THE EFFECTS OF SLOW DEEP BREATHING EXERCISE AFTER MODERATE-INTENSITY INTERVAL TRAINING ON AEROBIC RECOVERY PERIODS IN OVERWEIGHT AND OBESE YOUNG ADULTS

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THE EFFECTS OF SLOW DEEP BREATHING EXERCISE AFTER MODERATE-INTENSITY INTERVAL TRAINING ON AEROBIC RECOVERY PERIODS IN OVERWEIGHT AND OBESE YOUNG ADULTS

BY

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Title	THE EFFECTS OF SLOW DEEP BREATHING EXERCISE AFTER MODERATE-INTENSITY INTERVAL TRAINING ON AEROBIC RECOVERY PERIODS IN OVERWEIGHT AND OBESE YOUNG ADULTS
Author	KATAVUT PHIWJINDA
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Obesity and overweight have become risk factors for health issues, among various weight control strategies, regular aerobic exercise is the recommended. Exercise interventions showed the benefits of weight loss. However, activities with more enjoyment and less exhaustion are required for the overweight. Therefore, the focus was to find an exercise activity suitable for beginners and finding a new technique to reduce the exhaustion of exercise. Recently, moderate-intensity interval training (MIIT) was introduced to the beginning of an exercise. MIIT intervention contains a couple of cycles of brief, moderate exercise intensity, following by a brief interval of low exercise intensity, the benefits of less exhaustion and more excitement. Therefore, the introduction of MIIT to an overweight population is suitable for exercise engagement. Another modality to reduce exhaustion is a slow, deep breathing technique. Besides enhancing cardiorespiratory function, slow deep breathing techniques can modulate the autonomic nervous reflex that can enhances exercise recovery. Therefore, this study characterized the exercise modality of moderate-intensity interval training (MIIT), in 25 young, adult and overweight participants with (BMI > 23 kg/m^2). The effects of the additional slow, deep breathing technique in the cool down phase of exercise programs to demonstrate whether could enhance the rate of exercise recovery. There were 25 participants (13 men and 12 females, 16 overweight and nine obese) performed 45 minutes of a cycling program (warm up for five minutes; three intervals of five minutes at 20-40% and three intervals of five minutes at 50-60%; cool down: 10 minutes) followed by 30 minutes of recovery. Each participant had three exercise trials: (1) oxygen consumption measurement; (2) slow, deep breathing (SDB) during 10-minute cool down; or (3) without slow deep breathing (as control). Heart rate variability evaluated changes in cardio-autonomic activity. The results demonstrated that MIIT exercise program consumed oxygen 0.71 L/min similarly between males and females. The results suggested that the slow deep breathing session in this study during cool down improved the fast phase with the parasympathetic reactivation. Unfortunately, the benefit of slow deep breathing was reverse to become the resistance to recovery during slow phase. This disadvantage might be due to the respiratory exhaustion resulted from controlled breathing effort.

Keyword : Slow deep breathing, Moderate-intensity interval training, Aerobic recovery period, Overweight, Heart rate variability

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CHAPTER 1
INTRODUCTION

Background

Overweight and obesity are resulted from a cumulative positive energy balance, in which slowly occurs on an average day-to-day basis for several years (Morgen & Sorensen, 2014). The prevalence of obesity has escalated worldwide in both developed and developing countries including Thailand (World Health Organization and Regional Office for the Western, 2000). Even though the prevalence of overweight and obesity was highest in middle and old age groups, the obese incidence in young adults were surprisingly elevated (Jitnarin et al., 2011). In Thai population study, the prevalence rate of overweight and obesity increased from 2.4% and 0.9% in 12-18 years old to 14.9% and 19.6% in 19-39 years old, respectively (Jitnarin et al., 2011). In addition, the progressively increase in obesity among young adults, especially college and university students, has been concerned (Butler, Black, Blue, & Gretebeck, 2004). It has been proposed that changing in lifestyle of young adults is the cause of overweight and obesity. The lifestyles of young adults aged between 18-25 years have been changed due to this age range in a period of 'transition' from adolescence to adulthood. Significant lifestyles including leaving home, attending university or college, starting work, developing relationships, and possibly cohabiting or marrying have been altered. These key points made young adults being vulnerable to control energy balance, leading to weight gain (Poobalan & Aucott, 2016). Therefore, energy uptake and energy utilization are needed to be focused in this group of population.

Overweight and obesity is well-known in inducing adverse metabolic conditions, including diabetes and cardiovascular disease, (Morgen & Sorensen, 2014). Obesity is associated with decreased physical performance leading to impaired activities in daily living. Adverse psychosocial conditions such as lowering self-esteem and body dissatisfaction were also established in obesity (Morgen & Sorensen, 2014). The adverse socioeconomic conditions in obesity were also reported in which the hospital cost for obesity was higher than that of non-obesity patients (Cawley & Meyerhoefer,

2012; Morgen & Sorensen, 2014; Muller-Riemenschneider, Reinhold, Berghofer, & Willich, 2008). All evidences imply that overweight and obesity have become the serious threats to global public health today. Therefore, the weight management program is a significant priority to prevent those health problems.

Besides calorie restriction, regular exercise is a potent weight control method in preventing metabolic disorders. Many exercise protocols have been developed for obesity. Although moderate-intensity continuous training is usually recommended by the American College of Sports Medicine (ACSM), high-intensity interval training (HIIT) is successful in preventing weight gain. HIIT consists of a brief period of high-intensity followed by longer bouts of low-intensity or rest (Su et al., 2019). High-intensity interval exercise (HIIT) is perceived to be more enjoyable (Bartlett et al., 2011) and has a time-efficient effect (Kong, Sun, Liu, & Shi, 2016) when compared to moderate-intensity continuous training. However, HIIT may not be suitable for the beginning program for overweight and obese population. Previous study demonstrated that obese people had low tolerable to maximal-level training than moderate-intensity training (Alkahtani, King, Hills, & Byrne, 2013). Obese women who perform walking exercise at 10% faster than the self-selected walking speed decreased the pleasure of training leading to poor adherence (Ekkekakis & Lind, 2006). Thus, a high-intensity regimen would not be recommended for the beginner. Recently, a moderate-intensity interval training is introduced for the initial. Due to brief period of moderate-intensity followed by longer bouts of low-intensity exercise, MIIT can be more enjoyable than HIIT and continuous exercise. MIIT becomes an alternative option when specific health risks or difficulties appear in applying HIIT or can be used as a pre-HIIT session within a long-term training program (Jimenez-Pavon & Lavie, 2017). MIIT has showed improving blood lipids and cardiovascular benefits (DeBusk, Stenestrand, Sheehan, & Haskell, 1990; Haskell et al., 2007). Therefore, MIIT might be suitable as an initial recommendation protocol for the overweight and obese population.

Besides enjoyable exercise, the rate of exercise recovery might impact the exercise engagement. In the view of psychological factors, obese people have not achieved their

exercise goals because of their physical discomfort (pain, fatigue or uneasiness) and too tried to exercise (Egan et al., 2013; Hussien, Brunet, Romain, Lemelin, & Baillot, 2020). This burden may lead to the negative experience of exercise (Ball, Crawford, & Owen, 2000). Exercise intolerance due to exertional dyspnea has been documented to decrease exercise capacity in obese persons (Parameswaran, Todd, & Soth, 2006). Higher fat distributions were documented and related to reduced cardiopulmonary endurance during exercise in obese women (J. Li, Li, Feuers, Buffington, & Cowan, 2001). The restriction of lung volume in overweight and obese was attributed by the increased chest wall fat (Babb, Wyrick, DeLorey, Chase, & Feng, 2008). Moreover, higher weight resistance involves a huge metabolic cost required for a given level of exercise. A previous study demonstrated that the energy expenditure per unit of workload on a bicycle ergometer was markedly high in obesity (Dempsey, Reddan, Balke, & Rankin, 1966). These lung restriction and weight resistance caused more exhaustion after exercise, and consequently dispirited to continuous the training. Based on this problem, faster recovery technique might help attenuating the despondent.

During post-exercise recovery, cardiopulmonary system works for delivering oxygen is used for energy restore and metabolic waste elimination (Di Prampero, Peeters, & Margaria, 1973; Gaesser & Brooks, 1984; Knuttgen, 1970). Sympathetic activation is reduced with an increase in parasympathetic activation during this cool down phase. However, the rate of autonomic changes was slow in obesity, in which the relative predominance of sympathetic activity was found in obesity (Rossi et al., 2015; Shenoy, Doreswamy, Shenoy, & Prakash, 2014). This alteration of autonomic balance could impair cardiopulmonary recovery after exercise in obesity (Lins, Valente, Filho, & Silva, 2015; Silva et al., 2017; Sung, Choi, & Park, 2006). Therefore, the intervention which can fasten aerobic recovery and leads to the engagement of exercise in overweight and obesity was concerned.

In our interest, slow deep breathing has been showed in improving pulmonary function and the balance of the autonomic nervous system. Previous studies reported that the acute effect of slow deep breathing (six breaths per minute for 2-10 minutes)

significantly increased pulmonary function when compared with regular spontaneous breathing in healthy volunteers (Shravya, Bandi, Suresh, & Mallikarjuna, 2013; Sivakumar, Prabhu, Baliga, Pai, & Manjunatha, 2011). In addition, deep diaphragmatic breathing exercises improved alveolar ventilation, tidal volume, and peak inspiratory volume in obese subjects (Olsen, Lonroth, & Bake, 1999). For the autonomic nervous system, slow deep breathing has reset the autonomic nervous system (ANS) through stretch-induced inhibitory signals and hyperpolarization currents propagated through both neural and non-neural tissue (connective tissue fibroblasts). The mechanism leads to a synchronization between neural elements in the heart, lungs, limbic system and cortex (Jerath, Edry, Barnes, & Jerath, 2006). During slow deep breathing, the respiratory modulation affects the autonomic nervous system, such that complete sympathetic inhibition was observed during early inspiration to mid-expiration (Russo, Santarelli, & O'Rourke, 2017). It is then possible that the autonomic disturbance found in overweight and obesity could be improved by slow deep breathing technique. As mentioned, it could be the be possible that slow, deep breathing technique at cool down and recovery phases might reduce autonomic disturbance in overweight and obese persons. It was then interesting to understand the effect of slow, deep breathing after exercise on the relationship between the rate of aerobic recovery and autonomic balance. Therefore, the present study aimed to test whether 1) the addition of slow deep breathing during a recovery state can enhance aerobic recovery and 2) the mechanism might be associated with the change in cardio-autonomic balance.

Objective of the Study

1. First objective was to investigate the effect of slow deep breathing on aerobic recovery periods after MIIT exercise cessation in overweight and obese young adult participants.

2. Second objective was to compare sympatho-vagal balance of overweight or obese young adults during exercise recovery between with and without slow deep breathing.

Hypothesis

1. The addition of slow deep breathing at cool down state after exercise can fasten aerobic recovery periods in overweight and obese young adult.

2. The addition of slow deep breathing at cool down state after exercise increases parasympathetic activation in overweight and obese young adult.

Significance of the Study

The information from the present study would support the use of slow deep breathing intervention in improving recovery state after moderate-intensity interval training (MIIT) exercise in overweight persons. The intervention could help diminishing exercise exhaustion leading to more physical exercise engagement.

Scope of the Study

The scope of the present study was determined the effect of breathing intervention on aerobic capacity during exercise recovery of young adult (18 to 25 years old) with the overweight or obesity (Body mass index > 23). Heart rate recovery and heart rate variability was be determined after moderate-intensity interval training (MIIT) exercise with or without slow deep breathing intervention during 10 minutes cool down. Twenty-nine obese participants were included in this study. Each participant performed three times of MIIT exercise including 1) MIIT with oxygen consumption analysis (1st visit), 2) MIIT exercise with slow deep breathing intervention during 10 minutes cool down, and 3) MIIT exercise without slow deep breathing. The experiment was set in laboratory at Srinakharinwirot University and Mahidol University, Thailand. Number of participants is calculated by power analysis based on previous information.

Definition of Terms

Acute effect

A physiological reaction in a human body resulting in symptoms that develop rapidly during short-term exposure to intervention.

Dyspnea

Difficulty breathing, shortness of breath.

Aerobic recovery period

The recovery of oxygen consumption and heart rate following aerobic exercise.

Heart rate recovery

The rate of heart rate decline following the cessation of exercise until reaching resting level.

Heart rate variability

The variation in time between each heartbeat for identifying autonomic balance.

Obesity

Abnormal or excessive fat accumulation that presents a risk to health. A crude population measure of obesity is the body mass index (BMI). In Asia, BMI of 27.5 or more is generally considered to be obese.

Parasympathetic activity

The action of the involuntary nervous system that decelerates the heart rate, increases intestinal and gland activity, and relaxes sphincter muscles in the gastrointestinal tract.

Slow deep breathing

The rate of breathing as any rate from 4 to 10 breaths per minute (0.07 - 0.16 Hz).

Sympathetic activity

The action of the involuntary nervous system that accelerates the heart rate, constricts blood vessels, and raises blood pressure.

List of Abbreviations

Abbreviation	Definition
% HR decline	Percentage of heart rate decline
% HR recovery	Percentage of heart rate recovery
% $\dot{V}O_2$ recovery	Percentage of oxygen consumption recovery
ACSM	American College of Sports Medicine
ACLS	Aerobics Center Longitudinal Study
ADL	Activities of daily living
AER	Albumin excretion rates
AMP	Adenosine monophosphate

List of Abbreviations (Continued)

Abbreviation	Definition
ANB	Alternate nostril breathing
ANOVA	Analysis of variance
ANS	Autonomic nervous system
ARCET	Astrand-Rhyming cycle ergometer test
ATP	Adenosine triphosphate
BAT	Brown adipose tissue
BD	Body dissatisfaction
BMI	Body mass index
BP	Blood pressure
bpm	Beats per minute
BUP	Bupropion
CAD	Coronary artery disease
CB1	Cannabinoid
CDK	Chronic kidney disease
cm	Centimeter
CO_2	Carbon dioxide
CON	Control trial
CPT1	Carnitine palmitoyl transferase I
CRH	Corticotropin-releasing hormone
CV	Cardiovascular
CVD	Cardiovascular disease
DA	Dopamine
DB	Deep breathing
DIO	Diet-induced obesity
DNL	De novo lipogenesis
EOSS	Edmonton Obesity Staging System
EPOC	Excess post-exercise oxygen consumption
ERV	Expiratory reserve volume
FA	Fatty acid
FRC	Functional residual capacity
FVC	Forced vital capacity

List of Abbreviations (Continued)

Abbreviation	Definition
GERD	Gastroesophageal reflux disease
HDL-C	High-density lipoprotein cholesterol
HFnu	High-frequency in normalized units
HIF1-alpha	Hypoxia inducible factor 1 alpha
HIIT	High-intensity interval training
HPA axis	Hypothalamic-pituitary-adrenal axis
HR	Heart rate
HRmax	Maximum heart rate
HRR	Heart rate recovery
HRV	Heart rate variability
Hz	Hertz
IPAQ-SF	Short form international Physical Activity Questionnaire
kg	Kilogram
kg/m^2	Kilogram per square metre
LDLC	Low-density lipoprotein cholesterol
LFnu	Low frequency in normalized units
MCP-1	Monocyte chemotactic protein-1
METs	Metabolic equivalent units
mg/dL	Milligrams per decilitre
MICT	Moderate-intensity continuous training
MIIT	Moderate-intensity interval training
min	minute
ML	Medial-lateral
ml/kg/min	Milliliters per kilogram per minute
mmHg	Millimetre of mercury
NAFLD	Nonalcoholic fatty liver disease
NE	Norepinephrine
OA	Osteoarthritis
OHS	Obesity-hypoventilation Syndrome
OSA	Obstructive sleep apnea

List of Abbreviations (Continued)

Abbreviation	Definition
PDH	Pyruvate dehydrogenase
PPAR	Peroxisome proliferator-activated receptor
R^2	R squared
RER	Respiratory exchange ratio
RMSSD	Root mean square of successive differences
RPE	Rate perceived exertion
RPM	Revolutions per minute
RSA	Respiratory sinus arrhythmia
RT	Resistance training
SD	Standard deviation
SDB	Slow deep breathing
SDNN	Standard deviation of the normal-to-normal intervals
SDRR	Standard deviation of the interbeat intervals for all sinus beats in milliseconds
SHBG	Sex hormone-binding globulin
SIT	Sprint interval training
T2DM	Type 2 diabetes
TG	Triglyceride
TLC	Total lung capacity
TNF	Tumor necrosis factor
VC	Vital capacity
VEGF	Vascular endothelial growth factor
$\dot{V}O_2$	Oxygen consumption
$\dot{V}O_2max$	Maximal oxygen consumption
$\dot{V}O_2peak$	Peak oxygen consumption
WAT	White adipose tissue
WHO	World Health Organization
WHR	Ratio of waist to hip circumference

Conceptual framework

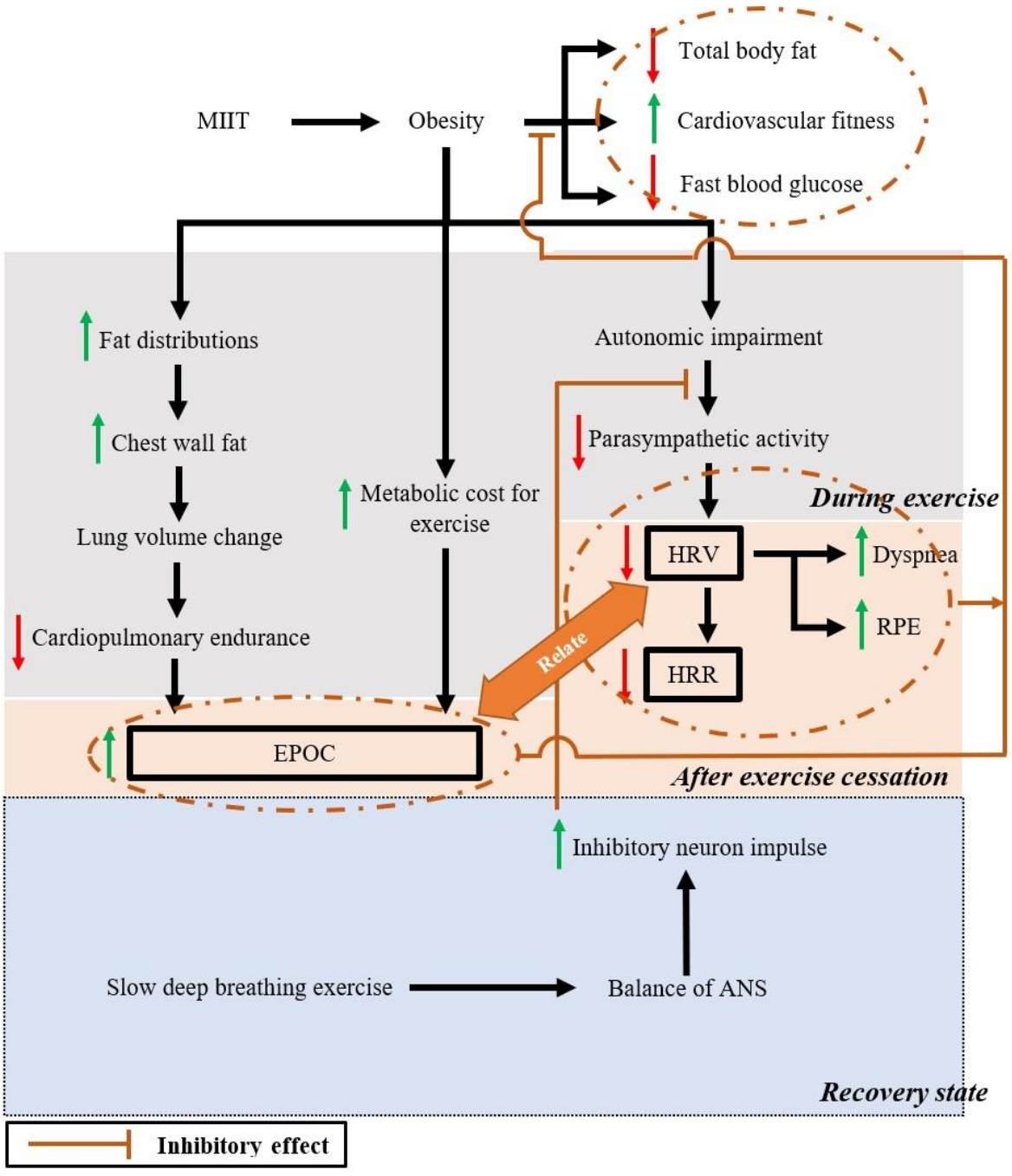


Figure 1 Conceptual framework of research dissertation.

CHAPTER 2
LITERATURE REVIEW

This chapter explains the background for this dissertation based on quality materials and relevant literature. There is extensive literature in both the medical and physiology related to the aspects of breathing mechanisms, and physical activity in obesity. This literature review includes books, journals and web encyclopedias. This chapter consists of all basic aspects and concepts that review the current knowledge relating to the study.

Obesity

According to Hubbard, in 2000, overweight and obesity were defined as the amount of excess body fat, in which health risks to individual is increased. In addition, the World Health Organization (WHO) expresses obesity as a chronic disease. It is widespread in developed and developing countries (Hubbard, 2000).

1. Epidemiology of obesity

Among Asia Pacific countries, the prevalence rates of overweight and obesity were represented to be low in Vietnam and India (1.7 % and 1.9 %, respectively), as shown in Figure 2. Malaysia and Thailand had a high prevalence rates of obesity among the south east Asia region, 14 % and 8.8 %, respectively (Cheong, 2014).

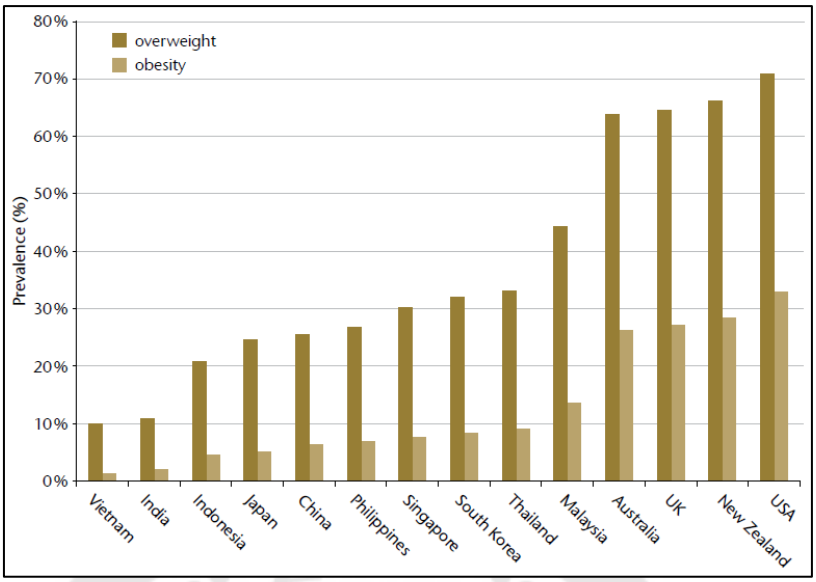


Figure 2 Prevalence rates of overweight and obesity in Asia Pacific countries compared to Australia, UK, New Zealand and USA, 2008.

Source: Cheong, W. S. (2014). Overweight and Obesity in Asia. General Reinsurance AG: https://www.genre.com/knowledge/publications.

According to Figure 3, while the change in overweight and obesity prevalence had almost stabilized in the last five years in the USA, prevalence tended to increase at a faster rate in many Asian countries. Between 1980 and 2013, overweight and obesity prevalence in Chinese adults rose from 11.3 % to 27.9 % (Cheong, 2014). The increasing rate in obesity prevalence of Malaysian adults was also reported from 4.4 % to 14 % in 1996-2006 (Khor, 2012).

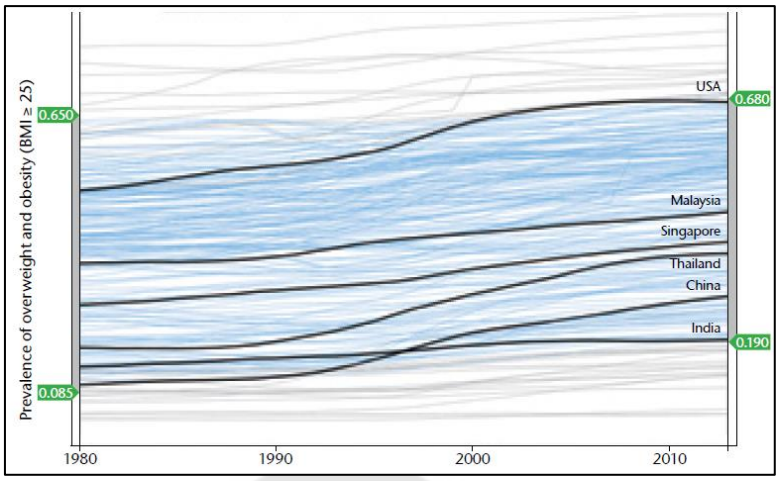


Figure 3 The change in overweight and obesity prevalence in some Asian countries in comparison to the United States, 1980 – 2013.

Source: Cheong, W. S. (2014). Overweight and Obesity in Asia. General Reinsurance AG: https://www.genre.com/knowledge/publications/uwfocus14-2-cheong-en.html.

Interestingly, the cross-sectional surveys of obesity among Thai adults in a rural community found that the prevalence of obesity increased from 33.9% in 2012 to 44.8% in 2018, as shown in Figure 4. The average BMI increased from 23.9 kg/m^2 in 2012 to 25.0 kg/m^2 in 2018 (Sakboonyarat *et al.*, 2020). Comparing all the regions across Thailand, the highest prevalence of overweight and obesity rates was highest in Bangkok (17.8% and 29.5%, respectively) (Jitnarin *et al.*, 2011).

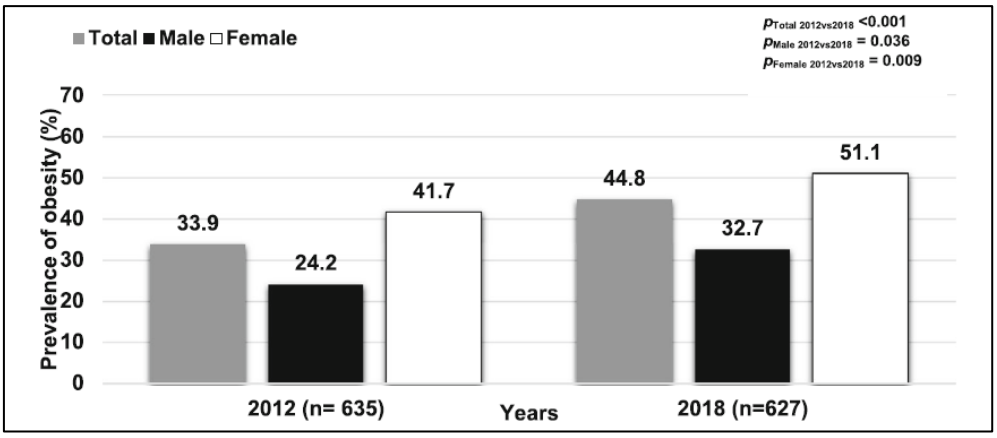


Figure 4 Trends in the prevalence of obesity stratified by sex in a rural community in Thailand, 2012 and 2018.

Source: Sakboonyarat, B., Pornpongsawad, C., Sangkool, T., Phanmanas, C., Kesonphaet, N., Tangthongtawi, N., Rangsin, R. (2020). Trends, prevalence and associated factors of obesity among adults in a rural community in Thailand: serial cross-sectional surveys, 2012 and 2018. BMC Public Health, 20(1), 850.

2. Measurement of obesity

2.1 Body mass index

Body mass index (BMI; in kg/m^2) is widely recognized as a weight-for-height index with a high correlation with adiposity. According to WHO Consultation in 2000, BMI was classified for treatment planning as shown in Table 1.

Table 1 Classification of overweight and obesity by BMI (WHO Consultation, 2000).

Classification	Obesity class	BMI (kg/m^2)
Underweight		< 18.5
Normal		18.5 – 24.9
Overweight		25.0 – 29.9
Obesity	I	30.0 – 34.9
Severe obesity	II	35.0 – 39.9
Morbid obesity	III	40.0 – 49.0
Severe morbid obesity	III	> 50

Source: WHO Consultation. (2000). Obesity: preventing and managing the global epidemic. Report of a WHO consultation. World Health Organ Tech Rep Ser, 894, i-xii, 1-253.

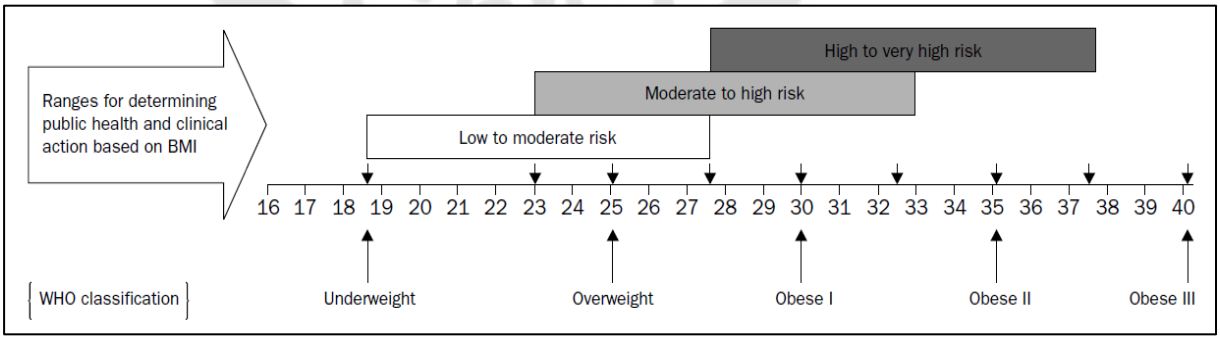


Figure 5 Body-mass index (BMI) cut-off points for public health action.

Source: WHO Expert Consultation. (2004). Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. Lancet, 363(9403), 157-163.

As shown in Figure 5, the body-mass index (BMI) cut-off points for public health action is presented. Many countries could make decisions to define BMI from the risk level for their population and public health action (WHO Expert Consultation, 2004). In Asian population, people had a higher risk of morbidity and mortality at the same BMI than the non-Asian populations (WHO Expert Consultation, 2004). Finally, the classification of BMI in the Asian population was 23 - 27.5 kg/m^2 in overweight and more than 27.5 kg/m^2 in obese. This classification of BMI may also correspond to the public health impact of overweight and obesity among Asian Americans (Jih *et al*., 2014)

2.2 Waist circumference and waist-hip ratio

Abdominal or visceral fat is related to cardiovascular risk factors, type 2 diabetes and hypertension. Therefore, the abdominal or visceral fat is a concern for the prediction or plan in the treatment of obesity. The ratio of waist to hip circumference (WHR) is used for the estimation of abdominal obesity. This is calculated as waist measurement divided by hip measurement (W/H). In Caucasians, a WHR of more than 1.0 for men and WHR of more than 0.85 for women indicate the abdominal fat accumulation (James, 1996). Waist circumference is also a convenient and simple method correlated with BMI, WHR, and abdominal obesity (WHO Consultation, 2000). South Asians such as Indians, people have a high prevalence of abdominal obesity, though they may not be determined obese by BMI criteria (McKeigue, Shah, & Marmot, 1991). According to WHO report in 2000, waist circumference ≥ 90 centimeters in men and ≥ 80 centimeters in women have been used as cut-off points for co-morbidities risk. The relation between levels of BMI and waist circumferences and co-morbidities level was shown in Table 2. It is then important to include waist measures in any assessment of obesity. Therefore, reduction in waist circumference with no weight change may result in significant risk reduction (World Health Organization and Regional Office for the Western, 2000).

Table 2 Co-morbidities risk associated with different levels of BMI and suggested waist circumference in adult Asians (World Health Organization and Regional Office for the Western, 2000).

Classification	BMI (kg/m^2)	Risk of co-morbidities	
		Waist circumference	
		< 90 cm (men)	≥ 90 cm (men)
		< 80 cm (women)	≥ 80 cm (women)
Underweight	< 18.5	Low (but increased risk of other clinical problems)	Average
Normal range	18.5 - 22.9	Average	Increased
Overweight:	≥ 23		
At risk	23 – 24.9	Increased	Moderate
Obese I	25 – 29.9	Moderate	Severe
Obese II	≥ 30	Severe	Very severe

Source: World Health Organization and Regional Office for the Western, P. (2000). The Asia-Pacific perspective: redefining obesity and its treatment: Sydney: Health Communications Australia.

2.3 Body fat percentage

By WHO definition, overweight and obesity are interpreted as an excessive or abnormal accumulation of fat in the body, creating impaired health (World Health Organization, 2020). Excessive adipose tissue is an essential factor affecting cardiovascular diseases (Akoumianakis & Antoniades, 2017). Some investigators believed that the body fat content is more effective indicator of true obesity (Ho-Pham, Lai, Nguyen, & Nguyen, 2015). Given of the clinical definition of obesity, the assessment

should be based on body fat percentage. Obesity is defined in young adults as body fat >25% in males and >35% in females (Lobman, Houtkooper, & Going, 1997; WHO Expert Committee, 1995).

1.3 Factors associated with obesity

1.3.1 Sociodemographic factors

Age and gender

The prevalence of obesity was related to increasing age in the USA, Europe and Asia (Cheong, 2014). For Thai adults, the increased prevalence was observed in populations aged 15–29 years (19.5%), 30 - 44 years (38.4%), and 45 - 59 years (42.4%), respectively. This trend was similar for both genders (Aekplakorn *et al*., 2014).

Cultural level

Some studies reported the relationship between cultural level and obesity prevalence. The cultural level in these papers included three aspects (beliefs, educational levels, and cultural eating habits). First, some obese people believe that big bodies or getting fat is a sign of wealth and happiness. Women's obesity is considered a sign of wealth in developing countries (Misra et al., 2001). Another belief was reported that happiness and contentment could be expressed by big bodies and excessive body fat in obese people (Garza & Zárate, 2007). Second, the prevalence of obesity was more elevated at lower educational levels (Serra-Majem & Bautista-Castano, 2013). For cultural eating habits, the act of eating or being fed by parents is an indicator of well-being and energy balance in obese babies through family culture (Garza & Zárate, 2007; Tschann et al., 2013).

Socioeconomic level

A study of socioeconomic levels including education and income was related to the increasing of body weight (McLaren, 2007). The behavior of obese persons was influenced by the socioeconomic level in different countries (Monteiro, Moura, Conde, & Popkin, 2004). A previous study in developing countries such as India suggested that the socioeconomic level, including high income in the city, was positively associated with diabetes and obesity (Serra-Majem & Bautista-Castano, 2013). On the other hand, school-based education was found be negatively associated (Ramachandran, Mary,

Yamuna, Murugesan, & Snehalatha, 2008). The reason about the difference in obesity prevalence between high and lower socioeconomic level in this population may be explained by three reasons including 1) food insufficiency and high energy expenditure activity in the poor people, 2) the greater ability to get sufficient food in the elite people and 3) the cultural belief of favoring fat body shapes in elite people (Monteiro et al., 2004).

1.3.2 Lifestyle related factors

Sedentary lifestyles

A high prevalence of obesity has been observed in sedentary persons who never play any sports. In theory, sedentary lifestyles include activities involving low energy expenditure between 1.0 to 1.5 metabolic equivalent units (METs) (Pate, O'Neill, & Lobelo, 2008). According to guidelines for data processing and analysis of the International Physical Activity Questionnaire (IPAQ) short form, continuous physical activity of less than 10 minutes with less than half an hour per day is classified as "Low physical activity" (Fan, Lyu, & He, 2014). Low regular physical activity or sedentary are usually involved in excess body weight (Serra-Majem & Bautista-Castano, 2013). Therefore, physical training is a necessary component of obesity treatment.

Diet

Although high energy expenditure from physical activity plays an important role in reducing weight gain (Prentice & Jebb, 1995), dietary intake is another important factor in the lightening of obesity (Serra-Majem & Bautista-Castano, 2013). Several human investigations concluded that high-energy foods and liquor encouraged overconsumption and obesity (Lawton, Burley, Wales, & Blundell, 1993; Sclafani, Weiss, Cardieri, & Ackroff, 1993). The study about the relationship between obesity and diet has usually focused on the role of energy-dense foods that contain a high proportion of fat (Poppitt & Prentice, 1996). Many reports confirmed that habitual alcohol, high fat and high carbohydrate intake induce excess body weight by increasing fat mass and adiposity (Macdiarmid, Cade, & Blundell, 1996; Serra-Majem & Bautista-Castano, 2013; Willett, 2003).

According to general research on obesity, diet-induced obesity (DIO) contains highly delicious calorie-dense foods in modern cultures. The causes of increasing DIO food intake are the luxurious attraction, food preferences, cannabinoid (CB1) signaling and hunger hormone such as ghrelin (Figlewicz & Benoit, 2009; Schwartz, Woods, Porte, Seeley, & Baskin, 2000). Opposing these factors are leptin action, parallel with other mediators, including promoting satiety feeling, insulin action and many gut-derived signals. Altering the leptin strength or sensitivity change the amount of food consumption and affects adiposity. While decreasing the strength of the leptin signal increases eating by decreasing a loss of appetite (anorectic drive), it increases in the deliciousness and/or food availability (Myers, Leibel, Seeley, & Schwartz, 2010). The mechanism of increased food intake in the DIO or pleasant food favors resulting in increased fat may be explained by creating a new baseline feeding with the chance of higher adiposity. In obese people, the cellular mediators of leptin resistance are promoted in the hypothalamus, which limit leptin action and increases the amount of leptin or adiposity for requiring to suppress feeding levels (Myers et al., 2010).

Smoking cessation

The increasing body mass index (BMI) has been demonstrated in persons who cease smoking. A case study reported that male and female weights gained 4.4 kg and 5 kg of boy weight respectively, after smoking cessation (Serra-Majem & Bautista-Castano, 2013). Because of the removal of nicotine's effects on the central nervous system, the smokers gain weight after smoking cessation (Audrain-McGovern & Benowitz, 2011; Komiyama et al., 2013). Some smokers try to manage nicotine withdrawal by eating to replacement the "hand to mouth" behavior of smoking that induces an increase in energy intake (Jo, Talmage, & Role, 2002; Komiyama et al., 2013).

Number of children (parity)

Gravida numbers are related to obesity. With the same habitat and age group, the women who had more children tend to increase their weight compared to nulliparous women (Serra-Majem & Bautista-Castano, 2013). Although the mechanisms of the association between obesity and parity are still inconclusive (Hajiahmadi, Shafi, &

Delavar, 2015), some studies suggest that high maternal glucose, free fatty acid and amino acid concentrations may play an important role in weight gain (Gunderson, 2009; Martínez et al., 2013). During pregnancy, the release of placental corticotropin-releasing hormone (CRH) may motivate the maternal hypothalamic-pituitary-adrenal axis (HPA axis: a complex set of direct influences and feedback interactions among hypothalamus, pituitary gland and adrenal gland) and increase cortisol concentrations (Magiakou et al., 1996). Increased cortisol plays a role in the pathophysiological mechanism of abdominal obesity (Pasquali, Vicennati, Cacciari, & Pagotto, 2006). Furthermore, psychosocial stress, socioeconomic, traits of depression, unhealthy lifestyles and anxiety during pregnancy may also lead to hypothalamic-pituitary-adrenal hyperactivity (Diamanti-Kandarakis & Economou, 2006). Therefore, a greater number of gestations are associate with the risk of obesity in women.

1.4 Mechanism of obese by adipose tissue

The cause of obesity is an imbalance of energy. When people intake calories diet more than expenditure energy, they become obese due to excess body fat under their skin and visceral area. The pathophysiology of adipose tissue expansion becomes a key aspect of comorbidity that contributes to cardiovascular disease, diabetes and cancer (Rajala & Scherer, 2003).

1.4.1 Adipose tissue depots and obesity

Adipose tissue is various mix of adipocytes, endothelium, stromal preadipocytes and immune cells (Halberg, Wernstedt-Asterholm, & Scherer, 2008). Adipose tissue functions like a complex endocrine organ secretes a host of factors together as adipokines (Halberg et al., 2008). The adipocyte can release leptin and the highly adipocyte-specific protein adiponectin (Chudek & Wiecek, 2006). Two types of adipose tissue are deposited in several parts of the body including white and brown adipose tissues. The white adipose tissue (WAT) expanded at visceral or abdominal organs is powerfully correlated to cardiovascular disease and insulin resistance in humans (Haffner, 2007). Compared with visceral area, subcutaneous WAT does not negatively effects on metabolism (Kim et al., 2007). In contrast, brown adipose tissue (BAT), is well-supplied with mitochondria and highly vascularized. BAT generates body heat in

infants (Seale, Kajimura, & Spiegelman, 2009), and can be called "good adipose" because of no adverse effects like visceral WAT (Nedergaard, Bengtsson, & Cannon, 2007; Seale et al., 2009).

1.4.2 Body fat distribution

The various fat depots contain unique characteristics dividing by the area of fat deposition. Central obesity is considered as excess fat stored in the abdominal area and peripheral obesity is the store of excess fat in the hips, buttocks and thighs (Aras, Ustunsoy, & Armutcu, 2015). In addition, an upper body or visceral fat distribution includes pericardial and buccal fat, and superficial and deep abdominal fat (Smith et al., 2001). A lower body fat distribution is the storage of all adipose tissue caudal to the inguinal ligament anteriorly and the ileac crest posteriorly, including gluteal, leg depots and adipose tissue in between the major muscle groups at legs (Goodpaster, Thaete, & Kelley, 2000). Most women have a higher percentage of body fat and a lower percentage of muscle mass than men. The fat depots in woman are likely to be lower body fat distribution with subcutaneous white adipose tissue in gluteofemoral regions (Varlamov, Bethea, & Roberts, 2014) as shown in Figure 6.

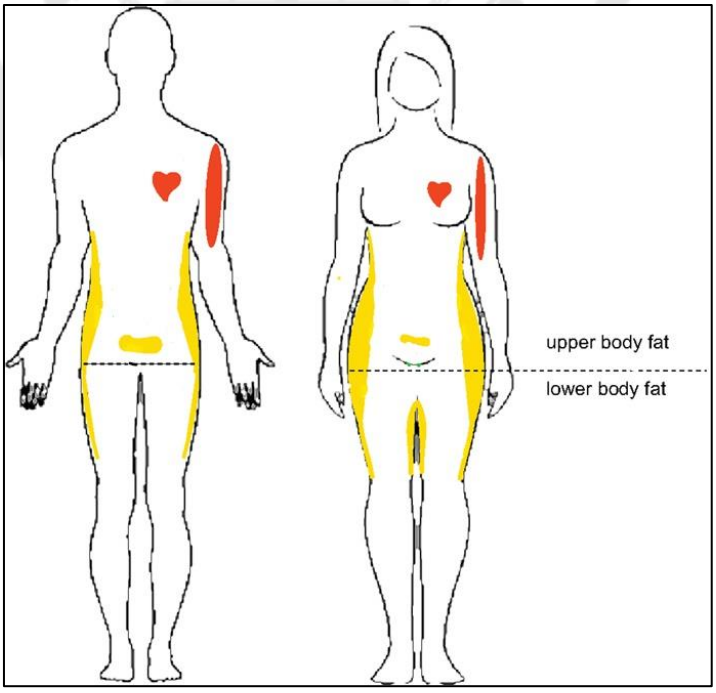


Figure 6 Genders and depot-specific differences in human.

Source: Varlamov, O., Bethea, C. L., & Roberts, C. T., Jr. (2014). Sex-specific differences in lipid and glucose metabolism. Front Endocrinol (Lausanne), 5, 241.

Interestingly, an upper body or visceral fat distribution is significantly associated with obesity-related metabolic complications in obesity (Jensen, 2008). There was hypothesized that visceral fat is an "ectopic fat depot" (Rasouli, Molavi, Elbein, & Kern, 2007). Visceral fat is the location of ectopic triglyceride accumulation from exceeded fat storage as the result of energy imbalance (Jensen, 2008). Upper body fat distribution could interrupt lipid metabolism by reducing the ability of take up triglycerides leading to postprandial hypertriglyceridemia (Evans et al., 2008). When compared between upper body obesity and lower body obesity, the greater postprandial free fatty acid concentrations could be totally considered by free fatty acid release from upper body fat (Guo, Hensrud, Johnson, & Jensen, 1999). Thus, upper body obesity is highly associated with cardiovascular risk. Based on gender difference in fat distribution, the higher lower fat deposition in women might relate with a lower cardiovascular problem as compared to men (Koutsari, Dumesic, Patterson, Votruba, & Jensen, 2008). Moreover, waist circumference is a generally representative marker for upper body fat and the associations between waist circumference and cardiovascular risk factors are similar for men and women (Grundy, Williams, & Vega, 2018).

1.4.3 Adipose tissue angiogenesis

Expansion of adipose tissue rapidly induces hypoxia and vascular angiogenesis (Figure 7) (Halberg et al., 2008). The expansion of WAT induced hypoxia and increasing levels of hypoxia inducible factor 1 alpha (HIF1-alpha), leading to the upregulation of the inflammatory adipokines IL-6, tumor necrosis factor-alpha (TNF-alpha), and monocyte chemotactic protein-1 (MCP-1) secretions. These pro-inflammatory secretory products were associated with insulin resistance (Trayhurn, Wang, & Wood, 2008) and tissue fibrosis (Halberg et al., 2009; Khan et al., 2009). Eventually, it increases the chance of metabolic disorders (Halberg et al., 2009).

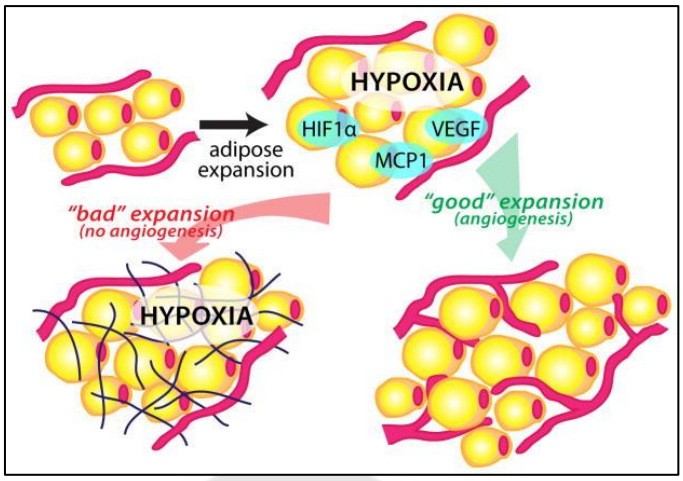


Figure 7 Adipose expansion results in tissue hypoxia.

Source: Rutkowski, J. M., Davis, K. E., & Scherer, P. E. (2009). Mechanisms of obesity and related pathologies: the macro- and microcirculation of adipose tissue. FEBS J, 276(20), 5738-5746.

There are different responses to hypoxia between two types of adipose tissue. A study described that hypoxia induced the major inflammatory response in the WAT with increasing expression and release of inflammation-related adipokines, blocking the differentiation of preadipocytes and stimulating glucose transport by adipocytes (Trayhurn et al., 2008). To counter hypoxia, WAT induced angiogenesis by secreting angiogenic factors as well as increasing macrophages, circulating progenitors and endothelial cells (Halberg et al., 2008). On the other hand, BAT increased vascular endothelial growth factor (VEGF), vascular density expansion and angiogenesis in response to hypoxia during exposure to cold temperature (Xue et al., 2009).

1.5 Morbidity and mortality associated with obesity

1.5.1 Obesity and its major comorbidities

Diabetes mellitus

It is well known that high body weight is associated to diabetes. Fifty percentage of obese persons in developing countries were diagnosed type 2 diabetes (T2DM) while the risk of T2DM increased by 20% for each 1 kg/m^2 increment of BMI (Abdelaal, le

Roux, & Docherty, 2017). The elevation of plasma free fatty acid levels in obesity could increase hepatic glucose production, obstruct muscle glucose utilization and stimulate insulin secretion from pancreatic beta (β) cells (Shulman, 1999). This pathophysiology involved hyperinsulinemia and hyperglycemia. Obesity induces insulin resistance by inducing high levels of tumor necrosis factor (TNF) and free fatty acid from excess adipocytes, as shown in Figure 8 (Khaodhiar, McCowen, & Blackburn, 1999).

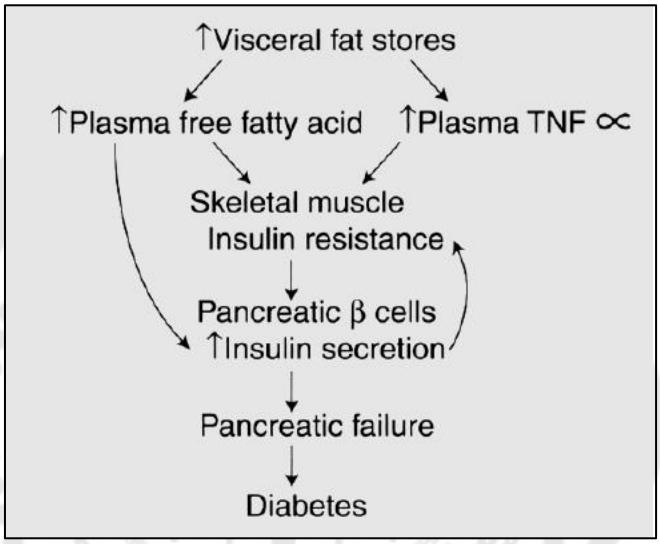


Figure 8 Pathogenesis of type 2 diabetes in obese individuals.

Source: Khaodhiar, L., McCowen, K. C., & Blackburn, G. L. (1999). Obesity and its comorbid conditions. Clin Cornerstone, 2(3), 17-31.

Airway and respiration

The airway and respiration complications were found in obese persons (Abdelaal et al., 2017; Parameswaran et al., 2006; Piper & Grunstein, 2011). The excess abdominal adipose tissue impacted on diaphragm leading to lung volume reduction such as expiratory reserve volume (ERV), functional residual capacity (FRC), and total lung capacity (TLC) (Abdelaal *et al.*, 2017). Obesity hypoventilation Syndrome (OHS) is defined as the combination of obesity and chronic daytime hypercapnia (Partial pressue of arterial carbon dioxide ≥ 45 mmHg). This disease could be associated with higher

morbidity and mortality rates than obstructive sleep apnea (OSA) or simple obesity (Piper & Grunstein, 2011).

Cardiovascular disease (CVD)

Obesity is common and associated with an increased risk for metabolic syndrome associated with CVD. Central obesity relates to high serum triglyceride (TG) levels, low serum high-density lipoprotein cholesterol (HDL), hypertension, and elevated fasting blood glucose levels. These changes induced cardiac dysfunction leading to cardiac hypertrophy and heart failure. (Abdelaal *et al.*, 2017). As shown in Figure 9, increased free fatty acid in obesity is the major risk factor in developing heart failure and other weight-related comorbidities (Gadde, Martin, Berthoud, & Heymsfield, 2018).

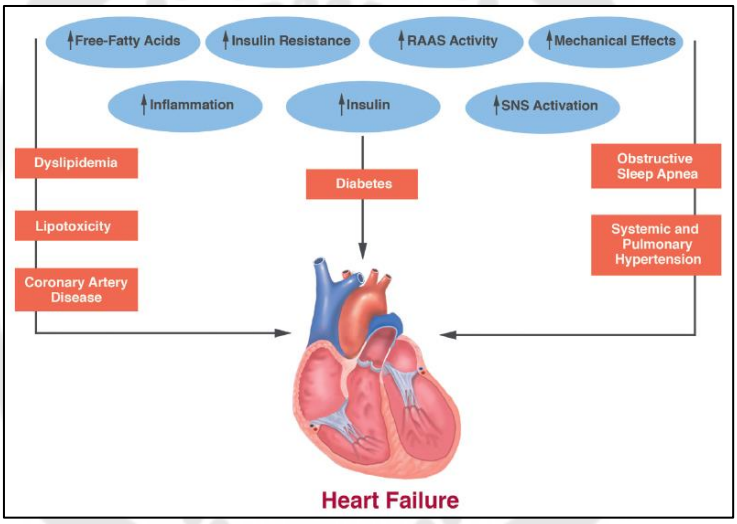


Figure 9 Adiposity-associated major risk factors for developing heart failure and other weight-related comorbidities.

Abbreviations: RAAS = renin-angiotensin aldosterone system, SNS = sympathetic nervous system.

Source: Gadde, K. M., Martin, C. K., Berthoud, H. R., & Heymsfield, S. B. (2018). Obesity: Pathophysiology and Management. J Am Coll Cardiol, 71(1), 69-84.

Hypertension

One of the comorbidities in obesity is hypertension. Previous studies reported that the prevalence of hypertension progressively increases with higher BMI in both genders

(C. D. Brown et al., 2000). The risk of developing hypertension was positively associated with both waist-hip ratio and waist circumference (Canoy et al., 2004). The underlying mechanism could explain by intravascular volume expansion resulting from sodium retention. Histologic change in renal medulla in obesity including deposition of non-cellular matrix and interstitial cell proliferation could lead to compression of tubules and vasa recta, then become increased sodium reabsorption (Hall, 1994). This factor induced an increase in cardiac output, and venous return, and peripheral vascular resistance (Hall, 1994; Kolanowski, 1999). Consequently, prolong hypervolemia with hypertension induces a changing of pressure natriuresis to higher blood pressure (Kolanowski, 1999). This mechanism modifies kidney and cardiovascular system, which may be related to the increase in sympathetic nervous activity, activation of the renin-angiotensin-aldosterone system and insulin resistance.

Kidney

Obesity is related to glomerulopathy and chronic kidney disease (CKD) because early renal impairment and elevated risk of cardiovascular (CV) morbidity are induced by increasing albumin excretion rates (AER) (Gilardini et al., 2010; Tsuboi et al., 2012). The mortality rate was reported to associate with kidney diseases as the increasing 5 kg/m^2 of BMI (Prospective Studies et al., 2009). Moreover, dyslipidemia and lower concentrations of high-density lipoprotein (HDL) in obesity induce inflammation and endothelial dysfunction leading to CKD (Goek et al., 2012; Ruan, Varghese, & Moorhead, 2009).

Liver

Non-alcoholic fatty liver disease (NAFLD) consists of non-alcoholic steatohepatitis (NASH), hepatic steatosis, cirrhosis and fibrosis (Anstee, Targher, & Day, 2013; Loomba & Sanyal, 2013). A report described that obesity and insulin resistance were main factors of NAFLD (Machado, Marques-Vidal, & Cortez-Pinto, 2006). Hepatic steatosis is caused by an imbalance between triglyceride (TG) production and utilization. In addition, the hepatic production of TG consists of free fatty acids derived from food, de novo lipogenesis (DNL: the creation of fat from non-fat sources) and adipose tissue lipolysis (Z. Li, Clark, & Diehl, 2002).

Gonadal

A cohort study suggested that androgen disorder, insulin resistance, and hyperinsulinism induced subfertility in obese patients (Gosman et al., 2010). Obesity induces the impaired secretion and activity of sex hormones on both estrogen and androgens (Abdelaal et al., 2017). An increase in aromatization, turning androgen to estrogen in obesity. In addition, there was a decrease in the hepatic synthesis of sex hormone-binding globulin (SHBG) due to the enlarged adiposity. These changes induced abnormal folliculogenesis and follicular atresia in obese women (Levens & Skarulis, 2008).

Gastroesophageal reflux

The high prevalence of gastroesophageal reflux disease (GERD) is related to obesity (Y. Y. Lee & McColl, 2013; Pandolfino, Kwiatek, & Kahrilas, 2008). BMI higher than 30 kg/ m^2 was associated with acid reflux episodes, pH less than 4 in the esophagus and reflux lasting longer than 5 minutes (El-Serag et al., 2007). The production of inflammatory mediators and the increased esophagogastric pressure gradient in obesity were underlined in the pathogenesis of GERD (Emerenziani, Rescio, Guarino, & Cicala, 2013).

Cancer

The prevalence of cancer is associated with obesity in which high-fat diet or de novo synthesized fatty acids turned into pro-tumorigenic signaling lipids leading to cancer cell induction (Nomura et al., 2010). Furthermore, the main source of estrogen biosynthesis in obese postmenopausal women is adipose tissue, which then activated breast tumors (Bezemer et al., 2005; Lukanova et al., 2004).

Body dissatisfaction

Body image is defined as a psychological factor of a person including body shape, size, form of self-body, and the feeling about body components. Body dissatisfaction (BD) is depended on the reality and an individual's ideal of body image (Slade, 1994; Williams, Hudson, Whisenhunt, & Crowther, 2014). The sedentary lifestyle causes obese people to be socially isolated, depressed or discriminated against

(making them to be the target of bullying, poor self-esteem or body image distortions) (Dietz, 1998; Trull, Verges, Wood, Jahng, & Sher, 2012).

1.5.2 Mortality associated with obesity

As mentioned above, obesity is associated with a risk of cardiovascular disease occurrence and mortality. Even the relationship between BMI and mortality rate substantially varies among populations, obesity is becoming the leading cause of declining well-being over time. There are a few criteria of obesity related to mortality that help medical personnel for healthcare management.

Edmonton Obesity Staging System (EOSS)

The EOSS classifies the impact of obesity on an individual into five stages of severity. Stage 0 represents the obese with no comorbidities (Padwal, Pajewski, Allison, & Sharma, 2011). The EOSS definition and modified study definition is shown in Table 3.

Table 3 Edmonton Obesity Staging System (EOSS) definition (Kuk *et al*., 2011)

Stage	Conceptual description	Modified study definition*
0	No apparent obesity-related risk factors (e.g., BP, serum lipids, fasting glucose, etc., within normal range), no physical symptoms, no psychopathology, no functional limitations and (or) impairment of well being	No reported EOSS factors
1	• Presence of obesity-related subclinical risk factors (e.g., borderline hypertension, impaired fasting glucose, elevated liver enzymes, etc.) • Mild physical symptoms (e.g., dyspnea on moderate exertion, occasional aches and pains, fatigue, etc.) • Mild functional limitations • Mild psychopathology and (or) • Mild impairment of well being	• BP ≥ 130/85 and (or) < 125/75 mmHg for individuals with type 2 diabetes • Fasting glucose ≥ 100 and < 125 mg/dL • Cholesterol ≥ 200 and < 240 mg/dL • Triglycerides ≥ 150 and < 200 mg/dL • High-density lipoprotein < 60 mg/dL • Shortness of breath during physical activity

Table 3 (Continued)

Stage	Conceptual description	Modified study definition*
2	• Presence of established obesity-related chronic disease (e.g., hypertension, type 2 diabetes, sleep apnea, osteoarthritis, reflux disease, polycystic ovary syndrome, anxiety disorder, etc.) • Moderate limitations in activities of daily living and (or) • Moderate impairment of well being	• Diagnosed hypertension or hypertension medication • Blood pressure ≥ 140/90 mmHg or 130/80 for individuals with type 2 diabetes • Type 2 diabetes • Fasting glucose ≥ 125 mg/dL • Diagnosed hypercholesterolemia • Cholesterol ≥ 240 mg/dL • Diagnosed hypertriglyceridemia • Triglycerides ≥ 200 mg/dL • High-density lipoprotein < 40 mg/dL • Gout • Depression • Fatigue • Urinary leakage • Low back pain • Joint stiffness • Reported emotional outlook of "generally sad," or • Self-reported health of "fair"

Table 3 (Continued)

Stage	Conceptual description	Modified study definition*
3	• Established end-organ damage (e.g., myocardial infarction, heart failure, diabetic complications, incapacitating osteoarthritis, etc.) • Significant psychopathology • Significant functional limitations and (or) • Significant impairment of well being	• Reported chest pain • Chest pain during exercise • Heart attack • Calf pain during exercise • Stroke • Shortness of breath when sleeping • Shortness of breath when sitting • Psychiatric or psychological counseling, or • Moderate or severe cardiomegaly • Reported emotional outlook of “often depressed”, or • Self-reported health of “poor”
4	• Severe (potentially end-stage) disabilities from obesity-related chronic diseases • Severe disabling psychopathology • Severe functional limitations and (or) • Severe impairment of well being	This stage was not examined as these factors were not available in the ACLS database

***** Based on the availability of data in the Aerobics Center Longitudinal Study (ACLS) dataset.

Source: Kuk, J. L., Ardern, C. I., Church, T. S., Sharma, A. M., Padwal, R., Sui, X., & Blair, S. N. (2011). Edmonton Obesity Staging System: association with weight history and mortality risk. Appl Physiol Nutr Metab, 36(4), 570-576. doi:10.1139/h11-058

Physical performance

Physical performance is described as an objective measurement of whole-body function related to mobility (Beaudart et al., 2019). Physical performance is depended on muscle function including muscle power and endurance, producing activities of daily living (ADL). The impairment of physical performance is the inability to perform ADL, which may be a sign of the beginning of disability (Beaudart et al., 2019).

A decline in physical performance in obese people affects organ systems during exercise. A decline in physical performance is related to the regional distribution of fat in the chest wall, which promotes lower pulmonary function. In comparison to normal-weight subjects, obese persons had lower tidal volume and minute ventilation, leading to exercise intolerance (Littleton, 2012). The limitation of exercise performance may be due to a rapid, shallow breathing pattern from the increased dead space ventilation in obesity. As a result, it is difficult to achieve demanded minute ventilation during exercise (Ofir, Laveneziana, Webb, & O'Donnell, 2007). In cardiovascular function, decreased arterial compliance may lead to increased cardiac afterload and eventually induce left ventricular hypertrophy and cardiovascular mortality risk (Davison *et al.*, 2010). In metabolic activity, obese people required more energy against body resistance during a rising level of exercise, which is another effect of obesity on exercise performance (Dreher & Kabitz, 2012).

1. Physical functioning in obesity

A powerful factor in the prevention and treatment of health conditions is physical function. It is defined as the ability to fulfil activities that require physical actions, from ADLs to more complex activities (Beaudart et al., 2019). The physical function in obesity for this review includes the factors of body function and functional mobility, as detailed below.

1.1 The factors to body functional decline in obesity

Body functions are the physiological functions of the body systems and structures, including limbs, organs and their components (Cowan et al., 2012). The study of body functions and structures associated with bones and joints, neurocognitive function and pain.

Impact on bones and joints

The severely obese patients showed impairment in daily living activities such as bathing, climbing stairs, and walking, involving the impairment of bone and joints. These problems caused very suffering or distressing for them (Al-Agha, Al-Ghamdi, & Halabi, 2016; Wadden et al., 2007) due to an increase in osteoarthritis (OA) and the risk of musculoskeletal pain (Peltonen, Lindroos, & Torgerson, 2003). There is some evidence suggesting that obesity has been associated with the development of osteoarthritis and gout, leading to pain in weight-bearing joints (Carman, Sowers, Hawthorne, & Weissfeld, 1994). Hyperuricemia in obesity may play an important role in the increased risk of gout (Hikita et al., 2007). The skeletal health at the knee and hip joints in obesity could be maintained by the altered walking pattern in all ages of obese individuals. Change in gait pattern led to a decrease in knee torque and a reduction of impact on proximal leg knee joint and hip joint during walking (DeVita & Hortobágyi, 2003). However, this mechanism may just maintain skeletal health in the short term if their body weight still increases (M. Forhan & Gill, 2013).

Pain

Osteoarthritis with knee pain is related to the increase in BMI and eventually develops into gait abnormalities in obesity (Marks, 2007). The great increase in the risk of low back pain has been observed in women with overweight or large waist circumference (Han, Schouten, Lean, & Seidell, 1997). A prolonged load of excess weight may cause low back pain through increased load compression on the intervertebral discs (Eklund & Corlett, 1984). An increase in mechanical load on spine by obesity reduces the efficiency of the shock absorber and increases stress on the spine when bending to lift objects or stepping downstairs (World Health Organization and Regional Office for the Western, 2000).

Neurocognitive function

Previous study reported that obese adults who have BMI more than 35 km/m^2 performed poorly on tasks of executive function, planning and mental flexibility when compared to lean adults (Boeka & Lokken, 2008). It is possible that impaired metabolic processing impacted on brain function on planning and organization at cerebellum

(Wolpert & Miall, 1996). In addition, a decreased oxygen flow to the brain, which is induced by physical inactivity in obesity, could promote impaired spatial abilities needed for the motor plan (M. Forhan & Gill, 2013).

1.2 Functional mobility

Functional mobility is the method which people are able to move around in the environment for activities of daily living and move from place to place (World Health, 2001). The effects of obesity on functional mobility for this review include stability and walking, and the difficulties with activities of daily living.

Stability and walking

Obesity is associated with decrease in stability and postural control during walking. The main factor is the abnormal body fat distribution, especially in the abdominal area. This factor induces their weight moving toward the front of their feet and anterior-posterior instability during static and dynamic balance (Capodaglio, Cimolin, Tacchini, Precilios, & Brunani, 2013). In addition, instability caused by excessive fat accumulation in the abdominal area induces gait parameter compensation, such as the number of steps per minute and distance between steps (Wearing, Hennig, Byrne, Steele, & Hills, 2006). The previous study reported that obese people walked at a slower speed with shorter step lengths and spent more time with their feet contacting the ground through increased double support (amount of time that both feet are on the ground at the same time while walking) and stance time (amount of time that one or both feet are in contact with the ground during walking) (Lai, Leung, Li, & Zhang, 2008). Instability with a loss of balance could lead to high risks of falls and injury (Gill & Narain, 2012).

The difficulties with activities of daily living (ADLs)

Obesity people spended more time on self-care activities, especially time to complete activities of daily living including getting dressed, meal preparation, bathing and moving from place to place when compared to the same age groups in lean people (Mary Forhan, Law, Vrkljan, & Taylor, 2011). In addition, several evidences suggest that Class II and Class III obesity is related to the increased chances of impairment in activities of daily living such as eating, bathing, dressing and getting in or out of bed

(Alley & Chang, 2007; Mary Forhan et al., 2011). Moreover, they could have hygiene defect due to a difficulty to reach areas of their body (Rose & Drake, 2008).

2. Advantage of weight loss

A systematic review of weight loss intervention in obese adults reported that physical activity with a dietary diet was more effective in reducing blood lipids and blood pressure than dietary diets alone (Ma *et al.*, 2017). Weight loss was also associated with the improvement of many organ systems, such as reduced blood pressure, increased lung volume and increased autonomic nervous system balance in obesity (Ma et al., 2017; Poirier & Després, 2001).

2.1 Cardiovascular system

Weight loss of more than 10 kg could reduce hypertension risk by 26% (Huang et al., 1998), while weight loss of 1 kg could be lower blood pressure by 0.3 to 1 mmHg on average. Weight loss was related to a decrease in blood pressure in obese patients with hypertension and obese patients without hypertension (Cutler, 1991; Wassertheil-Smoller et al., 1992). The reasons for blood pressure reduction were explained by the effects on reduction in peripheral vascular resistance, improvement of an insulin resistance, a better in blood lipids, a reduction in sympathetic nervous activity, and decrease in renin-angiotensin aldosterone system (Reisin, 1986; Reisin et al., 1983). A previous study in obese women found that a reduction by 5% of initial body weight significantly decreased angiotensinogen expression by 20% in adipose tissue, angiotensin-converting enzyme activity by 12%, aldosterone by 31%, renin by 43% and angiotensinogen levels by 27% (Engeli et al., 2005).

2.2 Respiratory system

The respiratory abnormalities in obesity are mainly due to the mechanical load of excess adipose tissue on the chest wall and loss of physical fitness. It has been found that the expiratory reserve volume (ERV) was improved in relating to a significant weight loss (Babb et al., 2011; El-Gamal, Khayat, Shikora, & Unterborn, 2005). Another study reported that BMI reduction from 35 to 32 kg/m^2 induces moderate improvement in ERV (Babb et al., 2011). Moreover, functional residual capacity (FRC) and total lung capacity (TLC) could be improved after weight loss (Hakala, Mustajoki, Aittomaki, & Sovijarvi,

1995; Weiner et al., 1998). The study in jejunoileal bypass in obese patients suggested that the difference between the oxygen concentration in the alveoli and arterial system (The A-a O_2 gradient) tended to improve when BMI reduction of more than 20 kg/m^2 (Farebrother, McHardy, & Munro, 1974). A widened A-a O_2 gradient in obesity is caused by the ERV decline. (Parameswaran et al., 2006). The weight on the lower thorax and the abdomen compresses the lungs reduce the chest wall compliance and eventually creates a delay of gas wash-out from the lungs (Caro, Butler, & Dubois, 1960). This delay may cause by air trapping in poor ventilated units of the lung, which consequently caused a decrease in partial pressure of O_2 in arterial blood (PaO_2) and an increase in ventilation-perfusion (V/Q) mismatch lastly (Parameswaran et al., 2006).

2.3 Autonomic nervous system

A study found that people who gain weight about 10% of their initial body weight increased sympathetic activity and decreased parasympathetic activities. On the other hand, a weight loss of 10% of initial weight decreased in sympathetic activity and increased in parasympathetic activities (Arone, Mackintosh, Rosenbaum, Leibel, & Hirsch, 1995). The reduction in sympathetic activity after weight loss may be explained by catecholamine data (Landsberg & Young, 1978). The study demonstrated that mild weight loss effects of bupropion (BUP), a dopamine (DA) and norepinephrine (NE) reuptake inhibitor in humans. Moreover, the inhibition of DA and NE reuptake increased energy expenditure without a compensatory increase in food intake (Billes & Cowley, 2008).

3. Exercise program for weight loss

3.1 Moderate-intensity continuous training (MICT)

Moderate-intensity continuous training describes the continuous perform of exercise at a steady intensity state for a set duration (usually 20-60 min). Moderate-intensity activity is defined as an intensity that elicits a heart rate response of 55-69% of maximum heart rate (HR_{max}) or elevates the rate of oxygen consumption to 40-59% of $\dot{V}O_2max$ (Norton, Norton, & Sadgrove, 2010). Fourteen weeks of daily exercise for 30 minute at heart rate at 40 - 50% of the corresponding heart rate at their $\dot{V}O_2max$. Resulted in a significant decrease in bodyweight and percentage fat mass in the diet

plus exercise group when compared with diet alone (Okura, Nakata, Lee, Ohkawara, & Tanaka, 2005). All aerobic exercise either walking on land, swimming or walking in water for group for 40 minutes and 4 times per week at 70% of HR_{max} were significantly decreased body weigth and fat mass (Gappmaier, Lake, Nelson, & Fisher, 2006).

3.2 Interval training

Interval training is a type of exercise in repeating bouts of relatively intense exercise with short periods of recovery between bouts, such as high intensity interval training (HIIT) and sprint interval training (SIT). Both forms of interval training can increase aerobic capacity and mitochondrial content (MacInnis & Gibala, 2017).

The high intensity interval training (HIIT) protocol is defined as aerobic intervals with targets intensities between 80% and 100% of peak heart rate or aerobic capacity (Keating, Johnson, Mielke, & Coombes, 2017). This protocol typically employs bursts of activity lasting between 60 and 240 s, at the aerobic capacity of the individual (sub-maximal), but extremely strenuous (Keating *et al.*, 2017). Interval training is an aerobic and/or anaerobic workout that consists of three elements: (1) a selected work interval, (2) a target time for that work interval, and (3) a programmed recovery or relief period before the next work interval (Plowman & Smith, 2011). The previous study in men with class II and III obesity performed 10 x 60 seconds cycling intervals at workload of about 90% of $\dot{V}O_2max$ combined with 60 seconds recovery at 50 Watt for 14 days. The results reported that body weight and percentage of fat mass were significantly decreased when compared between before and after the exercise program (Lanzi et al., 2015).

Moderate intensity interval training (MIIT) is aerobic interval exercise with moderate intensity between 45 to 65% maximal oxygen consumption (VO_2max) (Alkahtani et al., 2013) or 65 to 80% maximal heart rate (HR_{max}) (Reljic, Frenk, Herrmann, Neurath, & Zopf, 2021) . Peak oxygen uptake (VO_2peak) and maximal workload were increased after exercise for 4 weeks (Alkahtani et al., 2013). Moreover, previous studies reported that body weight, BMI, percentage of body fat, fat mass, a waist circumference were decreased after MIIT exercise for 12 weeks (Racil et al., 2013; Reljic et al., 2021).

3.3 Comparison of MICT, HIIT and MIIT

According to systematic review and meta-analysis, while MICT and HIIT were similarly efficient for decreasing fat mass and waist circumference, HIIT training required about 40% less time engagement (Keating et al., 2017; Wewege, van den Berg, Ward, & Keech, 2017). Likewise, Kong *et al*. in 2016 also reported that short-term HIIT in overweight and obese young women was more time-efficient and better in improving cardiorespiratory fitness and fasting blood glucose when compared with MICT (Kong et al., 2016). HIIT was also a time-efficient strategy to improve both whole-body fat mass and abdominal (visceral) fat mass (Maillard, Pereira, & Boisseau, 2018). Furthermore, the comparison of MICT, HITT and HITT plus resistance training (RT) in postmenopausal women with overweight and obesity at 3 days per week for 12 weeks demonstrated that body weight and total fat mass were decreased in all three exercise groups. but significant reduction of abdominal or visceral fat mass was observed only in HIIT and HIIT plus RT groups (Dupuit et al., 2019). HIIT is a time-efficient strategy to decrease visceral fat and waist circumference in obese person and it is an effective exercise program for improving cardiorespiratory fitness while body fat reduction is still a therapeutic target (Keating *et al*., 2017).

Nevertheless, Alkahtani 2013 proposed that achieving training at maximal levels was found to be less tolerable compared with moderate-intensity training in obesity (Alkahtani et al., 2013). The previous study found that receiving 10% higher than the self-selected walking speed decreased the pleasure of exercise in obese sedentary women (Ekkekakis & Lind, 2006). Furthermore, the supramaximal exercise operation in the obese population may increase the risk of adverse effects, for example, an acute cardiovascular event during increasing exercise intensity, especially in people who are unaccustomed to exercise (Whyte, Gill, & Cathcart, 2010). To solve this problem, MIIT was therefore introduced. The cross-over study of 4-week MIIT and HIIT training suggests that both HIIT and MIIT could similarly increase fat oxidation. But blood lactate and rating of perceived exertion decreased after HIIT at a greater extent than MIIT

(Alkahtani et al., 2013). Another study found that body mass, body mass index and percentage fat mass were significantly decreased by both exercises with increases in peak oxygen consumption and maximal aerobic velocity. Furthermore, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDLC), adiponectin levels and insulin resistance index were improved by both HIIT and MIIT when compared between pre- and post-protocol (Racil et al., 2013). Although exercise is done at moderate intensity, it was sufficiently effective in improving body composition, blood lipid and cardiorespiratory in obese (Reljic et al., 2021). MIIT thus becomes an alternative option when any specific health risks or difficulties appear in applying HIIT or can be used as a pre-HIIT session within long term training (Jimenez-Pavon & Lavie, 2017).

4. Recovery state in obesity

4.1 Oxygen uptake and ventilator response in obesity ($\dot{V}O_2$)

Oxygen uptake or consumption ($\dot{V}O_2$) is a measure of a person's ability to intake oxygen via the respiratory system, deliver it to the working tissues via the cardiovascular system, and the ability of working tissues to use oxygen (Haff & Triplett, 2015). At rest, obesity is related to increased oxygen consumption due to the high basal metabolic rate. Similarly, oxygen consumption is more enhanced during exercise at the same work rate in obesity as compared to lean body mass (Babb, Korzick, Meador, Hodgson, & Buskirk, 1991; Salvadori, Fanari, Mazza, Agosti, & Longhini, 1992; Salvadori et al., 1993). Even though obesity induced the widened A-a O_2 gradient and reduced PaO_2 at rest from atelectasis in some parts of the lung, the improved A-a O_2 gradient and normal PaO_2 are demonstrated during exercise by increased tidal volume and respiratory rate. However, their body still needs more oxygen to avoid expiratory flow limitation. Obesity people must increase their minute ventilation greater than normal weight people. This phenomenon promotes a higher respiratory rate as well as a reduced tidal volume (Dempsey, Reddan, Rankin, & Balke, 1966; J. Li et al., 2001). When compared to lower body distribution, a study suggested that an upper body distribution (waist-hip circumference ratio more than 0.80) had a lower anaerobic threshold, higher maximal

oxygen consumption, higher respiratory rate and minute ventilation (J. Li et al., 2001). These results implied that the upper body fat irritated the increase in tidal volume and minute ventilation. However, the increase in minute ventilation was insufficient to match metabolic demands. The higher oxygen consumption in the obese group with upper body fat may be explained by a higher work of breathing (Littleton, 2012). A study informed the elevation of oxygen uptake ($\dot{V}O_2$) and heart rate for two minutes of the recovery stage after 60% of maximal heart rate of cycling study in obesity as compared to the non-obese group. Interestingly, this study also reported that obese people had a longer recovery time of $\dot{V}O_2$. Because of the sustainable exercise needed more oxygen to completely oxidize fat during the recovery stage of exercise in obesity. Then the decreasing respiratory exchange ratio (RER) was observed during recovery (Cavuoto & Maikala, 2016). The oxygen requirement after exercise is occurred for anaerobic alactic (ATP-CP) resynthesis and lactate removal (Plowman & Smith, 2011). The high fatty acid (FA) oxidation in skeletal muscle during the recovery stage of exercise seems to be managed at several steps, including an enhanced FA uptake into the mitochondria through the carnitine palmitoyl transferase I (CPT1) reaction. Furthermore, adenosine monophosphate (AMP)-activated protein kinase mediates pyruvate dehydrogenase (PDH) inhibition of glucose oxidation that induces high FA oxidation. The enhanced level of FA within the myocytes could be critical signaling molecules, for example, peroxisome proliferator-activated receptor (PPAR) signaling in the order of the exercise-induced FA oxidation program in skeletal muscle during exercise recovery (Lundsgaard, Fritzen, & Kiens, 2020). Therefore, these processes are attended by excess post-exercise oxygen consumption (EPOC) which the magnitude and time depend on exercise duration and intensity (Borsheim & Bahr, 2003).

4.2 Excess post-exercise oxygen consumption (EPOC)

At the onset of exercise, the body needs a higher amount of energy. The body has three different energy systems to supply working muscles with ATP (adenosine triphosphate). All three energy systems are involved in this response, including

anaerobic alactic (ATP-CP), anaerobic lactic (Glycolytic), and aerobic energy system. Their dependent contributions are related to activity's intensity and duration.

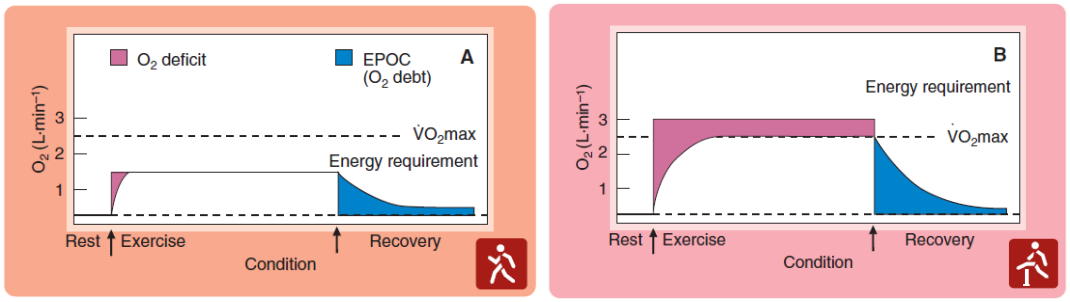


Figure 10 Oxygen deficit and excess post exercise oxygen consumption (EPOC) during submaximal exercise and supramaximal exercise.

Source: Plowman, S. A., & Smith, D. L. (2011). Exercise Physiology for Health, Fitness, and Performance (3): Wolters Kluwer Health/Lippincott Williams & Wilkins.

According to Figure 10, the area under the smoothed curve during both exercise and recovery represents oxygen used. However, oxygen consumption is an initial delay. It represents the supplied and consumed oxygen below the oxygen requirement for providing energy. This difference between the oxygen required and the oxygen supplied and consumed during exercise is called the oxygen deficit. EPOC was also defined by Plowman & Smith in 2011 as oxygen consumption during recovery above average resting values. It is a result of ATP-CP resynthesis, lactate removal, restoration of O_2 stores, enhanced physiological functions such as cardiovascular and respiratory function, hormonal levels and body temperature. During submaximal exercise, oxygen deficit and EPOC are small as shown in Figure 10 (at the left). The energy supplies in this condition rely on stored ATP-CP in muscle, anaerobic glycolysis, and oxygen in capillary blood and bound to myoglobin. During supramaximal exercise, both the O_2 deficit and the EPOC are large. The graph in Figure 10 (at the right) shows plateaus or levels off of O_2 consumption representing maximal oxygen consumption ($\dot{V}O_2max$) and more energy is still needed if exercise is to continue at supramaximal exercise (Plowman

& Smith, 2011) . Although all three-energy system are utilized at this intensity, the reliance on anaerobic glycolysis is more remarkable.

Emphasis on EPOC, the bowl-shaped curves obtained in the recovery state displays two components, fast and slow components, as shown in Figure 11 (Knuttgen, 1970) . The first curve, lasting 2-3 minutes and declining quickly, is called the fast component. It is for the production of phosphocreatine (Di Prampero et al., 1973; Plowman & Smith, 2011). And then, the graph slightly decreased for 3-60 minutes in the slow component. In this period, the lactic acid is removed, and oxymyoglobin is restored with muscle glycogen (Di Prampero *et al*., 1973; Plowman & Smith, 2011). This recovery oxygen consumption represents additional calories spent like during exercise for an hour or two after exercise cessation (Speakman & Selman, 2003).

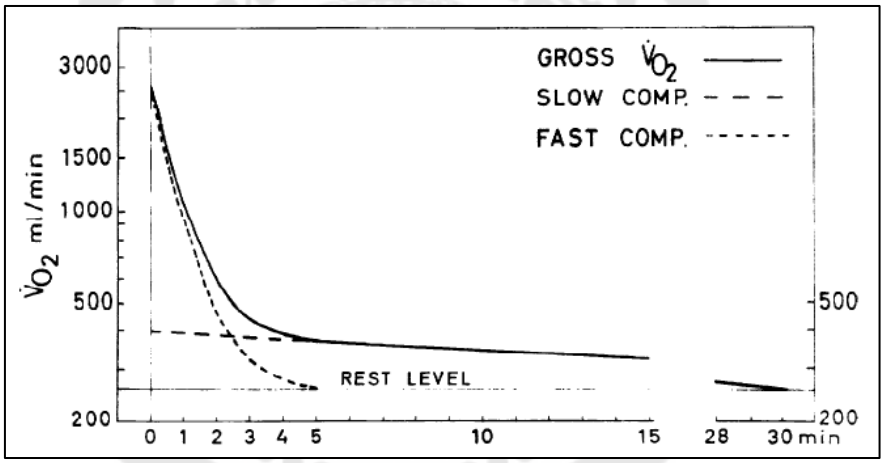


Figure 11 Oxygen uptake during recovery after 15 minutes work at 180 watts.

Source: Knuttgen, H. G. (1970). Oxygen debt after submaximal physical exercise. J Appl Physiol, 29(5), 651-657.

In addition, the rate of change in post exercise $\dot{V}O_2$ ($\dot{V}O_2$ % recovery) can be observed during EPOC. It shows the post-exercise time for $\dot{V}O_2$ to return percentage of the base-line resting value after exercise cessation (Hagberg, Hickson, Ehsani, & Holloszy, 1980). This value represents speed of metabolic recovery. $\dot{V}O_2$ % recovery is calculated as the percentage difference between the end-exercise value (assigned a

value of 0%) and resting baseline value (100% recovery) for each measurement time during recovery periods (Short & Sedlock, 1997).

4.3 Heart rate variability in recovery stage (HRV)

The autonomic nervous system plays an important role in the cardiovascular responses to exercise such as the increased metabolic requirements of the active skeletal muscle. The effect of the autonomic nervous system during exercise is the decrease in parasympathetic and an increase in sympathetic activity. Moreover, the effects include increased the autonomic nervous system plays an important role in the cardiovascular responses to exercise such as an increase in metabolic requirements of the active skeletal muscle. The effect of the autonomic nervous system during exercise includes decrease in parasympathetic and an increase in sympathetic activity.The effect includes increased heart rate, increased stroke volume as well as cardiac output and facilitated blood flow to the active skeletal muscles (Mitchell, 1990).

Heart rate variability (HRV) or the beat-to-beat alteration in heart rate provides a noninvasive measurement of autonomic nervous system. Sustained passive stretches of triceps surae muscle in a human calf influenced a temporary increase in heart rate of about 5-6 beats/min during the first 20 cardiac cycles of observation (Gladwell & Coote, 2002) . This activity caused a reduction in HRV and affected the index of cardiac parasympathetic activity (standard deviation of successive differences in R–R interval). In addition, the heart rate response to passive calf stretch was essentially eliminated by the administration of a muscarinic cholinergic blocker. Thereby muscle mechanoreceptor activation can produce an increase in heart rate by inhibiting cardiac parasympathetic activity (Gladwell et al., 2005).

During dynamic exercise, muscle metaboreflex activation influences the autonomic control of the heart. The previous study reported that enlarged muscle metaboreflex activation during leg cycling with partial flow restriction increased heart rate. This change was reduced with β1-adrengeric blockade by about 50% . Moreover, the elevation in heart rate was found during postexercise ischemia following leg cycling exercise (Hartwich, Dear, Waterfall, & Fisher, 2011).

As mentioned above, most HRV measures are considerably reduced during exercise. HRV has been used as a tool to investigate post-exercise autonomic (mainly parasympathetic) activity (Goldberger et al., 2006). HR and HRV show a time-dependent recovery and finally return to pre-exercise levels after exercise cessation (Stanley, Peake, & Buchheit, 2013). Change in HRV during recovery is depended on exercise intensity and duration. For exercise intensity, the studies concluded that higher exercise intensity is related to slower recovery of cardiac parasympathetic neuvous activty-HRV measures (P. Kaikkonen, Rusko, & Martinmaki, 2008; Seiler, Haugen, & Kuffel, 2007), the general acceptance of beat-to-beat measures as indicators of cardiac parasympathetic modulation (Parati, Mancia, Rienzo, & Castiglioni, 2006). According to Figure 12, a greater exercise intensity results in a slower HR and HRV recovery (Dupuy et al., 2012). From a mechanism viewpoint, the effect of intensity on HRV recovery might related to the amount of non-oxidative energy influence and the stimulation of the muscle metaboreflex (Buchheit, Laursen, & Ahmaidi, 2007).

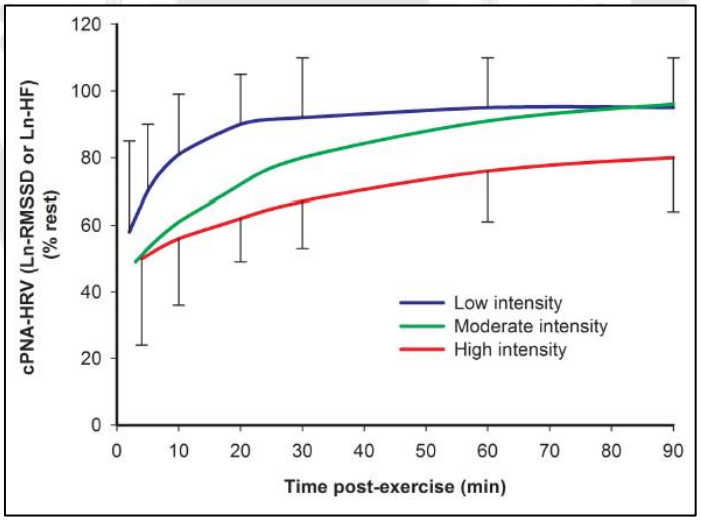


Figure 12 Time course of cPNA-HRV recovery following different intensities of preceding exercise.

Source: Michael, S., Graham, K. S., & Davis, G. M. O. (2017). Cardiac Autonomic Responses during Exercise and Post-exercise Recovery Using Heart Rate Variability and Systolic Time Intervals-A Review. Front Physiol, 8, 301.

For the effect of exercise duration on HRV, previous studies reported that a 100% increase in exercise duration (such as 60 vs 120 minutes for exercise with the same intensity) did not change HRV in recovery after exercise (Michael, Graham, & Davis, 2017; Seiler et al., 2007). Another study found that HRV recovery was slowed following a 300-400% increase in exercise duration (about 20 vs 90 minutes) (Piia Kaikkonen, Hynynen, Mann, Rusko, & Nummela, 2009). The concept of theoretical viewpoint suggested that exercise duration must be prolonged at some critical length, then an effect on HRV recovery might be observed (Michael et al., 2017).

4.4 Heart rate recovery (HRR)

Heart rate recovery (HRR) is a strong predictor of mortality measured by decreasing heart rate after cessation of exercise. HRR has been investigated and recognized as a predictor of coronary artery disease (CAD) (Lipinski, Vetrovec, & Froelicher, 2004; Morshedi-Meibodi, Larson, Levy, O'Donnell, & Vasan, 2002). Previous studies concluded that HRR at 2-minute represented the maximal predictive value for mortality (Gorelik, Hadley, Myers, & Froelicher, 2006; Lipinski et al., 2004; Shishehbor, Hoogwerf, & Lauer, 2004). HRR after acute exercise seems to be controlled by vagal reactivation and sympathetic withdrawal (Imai et al., 1994; Pierpont, Stolpman, & Gornick, 2000). In addition, the rate of heart rate return to baseline after exercise was dependent on physical fitness and health status (Shetler et al., 2001). HRR level is calculated from peak exercise to minute 1 or 2 of recovery (Nishime, Cole, Blackstone, Pashkow, & Lauer, 2000; Rosenwinkel, Bloomfield, Allison Arwady, & Goldsmith, 2001). A result of the decreased vagal activity is associated with reduced rate of heart rate recovery. In obese person, the impaired HRR was associated with higher BMI causing perhaps by vagus nerve dysfunction (Barbosa Lins, Valente, Sobral Filho, & Barbosa e Silva, 2015). HRR could then be a powerful predictor of overall mortality, changes in heart rate during exercise and myocardial perfusion defects (Cole, Blackstone, Pashkow, Snader, & Lauer, 1999). Abnormal HRR was classiflied when the declined heart rate is lower than 12 beats per minute (Cole et al., 1999).

Furthermore, the action of HRR has been investigated by many research groups which focus on different aspects. According to consideration of its kinetics, HRR can be correctly fitted by an exponential decay function (Pierpont et al., 2000). Thus, HRR can be divided into two phases, fast and slow recovery. The fast phase includes the 30 seconds (HRR30s) (Pecanha et al., 2017) and the first minute of exercise cassation (HRR60s) which there is a sudden and rapid decrease in HR (Coote, 2010). The major contribution of the fast HHR phase was occured by cardiac vagal reactivation. The slow phase occurred after the first minute of recovery until HR returns to resting values. This mechanism underlying included cardiac vagal reactivation and sympathetic withdrawal (Pecanha, Silva-Junior, & Forjaz, 2014) as Figure 13.

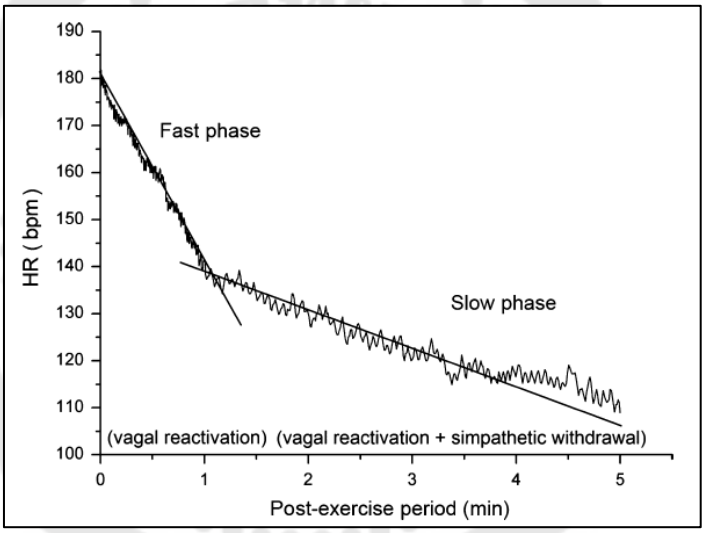


Figure 13 Phases of heart rate recovery (HRR).

Source: Pecanha, T., Silva-Junior, N. D., & Forjaz, C. L. (2014). Heart rate recovery: autonomic determinants, methods of assessment and association with mortality and cardiovascular diseases. Clin Physiol Funct Imaging, 34(5), 327-339.

The fast phase of HRR is calculated by taking the difference between peak HR at the end of exercise and the HR observed after 30 seconds (HRR30 s) 1 minute or (HRR60 s) of recovery. The studies demonstrated that there was the parasympathetic effect in the first 30 seconds after a submaximal exercise with no sympathetic

withdrawal during this time frame (Imai et al., 1994) and observed a parasympathetic effect for HRR60s after maximal exercise that delayed by parasympathetic blockade for heart rate recovery (Kannankeril, Le, Kadish, & Goldberger, 2004). The slow phase presented a slower decline of HRR due to autonomic role (Imai et al., 1994) and this phase was accompanied by a slow decrease in plasma norepinephrine (NE) indicating sympathetic withdrawal (Perini et al., 1989). There is the theoretical model (Figure 14) which hypothesizes that the fast phase of HRR is mostly regulated by the central command and mechanoreflex deactivations. A decrease in stimulation by the mechanical distortion of the receptive field via stretch or pressure produced a rapid recovery in HR without intensity-dependence of exercise. Whereas the change in the slow phase was depended on exercise intensity. The slow phase may be mainly influenced by metaboreflex, this reflex was stimulated by the chemical products of muscle contraction, and thermoregulatory deactivations resulting in a decrease in cardiac sympathetic activity (Pecanha et al., 2014).

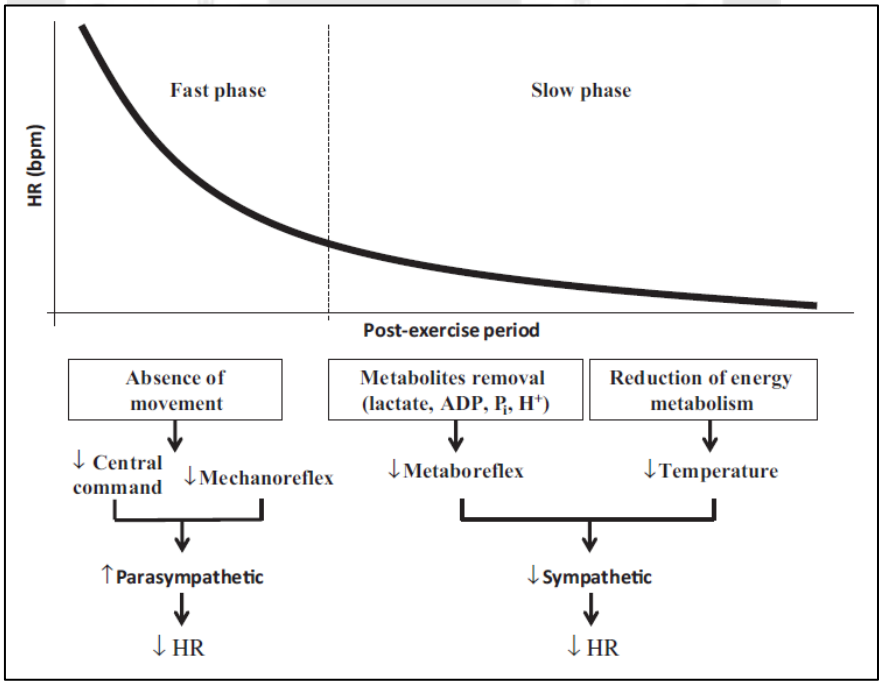


Figure 14 Theoretical model to explain the role of the cardiovascular control mechanisms in heart rate recovery.

Source: Pecanha, T., Silva-Junior, N. D., & Forjaz, C. L. (2014). Heart rate recovery: autonomic determinants, methods of assessment and association with mortality and cardiovascular diseases. Clin Physiol Funct Imaging, 34(5), 327-339.

The study in recovery periods found that HRR in obese persons tended to be slow in the 4th minute during recovery periods when compared to control group. HRR was significantly delayed in overweight with obstructive sleep apnea throughout the recovery periods when compared to overweight without obstructive sleep apnea and control group (Hargens et al., 2008).

Slow deep breathing exercise

1. Definition and methodology

Slow breathing is essentially performed by controlling the breathing frequency from 4 to 10 breaths/minute to restore and enhance healthy (Russo et al., 2017). The previous study reported that autonomic and respiratory characteristics in a relaxed state from slow breathing are quite similar to patterned breathing during Zen meditation (Cysarz & Bussing, 2005), yoga or rosary prayers (Bernardi, Sleight, et al., 2001).

According to a review from Russo *et al.* in 2017, slow deep breathing techniques aim to improve health and wellbeing by alternating of respiratory, cardiovascular, and autonomic nervous systems. Slow deep breathing could be performed with continuous positive airway pressure machines or other breathing devices, respiratory load, other breathing techniques or meditation, yoga, tai chi and exercise (Russo et al., 2017). In the viewpoint of this dissertation on this topic, we just included the reviews of slow deep breathing with exercise except for yoga and tai chi.

The clinical applications of breathing exercises are very complicated because the suggestion for breathing modification must require subjective factors from the subject, such as self-awareness of breathing movements and the perception of breathing effort (Gilbert, 2003). Therefore practicing the skill, building motivation, individualized instruction and personal coaching for training must be needed (Kow et al., 2018). Previous studies suggested that device guide breathing could improve the respiratory

and cardiovascular systems (Gavish, 2010; Rosenthal, Alter, Peleg, & Gavish, 2001). The device is called Respi-Low; the treatment consisted of 15 minutes daily with a musical guide. It consists of a belt of respiration-movement sensors fixed on the upper abdomen or chest attached to an onscreen control unit and headphones. The device can guide breathing pattern with prolong sound patterns for inducing prolonged expiration, and then breathing pattern modification occurs as the user voluntarily follows the sound pattern (Rosenthal et al., 2001). In addition, there is a study about slow deep breathing by similar music CD guide. The subjects breathe deeply while hearing birds chirping sound for 4 seconds and breathe out slowly when hearing the stream flowing sound for 8 seconds, 5 breaths per minute for 15 minutes (Kow et al., 2018). Moreover, a metronome is used for slow breathing training with a duty cycle (inspiratory time: total respiratory time) of 0.4 with a total respiratory time of 10 seconds for 6 breaths per minute. It was used as a breathing guide in laboratory until subjects performed reliably without the metronome as their home program (Jones, Sangthong, Pachirat, & Jones, 2015) and some studies used metronome as a tool for investigation of breathing rate on autonomic activity (De Souza et al., 2018; Driscoll & Dicicco, 2000) and investigation of breathing training on stress level (Naik, Gaur, & Pal, 2018).

However, devices which combine with sensors and breathing monitors and automatically adjust the music rhythm to guide the user to the target breathing rate may be too expensive (Kow et al., 2018) and not worthy for most people. In addition, our study was applied at exercise cessation and investigate pulmonary function in obesity during exercise and recovery state which elastic belt sensor may affect our outcomes. For the music CD guide, this method may be suitable for a quiet or relaxed environment, and the concentration of differential sound between inspiration and expiration guidance must be needed. Therefore, we choose the metronome as a breathing guide with an inspiratory time of 4 seconds and expiratory time of 6 seconds for a frequency of 6 breaths per minute after exercise cessation in obesity.

2. Advantage of slow deep breathing exercise

2.1 The acute effect on pulmonary function

Pulmonary function test after immediate slow deep breathing was investigated in healthy young volunteers. Shravya *et al*. in 2013 investigated the effect of slow deep breathing on pulmonary function tests in healthy volunteers. After slow deep breathing of 6 breaths/min for 10 minutes, a significant improvement in pulmonary function compared to regular spontaneous breathing was found (Shravya et al., 2013). In another study, deep breathing exercise in short duration (for 2, 5 and 10 minutes) at the rate of 6 breaths per minute significantly increased in forced vital capacity (FVC) after 2 minutes and significantly increased in vital capacity (VC) after 2 and 5 minutes of deep breating exercise (Sivakumar et al., 2011). It could be implied that deep breathing exercise could improve pulmonary function even after a few minutes.

The mechanism underlined by slow deep breaths is the raise of intrapulmonary pressure during the inspiratory phase, and the then the alveoli in the lung apices are filled with air leading to the breathing benefits of gaseous exchange (Subbalakshmi, Saxena, & D'souza, 2005). Stretching of lung fibroblasts leads to the generation of the slower wave brain activity and the parasympathetic autonomic shifts during slow deep breathing exercises (Shankarappa, Prashanth, Nachal, & Varunmalhotra, 2012). The increase in parasympathetic activity was then found when low breathing frequency.

2.2 Heart rate variability and baroreflex indices

The effect of patterned breathing on heart rate variability in obese people has been reported by Paprika *et al*. in 2014. Participants performed 3 breathing sessions at 6 breaths/minute for 3 minutes of each frequency with 5:5, 3:7, and 7:3 inspiration to expiration ratios. Results reported a reduction in immediate responses of arterial pressure and heart rate. The time domain parameters of heart rate variability (HRV) were significantly increased with patterned breathing. The major determinant of autonomic responses induced by slow patterned breathing was the breathing rate (Paprika, Gingl, Rudas, & Zollei, 2014). Other studies have represented that slow deep breathing exercises increased parasympathetic tone, as indicated by increased root mean square of successive differences (RMSSD: time-domain method of HRV) and high-frequency

(HF: frequency-domain method of HRV) (Sacha, 2014; Tharion, Samuel, Rajalakshmi, Gnanasenthil, & Subramanian, 2012). Deep breathing exercises were also used for individual calm down, presumably by activating the parasympathetic nervous system (Levin & Swoap, 2019).

2.3 Haemodynamic fluctuations, heart rate and blood pressure

The effect of respiration on the cardiovascular system may be discussed in terms of hemodynamics. During normal inspiration, the pressure gradient between the right heart and the systemic circulation was increased because of the decrease in intrathoracic pressure, which resulted in an increase in venous return then filling of the right atrium and right ventricular stroke volume (Shekerdemian & Bohn, 1999; Wise, Robotham, & Summer, 1981). At the same time, inspiration increased pulmonary resistance. pulmonary venous return was decreased resulting in a reduction in filling of the left heart (Shekerdemian & Bohn, 1999). These effects increase the storage of blood in the right heart and pulmonary circulation leads to an increase in cardiac output during the expiration (Russo et al., 2017). The study of deep breathing at 20, 15, 10 and 6 breaths per minute reported that the rate of respiration affected the harmonic modification of arterial pulse (Hsieh, Mao, Young, Yeh, & Yeh, 2003). Slow breathing at 6 breaths per minute has been suggested to result in increased venous return (Dick, Mims, Hsieh, Morris, & Wehrwein, 2014). In view of the effect on venous return, slow abdominal or diaphragmatic breathing patterns can enhance the collapsibility of the inferior vena cava during inspiration by greater diaphragmatic excursion, which positively affected venous return (Byeon et al., 2012). The study by Kimura et al. in 2011 also reported that a reduction in inferior vena cava diameter and lower extremity venous return were found during diaphragmatic breathing (Kimura et al., 2011).

Some studies in healthy subjects concluded that controlled slow breathing at 6 breaths per minute was related to an increase in modification of both blood pressure and heart rate when compared to breathing at a typical rate (Bernardi, Gabutti, Porta, & Spicuzza, 2001; Chang, Liu, & Shen, 2013; Radaelli et al., 2004). The hypothesis is that results reflect the buffering of respiratory-related hemodynamic modifications because

of the synchronization of the pulsating blood flow toward the rhythm of the heartbeat (Chang et al., 2013). Furthermore, several studies reported that mean blood pressure significantly decreased during slow respiration which would support this hypothesis (Chang et al., 2013; Dick, Mims, et al., 2014; Joseph et al., 2005). The studies of slow breathing at 6 breaths per minute have also reported that this breathing frequency affects heartbeats within the inspiratory phase (Lopes, Beda, Granja-Filho, Jandre, & Giannella-Neto, 2011; Mortola, Marghescu, & Siegrist-Johnstone, 2016). Therefore, the relationships between blood pressure, heart rate and respiration are also known as cardiorespiratory coupling (Dick, Hsieh, et al., 2014).

3. Slow deep breath exercise in obesity

Many studies on the effects of breathing exercises in overweight and obesity were reported. A study by Viskoper et al. 2003 investigated the effect of slow breathing protocol of 15 minutes per day for 8 weeks in overweight elderly with hypertension, who had average BMI of 28 km/m^2 (Viskoper et al., 2003). Anderson et. al. 2010 also studied adult hypertension who had a mean of BMI 27.9 km/m^2 with the slow breathing program of 6 breaths per minute of breathing, 15 minutes per day for 4 weeks (Anderson, McNeely, & Windham, 2010). Results concluded that slow breathing exercises might be a beneficial non-pharmacologic assistant in treating hypertension (Viskoper et al., 2003) by decreasing the resting blood pressure for short-term period (Anderson et al., 2010). Moreover, another study that Thai obese adults with hypertension who had a mean BMI of 27.3 km/m^2 performed slow breathing at 6 deep breaths per minute, 30 minutes, twice a day, every day for 8 weeks. After the training session, a smaller pressor response to the handgrip test with a more rapid recovery of systolic blood pressure and HR was detected as compared to before the training session (Jones et al., 2015). This finding confirmed that the acute effects of slow breathing was to increase baroreflex sensitivity (Joseph et al., 2005). These results are consistent with the finding that slow breathing training modified central mechanisms regulating cardiovascular function. (Jones et al., 2015). Obese subjects, with a mean BMI of 43 km/m^2 were instructed to take deeper breaths at a comfortable rhythm 10 breaths per session for 3 sessions with 3 minutes

pause between sessions. Results reported that tidal volume, alveolar ventilation and peak inspiratory volume were significantly higher than breathing programs with resistance devices. The investigator proposed that atelectasis in severe obesity may be prevented by the opening up of closed airways when a high peak inspiratory volume and increased expiratory airflow (Olsen et al., 1999). All evidences suggested that acute and chronic effects of slow deep breathing helped improving pulmonary, cardiovascular and autonomic nervous systems in overweight and obese.

4. Combination of exercise with slow deep breathing in obesity

A few studies on the effects of breathing training with exercise have been reported. A study of deep breathing on pulmonary function in mildly obese men, with a mean BMI of 29.9 km/m^2 with performed 30 minutes walking training by treadmill for two months with deep breathing (DB) or breathing with tube expiration resistance (BT). In group with DB, all subjects breathed through the nose using of the diaphragm; inspiration and expiration time lasted both about 4-8 seconds. Deep breathing program was done during walking for 10 - 15 minutes every day for 2 months, same as BT group. Results showed that RER was lower in DB than in BT after training, in which a decrease in respiratory frequency and increases in tidal volume and FVC was also found only in DB group. The authors suggested that the decrease in RER could be due to an increased contribution of fat oxidation (Kruk, Pekkarinen, & Litmanen, 2006). Deep breathing exercise activating fat oxidation could then enhance physical fitness during exercise (Ramos et al., 2008). The metabolic improvement in fat oxidation capacity could also increase the removal of plasma free fatty acids resulting in enhanced insulin sensitivity and mitochondrial ATP synthesis rate in obesity (Daniele et al., 2014).

CHAPTER 3
RESEARCH METHODOLOGY

Study design

This study was a crossover randomized controlled trial. The study was conducted by evaluating heart rate recovery and cardio-autonomic function between slow deep breathing and control trials after moderate-intensity intermittent training in overweight and obese young adults.

Participants

Twenty-nine overweight and obese young adults who live in Bangkok and their vicinity participated in this study. They completed confidential health screening questionnaires and physical assessments by physical therapist.

Inclusion criteria

All participants who met the inclusion criteria were:

1. Male and female participants
2. Ages between 18 – 25 years
3. Body mass index (BMI) between 23 and 32.5 kg/m^2 (WHO Expert Consultation, 2004) and waist circumference ≥ 85 cm in males and ≥ 80 cm in females (Obesity in Asia et al., 2007) or a percentage of fat mass > 22 % in males and > 32 % in females (Pescatello, 2014)
4. People with low physical activity levels, classified by short-form International Physical Activity Questionnaire (IPAQ)
5. People who had good communication and cooperation

Exclusion criteria

Exclusion criteria included:

1. Participant with severe orthopedic conditions or other major health problems that may reduce safe cooperation in exercise.

2. Participant who had clinical diagnoses of chronic diseases such as hypertension, diabetes mellitus, cancer, endocrine disorders, neurological disorders, heart disease, respiratory disease, or kidney failure.

3. Participants who had been using drugs or therapies for obesity.

4. Smoking persons.

5. Participants who planned to undergo surgery during the research period.

6. Participants who were reluctant to sign the consent form.

Discontinuation criteria

Discontinuation criteria during research periods were:

1. Participants who could not attend the completion of research periods.

2. Participants who could not follow up on the requirement along the study.

Sample size

Our sample size calculation focused on the effect of deep breathing on heart rate deviation. We chose the primary outcome, " The root mean square of successive differences between normal heartbeats (RMSSD)", from previous research done by Charles J Levin and Steven J Swoap in 2019 to determine the proper sample size (Levin & Swoap, 2019). The sample size was calculated by G*power program (version 3.1.9.4). We used data from control and treatment groups, yielding 48.9 milliseconds (SD, 35.2) and 68.7 milliseconds (SD, 40), respectively. Power and alpha were 80% and 5%, respectively. The sample size was estimated at 24 participants, as shown in Figure 15.

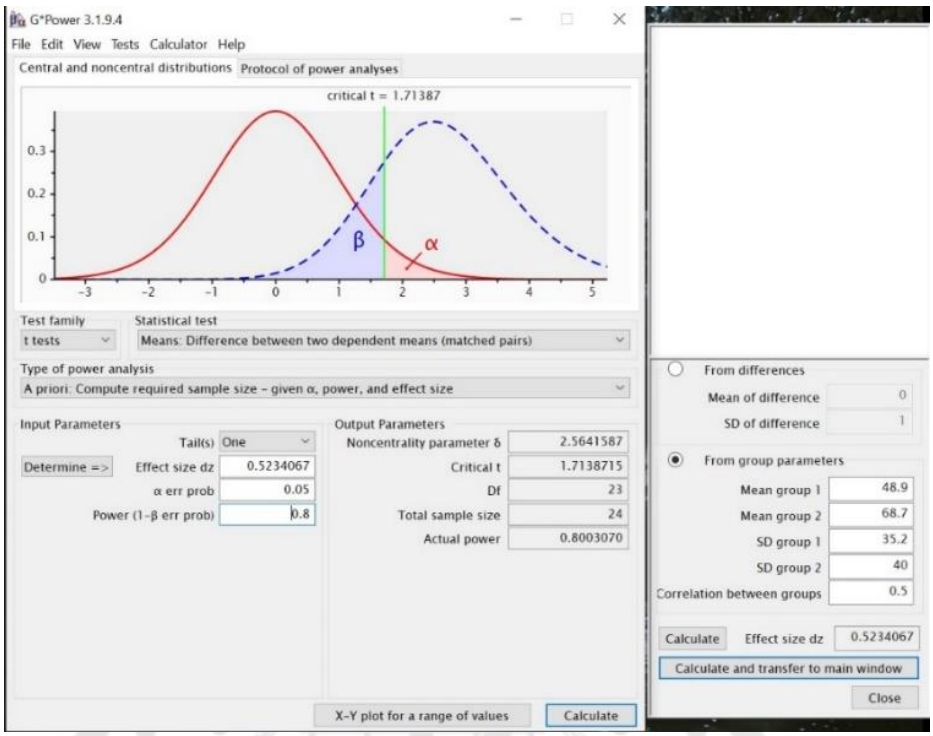


Figure 15 Sample size calculation by G*power program.

According to Figure 15, the sample size was at least 24 participants. To allow a margin of error in dropout cases, we expanded the sample size to 20%. Therefore, the sample size was increased to 29 participants in the present study, as calculated below.

Total sample size	=	24 * 0.20
	=	4.8
	=	4.8 + 24
	=	28.8 ~ 29

Ethical approval

The risks and benefits of all procedures in the study were explained to the participants. Informed consent was signed by all participants before experiment. The method of this study was reviewed and approved by ethic committee of Srinakharinwirot University.

Data collection and experiment protocol

1. Materials and outcome measurements

Oxygen consumption and Percentage of oxygen consumption recovery

Oxygen consumption ($\dot{V}O_2$) was measured throughout the experiment using a gas analyser (Vmax® Encore system 29n, Viasys Health Care Inc.) in the first visit. Resting condition was recorded 5 minutes before the start of exercise while sitting on the chair as $\dot{V}O_2$ baseline values (Short & Sedlock, 1997). The parameter was recorded during 45 minutes of moderate-intensity interval protocol.

The percentage of $\dot{V}O_2$ recovery ($\dot{V}O_2$% recovery) was the rate of change in $\dot{V}O_2$ post-exercise of the way to the baseline resting value after exercise cessation (Hagberg et al., 1980). It was calculated as the percent difference between exercise cessation (assigned a value of 0%) and $\dot{V}O_2$ baseline value (100% recovery) during recovery (Short & Sedlock, 1997).

Maximal oxygen consumption ($\dot{V}O_2$max) measurements

$\dot{V}O_2$max was measured by using Astrand-rhyming cycle ergometer test; this trial is the submaximal exercise test (Noonan & Dean, 2000). Before the cycle ergometer test, the participants were instructed to refrain from vigorous exercise for at least 2 hours or caffeine for 24 hours. All subjects performed cycling ergometry with a gradually increased workload until they reached the target heart rate range (125 – 170 beats/ minute) within 6 minutes (Pescatello, 2014). After that, Astrand-ryhming nomogram and calculation were used to determine $\dot{V}O_2$max. Then, the participants rested for 1 hour before testing MIIT protocol.

Heart rate variability (HRV)

Heart rate variability was measured by monitoring continuously the electrocardiogram (ECG) of standard limb leads all along the test using the PowerLab data acquisition system (ADinstruments Ltd) and Labchart Pro analyzing software (ADinstruments Ltd). Both time (the root mean square of successive differences (RMSSD) and standard deviation of the normal-to-normal intervals (SDNN)) and frequency (low frequency (LF) and high frequency (HF)) domains were analyzed.

Heart rate recovery (HRR)

Heart rate was measured by ECG heart rate sensor. Heart rate (HR) was monitored and recorded during the test. HRR was calculated as the decrease of HR per minute between the peak exercise period and time post-cool down ($HR_{at\ peak} - HR_{time\ post\text{-}cool\ down}$) at 30, 60, 120, and 300 seconds as HRR30, HRR60, HRR120, and HRR300, respectively (Gorelik et al., 2006).

Percentage of heart rate decline (% HR decline)

The HR after exercise was calculated as the percentage of heart rate decline at the 1st to 10th minutes post-exercise. The percentage of heart rate decline associated with peak HR was calculated as [(peak HR – min post-exercise HR)/ (peak HR)] ×100 (Dimkpa, Godswill, Okonudo, & Ikwuka, 2023).

Percentage of heart rate recovery (% HR recovery)

This variable relative to heart rate reserve (HRR) was computed by (peak HR – resting HR). Then, the percentage of heart rate recovery was calculated as [(peak HR – min post-exercise HR)/ (HRR)] ×100.

Blood pressure (BP)

BP measurement was measured using an automatic blood pressure monitor with an automatic blood pressure meter (Omron HEM-7221). The measurement was performed while sitting on a chair. The investigator put the participant's dominant arm on the bicycle handlebar, placed the area to be wrapped in the arm cuff on the same level as the participant's heart, and pressed start to monitoring BP. Blood pressure was monitored during checking resting blood pressure and recovery period for safety.

Rate perceived exertion (RPE)

The original category scale (6 to 20 scale) was used to ask the feeling and assess the intensity of the exercise during MIIT.

2. Pre - experimental procedures

The participants visited the laboratory three times, including the first visit for pre-experiment preparation and two later visits for exercise with intervention. Pre-experimental preparation was set for approximately one week before others. This procedure included:

1. Participants who met the criteria were included and signed the consent form. Anthropometrical and pre-experimental data, including height, body weight, waist-hip circumference, and fat percentage, were then recorded.

2. During the orientation session, the participants were informed of all testing procedures and exercise protocols. Moreover, all participants were trained to do slow deep breathing with metronome beats for breathing correctly. Participants were also asked to sleep adequately and instructed to refrain from vigorous exercise for at least 2 hours or caffeine for 24 hours prior to testing. The soft food diet was allowed to be consumed at least 2 hours before exercise to prevent discomfort and blood circulation insufficiency in large muscle groups during exercise. The participants were allowed to sip pure water before exercise for 30 minutes for dehydrate prevention but water volume for drinking was limited to 500 ml.

3. Maximal oxygen consumption ($\dot{V}O_2max$) was measured by using Astrand-rhyming cycle ergometer test. After that, participant rested for 1 hour before testing the MIIT protocol.

4. After a period of rest, participants were asked to perform MIIT of 3 moderate intensity and 3 low intensity intervals for a total of 30 minutes. This was meticulously designed to ensure their familiarity with the procedure. $\dot{V}O_2$ and heart rate, crucial indicators of their physiological response, were measured with precision during this exercise session. Similarly, these measurements were also meticulously monitored during the 30-minute recovery period, providing us with valuable data for the acute post-exercise recovery phase (Lanzi et al., 2014).

Two later experiments were randomly assigned to different sequences, with MIIT performed with slow deep breathing (SDB) or spontaneous breathing as control trials. The first intervention of all participants was randomly assigned by a computer program, and thereafter, participants alternated between two interventions, providing a balanced crossover design, as shown in Figure 16. The participants rested for one week between each intervention to allow sufficient time to wash out the effect of moderate-intensity continuous training and maintain their usual lifestyle patterns throughout the study.

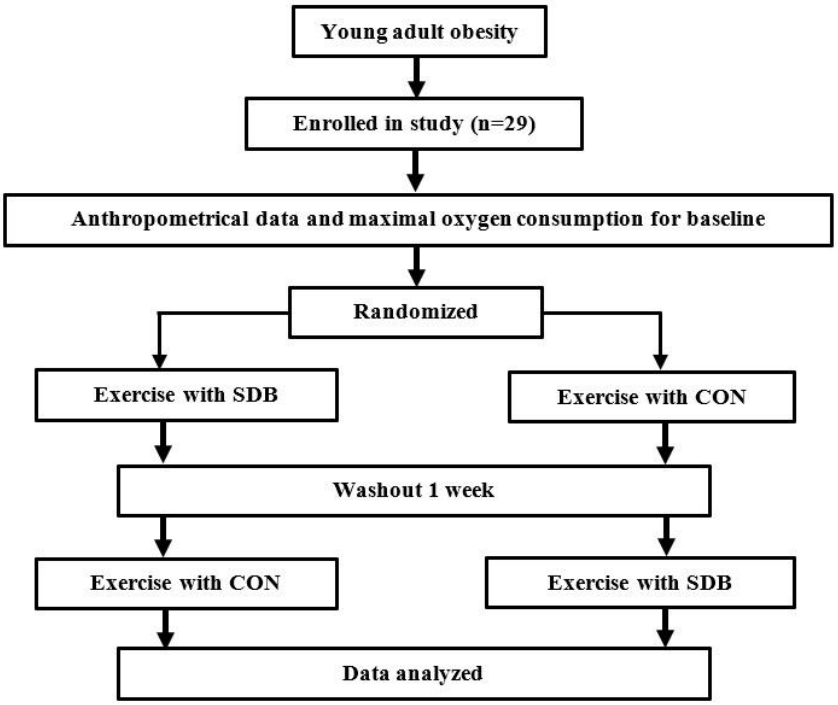


Figure 16 Flow of participants through the study.

3. Exercise training protocol

1. The participants were asked to rest in a quiet, separated room prior to exercise for at least one hour.

2. The participants stretched quadriceps femoris, hamstrings, and gastrocnemius muscles with guidance by the researcher for 10 seconds/muscle group 3 times with both legs. After that, participants sit on a cycle ergometer (Monark 818E Ergomedic bike, Varberg, Sweden) then warm up by cycling with no load for 5 minutes at 50 – 70 rpm (Dupuit et al., 2019).

3. Each exercise interval was performed for 5 minutes. After the warm-up, the workload was increased to reach 20-40% of heart rate reserve in the low-intensity session. Then, the workload was adjusted to reach 50-60% of heart rate reserve for the moderate-intensity sessions. Participants alternately undertook three intervals of 5-minute low intensity and three intervals of 5-minute moderate intensity (5 min/5 min × 3 sets). Participants then received uncontrolled freeload cycling for 6 minutes and resting

on Ergo bike for 4 minutes as a cool down, then sitting in a chair with armrests for 30 minutes in a recovery phase.

4. Interventions during cool down state

4.1 SDB trial

The participants were trained SDB before performing MIIT on the first visit. The participants sat on a chair, leaning on the backrest with relaxation. Then, the participant's dominant hand was put on the abdomen, and another was put on the front upper chest. The slow deep breathing was guided by metronome beats and supervised by physical therapist. The participants were asked to deep breathe through the nose with an inflating abdominal for 4 seconds and exhale out very slowly through nose for 6 seconds at 6 cycles per minute (Pirompol et al., 2015) (Figure 17). SDB interval was performed for 5 minutes (1st, 3rd, 5th, 7th and 9th minute of cool down). SDB with guidance was carried out during the cool down session for 1 minute then separated by resting for a minute with spontaneous breathing. The participant underwent SDB intervals until 10 minutes were completed.

4.2 Control trial

The participants were asked to do spontaneous breathing with uncontrolled freeload cycling for 6 minutes and sit on an Ergo bike for 4 minutes as a cool down.

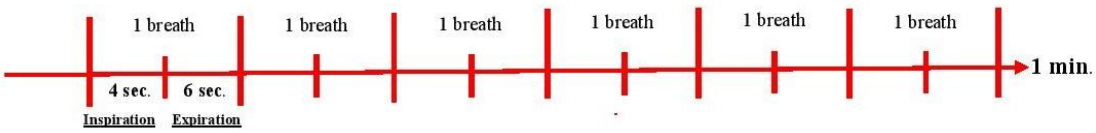


Figure 17 Timeline diagram of slow deep breathing for a minute.

5. Data achievement

Astrand-rhyming cycle ergometer and gas analyzer for $\dot{V}O_2max$ and $\dot{V}O_2$ recording were applied in the first visit, except HRV, HR, and HRR were recorded by electrocardiogram along the trials in three visits.

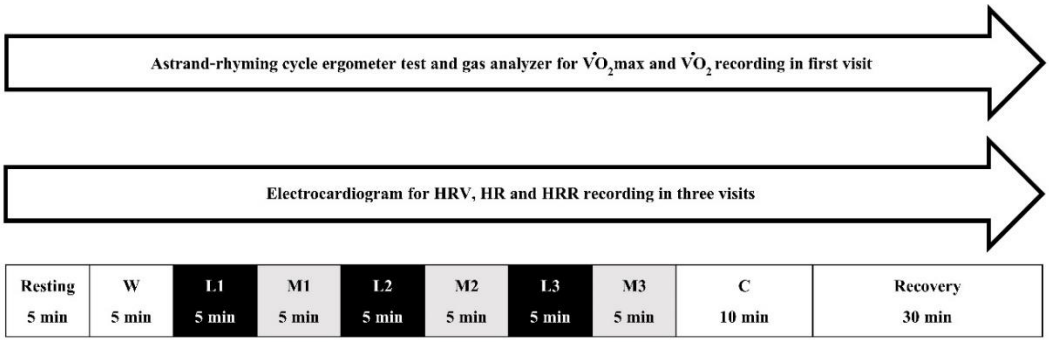


Figure 18 Diagram of MIIT protocol with data achievement.

Abbreviations: W = warm up, L1 = 1st low intensity, M1 = 1st moderate intensity, L2 = 2nd low intensity, M2 = 2nd moderate intensity, L3 = 3rd low intensity, M3 = 3rd moderate intensity, C = cool down.

Statistical Analyses

Data in this study was expressed as mean ± SD. SPSS Statistical software version 23.0 was used to perform the statistical analysis. The $\dot{V}O_2$-HR relationship of MIIT session was computed by linear regression analysis and represented by R squared (R^2). We used the Shapiro-Wilk test to indicate the normality of the distribution. The percentage of HR decline and HR recovery were compared between trials by a paired t-test. Moreover, HRV parameters of two trials were compared by Wilcoxon signed-rank test in case of non-parametric distribution. For comparing HR and HRR in cool down state for 10 minutes, Two-way repeated measures ANOVA was chosen to analyze between interventions at different time points and trials. P-value less than 0.05 was considered statistically significant.

Research place

The study was explored in a laboratory in the Department of Physiology, Mahidol University, and the Department of Physical Therapy, Srinakharinwirot University, Thailand.

CHAPTER 4
RESULT

One of obstacle to exercise in overweight and obese persons is lack of confidence in their ability to be physically active. Quickly exhaustion with the slow rate of recovery could be the main reason of those internal barrier. Therefore, any prevention of rapid exhaustion and any enhancement of recovery rate would motivate their adherence to exercise. In the present study, we aimed to test whether slow deep breathing during cool down might improve the rate of exercise recovery leading to the achievement in exercise. Overweight participants were performed three trials of moderate-intensity interval training (MIIT), in which heart rate and heart rate variability were continuously measured during exercise, cool down and recovery. The oxygen consumption was also monitored at first trial or first visit to indicate individual aerobic performance and make participants being familiarized with the protocol. In, the second and third trials, participants were performed MIIT session with and without slow deep breathing intervention during cool down, randomly. Heart rate recovery by electrocardiogram measurement represented aerobic recovery periods during cool down, while heart rate variability measurement indicated sympatho-vagal balance as potential mechanism underlying the effect of slow deep breathing.

Baseline characteristics and pre - experimental measurement

1. Participants

Baseline characteristics of the participants are showed in Table 4. The study included twenty-five healthy young adults (20.5 ± 2.0 years). All participants were central obesity with overweight to obese class I, moderate to high risk for determining public health and clinical action (body mass index in between 23 and 32.5 kg/m^2). Their physical activity was low level by short form international physical activity questionnaire or IPAQ (metabolic equivalent (MET) below 600 MET-minutes/week and 2 or less day of activities lower than 20 minutes/day). Estimated maximum oxygen consumption was calculated from the Astrand-Rhyming cycle ergometer test (ARCET) and all participants

was 41.7 ± 7.53 ml/kg/min (38.2 ± 6.37 ml/kg/min in males and 45.5 ± 6.98 ml/kg/min in females), aerobic capacity classification was fair fitness in males and average fitness in females.

2. Pre - experimental measurement

The descriptive characteristics of participants in three visits are provided in Table 5. There was no significantly difference between three visits except frequency domain of heart rate variability (HRV) in normalized units. Low frequency domain (LFnu) of heart rate variability at rest was significantly higher in both spontaneous breathing (Control) and slow deep breathing (SDB) trials than the first visit, while high frequency domain (HFnu) was significantly lower in both control and SDB trials. However, there was no significantly difference between control and SDB trails on HRV at rest indicating similar baseline of cardiac activation between trials.

3. Effect of MIIT session on oxygen consumption

In order to characterize the aerobic performance of participant during MIIT session and recovery, changes in oxygen consumption ($\dot{V}O_2$) were monitored. $\dot{V}O_2$ increased gradually during the warm-up and the first interval of low-intensity exercise (L1). At the first moderate intensity interval (M1), $\dot{V}O_2$ markedly raised 3 to 4 folds of L1 and then dropped suddenly in the second low intensity (L2). $\dot{V}O_2$ during L2 almost completely returned to the same level detected during L1. Interestingly, Peak $\dot{V}O_2$ in the second (M2) and the third moderate-intensity exercise (M3) became higher than the first (M1), respectively. At cool down (Figure 20A and B), $\dot{V}O_2$ suddenly decreased and approached near the resting levels within 10 minutes. The percentage of $\dot{V}O_2$ recovery after one minute was 29.1 ± 10.2 % and reaching 80.5 ± 9.7 % at minute 10^{th} (Table 6).

4. Effect of MIIT session on heart rate

During MIIT session, heart rate was also recorded. Heart rate was slightly increased during warm-up and further increased during the first low-intensity exercise (L1). Following moderate-intensity exercise (M1), the heart rate gradually raised and hit the target heart rate as calculated (141–153 beats/min). Upon shifting to low intensity, the heart rate dropped, but did not completely return to the heart rate zone throughout 5

minutes in both the second (L2) and the third low-intensity exercise (L3). Interestingly, the peak heart rate found during the second moderate intensity (M2) was higher than the peak of the first moderate-intensity exercise (M1) (M2: 155 ± 7.19 *vs*. M1: 149 ± 9.44 beats/min). Moreover, with the same cycling load, the peak heart rate of the third moderate-intensity exercise (M3) raised beyond the desired target heart rate zone (M3: 163 ± 9.86 beats/min). This finding suggests that the MIIT protocol in this study activated more effort in overweight persons than expected from the volume-load. During the cool down period, heart rate gradually decreased, however, the level after 10 minutes was still significantly higher than levels during L1. Figure 19B also presented that heart rate recovery of all participants progressively increased and was 53.5 ± 9.61 beats/min (68.0 ± 6.10 % of heart rate recovery) at 10 minutes after exercise cassation (Table 6).

The relationship of heart rate to oxygen consumption during MIIT session was also evaluated (Figure 19C). There is highly correlation between heart rate and oxygen consumption during MIIT session (R^2 = 0.716 ± 0.104) and cool down stage (R^2 = 0.611 ± 0.197). Therefore, changes in heart rate can indicate the change in oxygen consumption in this study.

5. Changes in heart rate variability at the peak exercise

In order to evaluate the effect of MIIT session on cardio-autonomic activation, both time-domain and frequency domain of HRV were calculated at the last five minutes of moderate intensity exercise (Table 5). The Borg rating of perceived exertion and peak heart rate were similar among three trials at last moderate intensity interval. During the last five minutes of exercise phase, the standard deviation of the interbeat intervals for all sinus beats (SDRR) and Root means square successive difference (RMSSD) were significantly decreased as compared to resting state. There was no significant difference in these time-domain parameters among three trials. For frequency domain of HRV. LFnu was increased while HF was decreased during exercise as expected. Results demonstrated slightly higher in LFnu of the control and SDB trials during exercise than that of the first visit. This difference might be due to the higher in LFnu in

both trails since resting. Nevertheless, both time and frequency domains of HRV were not significantly different between control trial and SDB trials.

Table 4 Baseline characteristics of participants.

Variables	
Participants	25 (13 M: 12 F)
Age (years)	20.5 ± 2.0
BMI (kg/m^2)	27.3 ± 2.6
Waist circumference (cm)	92.4 ± 6.9
Percentage of fat mass (%)	32.4 ± 9.3
Estimated $\dot{V}O_2max$ (kg/ml/min)	41.7 ± 7.53

Data shows mean ± standard deviation.

Abbreviations: BMI = body mass index, $\dot{V}O_2max$ = maximal oxygen consumption, SDB = slow deep breathing, M = male, F = female.

Table 5 The comparison of heart rate, blood pressure at rest and heart rate variability parameter between the first visit, control, and slow deep breathing at the last 5 minutes of moderate intensity interval training before cool down.

Variables	Visit 1 (n=25)	Control (n=25)	SDB (n=25)	P value
At rest				
Heart rate (beats/min)	80.2 ± 7.4	79.6 ± 9.2	80.2 ± 8.6	0.942
SDRR (millisecond)	53.6 ± 19.9	54.9 ± 24.0	52.7 ± 18.0	0.881
RMSSD (millisecond)	37.8 ± 19.9	37.4 ± 26.7	32.8 ± 15.5	0.392
LFnu	50.2 ± 27.0	62.6 ± 20.7*	64.2 ± 20.4*	0.003
HFnu	50.5 ± 25.7	37.6 ± 19.6*	35.8 ± 19.3*	0.001
LF/HF ratio	2.11 ± 2.59	2.66 ± 2.48	3.23 ± 3.47	0.075
The last 5 minutes of moderate intensity interval training before cool down				
The Borg rating of perceived exertion	14.0 ± 2.4	13.0 ± 2.1	13.2 ± 1.9	0.089
Heart rate (beats/min)	149 ± 10	147 ± 10	148 ± 10	0.497
SDRR (millisecond)	18.1 ± 31.3	12.5 ± 5.6	13.1 ± 6.5	0.428
RMSSD (millisecond)	13.0 ± 50.8	3.36 ± 2.47	2.83 ± 1.24	0.336
LFnu	60.9 ± 26.1	72.9 ± 18.6*	72.7 ± 22.7	0.035
HFnu	29.9 ± 21.8	24.1 ± 17.4	24.4 ± 18.4	0.315
LF/HF ratio	11.7 ± 32.1	5.66 ± 5.25	6.86 ± 6.80	0.396

Data shows mean ± standard deviation.

*$p < 0.05$ compared to Visit 1 and data were analyzed by repeated measure ANOVA.

Abbreviations: SDB = slow deep breathing, LFnu = low frequency in normalized units, HFnu = high frequency in normalized units, LF/HF ratio = the ratio of LF to HF, SDRR; The standard deviation of the interbeat intervals for all sinus beats in milliseconds, RMSSD = Root means square successive difference in milliseconds.

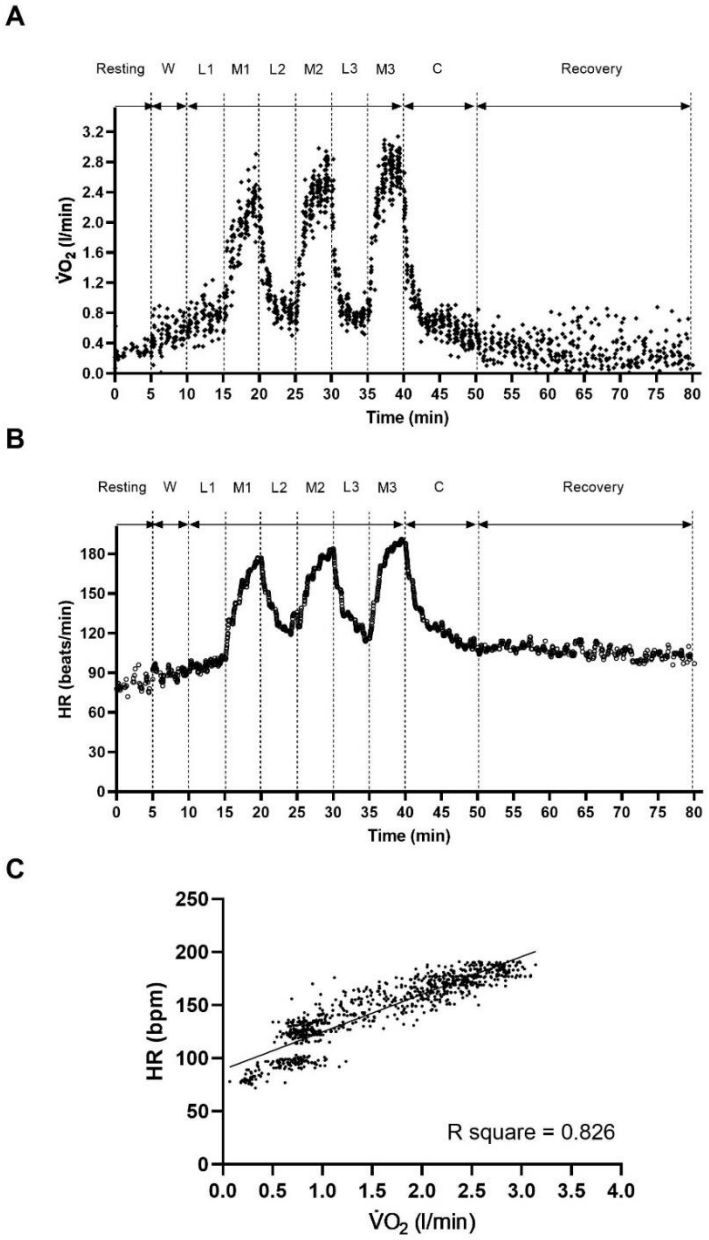


Figure 19 The example of a participant for oxygen consumption (A), heart rate (B) during MIIT to recovery after exercise and the relationship of both outcomes (C) during MIIT.

Abbreviations: W = warm up, L1 = 1st low intensity, M1 = 1st moderate intensity, L2 = 2nd low intensity, M2 = 2nd moderate intensity, L3 = 3rd low intensity, M3 = 3rd moderate intensity, C = cool down.

Table 6 The oxygen consumption, percentage of oxygen consumption recovery and heart rate recovery for 10 minutes of cool down after moderate intensity interval training at first visit (n=25).

Cool down	$\dot{V}O_2$ (L)	%$\dot{V}O_2$ recovery (%)	% HR recovery (%)
0 min	1.39 ± 0.41	0	0
1 min	0.983 ± 0.305	29.1 ± 10.2	32.4 ± 8.00
2 min	0.658 ± 0.198	52.9 ± 8.2	43.5 ± 7.83
3 min	0.562 ± 0.172	59.7 ± 9.3	49.6 ± 7.03
4 min	0.514 ± 0.167	63.1 ± 9.1	52.4 ± 7.37
5 min	0.488 ± 0.186	64.9 ± 10.9	54.3 ± 7.19
6 min	0.463 ± 0.173	66.7 ± 9.9	57.6 ± 6.32
7 min	0.360 ± 0.168	74.0 ± 11.2	66.7 ± 7.22
8 min	0.281 ± 0.137	79.7 ± 9.4	69.1 ± 7.78
9 min	0.239 ± 0.126	82.3 ± 8.1	68.8 ± 6.76
10 min	0.260 ± 0.135	80.5 ± 9.7	68.0 ± 6.10
Baseline	0.182 ± 0.091	100	

Data shows mean ± standard deviation.

Abbreviations: $\dot{V}O_2$ = oxygen consumption, %$\dot{V}O_2$ recovery = percentage of oxygen consumption recovery, HRR = heart rate recovery, % HR recovery = percentage of heart rate recovery.

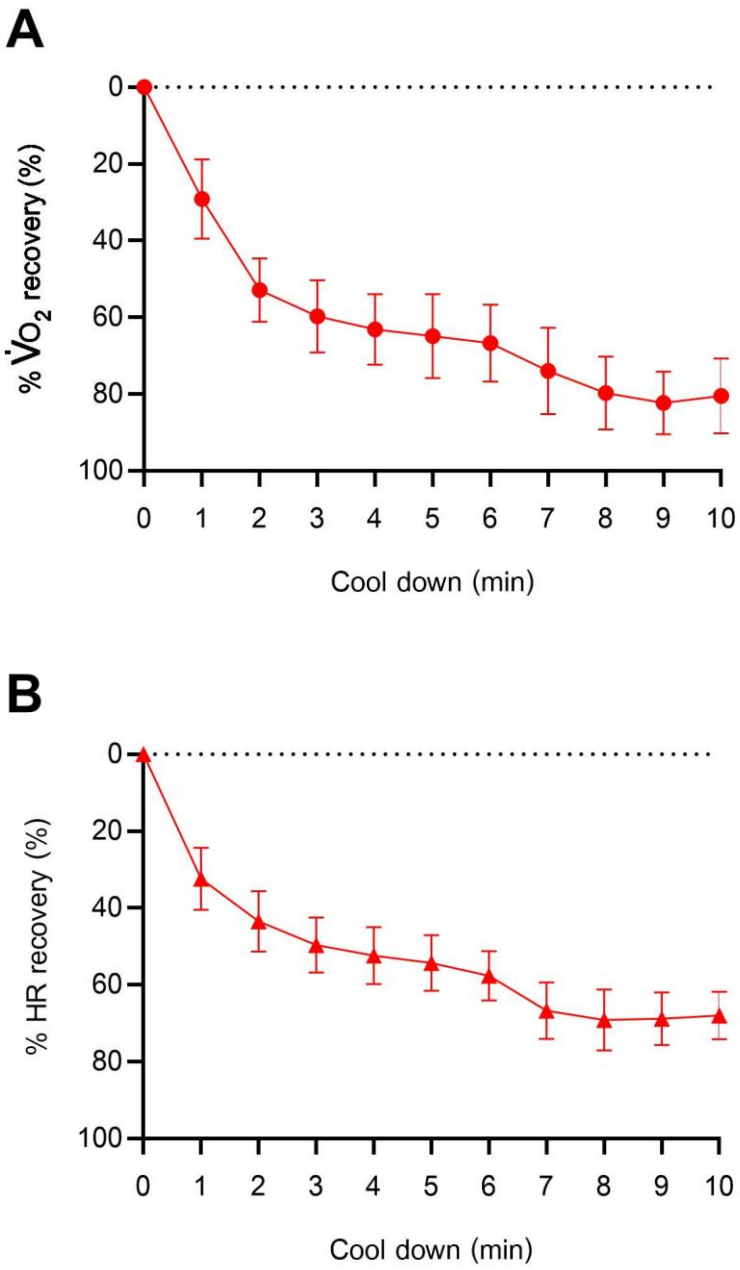


Figure 20 Percentage of oxygen consumption (A) and percentage of heart rate recovery (B) during cool down at first visit.

The effects of slow deep breathing on heart rate recovery

1. Heart rate recovery in cool down for 10 minutes

In order to evaluate the effect of slow deep breathing on enhancing cardiopulmonary routine, heart rate recovery was recorded after MIIT session with and

without show deep breathing during cool down (Table 7). Results demonstrated that the additional of SDB significantly increased the percentage of heart rate decline during cool down as compared to that during cool down without SDB on the first minute. However, the difference was disappeared in the second minute to minute fifth. Although Wilcoxon signed rank test revealed that the percentage of heart rate decline with SDB was significantly lower than that without SDB on minute 6^{th}, 8^{th} and 10^{th}, this phase of plateau (7^{th} to 10^{th} minute) had no any impact on recovery. Therefore, slow deep breathing technique used in this study during cool down helped improving fast phase recovery, without any significance on the slow recovery phase. In order to evaluate the effect of slow deep breathing on enhancing cardiopulmonary routine, heart rate recovery was recorded after MIIT session with and without show deep breathing during cool down (Table 7). Results demonstrated that the additional of SDB significantly increase the percentage of heart rate decline during cool down as compared to that during cool down without SDB on the first minute. However, the difference was disappeared in the second minute to minute fifth. Interestingly, Wilcoxon signed rank test revealed that the percentage of heart rate decline with SDB was significantly lower than that without SDB on minute 6^{th}, 8^{th} and 10^{th}. These results suggested that slow deep breathing technique used in this study during cool down helped improving fast phase recovery, but delayed the slow recovery phase.

2. Heart rate recovery for 30 minutes after cool down

The effect of slow deep breathing during cool down on recovery phase was also indicated. Heart rate was slowly declined throughout 30 minute of recovery period. No significant difference in percentage of heart rate recovery between control trial and SDB trial at all time (Table 8), except only at 24^{th} minute. Results suggested no effect of slow deep breathing during cool down on heart rate recovery.

Table 7 The percentage of heart rate decline in every minute during cool down state for 10 minutes within the control and slow deep breathing session (n=25).

Cool down	HRR (beats/min)		% HR decline (%)		Effect size *d*	95% confidence interval of difference	
	Control	SDB	Control	SDB		Lower	Upper
HRR30	14.5 ± 6.0	17.9 ± 8.2	9.33 ± 3.57	11.4 ± 4.6*	0.468	-3.951	-0.247
HRR1	22.0 ± 6.5	26.2 ± 8.0	14.3 ± 3.8	16.7 ± 4.2*	0.681	-3.959	-0.971
HRR2	30.9 ± 6.9	31.4 ± 8.8	20.1 ± 4.0	20.1 ± 4.5	0.006	-1.408	1.37
HRR3	34.9 ± 7.4	36.3 ± 8.9	22.6 ± 4.2	23.3 ± 4.6	0.180	-2.180	0.860
HRR4	36.6 ± 8.1	35.7 ± 9.1	23.7 ± 4.5	22.9 ± 4.9	0.238	-0.607	2.27
HRR5	38.2 ± 8.2	38.7 ± 8.6	24.8 ± 4.5	24.8 ± 4.5	0.023	-1.526	1.37
HRR6	40.7 ± 7.5	38.3 ± 9.2	26.4 ± 4.2	24.6 ± 4.9*	0.583	0.530	3.10
HRR7	48.0 ± 7.3	46.7 ± 8.6	31.2 ± 4.6	30.1 ± 4.4	0.224	-0.963	3.26
HRR8	49.5 ± 7.1	45.9 ± 9.5	32.2 ± 4.0	29.5 ± 5.0*	0.284	0.857	4.40
HRR9	49.6 ± 8.9	48.1 ± 9.7	32.2 ± 4.9	30.9 ± 5.3	0.249	-0.819	3.32
HRR10	48.0 ± 9.7	45.7 ± 8.3	31.1 ± 5.1	29.4 ± 4.5*	0.425	0.047	3.26

Data shows mean ± standard deviation.

*$p < 0.05$ compared to control in % HR decline and data were analyzed by paired t-test.

Abbreviations: SDB = slow deep breathing, % HR decline = percentage of heart rate decline, HRR = heart rate recovery, HRR30 = HRR at 30 seconds after exercise, HRR1 = HRR at 1 minute after exercise, HRR2 = HRR at 2 minutes after exercise, HRR3 = HRR at 3 minutes after exercise, HRR4 = HRR at 4 minutes after exercise, HRR5 = HRR at 5 minutes after exercise, HRR6 = HRR at 6 minutes after exercise, HRR7 = HRR at 7 minutes after exercise, HRR8 = HRR at 8 minutes after exercise, HRR9 = HRR at 9 minutes after exercise, HRR10 = HRR at 10 minutes after exercise.

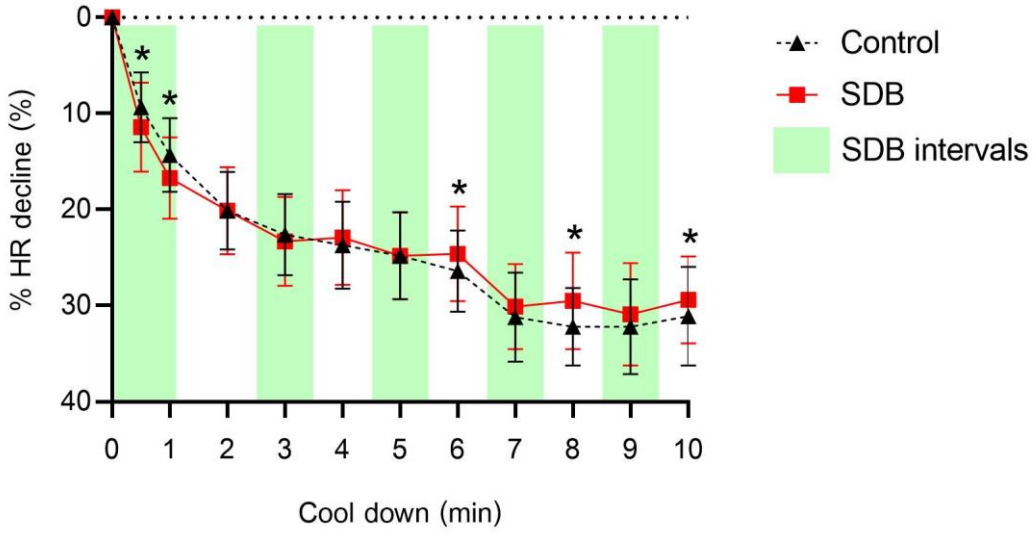
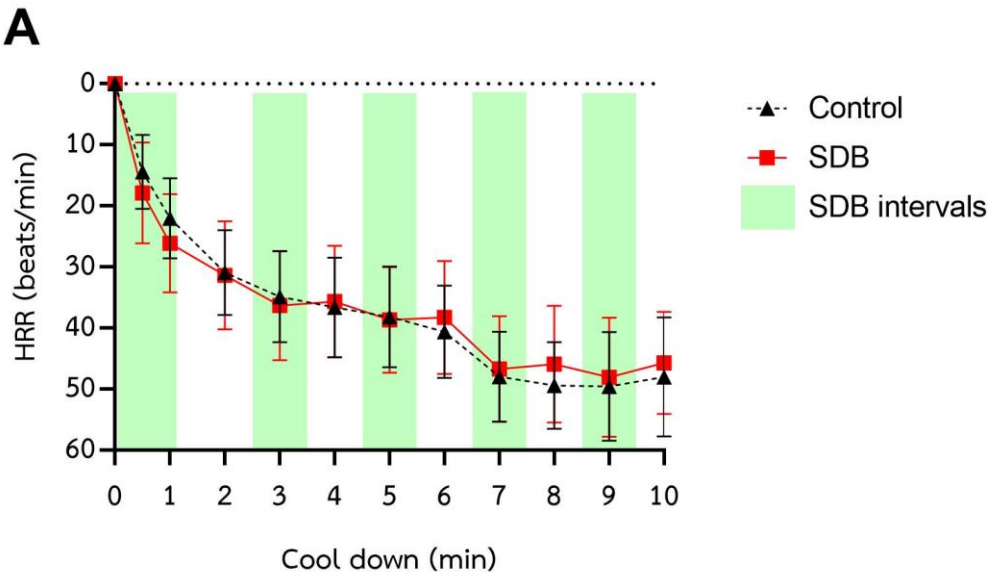


Figure 21 Mean of Heart rate and time during cool down state for 10 minutes between control and slow deep breathing session in 25 participants.

* $p < 0.05$ compared to control in % HR decline and data were analyzed by paired t-test.

Abbreviations: SDB intervals = slow deep breathing was performed at 1st, 3rd, 5th, 7th, and 9th minute period.



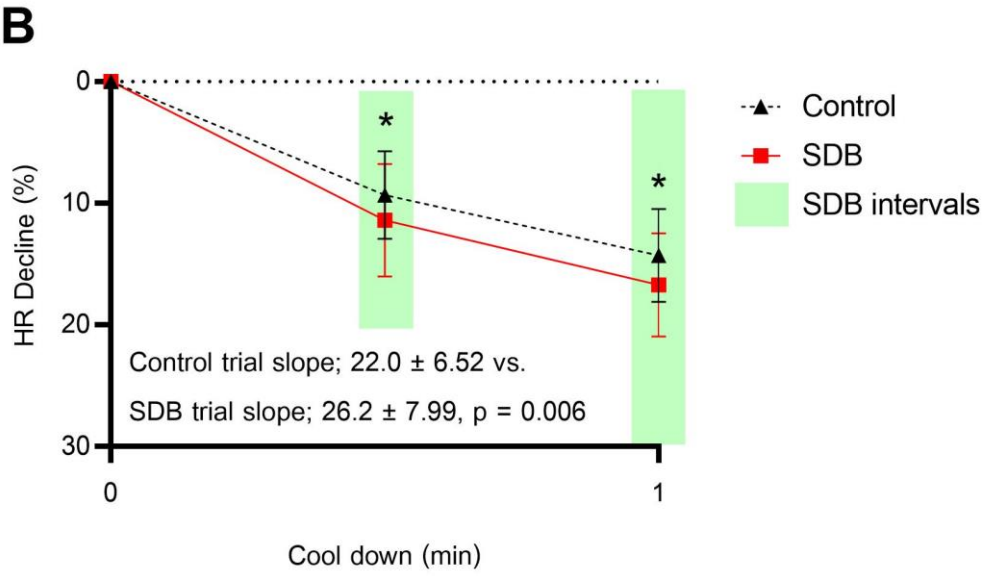


Figure 22 Mean of heart rate recovery (A) and the percentage of heart rate decline (B) during cool down state in control and slow deep breathing session in 25 participants.

HRR data were analyzed by 2-way repeated measure ANOVA

* $p < 0.05$ compared to control in the percentage of HR decline slope and data were analyzed by Wilcoxon signed rank test.

Abbreviations: SDB intervals = slow deep breathing was performed at 1st, 3rd, 5th, 7th, and 9th minute period.

Table 8 The comparison of percentage of heart rate decline during recovery 30 minutes after cool down between control and slow deep breathing (n=25).

Recovery phase	HR (beats/min)		% HR recovery		Effect size *d*	95% confidence interval of the difference	
	Control	SDB	Control	SDB		Lower	Upper
0-3 min	97.3 ± 9.64	99.6 ± 10.3	78.2 ± 8.25	76.3 ± 8.00	0.235	-1.49	5.18
3-6 min	96.0 ± 9.84	97.6 ± 10.5	80.2 ± 9.32	79.2 ± 8.14	0.104	-3.04	4.90
6-9 min	94.4 ± 9.80	96.5 ± 10.7	82.2 ± 9.32	80.7 ± 8.87	0.184	-1.91	4.80
9-12 min	92.2 ± 11.0	95.1 ± 10.6	85.4 ± 10.7	82.6 ± 9.21	0.287	-1.29	6.75
12-15 min	91.4 ± 11.1	94.0 ± 11.8	86.6 ± 10.7	84.5 ± 12.0	0.194	-2.40	6.52
15-18 min	90.5 ± 10.3	92.3 ± 11.6	87.6 ± 10.0	86.8 ± 11.5	0.069	-3.99	5.55
18-21 min	89.4 ± 10.2	92.6 ± 10.7	89.1 ± 9.78	86.3 ± 9.47	0.338	-0.571	6.25
21-24 min	88.9 ± 9.30	92.6 ± 10.4	89.7 ± 9.81	86.1 ± 9.90*	0.434	0.143	6.99
24-27 min	88.9 ± 10.6	91.9 ± 10.2	89.7 ± 10.2	87.1 ± 9.52	0.312	-0.868	6.04
27-30 min	89.9 ± 9.88	91.4 ± 9.49	88.3 ± 9.74	87.6 ± 9.23	0.061	-4.07	5.35

Data shows mean ± standard deviation

* $p < 0.05$ compared to control in % HR recovery and data were analyzed by paired t-test.

Abbreviations: HR = Heart rate, % HR recovery = percentage of heart rate recovery

The effects of slow deep breathing on cardio-autonomic activation

1. Sympatho-vagal balance in cool down for 10 minutes

To evaluate the possible mechanism underlying the effect of slow deep breathing technique during cool down on heart rate, time domain and frequency domain of heart rate variability were recorded. The time domain of HRV was analyzed minute to minute during cool down for 10 minutes (Table 9.1 and 9.2). Results demonstrated that SDRR gradually increased during cool down, in which the rate of SDRR increase in SDB trial was significantly higher than in that of control trial at 1st, 3rd, 4th, 5th, and 9th minute period. However, RMSSD in SDB trial was significantly higher only at 1st and 3rd minute period. This time domain of HRV suggests the potential recovery effect of slow deep breathing technique acting through the enhancement of parasympathetic activation.

The frequency domain of HRV, on the other hand, showed an inconsistency change during cool down in both trials (Table 9.3, 9.4 and 9.5, Figure 23). Interestingly, low frequency domain in SDB trial was significantly higher than that in control trial in almost period of cool down, while high frequency domain in SDB trial was significantly lower than that in control trial. LF/HF ratio was also higher in SBD trial than that in control trial. Results of frequency domain suggests the slow deep breathing technique has high potency in modulating sympatho-vagal activation by delay parasympathetic activation as previous mentioned (Sasaki & Maruyama, 2014).

2. Cardio-autonomic activation for 30 minutes after cool down

Heart rate variability was also calculated in every three minutes interval. Time domain of HRV significantly increased in both trials during this period as expected (Table 10.1 and 10.2). Similarly, LFnu decreased, and HFnu increased upon recovery in both trials. While no significant difference in SDRR between two trials was observed, RMSSD of SDB trial was significantly lower than that of control trial at the 24 to 27-minute interval of recovery phase (Table 10.2). For frequency domain of HRV, results demonstrated only few significant differences between two trials (Table 10.3, 10.4 and 10.5). LFnu of SDB trials was higher than control trial, while HF of SDB trial was lower than that of control trial only at first three-minute interval. Additionally, LF/HF was then higher in SDB trial at the first three-minute interval. The lower HFnu and higher LF/HF in

SDB trial was also observed at the 9 to 12-minute interval. With a very few differences observed between control and SDB trials during 30-minute recovery, it could be suggested no effect of slow deep breathing intervention during cool down on cardiopulmonary activity in the recovery period.

Table 9 The comparison of heart rate variability parameter from peak intensity (0 min) to 10 minutes during cool down state between control and slow deep breathing session.

Table 9.1 The comparison of the standard deviation of the interbeat intervals for all sinus beats in milliseconds (SDRR).

Cool down	SDRR (millisecond)	
	Control (n=25)	SDB (n=25)
0 min	4.99 ± 1.96	4.87 ± 1.92
1 min	15.2 ± 6.3	19.9 ± 9.4*
2 min	12.9 ± 6.1	15.3 ± 4.6
3 min	11.7 ± 6.5	18.4 ± 9.4*
4 min	10.7 ± 4.9	13.9 ± 5.7*
5 min	12.4 ± 5.0	18.3 ± 8.8*
6 min	18.1 ± 10.9	19.0 ± 11.0
7 min	22.0 ± 14.4	25.6 ± 11.2
8 min	20.2 ± 12.8	20.0 ± 7.3
9 min	20.5 ± 9.0	26.9 ± 9.7*
10 min	20.9 ± 11.8	22.3 ± 9.3

Data shows mean ± standard deviation.

* $p < 0.05$ compared to control and data were analyzed by Wilcoxon signed rank test.

Table 9. 2 The comparison of root means square successive difference in milliseconds (RMSSD).

Cool down	RMSSD (millisecond)	
	Control (n=25)	SDB (n=25)
0 min	2.73 ± 0.85	3.91 ± 6.41
1 min	4.81 ± 2.89	8.95 ± 13.30*
2 min	5.32 ± 3.66	5.72 ± 3.03
3 min	5.45 ± 3.79	7.90 ± 4.85*
4 min	6.03 ± 3.82	7.63 ± 13.36
5 min	6.15 ± 3.86	7.46 ± 4.55
6 min	7.98 ± 5.65	6.65 ± 4.37
7 min	10.8 ± 8.4	11.4 ± 7.4
8 min	10.2 ± 7.5	7.72 ± 4.01
9 min	10.5 ± 6.9	10.8 ± 6.4
10 min	8.84 ± 4.96	8.11 ± 5.68

Data shows mean ± standard deviation.

* $p < 0.05$ compared to control and data were analyzed by Wilcoxon signed rank test.

Table 9.3 The comparison of low frequency in normalized units (LFnu).

Cool down	LFnu	
	Control (n=25)	SDB (n=25)
0 min	74.4 ± 21.2	69.8 ± 26.0
1 min	72.9 ± 20.8	85.5 ± 17.9*
2 min	74.2 ± 19.9	79.4 ± 19.6
3 min	79.5 ± 16.7	85.6 ± 17.4
4 min	73.9 ± 20.9	81.6 ± 15.7
5 min	77.8 ± 16.6	91.1 ± 7.1*
6 min	77.8 ± 16.2	86.6 ± 9.7*
7 min	74.8 ± 15.2	85.3 ± 14.7*
8 min	77.4 ± 10.9	88.3 ± 7.9*

Table 9.3 (Continued)

Cool down	LFnu	
	Control (n=25)	SDB (n=25)
9 min	72.5 ± 18.2	86.8 ± 13.0*
10 min	73.3 ± 14.9	83.3 ± 12.2*

Data shows mean ± standard deviation.

* $p < 0.05$ compared to control and data were analyzed by Wilcoxon signed rank test.

Table 9.4 The comparison of high frequency in normalized units (HFnu).

Cool down	HFnu	
	Control (n=25)	SDB (n=25)
0 min	20.6 ± 17.5	25.0 ± 20.1
1 min	21.5 ± 12.8	14.7 ± 17.5*
2 min	23.5 ± 15.7	20.6 ± 18.3
3 min	18.8 ± 12.6	14.3 ± 17.0
4 min	23.4 ± 18.4	18.3 ± 14.4
5 min	20.5 ± 14.0	9.01 ± 7.00*
6 min	21.0 ± 13.8	14.4 ± 8.8
7 min	24.8 ± 13.0	14.9 ± 14.3*
8 min	23.1 ± 10.2	13.2 ± 7.6*
9 min	28.5 ± 17.0	13.6 ± 13.3*
10 min	27.1 ± 14.8	18.2 ± 11.5*

Data shows mean ± standard deviation.

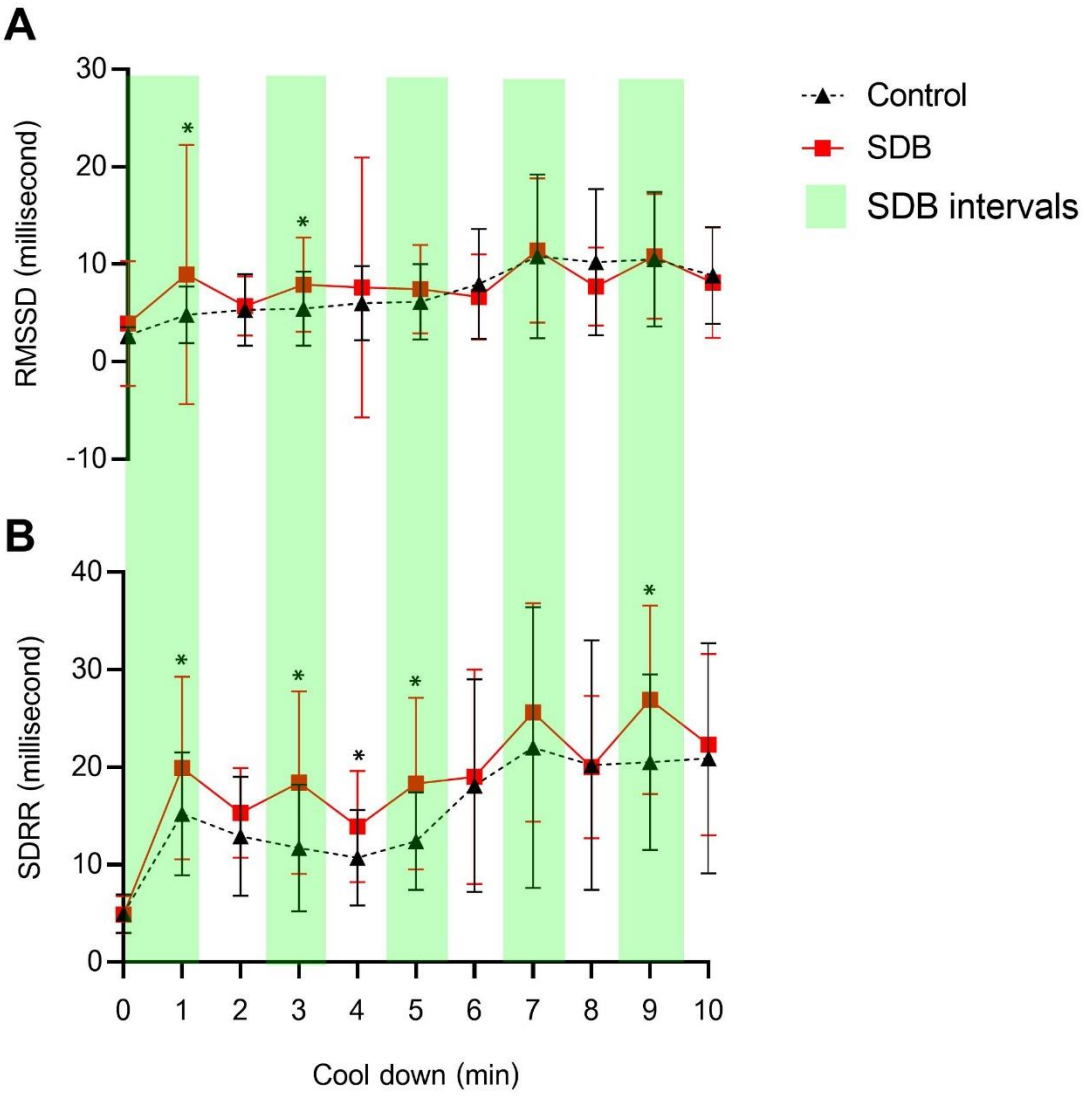
* $p < 0.05$ compared to control and data were analyzed by Wilcoxon signed rank test.

Table 9. 5 The comparison of the ratio of low frequency to high frequency in normalized units (LF/HF ratio).

Cool down	LF/HF ratio	
	Control (n=25)	SDB (n=25)
0 min	8.70 ± 9.81	8.1 ± 11.6
1 min	5.79 ± 5.30	21.4 ± 23.0*
2 min	6.36 ± 6.94	8.43 ± 6.87
3 min	7.44 ± 7.43	17.0 ± 17.6*
4 min	6.02 ± 5.51	8.95 ± 8.50
5 min	7.80 ± 9.80	16.6 ± 13.9*
6 min	6.97 ± 7.47	8.59 ± 5.22
7 min	4.33 ± 3.19	11.2 ± 7.4*
8 min	5.19 ± 6.54	9.96 ± 7.09*
9 min	4.27 ± 4.47	18.0 ± 17.0*
10 min	4.60 ± 4.78	11.6 ± 20.5*

Data shows mean ± standard deviation.

* $p < 0.05$ compared to control and data were analyzed by Wilcoxon signed rank test.


A
B
RMSSD (millisecond)
SDRR (millisecond)
Cool down (min)
Control
SDB
SDB intervals
30
20
10
0
-10
40
30
20
10
0
0 1 2 3 4 5 6 7 8 9 10
*

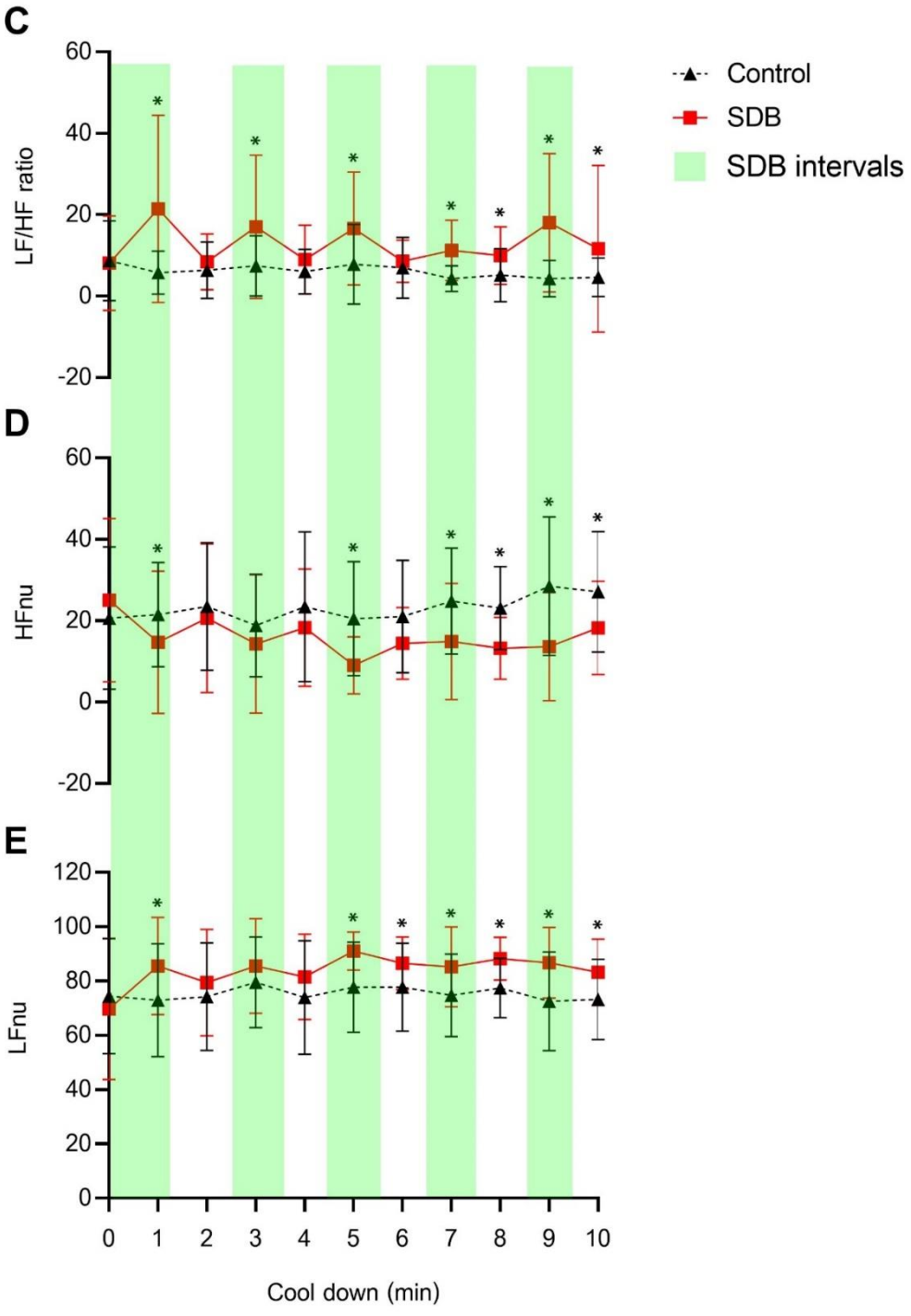


Figure 23 RMSSD (A), SDRR (B), LF/HF ratio (C), HFnu (D) and LFnu (E) of heart rate variability after exercise between control and slow deep breathing trials.

* $p < 0.05$ compared to control and data were analyzed by Wilcoxon signed rank test.

Abbreviations: SDB intervals = slow deep breathing was performed at 1st, 3rd, 5th, 7th, and 9th minute period.

Table 10 The comparison of heart rate variability parameter during recovery 30 minutes after cool down between control and slow deep breathing (n=25).

Table 10.1 The comparison of the standard deviation of the interbeat intervals for all sinus beats in milliseconds (SDRR) every 3 minutes of recovery 30 minutes.

Recovery phase	SDRR (millisecond)	
	Control (n=25)	SDB (n=25)
0-3 min	29.5 ± 12.6	27.6 ± 15.0
3-6 min	31.4 ± 14.0	27.8 ± 10.5
6-9 min	36.1 ± 16.7	31.2 ± 18.7
9-12 min	38.5 ± 19.0	34.2 ± 17.8
12-15 min	40.3 ± 19.3	36.2 ± 18.5
15-18 min	44.7 ± 23.7	39.8 ± 21.6
18-21 min	46.5 ± 22.5	42.7 ± 25.5
21-24 min	48.9 ± 22.1	40.7 ± 22.9
24-27 min	48.0 ± 23.6	41.2 ± 20.6
27-30 min	50.7 ± 26.9	41.8 ± 23.2

Data shows mean ± standard deviation.

Table 10. 2 The comparison of root means square successive difference in milliseconds (RMSSD) every 3 minutes of recovery 30 minutes.

Recovery phase	RMSSD (millisecond)	
	Control (n=25)	SDB (n=25)
0-3 min	14.4 ± 8.96	10.6 ± 6.05
3-6 min	15.1 ± 9.37	12.7 ± 7.75
6-9 min	17.9 ± 11.8	15.5 ± 11.3
9-12 min	20.8 ± 13.6	17.6 ± 13.6

Table 10.2 (Continued)

Recovery phase	RMSSD (millisecond)	
	Control (n=25)	SDB (n=25)
12-15 min	23.8 ± 16.2	19.9 ± 15.3
15-18 min	25.7 ± 19.5	22.2 ± 18.7
18-21 min	27.0 ± 18.8	21.4 ± 16.6
21-24 min	26.1 ± 14.6	22.7 ± 23.5
24-27 min	25.5 ± 13.5	21.5 ± 17.9*
27-30 min	26.2 ± 15.2	21.3 ± 13.3

Data shows mean ± standard deviation.

* $p < 0.05$ compared to control and data were analyzed by Wilcoxon signed rank test.

Table 10.3 The comparison of low frequency in normalized units (LFnu) every 3 minutes of recovery 30 minutes.

Recovery phase	LFnu	
	Control (n=25)	SDB (n=25)
0-3 min	72.5 ± 19.5	81.7 ± 15.8*
3-6 min	75.6 ± 17.4	78.7 ± 14.8
6-9 min	71.2 ± 19.0	77.8 ± 13.8
9-12 min	69.0 ± 20.7	75.6 ± 19.3
12-15 min	66.7 ± 20.7	74.0 ± 16.5
15-18 min	68.6 ± 18.1	73.2 ± 17.0
18-21 min	68.0 ± 18.9	73.4 ± 17.8
21-24 min	70.3 ± 20.7	69.0 ± 22.2
24-27 min	68.9 ± 16.9	70.0 ± 19.0
27-30 min	67.5 ± 17.8	73.0 ± 13.5

Data shows mean ± standard deviation.

* $p < 0.05$ compared to control and data were analyzed by Wilcoxon signed rank test.

Table 10.4 The comparison of high frequency in normalized units (HFnu) every 3 minutes of recovery 30 minutes.

Recovery phase	HFnu	
	Control (n=25)	SDB (n=25)
0-3 min	27.6 ± 18.3	19.3 ± 15.4*
3-6 min	25.1 ± 16.7	21.8 ± 14.1
6-9 min	29.8 ± 18.3	23.0 ± 13.1
9-12 min	31.1 ± 19.4	22.8 ± 16.2*
12-15 min	32.8 ± 18.7	26.4 ± 15.7
15-18 min	31.5 ± 17.2	27.0 ± 16.1
18-21 min	31.8 ± 18.0	28.0 ± 16.6
21-24 min	30.2 ± 19.7	31.9 ± 21.2
24-27 min	31.5 ± 16.2	30.5 ± 18.5
27-30 min	33.4 ± 16.9	26.8 ± 13.2

Data shows mean ± standard deviation.

* $p < 0.05$ compared to control and data were analyzed by Wilcoxon signed rank test.

Table 10.5 The comparison of the ratio of low frequency to high frequency in normalized units (LF/HF ratio) every 3 minutes of recovery 30 minutes.

Recovery phase	LF/HF ratio	
	Control (n=25)	SDB (n=25)
0-3 min	4.25 ± 2.89	8.22 ± 6.92*
3-6 min	5.95 ± 6.67	6.58 ± 5.79
6-9 min	4.46 ± 4.84	5.11 ± 3.66
9-12 min	4.00 ± 4.00	5.90 ± 4.52*
12-15 min	3.88 ± 4.16	4.26 ± 3.23
15-18 min	3.63 ± 3.75	5.46 ± 6.02
18-21 min	4.80 ± 8.47	4.83 ± 4.95
21-24 min	4.07 ± 4.18	4.30 ± 4.40
24-27 min	3.62 ± 3.32	5.08 ± 5.73
27-30 min	2.87 ± 2.00	3.88 ± 3.51

Data shows mean ± standard deviation.

* $p < 0.05$ compared to control and data were analyzed by Wilcoxon signed rank test.

CHAPTER 5
SUMMARY DISCUSSION AND SUGGESTION.

The main objective of the present study was to investigate the effect of slow deep breathing on aerobic recovery periods and sympathovagal balance. We used heart rate recovery, the percentage of heart rate decline, and heart rate variability as our primary outcome measures. Twenty-five overweight and obesity young individuals who performed cycling ergometer for MIIT were included in the study. The finding showed that applying of slow deep breathing (SDB) technique during cool down in this study resulted in an enhanced recovery of heart rate during fast phase, but resulted a delay in the recovery during the slow phase of heart rate recovery (HRR). Moreover, heart rate variability (HRV) revealed that the potential recovery effect of slow deep breathing technique increased parasympathetic activation at early cool down but modulated sympathovagal activation toward sympathetic activation at the late cool down.

Baseline characteristics and pre-experiment

The study included twenty-five healthy young adults with high fat mass (WHO Expert Committee, 1995), central obesity (Obesity in Asia et al., 2007) and overweight to obese class I (WHO Expert Consultation, 2004). Their physical activity was low level by short form international Physical Activity Questionnaire (IPAQ-SF). However, the estimated maximum oxygen consumption from the Astrand-Rhyming cycle ergometer test (ARCET) showed that aerobic capacity classification based on the previous study of maximal oxygen consumption ($\dot{V}O_2max$) in Thai people (Tongprasert & Wattanapan, 2007) was fair and average fitness in males and females, respectively. A systematic review of IPAQ-SF as a self-reported questionnaire suggested that in the large majority of validation studies only a small correlation with objective measures of fitness level was achieved (P. H. Lee, Macfarlane, Lam, & Stewart, 2011). Previous studies of $\dot{V}O_2max$ measurement between the submaximal and peak exercise, the deviation was found to increase gradually in $\dot{V}O_2max$ 10% for the predicted by Astrand-Rhyming in healthy young men (Keren, Magazanik, & Epstein, 1980). Moreover, there were a few studies

revealed that the submaximal tests for $\dot{V}O_2$max estimation by cycling ergometry were higher than $\dot{V}O_2$max in treadmills (Al-horani, 2019; Peric & Nikolovski, 2017; Storer, Davis, & Caiozzo, 1990). Then, in cycle ergometry tests should be regarded for reporting the final outcome and our outcome of estimate $\dot{V}O_2$max was possibly overestimated.

As the results in pre-experimental data in three visits, the low LF of baseline in visit 1 indicated that more abatement than later visits. This finding might be due to more nervous in the testing trials. However, there was no significant difference between control and SDB trails on resting BP, HR, and HRV, indicating a similar baseline of cardiac activation between trials.

Effect of MIIT exercise on oxygen consumption and heart rate

We proposed that MIIT can be the starting program for overweight and obese persons to increase energy utilization and exercise engagement. The intensity of exercise is a one factor affecting exercise engagement in which continuous high-intensity exercise results in a more negative affective response when compared to moderate- or low-intensity continuous exercise (Jung, Bourne, & Little, 2014). Therefore, MIIT could initiate more engagement to exercise, especially in overweight people. The first presentation of our study, we would like to verify the use of heart rate monitor to indicate the exercise intensity during MIIT exercise in overweight participants. It is well-accepted that $\dot{V}O_2$ and heart rate are linearly relationship. In the present study, linear relationship between them was also found by whole MIIT exercise and recovery periods. $\dot{V}O_2$ and heart rate increased during moderated-intensity exercise and declined during low-intensity exercise. A low to moderate duration ratio of 1:1 was sufficient to return both $\dot{V}O_2$ and heart rate to their stage levels. Interestingly, when $\dot{V}O_2$ and heart rate was specifically observed in each period, the rate of change was different between them, in which rates of HR decline were slower than the rate of $\dot{V}O_2$ decline. This finding might be due to abruptly reduce in muscle oxygen extraction based on Fick equation. Unfortunately, muscle oxygen extraction was not evaluated in this study. In addition, we

observed a greater increase in heart rate and $\dot{V}O_2$ in the second and third intervals of moderate exercise than in the first interval suggested more cardiorespiratory demand in the following intervals. This result suggested that MIIT protocol gradually increased intensity without intension. However, the increase intensity level upon MIIT exercise was in the upper range of moderated intensity exercise. Rate of perceived exertion at the last moderate interval also confirmed at the somewhat hard to very hard intensity range of 12-17 (based on Borg scale 6-20). Results suggested the MIIT protocol used in the study was sufficient to activate vast energy utilization and consequently reduce weight gain.

The effects of slow deep breathing on heart rate recovery

By addition of slow deep breathing during cool down, a faster rate of heart rate decline, as main outcome, was expected. The present finding supported that additional of SDB significantly enhanced the heart rate decline at 30 seconds and 1 minute after MIIT exercise as compared to the control trial. In contrast, an addition of SDB delayed HRR at the later phases (phase 6, 8, 10) as compared to the control trial. A previous report (Emily, 2019) showed that the continuous slow breathing at six breaths per minute increased HRR during 5 minutes of cool down.

The enhanced percentage of heart rate decline at the first minute due to slow deep breathing may result from increased parasympathetic reactivation. According to the phases of HRR, the fast phase comprises the first minute of recovery, showing an abrupt decrease in HR (Coote, 2010; Imai et al., 1994). This characteristic was promoted predominantly by cardiac vagal reactivation (Pecanha et al., 2014). It has been proposed that slow deep breathing boosts the parasympathetic nervous system via pulmonary reflex. Stretch receptors of the lungs generated inhibitory impulses to reduce neural action potentials and increased parasympathetic dominance (Jerath et al., 2006). With a greater increase in the time domain of HRV at the 1 minute in SDB trial than in control trial, performing in slow deep breathing potentially enhanced the vagal effect during the fast phase. However, we found that the increase in the frequency domain of HRV during slow breathing was strongly influenced by breathing frequency, which was

lower than 7 bpm. Data on the frequency domain of HRV would not be used for interpretation in this study. (More detail was discussed below.)

On the other hand, the lower HRR decline in the late phase of cool down (HRR6, 8 and 10) in the SDB trial suggested an ineffective sympathetic withdrawal after long-term slow deep breathing activity. The time period mentioned is characterized by a gradual decrease in heart rate recovery (HRR) after 2 minutes of exercise cessation, and the mechanisms rely on vagal reactivation and sympathetic withdrawal (Pecanha et al., 2014). Continuous slow deep breath performed during slow phase of cool down possibly resulted in higher respiratory muscle metabolism and limitation in the elimination of carbon dioxide. Carbon dioxide and waste retention could then activate the chemoreceptors and led to a greater sympathetic activity (Katayama & Amann, 2012). More specifically, the high accumulation of fat in the mediastinum and abdominal cavities in overweight and obese can lead to a decrease in respiratory compliance (Parameswaran et al., 2006). Hence, slow deep breathing performance while exhaustion after exercise was difficult to enhance enough tidal volume. Fatigue of respiratory muscles associated with CO_2 retention in this population (Chlif et al., 2007). Moreover, repeated deep inspiration affect thoracic pressure that might disturb baroreceptors activity and its reflex (Tzeng, Sin, Lucas, & Ainslie, 2009).

Notably, Although the impaired HRR was associated with higher BMI (Barbosa Lins et al., 2015), mean HRR during the first minute over 20 bpm in both control and SDB trials implied a normal cardiopulmonary response in all participants. The HRR that lower than 12 bpm at the first minute indicate the high risk of mortality (Cole et al., 1999; Maddox et al., 2008; Nishime et al., 2000).

The effects of slow deep breathing on sympathovagal balance

The mechanism underlying the effect of the slow deep breathing technique on heart rate recovery via sympathovagal balance was determined. It is well-accepted that the fast phase of heart rate recovery is predominantly promoted by cardiac vagal reactivation, while the rate of recovery at slow phase is alternately upheld by vagal reactivation and sympathetic withdrawal (Pecanha et al., 2014). A significant higher in

time domain of HRV in SDB trial as another main outcome in both fast and slow phases indicated an increase in parasympathetic to the heart. This finding is further supported by the work of Paprika and co-worker (2014), who reported that a breathing program with 6 breaths per minute in 3:7 inspiration to expiration ratio for 3 minutes significantly increased RMSSD when compared to the resting baseline (Paprika et al., 2014). Similar to the study of Remko Soer *et. al.* (2021) who demonstrated that breathing at a frequency of 5 to 7 breaths per minute for 2 minutes induced a highest HRV values, especially on the time domain (Soer, Six Dijkstra, Bieleman, Oosterveld, & Rijken, 2021). Systematic review also concluded that the voluntary slow breathing induced a significant increase in parasympathetic activity (Laborde et al., 2022). These reports support our finding that additional of SDB during cool down helped fasting heart rate return especially in the fast phase by enhancing parasympathetic activation.

Mechanism underlying the increase in parasympathetic activation by slow breathing was interesting. It has been reported that autonomic responses were favorable to vagal modulation with the lower breathing rate maneuvers (De Souza et al., 2018). Slow deep breathing slowly activates stretch receptors of the lungs at above tidal inhalation volume and then generates inhibitory impulse as the Hering–Breuer reflex. The neural inhibitory signals from respiratory neurons links to the autonomic nervous system, increasing parasympathetic dominance (Jerath et al., 2006; Russo et al., 2017). In addition, it has been suggested that SDB increases the perturbation of intrathoracic pressure (more negative pressure during inspiration) and venous return, which is associated with larger fluctuations in blood pressure (BP) and, consequently increasing stimulation of the baroreceptors located in the wall of the aortic arch (Bernardi et al., 2002). Therefore, these two potential mechanisms significantly enhanced parasympathetic activation.

Interestingly, the frequency domain of HRV fluctuated due to slow deep breathing. As found in Figure 24C, the ratio of low frequency to high frequency in normalized units (LF/HF ratio) in SDB trial significantly rose in every SDB interval. This finding is particularly significant as it suggests that deep breathing with 6 breaths/min can generate a resonant frequency at 0.1 Hz, known as coherence breathing (Nagarajan,

2022; Sasaki & Maruyama, 2014). Moreover, breathing at 0.1 Hz as low frequency rate (LF) reduces the high frequency rate (HF) of HRV and increases the LF/HF ratio. The LF/HF ratio is a measure of the balance between the sympathetic and parasympathetic nervous systems, with a higher ratio indicating a shift towards sympathetic dominance. However, it's important to note that these results may lead to the incorrect hypothesis of vagal and sympathetic nerve activity (Sasaki & Maruyama, 2014). Angelone and Coulter in 1964 demonstrated that respiratory rate reduction produced maximal HRV amplitude (Angelone & Coulter, 1964). The HRV synchronization with the respiration stages, by which R-R intervals of ECG are reduced during inspiration and extended during expiration and this HRV is called respiratory sinus arrhythmia (RSA) (Berntson, Cacioppo, & Quigley, 1993; Yasuma & Hayano, 2004). Moreover, about 6 breaths per minute has been confirmed that this frequency creates the maximization of RSA (Ben-Tal, Shamailov, & Paton, 2014; T. E. Brown, Beightol, Koh, & Eckberg, 1993) and baroreflex sensitivity (Bernardi, Gabutti, et al., 2001; Radaelli et al., 2004; Vaschillo, Vaschillo, & Lehrer, 2006). The breathing at 0.1 Hz referred to resonant frequency effect (Angelone & Coulter, 1964; Vaschillo et al., 2006) and RSA also resonates with the LF baroreflex integration frequency (Julien, 2006; Russo et al., 2017). According to these evidences, we implied that high LF/HF

Previous study suggested that the increased LF component during conscious controlled slow respiration was highly influenced by mental activities leading to the inhibition of parasympathetic nerve activity (Sasaki & Maruyama, 2014). The breathing voluntary with imposed breathing frequency creates the spectral energy affecting autonomic nervous system. In this situation, the LF/HF ratio does not faithfully explain the spontaneous sympathovagal balance. The mechanical effect of the controlled breathing frequency may cause misinterpretation similar to those changes in physiological fatigue or overtraining syndrome (Saboul, Pialoux, & Hautier, 2014). This mental effect and muscle exhaustion might be increased after repeated SDB leading to a significantly high LF at the late phase of cool down, which associated with a lower rate of HRR, in SDB trial. This mental situation could lead to sympathetic dominance. However, the effect of

SDB on sympathovagal activity was disappeared immediately after stop as demonstrated during recovery. These HRV findings suggested that change in time-domain of HRV is more associated with the potential mechanism of SDB on HRR.

Strength and limitation

The strength of this study was the first investigation in intermittent training combined with slow deep breathing for recovery booster. The crossover study in which the heart rate recovery analysis was gone together with mechanism via heart rate variability analysis. According to these present results, slow deep breathing at cool down stage appears to influence autonomic nervous system and then heart rate recovery. Slow deep breathing promoted fast heart rate recovery and parasympathetic activation enhanced at the early minute.

Lacking of minute ventilation value measurement during SDB is a major limitation of the present study. A significant increase in spontaneous breathing frequency in between SDB intervals during cool down was obviously observed in all participants. Therefore, it was difficult to estimate whether alveolar ventilation during cool down was equal between control and additional SDB trials. The difference between alveolar ventilation and then partial pressure of blood gases highly influences cardio-autonomic activation (Botek, Krejčí, De Smet, Gába, & McKune, 2015). Unfortunately, gas analyzer measurement could not be performed during experimental trials. The mask and mouthpieces caused difficult breathing due to an increase in airway resistance especially in obese person. Additionally, the use of uncomfortable mouthpieces could affect heart rate variability results by mental stress (Bernardi et al., 2000). Previous study also reported that HRV was influenced by the dead space created by using a face mask (Furutani et al., 1997). Based on technical burden, oxygen consumption and respiratory parameter measurements were not performed in the experimental trials.

Implication for practice in overweight and obesity

In agreement with changes in autonomic nervous system observed in overweight and obesity, there was a decrease in cardiac parasympathetic activity, which was

associated with high visceral adiposity (Yadav et al., 2017). Additionally, the heart rate recovery (HRR) following aerobic exercise was negatively correlated with body mass index (BMI) (Rahul et al., 2020). Result of present study showed the high percentage of heart rate decline and the improvement in time domain of HRV in fast phase of HRR of slow breathing trials compared to control. This finding recommended that only a few minutes of slow deep breathing is suitable enough to activate parasympathetic activation duration for recovery in overweight and obese young adults. Slow deep breathing can induce stretch receptors of the lungs. Subsequently, the inhibitory impulses produced in brain tissue decrease action potentials to enhance parasympathetic dominance (Jerath et al., 2006). However, slow deep breathing at slow phase of HRR could produce adverse effects. The conscious controlled slow respiration was led to the inhibition of parasympathetic activity by mental activities (Sasaki & Maruyama, 2014). This situation disturbed vagal reactivation plus sympathetic withdrawal and led to delayed HRR during late cool down. Therefore, slow deep breathing after exercise at 6 breaths/min with inspiration 4 sec and expiration 6 sec, total time less than 2 minutes is suitable breathing program for recovery booster in overweight and obesity class I.

Summary and further studies

Heart rate recovery rate and parasympathetic activation were significantly increased at the fast phase recovery by the additional of slow deep breathing session upon cooling down. However, additional of slow deep breathing had no any benefit during the late cool down stage. The present study further improved the understanding on the relationship between breathing pattern and autonomic nervous response in which the controlled deep and slow breathing increases sympathetic activity predominance. In consideration, many slow deep breathing technique variations have been introduced for athletes. Further studies regarding these breathing variations affecting on physiological response are definitely requested.

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