-1.12)
	Left	1.02±0.05	1.02±0.04	1.01±0.04	1.01±0.04
		(0.96-1.14)	(0.96-1.12)	(0.98-1.14)	(0.96-1.14)
	Mean	1.02±0.05	1.02±0.05	1.01±0.05	1.01±0.04
		(0.95-1.14)	(0.95-1.13)	(0.98-1.15)	(0.96-1.13)

CAVI: Cardio ankle vascular index, ABI: Ankle brachial index. There were no significant differences between the groups for CAVI and ABI.

Discussion

The findings of this study showed that slow breathing exercise at home could improve SDNN, RMSSD, HFnu together with decreased LF and LF/HF ratio. Moreover, the improvement of MIP and SBP were observed. DBP, VO_2max, MEP, CAVI and ABI were not significantly changed when compared to the control group (p>0.05).

Healthy obese individuals are more likely to experience dyspnea at rest or on exertion. Fat in the visceral organ may be attributed to displaced diaphragmatic excursion, respiratory muscle inefficiency and workload to the respiratory muscle.[37] Furthermore, previous research showed that a fatiguing diaphragm led to increased sympathetic outflow.[38] In the theory, HRV is a measurable reflection of autonomic balance and has been used as a marker for cardiac mortality from cardiovascular disease (CVD).[4] Previous results found autonomic imbalance in young obese people, characterized by an increase in sympathetic activity and decreasing parasympathetic activity.[39] SDNN and RMSSD were significant improved by slow breathing which reflected the improvement of autonomic balance. Moreover, slow breathing exercise at home improved autonomic function, by improving HF, decreasing LF and LF/HF, represented the sympathetic and parasympathetic balance. The study of Joseph showed that slow breathing at 6 breaths/min increased baroreflex sensitivity, reduced sympathetic activity and chemoreflex activation, therefore, it can be supposed a potentially beneficial effect of slow breathing on autonomic function.[40] Thus, it can be possibly suggested that slow deep breathing decreased sympathetic and increased parasympathetic activity in physically inactive normal weight obese participants.

Inspiratory muscle weakness may induce several impairments in both healthy and athletic individuals.[41] The balance of ANS plays an important role in respiratory performance. A fatiguing inspiratory muscle work causes sympathetic activation.[38] This study also showed improvement in MIP and observed lesser sympathetic activity in participants with NWO receiving breathing exercise. The improvement of autonomic regulation by slow breathing exercise, mediated by lesser sympathetic modulation, may help to improve respiratory muscle strength. However, this study did not measure baroreflex and/or chemoreflex sensitivity, therefore reasonable hypothesize for decreased the sympathetic activity effecting on both sensitivities by slow deep breathing exercise cannot be concluded. Further study between respiratory strength and sympathetic activity should be further investigated.

Excessive fat storage aggravates autonomic imbalance by increasing sympathetic activation and decreasing parasympathetic activity, which contribute to increased risk of cardiovascular diseases.[7] Slow breathing exercise has been demonstrated in improving autonomic function and arterial dysfunction by reduction in sympathetic activity in obese children.[42] Slow breathing exercise in our study reduced sympathetic activity and SBP, but not alternations of DBP, CAVI and ABI when compared with the control group. It suggested that SBP was reduced by decreasing sympathetic activation, but not change in DBP, which possibly depend on elasticity of conductance vessels.[43] NWO participants who have an excessive body fat percentage increase risk of subclinical arteriosclerosis.[44] Fat in blood circulation possibly played an important role in vessel elasticity.[45] Thus, our study did not found the alternation of DBP, CAVI and ABI. Nevertheless, some study showed that slow breathing for 12 weeks reduced of both SBP and DBP in healthy participants.[46] Further studies are needed to assess the long-term outcomes of slow breathing on vascular function.

Obese people mostly have difficulty to control breathing while moving body during exercise, due to increased workload, and this can lead easily to dyspnea and eventually avoidance of exercise.[47] Our results also show improvement of autonomic function and inspiratory muscle strength, but not in VO_2max or aerobic capacity after slow and deep breathing implemented completely. Additionally, the use of the submaximal Astrand-Ryhming protocol in order to predict the VO_2max may less acute value when compared to a treadmill VO_2max. Previous study in hypertensive people showed that inspiratory muscle training with the load set at 30% of MIP, for 30 minutes, 7 days per week for 8 weeks, reduced blood pressure, improved autonomic function and MIP from 82.7±28.8 to 121.5±21.8 cmH_2O, but did not change maximal functional capacity.[48] The slightly improvement of inspiratory muscle strength in this study because of no resistive loading during breathing exercise; therefore, breathing exercise should be performed with resistive load for further improving respiratory muscle strength. This study showed the importance of exercise, as just slow breathing without resistance could improve autonomic and inspiratory muscle strength. Slow breathing in obese participants help to control breathing before initially doing other types of exercise. Limitations of our study include the small sample size and lack of included male participants. The recruitment of physically inactive female adults aged 20-30 years; therefore, our findings should not be generalized to the female population as a whole.

Conclusion

Slow breathing exercise is a challenged strategy for promoting health by improving autonomic function and inspiratory muscle strength in physically inactive people that can be served as a clinical home-based program in the future.

Conflict of interest

The authors declare no conflict of interest.

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