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Original Article

Effect of previous diagnoses of depression, menopause status, vasomotor symptoms, and neuroticism on depressive symptoms among climacteric women: A 30-month follow-up

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ABSTRACT

Objectives: The research was designed to examine the impact of the previous diagnoses of depression, menopause status, vasomotor symptoms, and neuroticism on depressive symptoms among menopausal women in Taiwan over a 30-month follow-up.**Materials and Methods:** A community-based sample of 190 middle-aged women was enrolled. The Menopausal Symptoms Scale, Neuroticism Extraversion Openness Five Factor Inventory—Chinese version, and Ko's Depression Inventory were applied, and results were assessed. In addition, each woman underwent a semistructured diagnostic interview with the Chinese version of the Modified Schedule of Affective Disorders and Schizophrenia—Lifetime to obtain her lifetime psychiatric history. After 30 months, 111 participants completed follow-up questionnaires.**Results:** Results of the hierarchical multiple regression analyses showed that depressive symptoms during the menopause transition predicted depressive symptoms over 30 months. After controlling for depressive symptoms during the menopause transition, the previous diagnoses of depression, menopause status, and vasomotor symptoms could not predict depressive symptoms over 30 months, whereas neuroticism still predicted depressive symptoms over 30 months.**Conclusion:** The research suggested that neuroticism plays an important role in the persistence of depression among climacteric women after 30 months.

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Introduction

Menopause is a major turning point in a woman's life in which various physiological changes are experienced by all women who reach 40–60 years of age. The gradual decrease in estrogen levels that occurs during the menopause transition may cause women to experience menopausal symptoms, which include aspects of somatic symptoms, sexual dysfunction, psychological symptoms, and

vasomotor symptoms [1]. In addition, many women experience depressive symptoms and mood disorders associated with the low-estrogen phase of the menstrual cycle during menopause [2].

A potential association between depressive symptoms and menopause transition has been postulated. Estrogen deficiency has been suggested to predict depressive symptoms during the climacteric duration [2]. Previous research on this topic has indicated that menopause status is related to depressive symptoms [1–3]. However, other studies have not supported the relationship between menopausal status and depressive symptoms [2]. The incompatible findings may be attributable to the lack of consideration of confounding variables, such as surgical menopause, vasomotor symptoms, receiving hormone therapy (HT), previous depression, and neuroticism.

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Women who have undergone surgically induced menopause have been demonstrated to have higher rates of depression than those who experience the natural menopause process [2–4]. Moreover, according to the symptom hypothesis, depressive symptoms are explained by vasomotor symptoms associated with changing estrogen levels [1,2]. In addition, women who experience unstable and irregular estrogen patterns may choose to receive HT in order to reduce menopausal symptoms. Receiving HT has also been found to negatively correlate with menopause symptoms or positively associate with depressive symptoms [1]. Premenstrual dysphoric disorder (PMDD) [5,6], history of major depressive disorder (MDD) [7,8], and previous dysthymic disorder (PDD) [1,2,7] have been shown to be highly associated with depressive symptoms in midlife. Furthermore, neuroticism has been shown to be the best predictor of depressive symptoms in middle-aged women [9]. Kuh et al [10] indicated that women in midlife with high levels of neuroticism experience more psychological symptoms, including depression or anxiety, irritability, tearfulness, and feelings of panic.

After controlling for confounding variables, such as receiving HT, PMDD, history of MDD, PDD, and vasomotor symptoms, Lin et al [11] found menopausal status as a predictor of depressive symptoms among climacteric women during the menopause transition, and found this to be mediated by neuroticism. However, the study adopted a cross-sectional design; therefore, no causal relationship can be inferred clearly. The cross-sectional nature of the research referenced above suggests a need for further examination of the causal association among menopause status, neuroticism, and depressive symptoms. In addition, participants who are experiencing depressive symptoms may overestimate or underestimate the frequency or severity of the risks for depressive symptoms, particularly if the assessment is retrospective in cross-sectional research. Thus, the aim of the present prospective study was to determine whether menopause status and neuroticism in menopausal women, including those with pre-, peri-, and postmenopausal statuses, predicted a higher occurrence of depressive symptoms after 30 months in a community sample of midlife women in Taiwan when controlling for depressive symptoms measured during the menopause transition and confounding variables such as receiving HT, PMDD, a history of MDD, PDD, and vasomotor symptoms.

Materials and methods

Participants and procedures

The participants in the study were recruited from three districts in Tainan City, Taiwan, using cluster sampling. We collaborated with the three district officials, who collected the relevant materials for us, including informed consent, demographic variables (menopause status, HT status, history of psychiatric treatment, years of education, employment status), self-reported vasomotor symptoms, depressive symptoms, and menopausal symptoms. Initially, 316 women responded to our study.

We invited the respondents to complete the Neuroticism Extraversion Openness Five Factor Inventory—Chinese version (NEO-FFI-C); these women were then interviewed by trained research associates using the Modified Schedule of Affective Disorders and Schizophrenia—Lifetime (MSADS-L) to identify whether they had suffered from PMDD or had a history of MDD or dysthymic disorder. We excluded participants who were illiterate as well as those with surgically induced menopause because of hysterectomy and/or bilateral ovariectomy. Eventually, 130 women were excluded from the study; the final sample was 190 women, yielding a response rate of 60.13%. During the menopause transition (time 1), we further classified the participants into three groups according to

menopause status: pre-, peri-, and postmenopausal. After 30 months (time 2), 111 participants completed the follow-up questionnaires (follow-up rate, 35.13%). The demographic characteristics and related variables for the follow-up and the nonfollowed sample are presented in Table 1. There were no significant differences among all variables between the follow-up and the nonfollowed participants.

Measurements

Menopause status

We adapted the menopause status classification defined by the World Health Organization [12]. In the study, participants self-reported their menopause status according to the following definitions: premenopausal was defined as an unchanged menstrual pattern; premenopausal was defined as an irregular menstrual pattern with at least one menstrual period in the preceding 12 months; and postmenopausal was defined as experiencing amenorrhea for at least 1 year.

Menopausal Symptoms Scale

The Menopausal Symptoms Scale is a self-reported questionnaire that includes 25 items comprising four domains: somatic, psychological, sexual, and vasomotor symptoms. Each item is rated on a 4-point scale. Possible scores on the menopausal symptoms range from 0 to 75, with higher scores indicating a greater severity of menopausal symptoms. The internal consistency coefficient was 0.84 for somatic symptoms, 0.83 for psychological symptoms, 0.87 for sexual symptoms, and 0.78 for vasomotor symptoms [11].

Neuroticism

The levels of neuroticism in the present study were assessed using the NEO-FFI-C. The NEO-FFI-C is a Chinese modified version of the Neuroticism Extraversion Openness Five Factor Inventory [13], which is composed of 60 items and rated on a 5-point scale. The coefficient of Cronbach α for the five dimensions ranged from 0.44 to 0.65 [11].

Depression

The depression levels of participants over the previous week were assessed using Ko's Depression Inventory (KDI), which was developed by Ko [14] in 1989. The KDI, a self-reported depression scale containing 26 items, has an internal consistency of 0.87 and split-half reliability coefficients averaging 0.87 [15]. A sensitivity of 85.53% and a specificity of 91.80% were obtained when it was administered to screen for major depression [14]. A sensitivity of 84.62% and a specificity of 82.36% were obtained when it was administered to screen for dysthymic disorder [16]. Moreover, a highly positive correlation of 0.90 was revealed between the KDI and Beck Depression Inventory among 276 psychiatric patients [15], and a highly positive correlation was also found among the KDI, Adult Suicidal Ideation Questionnaire—Revised, and depressive symptoms to the Symptom Check List-90 Revised among college students [15,17,18].

Lifetime psychiatric history

All participants were personally interviewed by a research associate for an initial evaluation. A more detailed subsequent interview to determine the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Revision* diagnoses was conducted using the semistructured Chinese version of the MSADS-L, which was based on the MSADS-L from Merikangas et al [19]. The interrater reliability of the Chinese version of the MSADS-L has been investigated by comparing the diagnoses derived from independent administration by psychiatrists and research associates. High

Table 1

Characteristics and related variables between the follow-up group and nonfollow-up group over 30 months of follow-up.

Variable	Nonfollow-up (n = 79)	Follow-up (n = 111)	t/ χ^2	p
Age, mean (SD)	49.96 (5.41)	50.13 (5.78)	−0.20	0.69
Menopause status			3.14	0.52
Premenopausal	38 (48.10)	56 (50.45)		
Perimenopausal	19 (24.05)	16 (14.41)		
Postmenopausal	22 (33.66)	39 (35.14)		
Had psychiatric treatment			0.26	0.12
Never	68 (96.20)	105 (94.59)		
Ever	11 (3.80)	6 (5.41)		
Had received HT			0.57	0.20
Never	68 (86.08)	91 (81.98)		
Ever	11 (13.92)	20 (18.02)		
Neuroticism, mean (SD)	28.91 (6.45)	28.87 (7.32)	−0.04	0.87
Vasomotor symptoms (time 1), mean (SD) ^a	−0.24 (4.72)	0.11 (3.53)	−0.58	0.46
Depressive symptoms (time 1), mean (SD) ^b	9.80 (8.81)	10.25 (7.15)	−0.39	0.57
Depressive symptoms (time 2), mean (SD) ^c	—	9.57 (7.66)	—	—

Data are presented as n (%) unless otherwise indicated.

HT = hormone therapy; SD = standard deviation.

^a Vasomotor symptoms during the menopause transition.^b Depressive symptoms during the menopause transition.^c Depressive symptoms over a 30-month follow-up.

correlations between the Chinese version of the MSADS-L and clinical diagnoses have been reported. The κ values for schizophrenia, affective disorder, anxiety disorder, and alcohol abuse and dependence ranged from 0.71 to 1.0 between psychiatrists and psychologists [20].

Statistical analysis

First, independent samples *t* test and Chi-square test were used to identify statistically significant differences between the follow-up and nonfollow-up groups with respect to whole variables. Second, to verify correlations between measures in the study, Spearman and Pearson correlations were calculated. Third, a series of hierarchical multiple regression analyses were conducted to examine the role of neuroticism in the relationship between menopause status and depressive symptoms over a 30-month follow-up when controlling for depressive symptoms at time 1 and the possible confounders. All statistical analyses were performed using PASW Statistics 18 for Windows. In the present study, $p < 0.05$ was interpreted as statistically significant.

Results

In Table 2, the correlation coefficients are given for clinical measures, neuroticism, vasomotor symptoms, depressive

symptoms (time 1), and depressive symptoms (time 2). We found PMDD, history of MDD, vasomotor symptoms, and neuroticism to be positively associated with depressive symptoms (time 1). Moreover, we found history of MDD, vasomotor symptoms, and neuroticism to be positively associated with depressive symptoms (time 2). There was a high correlation (0.72) in the relationship between depressive symptoms (time 1) and depressive symptoms (time 2). Furthermore, history of MDD was correlated with neuroticism, depressive symptoms (time 1), and depressive symptoms (time 2). Menopause status was positively associated with receiving HT and vasomotor symptoms. Vasomotor symptoms were positively correlated with PMDD, menopause status, neuroticism, depressive symptoms (time 1), and depressive symptoms (time 2). Results of the hierarchical multiple regression analyses are presented in Table 3. As presented in Table 3, depressive symptoms (time 1) that predicted depressive symptoms (time 2) was entered into step 1 as a control. In step 2, the impact of PMDD, history of MDD, and PDD on depressive symptoms (time 2) were considered, but were not found to be significant. Depressive symptoms (time 1) remained significant in predicting depressive symptoms (time 2). Furthermore, receiving HT, vasomotor symptoms, and menopause status were added into step 3. The results showed that only depressive symptoms (time 1) reached significance in step 3. In step 4, neuroticism was entered as a predictor of depressive symptoms (time 2) and showed that depressive symptoms (time 1)

Table 2

Correlational matrix of clinical measures, neuroticism, vasomotor symptoms, and depressive symptoms (n = 111).

	1	2	3	4	5	6	7	8	9
(1) PMDD	1								
(2) History of MDD	0.13	1							
(3) PDD	0.22*	−0.03	1						
(4) Menopause status	−0.02	−0.04	0.02	1					
(5) Had received HT	0.00	−0.06	−0.05	0.46**	1				
(6) Neuroticism	0.24*	0.36**	0.05	0.15	−0.02	1			
(7) Vasomotor symptoms (time 1) ^a	0.22*	0.05	0.13	0.34**	0.07	0.48**	1		
(8) Depressive symptoms (time 1) ^b	0.19*	0.26**	0.09	0.17	0.09	0.59**	0.44**	1	
(9) Depressive symptoms (time 2) ^c	0.12	0.30**	0.13	0.14	0.02	0.65**	0.36**	0.72**	1

* $p < 0.05$.** $p < 0.01$.

HT = hormone therapy; MDD = major depressive disorder; PDD = previous dysthymic disorder; PMDD = premenstrual dysphoric disorder.

^a Vasomotor symptoms during the menopause transition.^b Depressive symptoms during the menopause transition.^c Depressive symptoms over a 30-month follow-up.

Table 3

Hierarchical regression analyses in predicting the depressive symptoms over a 30-month follow-up ($n = 111$).

	Beta	SE	B	p	ΔR^2
Step 1					0.515
Depressive symptoms (time 1) ^a	0.770	0.073	0.718	<0.001*	
Step 2					0.536
Depressive symptoms (time 1) ^a					
PMDD	-0.863	1.482	-0.041	0.562	
History of MDD	3.381	1.851	0.128	0.071	
PDD	6.608	5.545	0.082	0.236	
Step 3					0.540
Depressive symptoms (time 1) ^a	0.715	0.084	0.666	<0.001*	
PMDD	-0.922	1.525	-0.044	0.547	
History of MDD	3.407	1.882	0.129	0.073	
PDD	6.106	5.621	0.076	0.280	
Had received HT	0.086	0.178	0.040	0.630	
Vasomotor symptoms (time 1) ^b	0.348	0.712	0.041	0.626	
Menopause status	-1.187	1.578	-0.059	0.454	
Step 4					0.605
Depressive symptoms (time 1) ^a	0.565	0.087	0.526	<0.001*	
PMDD	-1.531	1.428	-0.073	0.286	
History of MDD	1.261	1.832	0.048	0.493	
PDD	7.035	5.240	0.088	0.182	
Had received HT	-0.110	0.173	-0.051	0.526	
Vasomotor symptoms (time 1) ^b	0.160	0.665	0.019	0.811	
Menopause status	-0.487	1.480	-0.024	0.743	
Neuroticism	0.369	0.091	0.351	<0.001*	

* $p < 0.001$.

HT = hormone therapy; MDD = major depressive disorder; PDD = previous dysthymic disorder; PMDD = premenstrual dysphoric disorder; SE = standard error.

^a Depressive symptoms during the menopause transition.

^b Vasomotor symptoms during the menopause transition.

remained significant. In addition, after controlling for depressive symptoms (time 1) and confounding variables, neuroticism remained a predictor of depressive symptoms (time 2).

Discussion

In our study, we found neuroticism to be significantly associated with depressive symptoms during the menopause transition. Furthermore, after controlling for depressive symptoms during the menopause transition and for other confounders, neuroticism still predicted symptoms over 30 months, which suggests that neuroticism plays an important role in the persistence of depression among climacteric women after 30 months. In the present study, the positive relationship found between neuroticism and depressive symptoms stands in accordance with the results of previous research on this topic [9–11,21–24]. A possible explanation for the casual relationship between neuroticism and depressive symptoms is that people with high levels of neuroticism tend to experience more distress. Climacteric women with high neurotic tendencies are more likely to experience psychological pain and therefore may be more predisposed to experience more depressive symptoms in the future [11,23]. Second, the results are probably because climacteric women with high neurotic tendencies are more likely to have false information about and negative attitudes toward estrogen deficits, climacteric symptoms (hot flashes, night sweats, vaginal dryness, and dyspareunia), sexual dysfunction, aging, and major life changes (retired from the workplace, empty nest, etc.)—thus increasing and maintaining their tendency to experience subjective and perceived stress, and therefore they may be more predisposed to experience more depressive symptoms in the future [1,22,24]. Third, neuroticism may presumably play an important role in chronic depression. Individuals with high levels of neuroticism exhibit sensitivity to negative stimuli, resulting in a range of

negative moods that are likely to predispose them to chronic depression [25].

Our findings indicated that after controlling for depressive symptoms during the menopause transition and confounding variables, menopausal status was not a predictor of depressive symptoms over 30 months. The results were in accordance with previous findings [1,2,6,7,22]. One probable explanation for these findings was the fact that the presence of both menopausal symptoms and depressive symptoms during the menopausal transition may illustrate a dissimilar phenomenon caused by changes in the reproductive hormonal milieu, such as estrogen and/or serotonin, to which some women are particularly impressionable [7]. Thus, unexpected changes in neuromodulator function and/or in reproductive hormone levels could contribute to the constellation of depressive and menopausal symptoms seen in some menopausal women. The second probable explanation for these findings is that depressive symptoms during the menopause transition may be associated with an earlier decline in ovarian function, whereas depressive symptoms over 30 months may not. Freeman [6] suggested that the greatest risks for new-onset depression in midlife women may be in the transition to menopause rather than in the postmenopausal years. Thus, menopause status did not predict the depressive symptoms over 30 months after controlling for depressive symptoms during the menopause transition and for confounding variables. However, our findings were not consistent with the conclusions of Lin et al [11]. A probable explanation for this may be attributable to the longitudinal design of the present study. In the cross-sectional results of Lin and colleague [11], females who pointed out more menopausal distresses and vasomotor symptoms and who also had previous depression expressed more depressive symptoms. Conversely, individuals who experienced more depressive symptoms were shown to be more likely to indicate an increase in menopausal reactions, additional vasomotor symptoms, or to have a history of depression. Therefore, the relationships between menopause status and depressive symptoms need to be considered in prospective research. In our prospective study, we found a causal relationship between menopause status and depressive symptoms over 30 months.

In the present study, we found a history of MDD to be positively associated with depressive symptoms during the menopause transition as well as with depressive symptoms after 30 months. These findings have been suggested from the results of previous studies [1,2,6]. The probable explanation for the findings is that the reproductive hormones are associated with mood among women with a history of depression. Compared to women with no history of depression, increased levels of Follicle-stimulating hormone (FSH) and Luteinizing hormone (LH), decreased levels of inhibin b, and increased variability of FSH and estradiol have been significantly correlated with the occurrence of depression among the women who met the diagnostic criteria for depressive disorder [6]. However, after controlling for the impact of depressive symptoms during the menopause transition on depressive symptoms after 30 months, the relationship between a history of MDD and depressive symptoms after 30 months was not significant. An explanation for this is that the history of depressive disorder may have varying impacts toward different stages of menopausal status. According to Freeman's findings, it was suggested that a history of depression may be associated with an earlier decline in ovarian function during the menopause transition [6]. Thus, after the menopausal transition, women in midlife with a more stable status in ovarian function will experience less climacteric symptoms, and therefore will not exhibit depression.

In our findings, vasomotor symptoms were positively correlated with menopause status, depressive symptoms during the

menopause transition, and depressive symptoms after 30 months. These findings stand in accordance with the results found in previous studies [1,2,6,7,22]. The probable reason for this is the domino effect. During the menopause transition, vasomotor symptoms may disturb sleep, and sleep disruption has been found to cause negative moods [2,6]. However, after controlling for the impact of depressive symptoms during the menopause transition on depressive symptoms after 30 months, the relationship between vasomotor symptoms and depressive symptoms after 30 months was not significant. We found that the impact of vasomotor symptoms on depressive symptoms during the menopause transition was stronger as compared to the depressive symptoms after 30 months. In accordance with Freeman's [6] findings, a probable explanation may be attributable to the impact of reproductive hormones on menopausal transition, which suggested that vasomotor symptoms may be correlated with an earlier decline in ovarian function during the menopause transition. After the menopausal transition, women in midlife with a more stable status in ovarian function will experience less climacteric symptoms or will adjust to the vasomotor symptoms, and therefore will not develop depression.

This study has several limitations. First, our sample size was small ($n = 111$); thus, caution is required when generalizing our findings to large samples of middle-aged females undergoing menopausal transition. Second, menopausal status was collected from a pencil-and-paper self-report, which raises the possibility of response bias. Recall bias and pressure to give socially desirable answers might also be sources of error. In the future, we suggest determining estrogen levels in prospective studies to investigate the impact of menopause status on depressive mood in climacteric women. Then, researchers can clearly evaluate the gradual decrease in estrogen levels during and after the menopausal transition and diminish recall bias in regard to reporting the actual menopausal transition phase.

Despite the limitations discussed above, five strengths of this study include: (1) the 30-month follow-up; (2) the use of randomly selected, community-based participants from the general population; (3) an age group covering the midlife years; (4) distinction between natural and surgically caused menopause in women; and (5) controlling for probable confounding variables correlated with depressive symptoms, such as vasomotor symptoms, receiving HT, previous diagnoses of depression, and depressive symptoms during the menopausal transition phase. The present study revealed that neuroticism plays an important role in the persistence of depression among climacteric women after 30 months. Thus, clinical approaches should assist climacteric women in Taiwan to build skills, including adaptive emotion regulation strategies, mindfulness [26,27], and yoga [28].

Conflicts of interest

The authors have no conflicts of interest relevant to this article.

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