

# 行政院國家科學委員會專題研究計畫 期末報告

呼吸訓練對併有憂鬱症狀之停經婦女之成效探討(第3年)

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計畫主持人：蔡佩珊  
共同主持人：王美業、邱震寰、許淳森  
計畫參與人員：碩士級-專任助理人員：潘潔馨  
學士級-專任助理人員：顏嘉瑩

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中文摘要：目的：本研究旨在探討竇性心律不整生物回饋引導之呼吸訓練對於改善停經後憂鬱婦女憂鬱症狀與自主神經功能之成效。

方法：本研究招募 45 至 64 歲自然停經滿 12 個月且貝克憂鬱量表 (BDI-II) 得分 10 分 (含) 以上的婦女參與研究。個案經隨機分派至實驗組與對照組。實驗組接受一週一次、每次一小時、為期八週以竇性心律不整生物回饋引導之呼吸訓練課程，而對照組會在相同時間點接受壓力管理訓練課程以作為控制。主要結果測量包括憂鬱症狀 (BDI-II) 及自主神經功能 (以功率頻譜分析之心跳變異性)。個案在第 0 (治療前)、9 (第一次後測) 及 16 週 (第二次後測) 接受測量，以重複測量變異數進行資料分析。

結果：共有 81 位 45~64 歲的研究對象 (平均年齡 56.49 歲) 納入分析。BDI-II 得分平均為  $21.16 \pm 11.13$  分 (範圍 10~57 分)。重複測量變異數結果顯示，接受竇性心律不整生物回饋引導之呼吸訓練個案，其憂鬱症狀的改變幅度與控制組的改變幅度間無顯著差異，亦無時間與組別間的交叉效應；自主神經功能參數隨時間的改變量亦無顯著的組間差異或時間與組別間的交叉效應。然而實驗組的憂鬱症狀與前測值相較，在訓練後一週 ( $p < 0.001$ ) 及八週 ( $p < 0.001$ ) 均顯著的下降。接受呼吸訓練之婦女其憂鬱症狀降低程度與心跳速率應激反應的改善程度有顯著相關 ( $r = .35, p = .039$  及  $r = -.37, p = .030$ )。

結論：竇性心律不整生物回饋引導之呼吸訓練對於改善憂鬱症狀及自主神經功能並未能優於壓力管理教育，但在竇性心律不整生物回饋引導之呼吸訓練完一週及八週後均可有效改善停經婦女的憂鬱症狀。接受呼吸訓練之停經婦女其憂鬱症狀的改善可能與自主神經功能的改善有關。

中文關鍵詞：停經、憂鬱症狀、自主神經功能

英文摘要：Purpose: To examine the effects of respiratory sinus arrhythmia (RSA) biofeedback-assisted breathing training on depressive symptoms and autonomic functioning in postmenopausal women with depressive symptoms.

Methods: Participants, aged from 45 to 64, reporting cessation of menstrual cycles with natural causes for more than 12 consecutive months and scored 10 or greater on the Beck Depression Inventory-II (BDI-II) were eligible for participation. Participants were randomly assigned to either an experimental or an

active control group. Participants assigned to the experimental group underwent 8 one-hour weekly RSA-biofeedback assisted breathing training sessions. Participants randomized as controls underwent stress management training during the same time period for controlling the attention effect. Primary outcomes included depressive symptoms assessed using the BDI-II and autonomic function determined by power spectral analysis of heart rate variability (HRV). Outcomes were assessed at Week 0 (pretest), 9 (posttest 1), and 16 (posttest 2). Data were analyzed using Repeated Measures Analysis of Variance (ANOVA). Results: Eighty-one participants aged from 45 to 64 with a mean of 56.49 years were analyzed. The mean BDI-II score, ranging from 10 to 57, was  $21.16 \pm 11.13$ . Repeated measures ANOVA revealed that the magnitude and pattern of changes in depressive symptoms over time from baseline to posttest were not significantly different between the group receiving RSA biofeedback-assisted breathing training and the group receiving the control condition. Similarly, the magnitude and pattern of changes in autonomic parameters over time were not significantly different between groups. The decrease in the severity of depressive symptoms is significantly associated with the improvement in autonomic functioning as determined by HR stress reactivity in postmenopausal women who receive breathing training ( $r = .35$ ,  $p = .039$  and  $r = -.37$ ,  $p = .030$ , respectively). Conclusion: RSA biofeedback-assisted breathing training is not better than the control condition (i.e., stress management education) for improving depressive symptoms and autonomic function. Nevertheless, RSA biofeedback-assisted breathing training is effective in reducing depressive symptoms 1 week and 8 weeks after completion of training. The improvement in depressive symptoms associated with breathing training in postmenopausal women may be related to the improvement in autonomic function.

英文關鍵詞： postmenopause, depressive symptoms, autonomic functioning



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## **INTRODUCTION**

Menopause is the permanent cessation of menstruation which is caused by the natural or surgical termination of hormone production by the ovaries <sup>1</sup>. For women with severe menopausal symptoms, not only their personal, social functioning, and quality of life may be deeply affected <sup>2</sup>, the presence of menopausal symptoms also increase the likelihood of poor health-related quality of life and healthcare resource utilization <sup>3</sup>. The Department of Health, Executive Yuan, R.O.C. indicated that heart and hypertensive diseases were the 2<sup>nd</sup> and 6<sup>th</sup> leading causes of death among women in 2011 <sup>4</sup>. Thus, menopausal symptoms may act as an important role related to menopause and cardiovascular morbidity and mortality.

Menopausal women experience not only physical changes but also such a psychological problem as depression. Depressed mood may also be associated with a history of depressed mood earlier in life, a longer menopause transition, or severe menopause related symptoms such as hot flashes, and other causes of mood disturbances include thyroid disorders, medication side effects, and life stresses <sup>5,6</sup>. Depressive symptoms are related to altered autonomic function <sup>7,8</sup> which has prognostic importance in cardiac events <sup>9</sup>. To maximize the improvement of health outcomes in postmenopausal women with depressive symptoms, interventions that target at both depression and autonomic functions are imperative. Previous studies suggested that a treatment-related improvement in cardiac autonomic function among depressed patients could play a major role in reducing cardiac risk <sup>10</sup>. Accordingly, the development of a training protocol that targets on depressive symptoms and autonomic functioning is of clinical importance to postmenopausal women with depressive symptoms.

## **AIMS OF THE STUDY**

The specific aims of this study are:

1. To develop a breathing training protocol specifically designed to improve HRV and psychosocial functioning for postmenopausal women with depressive symptoms,
2. To examine the immediate effects of an 8-week breathing training program on HRV and psychosocial end points in postmenopausal women with depressive symptoms,
3. To examine the intermediate-term effects of an 8-week breathing training program on HRV and psychosocial end points in postmenopausal women with depressive symptoms, and
4. To determine whether the change in depressive symptoms with breathing training in postmenopausal women is associated with the change in HRV.

## **SIGNIFICANCE OF THE STUDY**

Postmenopausal women without hormone replacement therapy are associated with higher risk of cardiac morbidity and mortality. They are likely to experience depressive symptoms after menopause, and the comorbidity of depression are related to altered autonomic function, as determined by spectral analysis of HRV, which have prognostic importance in cardiac events. Heart rate variability is one of the most widely used methods for measuring cardiac autonomic activity, and cost-effective and non-invasive procedures with standardized measurements are available. Treatments of depression or improvement in depressive symptoms contribute to enhanced heart rate variability. Breathing

technique is a trainable and convenience approach to help deal with depressive symptoms. We expect that postmenopausal women with depressive symptoms who receive breathing training will demonstrate decreased depressive symptoms, general psychological distress and increased heart rate variability immediately upon and later after completion of training, and optimally contributing to improve autonomic nervous regulation in their later life to prevent undesired cardiac outcomes.

## **LITERATURE REVIEW**

### Cardiovascular Mortality and Morbidity in Postmenopausal Women

According to the North American Menopause Society (NAMS), postmenopause is defined as the span of time after menopause (the final menstrual period) which can be confirmed after going 12 consecutive months without a period<sup>11</sup>. Menopause is the permanent cessation of menstruation which is caused by the natural or surgical termination of hormone production by the ovaries. Women experience various symptoms caused by changes in the endocrine and autonomic activity starting from the perimenopausal period, and then extending to the postmenopausal periods. Many cardiovascular diseases, such as high blood pressure, myocardial infarction, coronary artery disease, are known to increase in women during menopause and postmenopause than premenopause when the female hormone acts as a cardio-protective effect in women's lifespan<sup>11,12</sup>. Although heart diseases are more prevalent in men than in women, cardiovascular diseases are the number-one killer of women in North America. A woman's risk for heart disease increases after menopause regardless of her age, and after age 55, more than half of all deaths in women are caused by cardiovascular disease<sup>11</sup>.

### Depression in Postmenopausal Women

Postmenopausal women experience not only physical changes but also such a psychological problem as depression. Few scientific studies support the belief that menopause contributes to true clinical depression; however, some perimenopausal and postmenopausal women did report symptoms of depressed mood such as tearfulness, mood swings and feeling blue or discouraged<sup>6,11</sup>. Depressed mood may also be associated with a history of depressed mood earlier in life, a longer menopause transition, or severe menopause related symptoms such as hot flashes, and other causes of mood disturbances include thyroid disorders, medication side effects, and life stresses<sup>5,6,11</sup>. Therefore, clinical depression may not be caused by menopause, but it is understandable how they can arise or get worse during this period. In the United States, the prevalence of depressive symptoms was approximately 15.8% in the Women's Health Initiative Observational Study that followed up 93676 postmenopausal women with multiethnic groups<sup>12</sup>. These data suggest that depressive symptoms are common in postmenopausal women and may be associated with reduced quality of life. Hence, the management of depressive symptoms becomes an important issue among this group.

### Depression and Autonomic Dysfunction

Depression is significantly related to risk factors for cardiovascular morbidity and mortality<sup>7,10,12,13</sup>. Wassertheil-Smoller (2004) also found that there was a 50% increase in risk of cardiovascular disease-related deaths associated with depression among women with no previous cardiovascular disease history after adjusting for age, race, education and income. Although studies suggested that depression might be an independent predictor of cardiac morbidity and mortality after controlling for several major cardiac risk factors, such as hypertension, smoking, diabetes and reduced exercise

capacity; however, depression might also potentiate the effects of other cardiac risk factors<sup>7,14</sup>.

Many studies found that the autonomic reflex was impaired with decreased parasympathetic and increased sympathetic controls in either depressed patients with/without cardiovascular disease or in postmenopausal women<sup>15</sup>. HRV analysis is an inexpensive, noninvasive technique and most widely used methods for studying cardiac autonomic tone. Beat-to-beat variability in the heart's rhythm is under the control of the autonomic nervous system (ANS)<sup>9</sup>. Time domain measures of HRV are assessed with calculations based on statistical operations on R-R intervals, and frequency domain measures use spectral analysis of a sequence of R-R intervals and provide information on how power (variance) is distributed as a function of frequency<sup>9,16</sup>. In patients with cardiovascular diseases, autonomic dysfunction, as evidenced by decreased HRV, is a risk factor for negative cardiovascular events and mortality<sup>9</sup>. Moreover, depression also appears to be strongly associated with decreased HRV, which in turn can affect cardiovascular regulation<sup>17,18</sup>. Therefore, it is reasonable to speculate that impaired autonomic function may explain the link between depression and risk of cardiovascular events and mortality. Thus, employing strategies to manage depression or depressive symptoms in postmenopausal women may help decrease the risk of cardiovascular diseases and/or mortality in this population.

#### Impact of Pharmacological and Non-pharmacological Treatment of Depression on HRV

To maximize the improvement of health outcomes in postmenopausal women with depressive symptoms, interventions that target at both depression and autonomic functions are imperative. There are numerous treatments for depression. The options include antidepressant drugs, cognitive behavioral therapy (CBT) and physical activity such as aerobic exercise and cardiac rehabilitation<sup>19</sup>. Many treatments for depression have been studied in relation to their effect on heart rate and heart rate variability. According to the recommendations for treatment of depression and coronary heart disease (CHD) endorsed by the American Psychiatric Association, antidepressant use has been associated with both increased and decreased cardiac risk. Selective serotonin reuptake inhibitor (SSRI) antidepressants are safe for patients with CHD and effective for depression, but tricyclic antidepressants (TCA) and monoamine oxidase inhibitors are contraindicated for many patients with heart disease because of their cardiotoxic side effects<sup>8,19</sup>. Findings regarding the effects of SSRI antidepressants, cyclic antidepressants or other antidepressant/stress-reducing pharmacotherapies on the changes of HRV were found to be inconsistent across studies<sup>19-21</sup>.

For non-pharmacological treatment, CBT is a scientifically well-established and effective treatment for depression. It may be an alternative for cardiac patients who cannot tolerate antidepressants or who prefer non-pharmacological treatment<sup>19,20</sup>. CBT may benefit depression from the post-treatment and the follow-up evaluations<sup>20</sup>, but there is little evidence of RCTs showing that treatment of depression would improve cardiac events or mortality<sup>19,22</sup>. Exercise training is associated with several beneficial physiologic changes such as improvements in autonomic nervous system<sup>22</sup>, and a study concluded that exercise and stress management training reduced emotional distress and improved markers of cardiovascular risk as compared to usual medical care alone for patient with stable ischemic heart disease<sup>23</sup>. It must be born in mind that the prescription of exercise needs to be individually assessed based on the individual's medical status and exercise capacity<sup>19</sup>. One study has found that Sudarshan Kriya Yoga (SKY) effective in the treatment of depression<sup>24</sup>. Many breathing

and relaxation trainings sprouted in this decade. De Meersman, *et al.* (2007) suggested that a treatment-related improvement in cardiac autonomic function among depressed patients could play a major role in reducing cardiac risk.

In a quantitative review related to the effects of antidepressant treatment on HRV in major depression<sup>25</sup>, findings revealed that TCAs were associated with a large decrease in HRV and increase HR, but treatment effects with SSRIs were variable. Another study found that treatment of depression with CBT in patients with CHD was associated with decreased HR and increased short-term HRV<sup>20</sup>.

#### Effect of Breathing Training on Psychological Functioning

There are many integrated breathing and relaxation exercises implemented to treat various psychiatric, somatic and psychosomatic diseases<sup>26,27</sup>. When performing abdominal breathing, also named diaphragmatic breathing, individuals are trained to take deep, even and steady breaths, using diaphragm, with minimum possible movement of the chest, minimizing the pause between the breaths in supine position with the hands along the sides of the body, palms upward and legs slightly apart. Kaushik, *et al.* (2005) conducted a randomized control trial of diaphragmatic breathing combined with systematic relaxation in individuals with migraine and found that there was a significantly long-term prophylactic effect than propranolol in migraine headache. Holloway *et al.* (2007) integrated breathing and relaxation training for adults with asthma in a randomized control trial and found that anxiety and depression scores were reduced to a clinically meaningful degree after training.

#### Effect of Breathing Training on HRV

Slow breathing enhanced baroreflex sensitivity, and sympathetic activity was increased with faster breathing rate in patients with congestive heart failure<sup>28</sup>. HRV biofeedback with paced breathing and brief cognitive-behavioral training was found to enhance vagal heart rate regulation after stress countering<sup>21</sup>. A breathing training combined with biofeedback technique has been used to improve HRV in medical conditions, such as depression, CHD, asthma, hypertension, and fibromyalgia<sup>21,29-33</sup>. In a study of the effect of HRV biofeedback on depression, participants were instructed to breathe deeply and slowly at their resonant frequency and the results showed that depressed patients significantly reduced anxiety, decreased heart rate and increased HRV after HRV biofeedback training as compared with those receiving active control condition<sup>33</sup>. In an open label study<sup>31</sup> of HRV biofeedback with breathing at one's resonant frequency for the treatment of major depression, significant improvements of depressive symptoms and HRV parameters were noted. Patients receiving HRV biofeedback reduced symptoms of psychological stress and depression, and this improvement was associated with enhanced vagal HR modulation<sup>21</sup>. Another study<sup>32</sup> found that the pursed-lips abdominal breathing training can obliterate the effects of age on HRV changes during biofeedback in asthma patients. Taken together, abdominal breathing training might be beneficial in improving autonomic function. Thus, the development of a training protocol that targets on depressive symptoms and autonomic function is of clinical importance to postmenopausal women with depressive symptoms.

## **METHODOLOGY**

### Study Design

In this prospective randomized controlled study, participants will be randomly assigned to either

an intervention group or a control group using computer generated permuted block randomization schedule.

### Study Participants

This study will include postmenopausal women who meet the inclusion and exclusion criteria specified as follow.

*Inclusion criteria:* (1) Permanent termination of menstruation of natural cause. (2) Cessation of menstrual cycles for more than 12 consecutive months. (3) A score of the Chinese version of Beck Depression Inventory-II (BDI-II) of greater than 10. (4) Able to speak Mandarin or Taiwanese. (5) Age from 45 to 64 years.

*Exclusion criteria:* (1) Subjects who are clinically diagnosed with history of cardiac arrhythmia, coronary heart disease, heart failure, kidney disease, hypertension, chronic low blood pressure, diabetic neuropathy, psychosis, mental deficiency. (2) Subjects who received hormone replacement therapy prescribed by gynecological physicians. (3) Subjects who took cardiac and/or psychotropic medications which may affect the autonomic functions.

### Study Participant Recruitment and Screening

Subjects will be referred from 3 outpatient departments (family medicine, gynecology, and psychiatry) in one University-affiliated hospital and 1 psychiatry clinic in northern Taiwan. Subjects will also be recruited by dissemination of flyers advertising this study. Potential subjects will be screened for eligibility by a trained interviewer. Approval from the institutional Human Subjects Committee will be obtained before implementing the study. Written informed consent will be obtained at interview.

### Protocol of the Assessment Session

The autonomic function both in the basal state and in response to psychological stressors (i.e., ANS reactivity), severity of depressive symptoms, and general psychological distress will be assessed at week 0, 9, and 16. Before and during the assessment sessions, nicotine, alcoholic and caffeinated beverages will be strictly forbidden. Participants will be advised to arrive at the study site at 1:00 PM. They will fill out the BDI-II and the Chinese Health Questionnaire-12 (CHQ-12) to assess depressive symptoms and psychological distress, respectively. Participants will then be asked to rest for 10 minutes in a reclining position. Toward the end of this resting period, 10-minute HRV measurements will be recorded to serve as the baseline value. The rest period will be followed by 2 stress tasks during which power spectral analysis of HRV will be employed in order to assess ANS reactivity.

Following the stress tasks, participants will be asked to rest in a reclining position during a recovery period lasting for 10 minutes. HRV recordings will also be made during the recovery period.

### Stress Tasks

Two acute time-limited stressors (the Stroop color and word test and the mirror star tracing task) will be administered consecutively to all study participants during each of the 3 assessment sessions.

### Protocol of Intervention Sessions

#### *Experimental group: Respiratory sinus arrhythmia breathing training*

Respiratory sinus arrhythmia breathing training is provided once a week in eight individual 1-hour sessions. The participants will be advised to avoid taking any caffeine or alcohol for at least 12 hrs prior to all sessions included assessment ones.

Participants will be trained by a therapist to take deep, even and steady breaths, using diaphragm, with minimum possible movement of the chest, minimizing the pause between the breaths in a semi-recumbent position at a frequency of 6 breaths per minute. Correctness of the breathing techniques will be ensured by using a Procomp Biograph Infiniti Biofeedback System (Thought Technology, Canada).

Home exercises with an instruction paper sheet containing the above-mentioned reminders of the breathing and relaxation techniques will be supplied at the first treatment session. The participants will be sent home with a pulse/breathing rate monitor (StressEraser) and asked to practice relaxation daily and to use breath monitoring and regulation for a 20-minute period twice a day. The StressEraser device also measures photoplethysmographic blood volume pulse and identify every pulse the moment it occurs.

#### *Control group: stress management education*

Participants in the control group then will receive eight once a week, one hour per session, education sessions over an 8-week period to enhance psychological well-being and to modify symptoms of stress.

#### Measurements

##### *Severity of depressive symptoms*

The Chinese version of Beck Depression Inventory-II (BDI-II) will be used to assess the depressive level of participants. The reliability and validity of the BDI-II have been tested and demonstrated good internal consistency with Cronbach's  $\alpha=.94$ , spilt-half=.91.

##### *General psychological distress*

The factor structure of the Chinese Health Questionnaire (CHQ) includes somatic symptoms, anxiety and worrying, social dysfunction, and depression and poor family relationship. The internal consistency of CHQ was investigated in 2 samples in Taiwan, with its Cronbach's alpha coefficient of 0.84 and 0.83 for the 12-item.

##### *Heart rate variability (HRV)*

Electrocardiogram (ECG) signals will be recorded under standardized conditions in lead II and simultaneously sampled at a sampling rate of 500 Hz using a data acquisition device (model MP100, Biopac Systems, Inc., Goleta, CA) and a Biopac System ECG 100C preamplifier. The ECG signals will be processed using an HRV analysis system (Nevrokard version 6.8.0, Slovenia). Frequency domain analysis will be performed based on the Fast Fourier Transformation algorithm.

#### Study Procedure

Demographic information, medical, reproductive, family history, and personal habits will be collected at baseline (week 0). An assessment session as mentioned earlier will also be administered at baseline to collect HRV, BDI-II, and CHQ-12. Subjects assigned to the intervention group will undergo breathing training once a week for 8 weeks (weeks 1 to 8). Subjects randomized as controls will undergo a stress management educational program during the same time period for controlling the attention effect. In order to examine the immediate and intermediate-term effects of the breathing training program on HRV and psychosocial end points in postmenopausal women with depressive symptoms, the assessment session will be repeated at week 9 and 16 follow-ups.

#### Statistical analyses

Continuous variables were presented as mean values and standard deviation. Categorical variables were presented as frequencies and percentages. Chi-square and *t* tests were used to compare the differences in demographic variables between groups. Paired *t*-test was used to examine the differences between baseline and post-treatment values. Person's correlation was used to examine the association between the changes in depressive symptoms and autonomic functioning. The Predictive Analytic Software (PASW) 17.0 was used for all analyses. A statistical significant level was set at  $p < 0.05$ .

## RESULTS

### Baseline Characteristics

#### *Demographics*

A total of 98 postmenopausal women with depressive symptoms, aged 45 to 64 (mean = 56.46, S.D. = 4.13), participated in the study and were assessed at Week 0 (pretest). The majority women enrolled in this study received higher education (54.10%), were married (76.50%), unemployed (63.30%), non-smokers (96.3%), non-exercisers (55.6%), and had never used of antidepressants (98.00%). The bio-demographic data of the study participants at baseline was presented in Table 1.

#### *Severity of depressive symptoms*

Descriptive analyses related to BDI-II score were shown in Table 2. The mean BDI-II score, ranging from 10 to 57, was  $20.53 \pm 10.55$  with inter-quartile range 12.00 to 26.00. There were then 27 participants (27.60%) grouped into the low score group ( $BDI-II \leq 12$ ) and 25 participants (25.50%) grouped into the high score group ( $BDI-II \geq 26$ ). Based on the cutoffs suggested from the Chinese BDI-II instruction manual, 31 participants (31.60%) were evaluated with minimal depression, 23 participants (23.50%) with mild depression, 27 participants (27.60%) with moderate depression and 17 participants (17.30%) with severe depression. Among them, 67 participants (68.40%) were grouped into subclinical depression.

### Comparisons between experimental group and control group

#### *Demographics*

The flow chart presented in Figure 1 showed the number of women screened, eligible for the study, and the refusals and reasons. Eighty-one (87.10%) participants, ranging from 45 to 64 years of age (mean =  $56.49 \pm 4.15$ ), completed the study and were analyzed. The baseline demographic characteristics among study completers were shown in Table 3.

#### *Psychological endpoints*

The mean BDI-II score, ranging from 10 to 57, was  $20.91 \pm 10.75$ . The mean CHQ-12 score, ranging from 0 to 12, was  $4.68 \pm 3.41$ . Results of BDI-II scores in different assessment weeks among the experimental group and the control group were summarized in Table 4. The experimental group demonstrated moderate depressive symptoms at baseline (Week 0) and minimal depressive symptoms at the first posttest (Week 9) and the second posttest (Week 16) (mean scores of BDI-II:  $20.34 \pm 9.73$ ,  $12.71 \pm 8.51$  and  $10.20 \pm 8.31$ , respectively), and the control group did as well (mean scores of BDI-II:  $21.35 \pm 11.56$ ,  $9.98 \pm 6.33$  and  $8.33 \pm 6.29$ , respectively). Table 4 and Figure 2 showed that the BDI-II scores reduced significantly from Week 0 to Week 9 and from Week 0 to Week 16 in both the experimental group and the control group (all  $p < 0.001$ ).

### *Autonomic functioning*

Results of all autonomic parameters in different assessment weeks among the experimental group and the control group were summarized in Table 4. There were no group differences on all autonomic functioning in either Week 0, Week 9 or Week 16 except LF/HF and lnLF both at Week 9 ( $p = 0.013$  and  $p = 0.038$ , respectively). The paired  $t$ -test revealed that LF/HF increased significantly from Week 0 to Week 9 ( $p = 0.013$ ) in the experimental group. There were also significant differences in the changes of LF/HF from Week 0 to Week 9 between groups where LF/HF in the control group decreased more compared to the experimental group (mean differences =  $-0.26 \pm 1.71$  and  $1.10 \pm 2.49$ , respectively;  $p = 0.007$ , 95% CI = 0.38~2.35).

### Stress tasks stimulation

#### *Stress Reactivity (mean task values – resting values)*

Results of cardiovascular reactivity from stress tasks among groups were summarized in Table 5. There were no group differences on all autonomic functioning in either Week 0, Week 9 or Week 16 except LF/HF at Week 16, lnHF at Week 16 and lnTP at Week 9 ( $p = 0.015$ ,  $p = 0.040$  and  $p = 0.046$ , respectively). The paired  $t$ -test revealed that stress reactivity on lnTP decreased significantly from Week 0 to Week 9 and Week 16 ( $p = 0.006$  and  $p = 0.003$ , respectively) in the experimental group. Stress reactivity on HR also decreased significantly from Week 0 to Week 9 ( $p = 0.012$ ) and from Week 0 to Week 16 ( $p = 0.012$ ) in the control group.

#### *Stress Recovery (mean recovery values – resting values)*

There were no group differences on all autonomic functioning in Week 0, Week 9 and Week 16 (all  $p > 0.05$ ).

#### *Association between the changes in depressive symptoms and cardiovascular reactivity measures*

For depressive symptoms, the changes on BDI-II scores from Week 0 to Week 16 were positively correlated with HR stress reactivity from Week 0 to Week 16 ( $r = 0.35$ ,  $p = 0.039$ ) in postmenopausal women who received breathing training. There was no association between the changes in depressive symptoms and any cardiovascular reactivity measures from Week 0 to Week 9 between the experimental group and the control group (all  $p > 0.05$ ). There were no associations between the changes on BDI-II scores and any spectral analysis of HRV measures from Week 0 to Week 16 between the experimental group and the control group (all  $p > 0.05$ ), neither.

## **DISCUSSION**

This study aimed to examine the immediate and intermediate-term effects of an 8-week RSA biofeedback-assisted breathing training on depressive symptoms and autonomic functioning in postmenopausal women with depressive symptoms. Results from this study showed that postmenopausal women in Taiwan represented moderate depressive symptoms. We also found that depressive symptoms significantly reduced 1 week and 8 weeks after completion of breathing training, and the control group did as well. However, there was no significant improvement on autonomic functioning 1 week and 8 weeks after completion of the breathing training, neither the resting values nor the stress responses.

Although menopause does not contribute to depression directly, the comorbid depressive symptoms were prevailed in postmenopausal women. Our finding revealed that not only the

experimental group but the control group also demonstrated declined severity of depressive symptoms from moderate to minimal levels immediately and intermediately after completion of treatment sessions. Depression might be an independent predictor of cardiac morbidity and mortality, and it might also potentiate the effects of other cardiac risk factors<sup>7,14</sup>. If postmenopausal women with severe depressive symptoms are not carefully screened and intervened, the comorbid depressive symptoms may deteriorate into true clinical depression and in turn aggravate an increased risk on cardiovascular diseases. Our breathing training is a feasible intervention for healthcare provider to apply for alleviating depressive symptoms in postmenopausal women with depressive symptoms due to the contribution of obviously reduction on BDI-II scores in our findings. Likewise, stress management conducted in the control group might also produce psychological treatment effects in women with depressive symptoms.

HRV is an important indicator of both how the central nervous system regulates the autonomic nervous system and of how peripheral neurons feed information back to the central level<sup>9</sup>. Reduced HRV seems to indicate the decreased cardiac vagal tone and elevated sympathetic activity in depressive patients<sup>17,34</sup>. We examined the effects of 8-week RSA biofeedback-assisted breathing training on the autonomic functioning as determined by power spectral analysis of HRV in postmenopausal women with depressive symptoms, and the results revealed that no significant improvement in resting HRV or HRV reactivity were found 1 week and 8 weeks after completion of breathing training. Previous study reported that a greater reduction in parasympathetic nerve activity as determined by HF-HRV to a psychological stressor from baseline was found in postmenopausal women<sup>35</sup>. A lower HF and higher LF and LF/HF were also observed in postmenopausal women as compared to premenopausal women<sup>36</sup>. This indicated that estrogen may play an important role in autonomic regulation in middle-aged women. However, results of this study did not concur with results of previous studies reported. Although LF/HF in the experimental group increased significantly 1 week after completion of training, it was not that worse as compared to the norm due to large standard deviation. The participants included in this study were healthy and free from cardiovascular diseases, and the resting autonomic functioning was not impaired. It should be noted that we did not evaluate menopausal symptoms experienced by postmenopausal women with depressive symptoms. It has been reported that LF/HF was significantly higher in women with postmenopausal symptoms as compared with asymptomatic women<sup>37</sup>. Detailed mechanisms linking menopausal symptoms and HRV warrant further exploration.

Cardiovascular reactivity refers to the rise in cardiovascular parameters when exposed to stress stimulation, and greater cardiovascular reactivity is one manifestation of impaired ANS activity<sup>38</sup>. This study examined HR and HRV to laboratory stress tasks to explore the association between depressive symptoms autonomic nerve regulation in postmenopausal women with depressive symptoms who received breathing training. Results showed that our breathing training did not improve HR and HRV reactivity neither 1 week nor 8 weeks after completion of training. Cardiovascular recovery could provide more information than cardiovascular reactivity<sup>39</sup>. Our results did not demonstrate improved HR and HRV recovery 1 week and 8 weeks after completion of training. Previous studies have reported the association between depressed mood and cardiovascular reactivity; however, the results were inconsistent. One investigation reported a positive association between

depressed mood and HR reactivity<sup>40</sup> while the others exhibited a negative association between depressed mood and HR reactivity and HR recovery<sup>39,41</sup>. To determine the mechanisms of postmenopausal depression, our study examined the association between the change in depressive symptoms and autonomic functioning with breathing training in postmenopausal women. Findings indicated that the decrease in the severity of depressive symptoms was significantly associated with the improvement in HR stress reactivity 8 weeks after completion of training in postmenopausal women who receive breathing training. Although there were no more parameters supporting the improvement in other autonomic parameters contributing to the mechanism investigation, evidence obtained from this study did demonstrate that remission of depressive symptoms was associated with improved heart rate reactivity among postmenopausal women with depressive symptoms. These results suggested a mechanism that may partially explain the association between the treatment of depressive symptoms and autonomic functioning.

A major strength of our study was that the RCT study design was rigorous and the treatment protocol was standardized. In addition, this is the first study examining the efficacy of RSA biofeedback-assisted breathing training on both depressive symptoms and autonomic functioning in postmenopausal women with depressive symptoms, and the results were exhilarating. However, several limitations of this study must be addressed. First, previously reported that menopausal symptoms<sup>42,43</sup>, especially vasomotor symptoms<sup>44,45</sup>, were associated with increased cardiovascular risks. We did not evaluate the impact of menopausal complaints on autonomic function and thus may result in insignificant or ineffective changes. Second, our dose prescription of this 8-week breathing training did not demonstrate significant improvement on ANS activity immediately and intermediately after completion of training. While some findings were encouraging, a larger clinical trial is needed to confirm the efficacy of RSA biofeedback-assisted breathing training on depressive symptoms and autonomic functioning in postmenopausal women with depressive symptoms.

Despite these limitations, we consider our results important because of their practical implications. Our breathing training significantly alleviated depressive symptoms in postmenopausal women with depressive symptoms immediately after completion of the 8-week training. The following 8-week home practice also helped its persistence of symptoms' alleviation. Additionally, our study also provided significant information by determining the association between the changes in depressive symptoms and autonomic functioning to which the slow respiration relaxation skill through RSA biofeedback may improve HR stress reactivity to stressors 8 weeks after completion of training and in turn improve the severity of depressive mood in postmenopausal women with depressive symptoms. Our intervention was feasible for depressive patients and the outcome measures were also inexpensive and noninvasive. Although RSA biofeedback may need a standardized office-based biofeedback training protocol, it is easily integrated into treatment for symptom alleviation. Healthcare providers are suggested to apply this breathing practice accompanying with home practice assignments as a non-pharmacological adjunctive treatment for postmenopausal women with depressive symptoms.

## CONCLUSIONS

RSA biofeedback-assisted breathing training is not better than the control condition (i.e., stress management education) for improving depressive symptoms and autonomic function. Nevertheless,

RSA biofeedback-assisted breathing training is effective in reducing depressive symptoms 1 week and 8 weeks after completion of training. The decrease in the severity of depressive symptoms is significantly associated with the improvement in autonomic functioning as determined by HR stress reactivity in postmenopausal women who receive breathing training. Healthcare providers are suggested to apply this breathing practice accompanying with home practice assignments as a non-pharmacological adjunctive treatment for postmenopausal women with depressive symptoms.

## REFERENCES

1. Nelson HD. Menopause. *Lancet*. Mar 1 2008;371(9614):760-770.
2. Chedraui P, San Miguel G, Avila C. Quality of life impairment during the female menopausal transition is related to personal and partner factors. *Gynecol Endocrinol*. Feb 2009;25(2):130-135.
3. Shyu YK, Pan CH, Liu WM, Hsueh JY, Hsu CS, Tsai PS. Health-Related Quality of Life and Healthcare Resource Utilization in Taiwanese Women With Menopausal Symptoms: A Nation-Wide Survey. *J Nurs Res*. Sep 2012;20(3):208-218.
4. Department of Health. Taiwan Public Health Report 2011: Ten leading causes of death. 2012; [http://www.doh.gov.tw/CHT2006/DM/DM2\\_2.aspx?now\\_fod\\_list\\_no=12336&class\\_no=440&level\\_no=4](http://www.doh.gov.tw/CHT2006/DM/DM2_2.aspx?now_fod_list_no=12336&class_no=440&level_no=4). Accessed November 9, 2012.
5. Juang KD, Wang SJ, Lu SR, Lee SJ, Fuh JL. Hot flashes are associated with psychological symptoms of anxiety and depression in peri- and post- but not premenopausal women. *Maturitas*. Oct 16 2005;52(2):119-126.
6. Amore M, Di Donato P, Berti A, et al. Sexual and psychological symptoms in the climacteric years. *Maturitas*. Mar 20 2007;56(3):303-311.
7. Carney RM, Freedland KE, Miller GE, Jaffe AS. Depression as a risk factor for cardiac mortality and morbidity: a review of potential mechanisms. *J Psychosom Res*. Oct 2002;53(4):897-902.
8. Carney RM, Freedland KE, Veith RC. Depression, the autonomic nervous system, and coronary heart disease. *Psychosomatic Medicine*. May-Jun 2005;67 Suppl 1:S29-33.
9. TaskForce. Heart rate variability. Standards of measurement, physiological interpretation, and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. *Eur Heart J*. Mar 1996;17(3):354-381.
10. De Meersman RE, Stein PK. Vagal modulation and aging. *Biol Psychol*. Feb 2007;74(2):165-173.
11. NAMS. *Menopause Guidebook*. 6 ed. Cleveland, OH: The North American Menopause Society; 2006.
12. Wassertheil-Smoller S, Shumaker S, Ockene J, et al. Depression and cardiovascular sequelae in postmenopausal women. The Women's Health Initiative (WHI). *Arch Intern Med*. Feb 9 2004;164(3):289-298.
13. van der Kooy KG, van Hout HP, van Marwijk HW, de Haan M, Stehouwer CD, Beekman AT. Differences in heart rate variability between depressed and non-depressed elderly. *Int J Geriatr Psychiatry*. Feb 2006;21(2):147-150.
14. Grippo AJ, Johnson AK. Biological mechanisms in the relationship between depression and heart disease. *Neurosci Biobehav Rev*. Dec 2002;26(8):941-962.
15. Horsten M, Ericson M, Perski A, Wamala SP, Schenck-Gustafsson K, Orth-Gomer K. Psychosocial factors and heart rate variability in healthy women. *Psychosom Med*. Jan-Feb 1999;61(1):49-57.
16. Lahiri MK, Kannankeril PJ, Goldberger JJ. Assessment of autonomic function in cardiovascular disease: physiological basis and prognostic implications. *J Am Coll Cardiol*. May 6 2008;51(18):1725-1733.
17. Nahshoni E, Aravot D, Aizenberg D, et al. Heart rate variability in patients with major depression. *Psychosomatics*. Mar-Apr 2004;45(2):129-134.
18. Catipovic-Veselica K, Galic A, Jelic K, et al. Relation between major and minor depression and heart rate, heart-rate variability, and clinical characteristics of patients with acute coronary syndrome. *Psychol Rep*. Jun 2007;100(3 Pt 2):1245-1254.

19. Lichtman JH, Bigger JT, Jr., Blumenthal JA, et al. Depression and coronary heart disease: recommendations for screening, referral, and treatment: a science advisory from the American Heart Association Prevention Committee of the Council on Cardiovascular Nursing, Council on Clinical Cardiology, Council on Epidemiology and Prevention, and Interdisciplinary Council on Quality of Care and Outcomes Research: endorsed by the American Psychiatric Association. *Circulation*. Oct 21 2008;118(17):1768-1775.
20. Carney RM, Freedland KE, Stein PK, Skala JA, Hoffman P, Jaffe AS. Change in heart rate and heart rate variability during treatment for depression in patients with coronary heart disease. *Psychosomatic Medicine*. Sep-Oct 2000;62(5):639-647.
21. Nolan RP, Kamath MV, Floras JS, et al. Heart rate variability biofeedback as a behavioral neurocardiac intervention to enhance vagal heart rate control. *American Heart Journal*. 2005;149(6):1137.e1131-1137.e1137.
22. Lett HS, Davidson J, Blumenthal JA. Nonpharmacologic treatments for depression in patients with coronary heart disease. *Psychosomatic Medicine*. May-Jun 2005;67 Suppl 1:S58-62.
23. Blumenthal JA, Sherwood A, Babyak MA, et al. Effects of exercise and stress management training on markers of cardiovascular risk in patients with ischemic heart disease: a randomized controlled trial. *JAMA*. Apr 6 2005;293(13):1626-1634.
24. Janakiramaiah N, Gangadhar BN, Naga Venkatesha Murthy PJ, Harish MG, Subbakrishna DK, Vedamurthachar A. Antidepressant efficacy of Sudarshan Kriya Yoga (SKY) in melancholia: a randomized comparison with electroconvulsive therapy (ECT) and imipramine. *J Affect Disord*. Jan-Mar 2000;57(1-3):255-259.
25. van Zyl LT, Hasegawa T, Nagata K. Effects of antidepressant treatment on heart rate variability in major depression: a quantitative review. *Biopsychosoc Med*. 2008;2:12.
26. Holloway EA, West RJ. Integrated breathing and relaxation training (the Papworth method) for adults with asthma in primary care: a randomised controlled trial. *Thorax*. Dec 2007;62(12):1039-1042.
27. Kaushik R, Kaushik RM, Mahajan SK, Rajesh V. Biofeedback assisted diaphragmatic breathing and systematic relaxation versus propranolol in long term prophylaxis of migraine. *Complement Ther Med*. Sep 2005;13(3):165-174.
28. Bernardi L, Porta C, Spicuzza L, et al. Slow breathing increases arterial baroreflex sensitivity in patients with chronic heart failure. *Circulation*. Jan 15 2002;105(2):143-145.
29. Del Pozo JM, Gevirtz RN, Scher B, Guarneri E. Biofeedback treatment increases heart rate variability in patients with known coronary artery disease. *American Heart Journal*. 2004;147(3):G1-G6.
30. Hassett AL, Radvanski DC, Vaschillo EG, et al. A pilot study of the efficacy of heart rate variability (HRV) biofeedback in patients with fibromyalgia. *Applied Psychophysiology and Biofeedback*. 2007;32(1):1-10.
31. Karavidas MK, Lehrer PM, Vaschillo E, et al. Preliminary results of an open label study of heart rate variability biofeedback for the treatment of major depression. *Applied Psychophysiology and Biofeedback*. 2007;32(1):19-30.
32. Lehrer P, Vaschillo E, Lu SE, et al. Heart rate variability biofeedback: effects of age on heart rate variability, baroreflex gain, and asthma. *Chest*. Feb 2006;129(2):278-284.
33. Siepmann M, Aykac V, Unterdorfer J, Petrowski K, Mueck-Weymann M. A Pilot Study on the Effects of Heart Rate Variability Biofeedback in Patients with Depression and in Healthy Subjects. *Applied Psychophysiology and Biofeedback*. 2008;33(4):195-201.
34. Licht CMM, de Geus EJC, Zitman FG, Hoogendijk WJG, van Dyck R, Penninx B. Association Between Major Depressive Disorder and Heart Rate Variability in the Netherlands Study of Depression and Anxiety (NESDA). *Archives of General Psychiatry*. 2008;65(12):1358-1367.
35. Gianaros PJ, Salomon K, Zhou F, et al. A greater reduction in high-frequency heart rate variability to a psychological stressor is associated with subclinical coronary and aortic calcification in postmenopausal women. *Psychosom Med*. Jul-Aug 2005;67(4):553-560.
36. Liu CC, Kuo TB, Yang CC. Effects of estrogen on gender-related autonomic differences in humans. *Am*

*J Physiol Heart Circ Physiol.* Nov 2003;285(5):H2188-2193.

37. Lee JO, Kang SG, Kim SH, Park SJ, Song SW. The Relationship between Menopausal Symptoms and Heart Rate Variability in Middle Aged Women. *Korean J. Fam. Med.* Jul 2011;32(5):299-305.
38. Kamarck TW, Lovallo WR. Cardiovascular reactivity to psychological challenge: conceptual and measurement considerations. *Psychosom Med.* Jan-Feb 2003;65(1):9-21.
39. Salomon K, Clift A, Karlsdottir M, Rottenberg J. Major depressive disorder is associated with attenuated cardiovascular reactivity and impaired recovery among those free of cardiovascular disease. *Health Psychol.* Mar 2009;28(2):157-165.
40. Kibler JL, Ma M. Depressive symptoms and cardiovascular reactivity to laboratory behavioral stress. *Int J Behav Med.* 2004;11(2):81-87.
41. York KM, Hassan M, Li Q, Li H, Fillingim RB, Sheps DS. Coronary artery disease and depression: patients with more depressive symptoms have lower cardiovascular reactivity during laboratory-induced mental stress. *Psychosom Med.* Jul-Aug 2007;69(6):521-528.
42. Cagnacci A, Cannoletta M, Palma F, Zanin R, Xholli A, Volpe A. Menopausal symptoms and risk factors for cardiovascular disease in postmenopause. *Climacteric.* Apr 2012;15(2):157-162.
43. Gast GC, Grobbee DE, Pop VJ, et al. Menopausal complaints are associated with cardiovascular risk factors. *Hypertension.* Jun 2008;51(6):1492-1498.
44. Thurston RC, Christie IC, Matthews KA. Hot flashes and cardiac vagal control during women's daily lives. *Menopause.* Apr 2012;19(4):406-412.
45. Thurston RC, Sutton-Tyrrell K, Everson-Rose SA, Hess R, Matthews KA. Hot flashes and subclinical cardiovascular disease: findings from the Study of Women's Health Across the Nation Heart Study. *Circulation.* Sep 16 2008;118(12):1234-1240.

## APPENDIX

Table 1. *Baseline bio-demographic data of all study participants (n=98)*

Variables	Value
Age (years, mean±SD)	56.46 ± 4.13
Education, n (%)	
Junior High school and below	10 (10.20)
Senior High school	35 (35.70)
College and above	53 (54.10)
Marital status, n (%)	
No	13 (13.30)
Yes	75 (76.50)
Others	10 (10.20)
Employment, n (%)	
No / retired / housewife	62 (63.30)
Yes / part time	14 (14.30)
Yes / full time	22 (22.40)
Smoking, n (%)	
No	96 (98.00)
Yes	2 (2.00)
Exercise, n (%)	
No	49 (50.00)
Yes	49 (50.00)
Antidepressant, n (%)	
No	94 (95.90)
Yes	4 (4.10)
BMI (mean±SD)	23.17 ± 3.14
BDI-II (mean±SD)	20.53 ± 10.55
CHQ-12 (mean±SD)	4.27 ± 3.42
SBP (mean±SD)	121.42 ± 15.70
DBP (mean±SD)	66.67 ± 7.08
HR (mean±SD)	69.43 ± 8.05
LF/HF (mean±SD)	1.45 ± 1.56
ln LF <sub>ms</sub> (mean±SD)	4.38 ± 0.93
ln HF <sub>ms</sub> (mean±SD)	4.37 ± 0.98
ln TP <sub>ms</sub> (mean±SD)	6.09 ± 0.80

Abbreviations: BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; ln, natural logarithm; LF, power in low frequency range; HF, power in high frequency range; TP, total power.

Table 2. Baseline BDI-II score of all study participants (n=98)

	Mean $\pm$ SD	n (%)
Mean score	20.53 $\pm$ 10.55	
Range		
Min	10.00	
Max	57.00	
Percentile		
25 <sup>th</sup>	12.00	
50 <sup>th</sup>	17.50	
75 <sup>th</sup>	26.00	
Group (BDI-II score) <sup>a</sup>		
Low score ( $\leq$ 12)		27 (27.60)
Average (13-25)		46 (46.90)
High score ( $\geq$ 26)		25 (25.50)
Severity (BDI-II score) <sup>b</sup>		
Minimal depression ( $\leq$ 13)		31 (31.60)
Mild depression (14-19)		23 (23.50)
Moderate depression (20-28)		27 (27.60)
Severe depression ( $\geq$ 29)		17 (17.30)
Subclinical depression (BDI-II score)		
No ( $\leq$ 13)		31 (31.60)
Yes ( $\geq$ 14)		67 (68.40)

<sup>a</sup> Grouping based on the BDI-II score at the 25<sup>th</sup> and 75<sup>th</sup> percentile

<sup>b</sup> Grouping based on cutoffs suggested from the Chinese BDI-II instruction manual

Table 3. *Distribution of Sample Characteristics among groups (n=81)*

Demographic variables	Experimental group (n =35)	Control group (n =46)	<i>p value</i>
Age (years, mean±SD) <sup>a</sup>	56.37 ± 4.45	56.59 ± 3.96	0.819
Education, n (%) <sup>b</sup>			0.290
Junior High school and below	2 (5.70)	5 (10.90)	
Senior High school	11 (31.40)	20 (43.50)	
College and above	22 (62.90)	21 (45.70)	
Marital status, n (%) <sup>b</sup>			0.599
No	4 (11.40)	7 (15.20)	
Yes	29 (82.90)	34 (73.90)	
Others	2 (5.70)	5 (10.90)	
Employment, n (%) <sup>b</sup>			0.575
No / retired / housewife	23 (65.70)	26 (56.50)	
Yes / part time	4 (11.40)	9 (19.60)	
Yes / full time	8 (22.90)	11 (23.90)	
Smoking, n (%) <sup>b</sup>			0.725
No	34 (97.10)	44 (95.70)	
Yes	1 (2.90)	2 (4.30)	
Exercise, n (%) <sup>b</sup>			0.246
No	19 (54.30)	19 (41.30)	
Yes	16 (45.70)	27 (58.70)	
Antidepressant, n (%) <sup>b</sup>			0.451
No	34 (97.10)	43 (93.50)	
Yes	1 (2.90)	3 (6.50)	
BMI (mean±SD) <sup>a</sup>	22.48 ± 2.99	23.55 ± 3.09	0.121

<sup>a</sup>Group comparison by independent *t*-test.

<sup>b</sup>Group comparison by Chi-square test.

Table 4. Results of psychological and autonomic functioning among groups (n=81)

	Experimental group (n=35)	Control group (n=46)	<i>P</i> -value <sup>a</sup>	95% CI <sup>a</sup>
<b>BDI-II</b>				
Week 0 (baseline)	20.34 ± 9.73	21.35 ± 11.56	0.680	-3.82 ~ 5.83
Week 9 (posttest 1)	12.71 ± 8.51	9.98 ± 6.33	0.101	-6.02 ~ 0.54
Week 16 (posttest 2)	10.20 ± 8.31	8.33 ± 6.29	0.251	-5.10 ~ 1.35
Differences (Week 9 - Week 0) <sup>b</sup>	-7.63 ± 7.02*	-11.37 ± 12.54*	0.093	-0.64 ~ 8.12
Differences (Week 16 - Week 0) <sup>b</sup>	-10.14 ± 8.67*	-13.02 ± 12.19*	0.239	-1.95 ~ 7.71
<b>HR</b>				
Week 0 (baseline)	67.40 ± 7.33	69.43 ± 8.67	0.267	-1.59 ~ 5.66
Week 9 (posttest 1)	67.90 ± 9.52	68.72 ± 7.26	0.662	-2.89 ~ 4.53
Week 16 (posttest 2)	66.83 ± 9.08	70.35 ± 8.45	0.076	-0.38 ~ 7.41
Differences (Week 9 - Week 0) <sup>b</sup>	0.50 ± 6.12	-0.72 ± 7.65	0.443	-1.92 ~ 4.36
Differences (Week 16 - Week 0) <sup>b</sup>	-0.57 ± 5.67	0.91 ± 8.11	0.360	-4.68 ~ 1.72
<b>LF/HF</b>				
Week 0 (baseline)	1.31 ± 1.20	1.56 ± 1.53	0.422	-0.37 ~ 0.88
Week 9 (posttest 1)	2.41 ± 2.37	1.29 ± 1.09	0.013	-1.98 ~ -0.24
Week 16 (posttest 2)	2.08 ± 4.10	1.12 ± 0.94	0.183	-2.39 ~ 0.47
Differences (Week 9 - Week 0) <sup>b</sup>	1.10 ± 2.49*	-0.26 ± 1.71	0.007	0.38 ~ 2.35
Differences (Week 16 - Week 0) <sup>b</sup>	0.77 ± 4.12	-0.44 ± 1.75	0.076	-0.13 ~ 2.55
<b>LnLF</b>				
Week 0 (baseline)	4.50 ± 0.71	4.39 ± 0.92	0.567	-0.48 ~ 0.26
Week 9 (posttest 1)	4.76 ± 1.10	4.28 ± 0.95	0.038	-0.94 ~ -0.03
Week 16 (posttest 2)	4.55 ± 1.01	4.14 ± 1.13	0.100	-0.88 ~ 0.08
Differences (Week 9 - Week 0) <sup>b</sup>	0.26 ± 1.03	-0.12 ± 0.95	0.094	-0.07 ~ 0.82
Differences (Week 16 - Week 0) <sup>b</sup>	0.05 ± 0.98	-0.25 ± 1.07	0.206	-0.17 ~ 0.75
<b>LnHF</b>				
Week 0 (baseline)	4.52 ± 0.82	4.30 ± 1.00	0.281	-0.64 ~ 0.19
Week 9 (posttest 1)	4.36 ± 1.07	4.37 ± 0.96	0.973	-0.44 ~ 0.46
Week 16 (posttest 2)	4.58 ± 0.96	4.44 ± 0.96	0.508	-0.57 ~ 0.29
Differences (Week 9 - Week 0) <sup>b</sup>	0.16 ± 0.81	-0.07 ± 0.86	0.218	-0.61 ~ 0.14
Differences (Week 16 - Week 0) <sup>b</sup>	-0.06 ± 0.76	-0.15 ± 0.77	0.633	-0.42 ~ 0.26
<b>LnTP</b>				
Week 0 (baseline)	6.18 ± 0.58	6.06 ± 0.79	0.430	-0.42 ~ 0.18
Week 9 (posttest 1)	6.30 ± 0.81	5.96 ± 0.75	0.053	-0.69 ~ 0.00
Week 16 (posttest 2)	6.28 ± 0.73	6.10 ± 1.20	0.430	-0.64 ~ 0.27
Differences (Week 9 - Week 0) <sup>b</sup>	0.12 ± 0.70	-0.10 ± 0.77	0.189	-0.11 ~ 0.55
Differences (Week 16 - Week 0) <sup>b</sup>	0.10 ± 0.70	0.04 ± 1.25	0.797	-0.41 ~ 0.53

Values are expressed as mean ± standard deviation; Ln, natural logarithm.

<sup>a</sup>. Examined by independent samples *t*-test

<sup>b</sup>. Examined by paired *t*-test of baseline and post-treatment values

\*. *p*<0.05

Table 5. Results of stress reactivity (mean task values – resting values) on autonomic parameters among groups (n=81)

	Experimental group (n=35)	Control group (n=46)	<i>P</i> -value <sup>a</sup>	95% CI <sup>a</sup>
<b>HR</b>				
Week 0 (baseline)	4.43 ± 3.75	5.42 ± 3.82	0.247	-0.70 ~ 2.68
Week 9 (posttest 1)	4.03 ± 3.27	4.34 ± 3.36	0.679	-1.17 ~ 1.79
Week 16 (posttest 2)	3.69 ± 3.29	4.23 ± 3.58	0.491	-1.01 ~ 2.08
Differences (Week 9 - Week 0) <sup>b</sup>	-0.40 ± 3.08	-1.08 ± 2.82*	0.302	-0.63 ~ 1.99
Differences (Week 16 - Week 0) <sup>b</sup>	-0.74 ± 2.86	-1.19 ± 3.08*	0.499	-0.88 ~ 1.79
<b>LF/HF</b>				
Week 0 (baseline)	1.01 ± 1.51	1.80 ± 2.16	0.071	-0.07 ~ 1.64
Week 9 (posttest 1)	0.26 ± 3.21	1.35 ± 1.62	0.071	-0.10 ~ 2.29
Week 16 (posttest 2)	-0.02 ± 4.17	1.67 ± 1.70	0.015	0.33 ~ 3.05
Differences (Week 9 - Week 0) <sup>b</sup>	-0.76 ± 3.53	-0.44 ± 2.52	0.642	-1.65 ~ 1.02
Differences (Week 16 - Week 0) <sup>b</sup>	-1.03 ± 4.24	-0.12 ± 2.58	0.237	-2.42 ~ 0.61
<b>LnLF</b>				
Week 0 (baseline)	1.11 ± 0.16	1.16 ± 0.22	0.238	-0.03 ~ 0.13
Week 9 (posttest 1)	1.09 ± 0.23	1.18 ± 0.24	0.091	-0.01 ~ 0.20
Week 16 (posttest 2)	1.12 ± 0.22	1.18 ± 0.24	0.311	-0.05 ~ 0.16
Differences (Week 9 - Week 0) <sup>b</sup>	-0.02 ± 0.26	0.02 ± 0.29	0.511	-0.17 ~ 0.08
Differences (Week 16 - Week 0) <sup>b</sup>	0.01 ± 0.25	0.02 ± 0.30	0.948	-0.13 ~ 0.12
<b>LnHF</b>				
Week 0 (baseline)	0.95 ± 0.12	0.95 ± 0.18	0.796	-0.06 ~ 0.08
Week 9 (posttest 1)	0.95 ± 0.15	1.00 ± 0.27	0.345	-0.05 ~ 0.14
Week 16 (posttest 2)	0.97 ± 0.15	0.91 ± 0.12	0.040	-0.12 ~ 0.00
Differences (Week 9 - Week 0) <sup>b</sup>	0.01 ± 0.14	0.04 ± 0.22	0.373	-0.12 ~ 0.04
Differences (Week 16 - Week 0) <sup>b</sup>	0.03 ± 0.19	-0.05 ± 0.18	0.089	-0.01 ~ 0.15
<b>LnTP</b>				
Week 0 (baseline)	1.11 ± 0.10	1.10 ± 0.11	0.757	-0.06 ~ 0.04
Week 9 (posttest 1)	1.06 ± 0.12	1.11 ± 0.11	0.046	8.13 ~ 0.10
Week 16 (posttest 2)	1.05 ± 0.09	1.08 ± 0.13	0.231	-0.02 ~ 0.08
Differences (Week 9 - Week 0) <sup>b</sup>	-0.05 ± 0.11*	0.00 ± 0.14	0.050	-0.12 ~ -5.11
Differences (Week 16 - Week 0) <sup>b</sup>	-0.06 ± 0.11*	-0.02 ± 0.16	0.232	-0.10 ~ 0.02

Values are expressed as mean ± standard deviation; Ln, natural logarithm.

<sup>a</sup>. Examined by independent samples *t*-test

<sup>b</sup>. Examined by paired *t*-test of baseline and post-treatment values

\*. *p*<0.05

Table 6. Results of stress recovery (mean recovery values – resting values) on autonomic parameters among groups (n=81)

	Experimental group (n=35)	Control group (n=46)	P-value <sup>a</sup>	95% CI <sup>a</sup>
<b>HR</b>				
Week 0 (baseline)	-0.19 ± 1.58	-0.56 ± 2.11	0.386	-1.22 ~ 0.48
Week 9 (posttest 1)	-0.67 ± 2.30	-0.98 ± 2.60	0.576	-1.42 ~ 0.79
Week 16 (posttest 2)	-0.35 ± 2.22	-0.60 ± 2.26	0.615	-1.26 ~ 0.75
Differences (Week 9 - Week 0) <sup>b</sup>	-0.48 ± 2.61	-0.42 ± 2.73	0.920	-1.26 ~ 1.13
Differences (Week 16 - Week 0) <sup>b</sup>	-0.16 ± 2.15	-0.04 ± 2.71	0.833	-1.23 ~ 0.99
<b>LF/HF</b>				
Week 0 (baseline)	0.18 ± 0.89	0.15 ± 1.48	0.925	-0.59 ~ 0.54
Week 9 (posttest 1)	0.45 ± 3.07	0.23 ± 1.58	0.699	-1.37 ~ 0.92
Week 16 (posttest 2)	0.49 ± 1.96	0.46 ± 1.27	0.919	-0.75 ~ 0.68
Differences (Week 9 - Week 0) <sup>b</sup>	0.27 ± 3.25	0.07 ± 2.06	0.744	-0.98 ~ 1.37
Differences (Week 16 - Week 0) <sup>b</sup>	0.31 ± 2.18	0.30 ± 1.83	0.982	-0.88 ~ 0.90
<b>LnLF</b>				
Week 0 (baseline)	1.03 ± 0.14	1.04 ± 0.14	0.808	-0.05 ~ 0.07
Week 9 (posttest 1)	1.05 ± 0.19	1.05 ± 0.17	0.934	-0.08 ~ 0.08
Week 16 (posttest 2)	1.10 ± 0.20	1.06 ± 0.20	0.468	-0.12 ~ 0.06
Differences (Week 9 - Week 0) <sup>b</sup>	0.02 ± 0.24	0.01 ± 0.19	0.822	-0.08 ~ 0.11
Differences (Week 16 - Week 0) <sup>b</sup>	0.07 ± 0.25	0.03 ± 0.24	0.467	-0.07 ~ 0.15
<b>LnHF</b>				
Week 0 (baseline)	0.95 ± 0.12	0.95 ± 0.18	0.796	-0.06 ~ 0.08
Week 9 (posttest 1)	0.95 ± 0.15	1.00 ± 0.27	0.345	-0.05 ~ 0.14
Week 16 (posttest 2)	0.97 ± 0.15	0.91 ± 0.12	0.040	-0.12 ~ 0.00
Differences (Week 9 - Week 0) <sup>b</sup>	0.01 ± 0.14	0.04 ± 0.22	0.373	-0.12 ~ 0.04
Differences (Week 16 - Week 0) <sup>b</sup>	0.03 ± 0.19	-0.05 ± 0.18	0.089	-0.01 ~ 0.15
<b>LnTP</b>				
Week 0 (baseline)	1.11 ± 0.10	1.10 ± 0.11	0.757	-0.06 ~ 0.04
Week 9 (posttest 1)	1.06 ± 0.12	1.11 ± 0.11	0.046	8.13 ~ 0.10
Week 16 (posttest 2)	1.05 ± 0.09	1.08 ± 0.13	0.231	-0.02 ~ 0.08
Differences (Week 9 - Week 0) <sup>b</sup>	-0.05 ± 0.11*	0.00 ± 0.14	0.050	-0.12 ~ -5.11
Differences (Week 16 - Week 0) <sup>b</sup>	-0.06 ± 0.11*	-0.02 ± 0.16	0.232	-0.10 ~ 0.02

Values are expressed as mean ± standard deviation; Ln, natural logarithm.

<sup>a</sup>. Examined by independent samples *t*-test

<sup>b</sup>. Examined by paired *t*-test of baseline and post-treatment values

\*. *p*<0.05

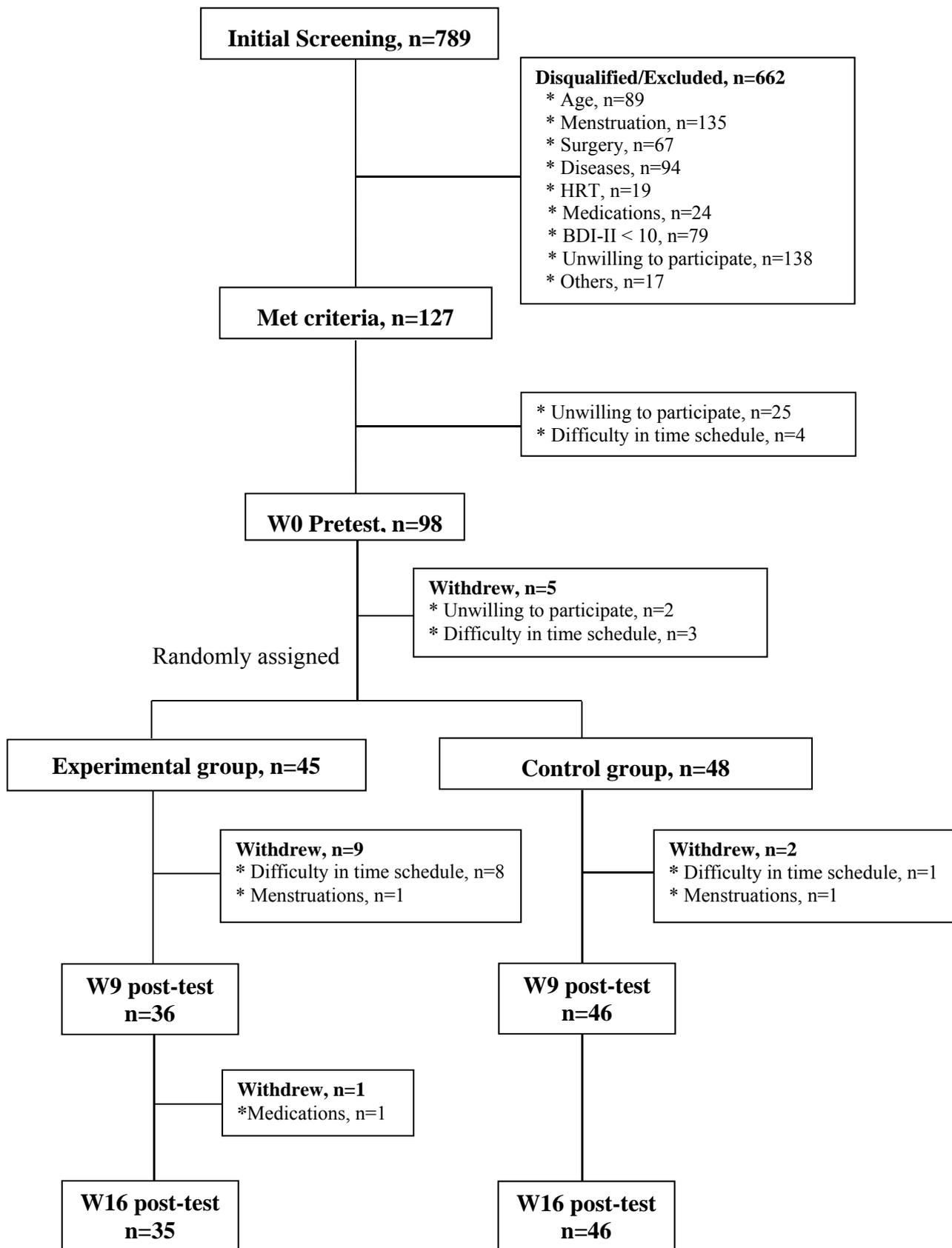


Figure 1. Flow of the participants

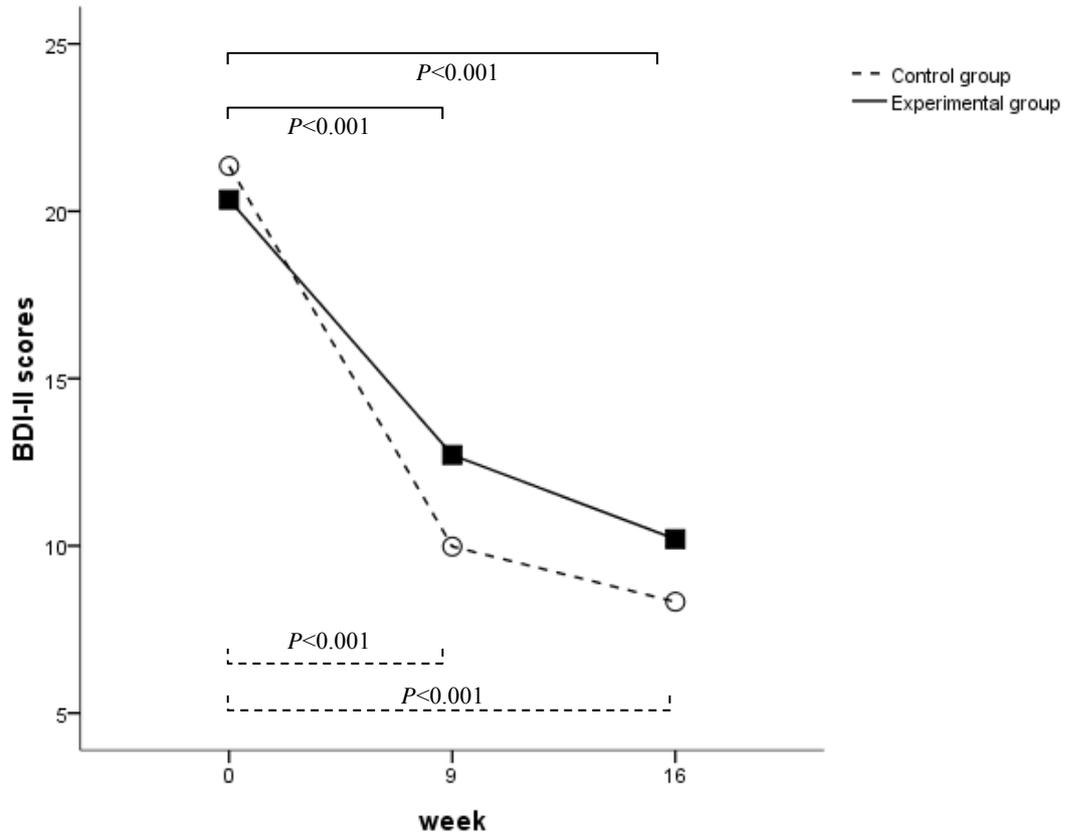


Figure 2. Changes of BDI-II scores across assessment weeks

# 國科會補助專題研究計畫項下出席國際學術會議心得報告

日期：\_101\_年\_8\_月\_24\_日

計畫編號	NSC 98-2629-B-038-002-MY3		
計畫名稱	呼吸訓練對併有憂鬱症狀之停經婦女之成效探討		
出國人員姓名	蔡佩珊	服務機構及職稱	臺北醫學大護理學研究所教授
會議時間	101年7月30日至 101年8月3日	會議地點	澳洲布里斯本
會議名稱	(中文) 國際護理榮譽學會第23屆國際護理研究大會 (英文) Sigma Theta Tau International, 23 <sup>rd</sup> International Nursing Research Congress		
發表論文題目	口頭報告題目：Psychometric testing of the Taiwan version of the Clinically Useful Depression Outcome Scale in patients with diabetes. 海報發表題目：Exploring Factors Associated with Postmenopausal Depression in Taiwanese Women		

## 一、參加會議經過

參與國際學術研討會口頭發表學術論文及海報發表學術論文。

## 二、與會心得

1. 此次國際護理研究會議，共有 700 多人參加，其中 200 多人來自台灣，由此可見台灣護理科學之蓬勃發展。
2. 此次報告主題之研究族群為糖尿病患者，被安排在 Culturally Diverse Health Behaviors: Global Perspectives on Diabetes 的 session 中，其他兩位報告者分別來自斯里蘭卡及越南的研究者，足見糖尿病不僅在先進國家盛行，同樣也是其他發展中國家的重要健康議題。
3. 報告的主題為憂鬱症狀量表在糖尿病族群的量表建構，議題引發與會學者的興趣，

討論熱烈，也證實了憂鬱症與慢性病的共病現象是現今重要的健康議題。

### 三、考察參觀活動(無是項活動者略)

略。

### 四、建議

參與此國際學術會議，能從海報論文展中見證最新研究趨勢，並且與國際學者切磋商交流，收穫良多。

### 五、攜回資料名稱及內容

攜回 23rd International Nursing Research Congress 大會手冊

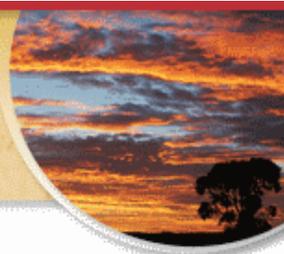
### 六、其他

無。

Honor Society of Nursing, Sigma Theta Tau International  
**23rd International Nursing Research Congress**  
Brisbane, Australia • 30 July-3 August 2012



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Psychometric Testing of the Chinese Version of the Clinically Useful Depression Outcome Scale in Patients with Diabetes

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*Thursday, 2 August 2012: 9:10 AM*

**Pei-Shan Tsai, PhD**

*Graduate Institute of Nursing, College of Nursing, Taipei Medical University, Taipei, Taiwan*

*Learning Objective 1:* describe the procedures used to examine the psychometric properties of an instrument.

*Learning Objective 2:* describe the validity and reliability of the Chinese version of the Clinically Useful Depression Outcome Scale.

**Purpose:**

The purpose of this study was to test the psychometric properties of the Clinically Useful Depression Outcome Scale (CUDOS) in Taiwanese diabetic patients.

**Methods:**

The CUDOS contains 18 items covering the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition symptoms of major depressive disorder and dysthymic disorder. Each item is scored a 5-point Likert scale (0 – 4) with a score ranging from 0 to 72. Following the standard translation-back translation procedure, the CUDOS was translated into Chinese. Internal consistency was examined by the Cronbach's alpha. All participants filled out the Beck Depression Inventory-II (BDI-II), Beck Anxiety Inventory (BAI), and the World Health Organization Quality of Life-BREF (WHOQOL-BREF), Taiwan Version, along with the CUDOS. To assess test-retest reliability, the participants were asked to fill out the CUDOS on a second occasion at a 7-day interval.

**Results:**

Included in the study were 153 diabetics, including 132 patients without and 21 patients with depression. The CUDOS scores were significantly different between patients with depression and those without ( $p < 0.001$ ). The Cronbach's alpha was 0.93 for the CUDOS. The test-retest correlation coefficient was 0.83. The CUDOS significantly and positively correlated to BDI-II ( $r = 0.88$ ,  $p < 0.001$ ) and BAI ( $r = 0.78$ ,  $p < 0.001$ ) and inversely correlated to the WHOQOL-BREF ( $r = -0.32$ ,  $p < 0.001$ ). The CUDOS score significantly and independently predicted quality of life after adjusting for possible confounders.

**Conclusion:**

The Chinese version of the CUDOS demonstrated satisfactory reliability and validity for use in the Taiwanese diabetic patients.

---

See more of: [Culturally Diverse Health Behaviors: Global Perspectives on Diabetes](#)  
See more of: [Research Sessions: Oral Paper & Posters](#)



[Start](#) | [Browse by Day](#) | [Author Index](#) | [Keyword Index](#)

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Exploring Factors Associated with Postmenopausal Depression in Taiwanese Women

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*Monday, 30 July 2012*

**Pei-Shan Tsai, PhD**

*College of Nursing, Taipei Medical University, Taipei, Taiwan*

*Learning Objective 1:* describe the distribution of the severity of depression in postmenopausal women.

*Learning Objective 2:* answer the question of whether postmenopausal depression can be explained by demographic factors, lifestyles factors, or autonomic nervous function.

**Purpose:**

The purpose of this study was to examine demographic, lifestyles, and physiological factors associated with the severity of depressive symptoms in postmenopausal women.

**Methods:**

Baseline data, including demographics, lifestyles variables, and heart rate variability (HRV) from a prospective randomized controlled trial examining the efficacy of a breathing training program on psychosocial functioning and HRV in postmenopausal women with depressive symptoms were analysed. Included in the analyses were 68 women who reported cessation of menstrual cycles for more than 12 consecutive months and scored 10 or greater on the Beck Depression Inventory-II (BDI-II).

**Results:**

The participants aged from 45 to 65 with a mean of 57.1 years. The BDI-II score ranged from 10 to 57. BDI-II was not significantly correlated with age ( $p = 0.974$ ), education ( $p = 0.944$ ), marital status ( $p = 0.839$ ), employment status ( $p = 0.501$ ), smoking ( $p = 0.824$ ), and exercise habit ( $p = 0.833$ ). BDI-II scored was significantly correlated to neither one of the parameters of HRV (all  $P > 0.05$ ).

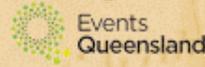
**Conclusion:**

Depressive symptoms in postmenopausal women may be unrelated to women's demographic factors, lifestyles factors, and autonomic function. The mechanisms of postmenopausal depression warrant further investigation.

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See more of: [Research Poster Session 1](#)  
See more of: [Research Sessions: Oral Paper & Posters](#)

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**23rd International Nursing Research Congress**  
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09 August 2012

Pei-Shan Tsai PhD  
Taipei Medical University  
250 Wu Hsing St  
Taipei, 110  
Taiwan

Dear Pei-Shan Tsai:

Congratulations again on your selection for presentation during Sigma Theta Tau International's 23rd International Nursing Research Congress. We look forward to your participation in this prestigious event. The opportunity to collaborate with nursing scholars from around the world involved in the advancement of nursing science promises to be rewarding and stimulating.

### **PRESENTATION(S) SCHEDULED**

- Abstract ID # 50695
- Presentation Type: Poster
- Abstract Title: Exploring Factors Associated with Postmenopausal Depression in Taiwanese Women
- Session Title: Research Poster Session 1
- Date, Session Start/End Time: Monday, 30 July 2012: 10:00 AM - 10:45 AM, BCEC ;  
Monday, 30 July 2012: 01:30 PM - 02:15 PM ;  
Tuesday, 31 July 2012: 10:00 AM - 10:45 AM ;  
Tuesday, 31 July 2012: 02:45 PM - 03:30 PM
- Abstract ID # 50616
- Presentation Type: Oral
- Abstract Title: Psychometric Testing of the Chinese Version of the Clinically Useful Depression Outcome Scale in Patients with Diabetes
- Session Title: Culturally Diverse Health Behaviors: Global Perspectives on Diabetes
- Date, Session Start/End Time: Thursday, 2 August 2012: 08:30 AM - 09:45 AM, BCEC, Mezzanine Level, M1

There will be a total of three presentations scheduled during the concurrent sessions and the total presentation time for each oral presentation is 15 minutes with an additional 5 minutes for questions from the audience. For a symposium, the organizer of the symposium will determine the order and length of each presentation. Poster presentations are scheduled at various dates and times during the program.

Please review the poster schedule on our [website](#).

## VIEW YOUR SESSION/ABSTRACT

A detailed schedule of presentations is available [online](#). You can view your submission, but changes cannot be made at this time.

## IMPORTANT INFORMATION

Important deadlines can be found through the [Speaker's Corner Form](#).

- **23 MAY 2012** — Presenter Registration Deadline
  - All presenters are required to register for at least the day(s) of presentation. Presenters must register and submit payment by the presenter registration deadline to be listed in the final program. Removal from the program will result if this deadline is not met.
  - [Register online NOW!](#)
- **27 JUNE 2012** — PowerPoint Upload Deadline — if your presentation is an oral presentation or symposium, upload your presentation by the PowerPoint Upload Deadline to avoid having to present from handouts. All presentations are preloaded to ease the transition between presenters. If we do not have a PowerPoint file preloaded, you will not be able to use PowerPoint to present and must present from handouts only. Please refer to the [Oral Presenter Information](#) available on our website for additional information.
- Please review the PowerPoint Requirements and Guidelines thoroughly as changes have been made on the type of files that will be supported and what can be included within a presentation.
- Please note that the honor society has instituted a [Presenter Acceptance Policy](#) that will be strictly adhered to for all of our events. Please review this document to ensure all required guidelines are met.

Additional important information to assist with any questions is available on our website for [speakers](#) and [poster presenters](#).

If you have any questions, please contact Machel Fisher (email: [abstracts@stti.org](mailto:abstracts@stti.org); phone: 888.634.7575 <sup>US/Canada</sup> or +1.317.634.8171 <sup>International</sup>).

Thank you,  
Cynthia Vlasich, MBA, BSN, RN  
Director, Education and Leadership  
Sigma Theta Tau International, Honor Society of Nursing

# 國科會補助計畫衍生研發成果推廣資料表

日期:2013/01/30

國科會補助計畫	計畫名稱: 呼吸訓練對併有憂鬱症狀之停經婦女之成效探討
	計畫主持人: 蔡佩珊
	計畫編號: 98-2629-B-038-002-MY3      學門領域: 護理
無研發成果推廣資料	

98 年度專題研究計畫研究成果彙整表

計畫主持人：蔡佩珊		計畫編號：98-2629-B-038-002-MY3				計畫名稱：呼吸訓練對併有憂鬱症狀之停經婦女之成效探討	
成果項目		量化			單位	備註（質化說明：如數個計畫共同成果、成果列為該期刊之封面故事...等）	
		實際已達成數（被接受或已發表）	預期總達成數（含實際已達成數）	本計畫實際貢獻百分比			
國內	論文著作	期刊論文	1	0	100%	篇	
		研究報告/技術報告	0	0	100%		
		研討會論文	1	0	100%		
		專書	0	0	100%		
	專利	申請中件數	0	0	100%	件	
		已獲得件數	0	0	100%		
	技術移轉	件數	0	0	100%	件	
		權利金	0	0	100%	千元	
	參與計畫人力（本國籍）	碩士生	0	0	100%	人次	
		博士生	1	0	100%		
		博士後研究員	0	0	100%		
		專任助理	2	0	100%		
國外	論文著作	期刊論文	0	0	100%	篇	
		研究報告/技術報告	0	0	100%		
		研討會論文	2	0	100%		
		專書	0	0	100%		章/本
	專利	申請中件數	0	0	100%	件	
		已獲得件數	0	0	100%		
	技術移轉	件數	0	0	100%	件	
		權利金	0	0	100%	千元	
	參與計畫人力（外國籍）	碩士生	0	0	100%	人次	
		博士生	0	0	100%		
		博士後研究員	0	0	100%		
		專任助理	0	0	100%		

<p>其他成果 (無法以量化表達之成果如辦理學術活動、獲得獎項、重要國際合作、研究成果國際影響力及其他協助產業技術發展之具體效益事項等，請以文字敘述填列。)</p>	無。
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	成果項目	量化	名稱或內容性質簡述
科 教 處 計 畫 加 填 項 目	測驗工具(含質性與量性)	0	
	課程/模組	0	
	電腦及網路系統或工具	0	
	教材	0	
	舉辦之活動/競賽	0	
	研討會/工作坊	0	
	電子報、網站	0	
	計畫成果推廣之參與(閱聽)人數	0	

# 國科會補助專題研究計畫成果報告自評表

請就研究內容與原計畫相符程度、達成預期目標情況、研究成果之學術或應用價值（簡要敘述成果所代表之意義、價值、影響或進一步發展之可能性）、是否適合在學術期刊發表或申請專利、主要發現或其他有關價值等，作一綜合評估。

1. 請就研究內容與原計畫相符程度、達成預期目標情況作一綜合評估

達成目標

未達成目標（請說明，以 100 字為限）

實驗失敗

因故實驗中斷

其他原因

說明：

2. 研究成果在學術期刊發表或申請專利等情形：

論文： 已發表  未發表之文稿  撰寫中  無

專利： 已獲得  申請中  無

技轉： 已技轉  洽談中  無

其他：（以 100 字為限）

徐育愷、王美業、蔡佩珊（2013）。臺灣婦女停經後憂鬱症狀之相關因素探討。新臺北護理期刊（已接受刊登）。

3. 請依學術成就、技術創新、社會影響等方面，評估研究成果之學術或應用價值（簡要敘述成果所代表之意義、價值、影響或進一步發展之可能性）（以 500 字為限）

本呼吸訓練在呼吸訓練完一週及八週之後，可有效改善憂鬱症狀，及產生與心跳速率及心率變異性之壓力應激反應等相關指標小的治療效果量。而接受呼吸訓練之婦女其憂鬱症狀嚴重度降低與心跳應激反應的改善有關。本呼吸方法容易學習，且憂鬱症狀與自主神功能參數的測量經濟又不具侵入性，臨床照護者可運用此呼吸方法合併居家練習，做為有憂鬱症狀的停經婦女之非藥物性輔助療法，進而改善其自主神經協調能力，預防心血管不良預後的發生。此外，本研究另計算在呼吸訓練後的治療效果量，對未來相關研究提供了重要的參考研究數據；但因自主神經功能未受壓力刺激前基礎值未因呼吸訓練而獲得改善，未來研究可再針對個案資料做更深入的軌跡分析或次族群分析，探討停經婦女的憂鬱相關機轉。