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ORIGINAL RESEARCH

Moderating and Mediating Effects of Resilience Together with Neuroticism on Depressive Symptoms in Adult Volunteers

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Background: Parenting quality experienced in childhood affects depressive symptoms in adulthood, and neuroticism and resilience are attracting attention as personality traits that mediate the effects of parental rearing quality experienced in childhood on adulthood depressive symptoms. However, the interaction between neuroticism and resilience remains unclear. In this study, we hypothesized that resilience and neuroticism are mediators between parental rearing quality experienced in childhood and depressive symptoms in adulthood, and furthermore, that resilience and neuroticism interact with each other in their effects on depressive symptoms. To test these hypotheses, we conducted structural equation modeling and hierarchical multiple regression analysis including interactions in adult volunteers.

Methods: A self-administered questionnaire survey was conducted on 528 adult volunteers recruited at Tokyo Medical University for 1 year from April 2017 to April 2018. The Parental Bonding Instrument (PBI), Eysenck Personality Questionnaire-revised short version, Connor-Davidson Resilience Scale, and Patient Health Questionnaire-9 were used as questionnaires, and their scores were analyzed using structure equation modeling. The interaction between resilience and neuroticism was analyzed using hierarchical multiple regression analysis.

Results: Structural equation modeling showed that parenting quality (care and overprotection) experienced in childhood had a significant indirect effect on the severity of depressive symptoms in adulthood, mediated by both neuroticism and resilience. Among the subscores of the PBI, "care" showed opposite effects to "overprotection". Structural equation modeling of "care" and "overprotection" explained 36.9% and 36.6% of the variability in depressive symptoms in adulthood, respectively. Hierarchical multiple regression analysis showed that the negative interaction between neuroticism and resilience had a significant effect on depressive symptom severity in adulthood.

Conclusion: The results of this study show that resilience and neuroticism are mediators of the effects from parental child-rearing to depressive symptoms in adulthood. Furthermore, resilience antagonizes the effect of neuroticism on adulthood depressive symptoms.

Keywords: parenting, resilience, neuroticism, depressive symptoms, structure equation modeling, hierarchical multiple regression analysis

Introduction

Major depressive disorder is a mental disorder, in which patients demonstrate symptoms of depressed mood, anxiety, insomnia, and decreased motivation, interest, activity, and appetite. The lifetime prevalence of major depressive disorder is estimated to be 5% to 17%.[1] Major depressive disorder has been suggested to be the second leading cause of years lived with disability in the Global Burden of Disease Study.[2] Furthermore, the prevalence of subthreshold depression, which does not fulfill the diagnostic criteria for major depressive disorder, is also high,[3] and a wide spectrum of depressive symptoms from mild to severe are major clinical and social problems. Although the etiology of depression has

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not yet been fully elucidated, external environmental factors, such as the experience of abuse, inappropriate parenting, and bullying victimization in childhood, and the experience of harassment victimization in adulthood have been pointed out as triggers for the onset of depression.[1,4–9] Regarding internal factors in patients with major depressive disorders, neuroticism and genetic vulnerability are risk factors for developing depression, although the effect of genetic vulnerability is relatively small.[7,9,10] The interaction of genetic vulnerability and neuroticism with stress is considered to play an important role in the mechanism of the onset of depression.[10,11]

The Parental Bonding Instrument (PBI) is a 25-item questionnaire to retrospectively evaluate parenting quality in childhood, which was developed by Parker et al[12] Factor analysis demonstrated that the PBI consists of the following 2 subscales: "care" and "overprotection". The "care" subscale has 1 pole comprising emotional warmth, affection, intimacy, and empathy, and the other pole comprising neglect, emotional coldness, and indifference. The "overprotection" subscale has 1 pole comprising overprotection, control, obstruction of self-reliance, and excessive contact, and the other pole comprising autonomy and the allowance of self-reliance.[12] Childhood parenting quality, particularly a lack of care, has been reported to be closely associated with depression and depressive symptoms.[5,9,13,14]

Many recent studies have suggested a close association between the personality trait of neuroticism and the onset of major depressive disorder or depressive symptom severity.[9,10,15,16] The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) clearly pointed out that neuroticism, which interacts with stressful life events, is a major risk factor for the development of major depressive disorder.[17] A previous study reported that neuroticism mediates the effect of paternal overprotection on depressive symptom severity, among only male patients with depression.[18] Our research group recently reported that parenting quality experienced in childhood affects depressive symptom severity in general adult male and female volunteers, through its effect on neuroticism.[15] Thus, it has been suggested that parenting quality experienced in childhood and neuroticism are interactively associated with depressive symptoms in adulthood.

Whereas the aforementioned neuroticism is a vulnerability to the onset of depression, the role of resilience in the prevention or amelioration of depression has recently attracted attention. Resilience is regarded as "the process of adapting well in the face of trauma, adversity, threats, significant stress sources, and tragedy".[19] In recent years, resilience has been regarded as a measure of stress coping, and an important therapeutic target for anxiety, depression, and stress responses.[20] Low resilience is reportedly associated with the development of depression, and resilience may ameliorate stress-induced depressive symptoms.[21,22] A meta-analysis of 38 controlled studies has reported that cognitive-behavioral interventions that enhance resilience are effective in improving depressive symptoms.[23] Interestingly, a decrease in resilience as evaluated by the Connor-Davidson Resilience Scale (CD-RISC), which is a self-rated scale, can be improved by pharmacological treatments, and predicts the responses to treatments for post-traumatic stress disorder.[20,24] Several studies to date have suggested that resilience is closely associated with depression and depressive symptoms, and plays a role in the prevention or improvement of depression and depressive symptoms. However, the clinical role of resilience in depression has not yet been fully elucidated.

We recently suggested that parental rearing quality experienced in childhood affects adulthood resilience and occupational stress, and that resilience mediates the effect of parental rearing quality experienced in childhood on occupational stress.[25] In other words, higher quality of parental care experienced in childhood increases resilience, decreases occupational stressors, and therefore decreases the physical and psychological stress response of workers.[25] On the other hand, parental overprotection in childhood has opposite effects to parental care, on resilience, occupational stressors, and physical and psychological stress responses.[25] Thus, these findings suggest that resilience is a mediator between parenting quality experienced in childhood and occupational stress, and has opposite effects to neuroticism, which is also a mediator between parenting quality experienced in childhood and occupational stress.[26] Several previous studies have shown that neuroticism mediates the effects of parental child-rearing experienced in childhood on depression and depressive symptoms.[15,18] Considering these theoretical models, it is assumed that resilience also acts as a mediator of the effects of parenting quality experienced in childhood on depression and depressive symptoms. Consistently, a previous study of people with congenital heart disease showed that the quality of parental child-rearing influenced resilience, and resilience influenced depressive symptoms, but a significant mediating effect of resilience between parental rearing quality and depressive symptoms was not verified.[27] Furthermore, to date, there has been no

study assessing the mediating effect of resilience between parenting quality experienced in childhood and the severity of depressive symptoms among the general adult population.

Considering the long interval from the experience of parental rearing in childhood to adulthood depressive symptoms, various personality traits have been hypothesized to act as mediators between these 2 factors.[15,28] Neuroticism reportedly mediates the effect of childhood parenting quality on depressive symptoms in the general population,[15] but there has been no study showing that resilience is a mediator between parental child-rearing quality experienced in childhood and the severity of depressive symptoms experienced in adulthood. As mentioned above, there is only 1 report demonstrating that resilience mediates the link between childhood parenting quality and occupational stress.[25] Furthermore, whether resilience and neuroticism interact with each other to affect depressive symptoms has not been analyzed to date. Therefore, we hypothesized that resilience acts as a mediator of the effect of parental child-rearing experienced in childhood on the severity of depressive symptoms in adulthood, and that resilience and neuroticism interact with each other to affect depressive symptoms. We conducted a cross-sectional study of adult volunteers utilizing a self-administered questionnaire survey and analyzed this hypothesis using structural equation modeling and hierarchical multiple regression analysis including interaction terms.

Subjects and Methods

Subjects

Self-report questionnaires were distributed to adult volunteers who were recruited using the convenience sampling method through our acquaintances at Tokyo Medical University for 1 year from April 2017 to April 2018. A total of 597 adult volunteers participated in the research. All participants gave written informed consent to participate in the research. Participant data were anonymized. A total of 528 general adult volunteers were included in the analyses, after excluding 69 volunteers with missing values. The following inclusion criteria were set: being over 20 years old. The following exclusion criteria were set: (a) having serious physical illnesses; and (b) having organic brain diseases. Individuals were informed that participation in this research was voluntary, their decision not to participate would not lead to any disadvantages, and that the data would be adequately managed and individual data would be protected. This study was conducted in accordance with the Declaration of Helsinki (amended in Fortaleza in 2013), and with approval from the Institutional Review Board of Tokyo Medical University (study approval no.: SH3502).

Questionnaires

The participants were asked to anonymously answer the following 4 questionnaires in addition to the questionnaire regarding their demographic and clinical characteristics.

Parental Bonding Instrument (PBI)

The PBI is a retrospective self-assessment measure of parental rearing attitudes experienced in childhood.[12] The Japanese version of the PBI was developed by Kitamura et al, and its validity has been confirmed.[29] The PBI consists of 2 subscales of "care" and "overprotection". A high score for "care" is regarded as indicating that a respondent was reared by their parents more appropriately (less rejection and indifference). A high score for "overprotection" is regarded as indicating that a respondent's parents were more overprotective (less encouragement of self-reliance). The PBI consists of 25 items (12 items for care and 13 items for overprotection), and each item is assessed using a 4-point Likert scale. In this study, the summary scores of each subscale for the father and mother were calculated, and the 4 subscores were used for analysis.

Eysenck Personality Questionnaire-Revised (EPQ-R) Short Version

The neuroticism subscale on the Japanese EPQ-R short version was used to assess neuroticism.[30] This subscale consists of 12 items, and each item is assessed using a 2-point scale of "yes" and "no". The reliability and validity of the Japanese EPQ-R short version was confirmed in our previous research.[31] The total score of the questionnaire was used for the study analyses.

Connor-Davidson Resilience Scale (CD-RISC)

The CD-RISC is a questionnaire for measuring resilience.[20] In this study, the Japanese version of the CD-RISC was used. The validity and reliability of the Japanese version of the CD-RISC was previously confirmed by Nakajima et al.[32] The CD-RISC consists of 25 items, and each item is evaluated using a 5-point Likert scale (not true at all [0 points], to true nearly all the time [4 points]). The total score was used for the analysis.

Patient Health Questionnaire-9 (PHQ-9)

The PHQ-9 is a self-reported scale used as a screening tool for major depressive episodes or as an indicator of depressive symptom severity.[33] The Japanese version of PHQ-9 was used in this study. The Japanese version of PHQ-9 was developed by Muramatsu et al, and its validity and reliability have been confirmed.[34] PHQ-9 consists of 9 items regarding depressive symptom severity during the previous 2 weeks, and each item is assessed using a 4-point Likert scale. The severity of depressive symptoms is evaluated using the total score on the PHQ-9, and the total score was used for the analysis.

Statistical Analyses

The associations of participants' clinical and demographic characteristics and questionnaire scores with the total score on the PHQ-9 were analyzed by the *t*-test or Pearson's correlation coefficient. Furthermore, the interaction between resilience and neuroticism was analyzed by hierarchical multiple regression analysis, with the dependent variable being the PHQ-9 summary score. These analyses were performed using SPSS for Windows version 27.0J (IBM, Armonk, NY, USA). Furthermore, to clarify whether resilience and neuroticism mediate the effect of parental rearing quality experienced in childhood on depressive symptom severity, these variables were analyzed by structural equation modeling using the robust maximum likelihood estimation method, and the standardized coefficients were calculated. For structural equation modeling, Mplus 8.5 software (Muthén & Muthén, Los Angeles, CA, USA) was utilized. Regarding goodness-of-fit indices for comprehensive judgment, the Root Mean Square Error Approximation (RMSEA) and Comparative Fit Index (CFI) were used. If the RMSEA was lower than 0.08, and the CFI was greater than 0.95, the model fit was regarded as being acceptable If the RMSEA was lower than 0.05, and the CFI was greater than 0.97, the model fit was regarded as being good.[35] A *p*-value of less than 0.05 was considered to indicate a statistically significant difference.

Results

Associations of Clinical and Demographic Characteristics and Questionnaire Scores with PHQ-9 Scores in the Study Population

Table 1 shows the associations of clinical and demographic characteristics and questionnaire scores with PHQ-9 summary scores in 528 adult volunteers. Women, lower education level, unmarried, living alone, current psychiatric

Table 1 Subject Characteristics, PBI, EPQ-R, and CD-RISC Scores and Their Association with PHQ-9 Score or Effect on PHQ-9 Score

Characteristic or Measure	Value (Number or Mean ± SD)	Correlation with PHQ-9 Score (*r*) or Effect on PHQ-9 Score (Mean ± SD, *t*-test)
Age (years)	41.4 ± 11.9	r = –0.026, p = 0.548
Sex (men: women)	233: 295	Men: 3.3 ± 3.8 vs women: 4.6 ± 4.3, $p < 0.001$ (*t*-test)
Education years	14.7 ± 1.8	r =–0.105, $p < 0.05$
Marital status (married: unmarried)	346: 178	Married: 3.6 ± 4.4 vs unmarried: 4.9 ± 3.9 $p < 0.001$ (*t*-test)

(*Continued*)

Table 1 (Continued).

Characteristic or Measure	Value (Number or Mean ± SD)	Correlation with PHQ-9 Score (*r*) or Effect on PHQ-9 Score (Mean ± SD, *t*-test)
Living alone (yes: no)	105: 423	Yes: 5.0 ± 4.8 vs no: 3.7 ± 3.9 $p < 0.05$
Number of children	1.4±1.3	$r = –0.028$, $p = 0.527$
Family history of psychiatric disease (yes: no)	52: 425	Yes: 4.3 ± 4.0 vs no: 4.0 ± 4.2 $p = 0.601$
Comorbidity of physical disease (yes: no)	159: 369	Yes: 4.3 ± 4.3 vs no: 3.9 ± 4.0 $p = 0.237$
Past history of psychiatric disease (yes: no)	64: 464	Yes: 6.6 ± 5.3 vs no: 3.6 ± 3.8 $p < 0.001$ (*t*-test)
Current psychiatric diseases (yes: no)	22: 497	Yes: 7.9 ± 5.0 vs no: 3.9 ± 4.0 $p < 0.001$ (*t*-test)
PHQ-9	4.00 ± 4.138	
PBI Paternal care Maternal care Paternal overprotection Maternal overprotection	 23.5 ± 8.1 28.1 ± 6.9 9.7 ± 6.9 9.6 ± 6.9	 $r = –0.177$, $p < 0.001$ $r = –0.260$, $p < 0.001$ $r = 0.219$, $p < 0.001$ $r = 0.240$, $p < 0.001$
EPQ-R score	4.4 ± 3.5	$r = 0.566$, $p < 0.001$
CD-RISC	55.0±17.4	$r = –0.391$, $p < 0.001$

Note: Data are presented as means ± standard deviation (SD) or numbers. *r* = Pearson's correlation coefficient.
Abbreviations: CD-RISC, Connor-Davidson Resilience Scale; EPQ-R, Eysenck Personality Questionnaire revised; PBI, Parental Bonding Instrument; PHQ-9, Patient Health Questionnaire-9.

disease, and psychiatric disease history showed significant associations with high PHQ-9 summary scores. Age, number of children, family history of psychiatric disease, and comorbidity of physical diseases were not significantly associated with PHQ-9 summary scores.

PBI care scores for the mother and father were significantly negatively correlated with PHQ-9 summary scores, and the higher the parental care received in childhood, the lower the depression scores in adulthood. PBI overprotection scores of the mother and father were significantly positively correlated with PHQ-9 summary scores, and the higher the parental overprotection in childhood, the higher the depression scores in adulthood. CD-RISC scores were significantly negatively correlated with PHQ-9 summary scores, and the higher the resilience, the lower the depression scores in adulthood. EPQ-R neuroticism scores were significantly positively correlated with the PHQ-9 summary scores, and the higher the neuroticism, the higher the depression scores in adulthood.

Analysis of the Interaction Between Neuroticism and Resilience by Hierarchical Multiple Regression Analysis

The interaction between EPQ-R (neuroticism) scores and CD-RISC (resilience) scores was analyzed by 2-step hierarchical multiple regression analysis with the dependent variable being PHQ-9 summary score (Table 2). In the first step, the EPQ-R neuroticism scores and the CD-RISC resilience scores were used as independent variables. EPQ-R scores and CD-RISC scores were significantly associated with PHQ-9 scores ($p < 0.001$). The adjusted coefficient of determination (R^2) was 0.342. In the second step, the interaction term between EPQ-R scores and CD-RISC scores was added as an independent variable. The interaction term between neuroticism scores and resilience scores was

Table 2 Hierarchical Multiple Regression Analysis of Depressive Symptoms (PHQ-9 Score) as the Dependent Variable with the Interaction Between Neuroticism and Resilience

Step 1			
	β	***p*-value**	**VIF**
Neuroticism (EPQ-R)	0.489	< 0.001	1.244
Resilience (CD-RISC)	–0.174	< 0.001	1.244
Adjusted R^2 = 0.342, F = 138.174, p < 0.001			
Step 2			
	β	*p*-value	VIF
Neuroticism	0.453	< 0.001	1.291
Resilience	–0.165	< 0.001	1.248
Neuroticism × resilience	–0.172	< 0.001	1.060
Adjusted R^2 = 0.369, F = 103.871, p < 0.001			
ΔR^2 = 0.0278, p < 0.001			

Note: β-value indicates the standardized partial regression coefficient.
Abbreviations: VIF, variance inflation factor; CD-RISC, Connor-Davidson Resilience Scale; PHQ-9, Patient Health Questionnaire-9.

significantly associated with PHQ-9 scores. The interaction term caused a significant R^2 change from the first step (ΔR^2 = 0.0278; p < 0.001).

Structural Equation Modeling

In Model 1, the latent variable of "care" consisted of 2 observed variables (maternal and paternal care) (Figure 1). Furthermore, CD-RISC score, neuroticism score on EPQ-R, and PHQ-9 summary score were used as observed variables in Model 1. The goodness of fit of the model was RMSEA = 0.000 and CFI = 1.000, which represents a good fit. Regarding the standardized coefficients from the latent variable to the observed variables, the standardized coefficient from "care" to maternal care (standardized coefficient β = 0.869) was greater than that from "care" to paternal care (β = 0.622). The latent variable "care" had a significant positive effect on CD-RISC score and significant negative effects on EPQ-R neuroticism

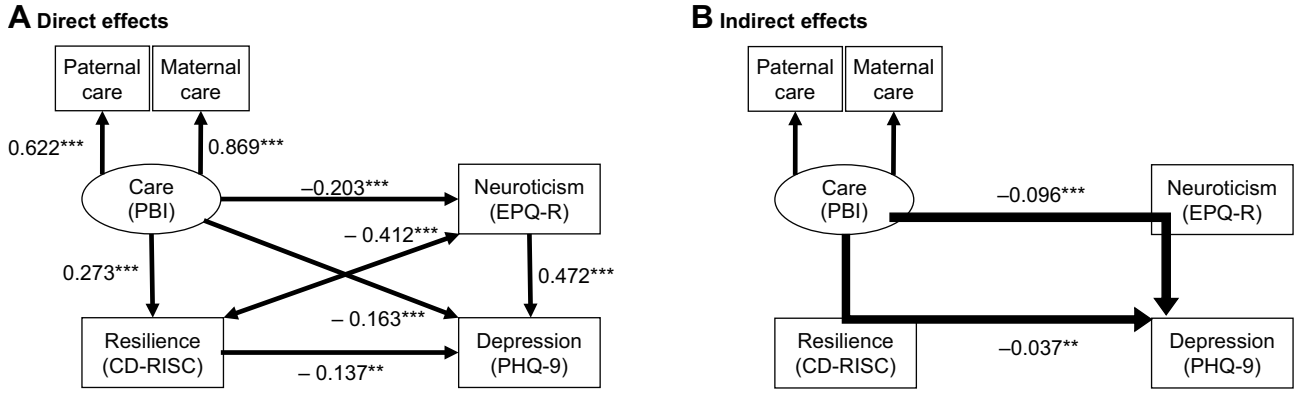

Figure 1 Results of the structural equation model with "care" as the latent variable, and resilience (CD-RISC), neuroticism (EPQ-R), and severity of depression (PHQ-9) as the observed variables. A structural equation model was created with the "care" subscale of the PBI, neuroticism evaluated by the shortened EPQ-R subscale, resilience evaluated by the CD-RISC, and depression evaluated by the PHQ-9 score. The latent variable is shown as an oval, and the observed variables are shown as rectangles. Direct effects (**A**) and indirect effects (**B**) between the variables are shown. The numbers show the standardized path coefficients (–1 ≤ β ≤ 1). **p < 0.01, ***p < 0.001.

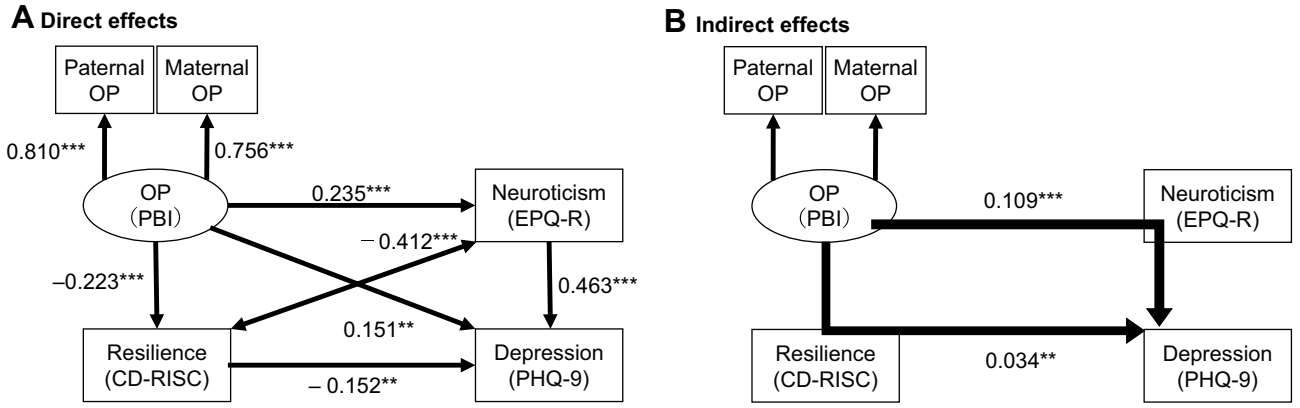

Figure 2 Results of the structural equation model with "overprotection (OP)" as the latent variable, and resilience (CD-RISC), neuroticism (EPQ-R), and severity of depression (PHQ-9) as the observed variables. A structural equation model was created with the "overprotection" subscale of the PBI, neuroticism evaluated by the shortened EPQ-R subscale, resilience evaluated by the CD-RISC, and depression evaluated by the PHQ-9 score. The latent variable is shown as an oval, and the observed variables are shown as rectangles. Direct effects (**A**) and indirect effects (**B**) between the variables are shown. The numbers show the standardized path coefficients ($-1 \leq \beta \leq 1$). $^{**}p < 0.01$, $^{***}p < 0.001$.

score and PHQ-9 score. CD-RISC score significantly decreased PHQ-9 score, although EPQ-R neuroticism score significantly increased PHQ-9 score. Furthermore, there was a significant negative correlation between EPQ-R neuroticism and CD-RISC scores. Regarding indirect effects, the latent variable "care" significantly decreased the PHQ-9 score through a positive effect on CD-RISC score ($\beta = -0.037$, $p = 0.002$). Furthermore, the latent variable "care" significantly decreased PHQ-9 score through a negative effect on EPQ-R neuroticism score ($\beta = -0.096$, $p < 0.001$). The adjusted R^2 was 0.369, indicating that this model explains 36.9% of the variation in depressive symptom severity.

In Model 2, the latent variable of "overprotection" consisted of 2 observed variables (maternal and paternal overprotection) (Figure 2). Furthermore, CD-RISC score, neuroticism score on EPQ-R, and PHQ-9 summary score were used as observed variables in Model 2. The goodness of fit of the model was RMSEA = 0.000 and CFI = 1.000, which indicates a good fit. Regarding the standardized coefficient from latent variable to observed variables, the standardization coefficient from "overprotection" to maternal overprotection ($\beta = 0.756$) was approximately equal to that of "overprotection" to paternal overprotection ($\beta = 0.810$). The latent variable "overprotection" had a significant negative effect on CD-RISC score and significant positive effects on EPQ-R neuroticism score PHQ-9 score. CD-RISC score significantly decreased PHQ-9 summary score, although EPQ-R neuroticism score significantly increased PHQ-9 summary score. Furthermore, EPQ-R neuroticism and CD-RISC scores showed a significant negative correlation. Regarding indirect effects, the latent variable "overprotection" significantly increased the PHQ-9 score through a negative effect on CD-RISC score ($\beta = 0.034$, $p = 0.003$). Furthermore, the latent variable "overprotection" significantly increased PHQ-9 score through a positive effect on EPQ-R neuroticism score ($\beta = 0.109$, $p < 0.001$). The adjusted R^2 was 0.366, which indicates that this model explains 36.6% of the variation in depressive symptom severity.

Discussion

In summary, the results of this study are as follows. The 2 structural equation models showed that overprotection significantly increased PHQ-9 score through attenuated resilience and enhanced neuroticism. On the other hand, care significantly decreased PHQ-9 score through enhanced resilience and attenuated neuroticism. In other words, resilience and neuroticism were found to be mediators of the effect of parental child-rearing on depressive symptom severity in adulthood. To our knowledge, this is the first study to date showing that resilience is a mediator between the quality of parental child-rearing experienced in childhood and depressive symptoms in adulthood, which is a strength of our research. Moreover, regarding the quality of parenting experienced in childhood, it is noteworthy that overprotection and care had opposite effects on both resilience and neuroticism. Furthermore, hierarchical multiple regression analysis showed that the interaction term between neuroticism and resilience scores was significantly associated with PHQ-9

summary score. This finding demonstrates that resilience acts on depressive symptoms by antagonizing neuroticism, which is a well-known risk factor for depressive symptoms in adulthood.[10]

There have been a few studies to date investigating the effects of parenting quality on depressive symptoms and resilience. In a study by Moon et al on adolescents with congenital heart disease, path analysis showed that parenting quality influences resilience, and resilience influences depressive symptoms.[27] Their results appear to show similar trends to our results. However, their study had limitations in that they were unable to clarify the statistically significant mediating effect of resilience between parental rearing and depressive symptoms, and furthermore, their findings are not applicable to the general public, because the study subjects were adolescents with congenital heart disease.

On the other hand, there have been several studies regarding the effects of childhood abuse and adverse childhood experiences (ACE) on resilience and depressive symptoms. Wang et al reported that neuroticism mediates the effects of childhood abuse on adulthood depressive symptoms in normative college students, and furthermore, resilience moderates the effect from childhood abuse to neuroticism.[36] Their study clarified the mediating effect of neuroticism, as was also suggested in our study, although their study did not analyze the moderating effect of resilience on the impact of neuroticism on depressive symptoms, which was a different point from our study.[36] Wingo et al found that resilience as a moderator alleviates the effects of various types of trauma experienced in one's lifetime on depressive symptoms, although their study also did not analyze the moderating effect of resilience on the impact of neuroticism on depressive symptoms.[37] As mentioned above, our study is the first to clarify that resilience moderates the effect of neuroticism on depressive symptoms, although there have been a few studies showing that resilience moderates the effects of abuse and trauma on depressive symptoms. On the other hand, Ward et al focused on ACE and analyzed whether resilience mediates the effect of ACE on depressive symptoms in adults aged 50 years and older.[38] However, a significant mediating effect of resilience was not detected in their study. There are 2 possible reasons why the results of the study by Ward et al differed from the results of the study by Wang et al and our study. First, Ward et al analyzed older people between 54 and 95 years, whereas the study by Wang et al and our study analyzed younger people and few older people. As Ward et al discussed, the discrepancies may be due to various events that ameliorate the effects of ACE on depressive symptoms over a long lifetime. The second reason for the discrepancies may be the difference in the questionnaires used to assess resilience. The study by Wang et al and our study used the CD-RISC to assess resilience, whereas the study by Ward et al used the resilience scale for older adults, which was developed by Hardy et al.[39]

There is also the idea that positive parenting (eg, providing warmth, support, and affection) is a resilience-enhancing factor.[40–43] A systematic review by Fritz et al reported that positive parenting had moderating and mediating effects on the effect of childhood adversity on mental health, although it was also suggested that there were discrepancies among previous reports.[40] Dubow et al showed that positive parenting has a moderating effect on the effects of ethnic-political conflicts on post-traumatic stress symptoms in adolescence.[43] Cui et al reported that a lack of positive parenting mediates the effect of parental marital problems on adolescent maladaptation, such as poor emotional well-being.[42] On the other hand, Masten et al reported that parenting quality did not show a moderating effect on the impacts of adverse life experiences on academic achievement, conduct, and peer social competence.[41] These previous studies suggest that positive parenting has a role as resilience. The possibility that positive parenting is a resilience-enhancing factor has also been suggested by several animal experiments.[40] Feder et al conducted a study in mice and suggested that appropriate parenting received from the mother may increase resilience through neurobiological mechanisms, although inappropriate parenting may cause epigenetic changes and decrease resilience, which may be transmitted to their offspring.[44] Thus, previous studies suggested that the parenting quality is closely associated with resilience, not only from a psychosocial point of view but also from a neurobiological point of view. Considering the findings of the above previous studies, a significant mediation effect of resilience between quality of parenting in childhood and depressive symptoms in adulthood, which we showed in this study, is considered to be evidence for the suggestion by Fritz et al that positive parenting is a resilience-enhancing factor.[40]

As mentioned above, Feder et al demonstrated that resilience is affected by a number of neurobiological and genetic factors, suggesting that interventions focusing on reversible factors might enhance resilience after birth.[44] Our findings that parenting quality influences adulthood depressive symptoms through resilience and neuroticism suggest that parental support and education are useful for reducing the risk of a child developing depressive symptoms after reaching

adulthood. Feder et al pointed out the possibility that cognitive-behavioral therapy can change a subject's tendency to optimism, which is a psychological attribute associated with resilience.[44] Although meta-analysis has shown that cognitive-behavioral therapy that enhances resilience improves depressive symptoms, it has not been investigated to date whether these interventions are effective in preventing the development and recurrence of depression.[23] Considering that resilience acts against neuroticism, which is a well-known risk factor for depressive symptoms,[9,10,17] it is expected that interventions for neuroticism, as well as increasing resilience will prevent the onset and exacerbation of depressive symptoms.

Limitations

There are several limitations to this study. First, although a study by Lu et al on adolescents suggested a close association between chronic disease and neuroticism,[45] we did not consider the influence of chronic diseases in adolescence on the association among parental rearing quality experienced in childhood, resilience, neuroticism, and depressive symptoms in adulthood. Furthermore, we did not consider the influence of several background factors, such as social status, which were shown to correlate with PBI scores in this study (data not shown). In other words, an alternative hypothesis is possible, in which background factors influenced our results. Secondly, although the path models used in our study appear to be acceptable when taking into account the chronological order of the targets of the questionnaires (ie, PBI for childhood, CD-RISC and EPQ-R for personality traits in adulthood, and PHQ-9 for present status), the design of our research is cross-sectional, and, therefore, a causal association between the variables remains unclear. A large-scale prospective investigation should be conducted in the future to confirm the causal association between these variables. Thirdly, because the subjects were adult volunteers in the community, our findings may not be applicable to patients with mood disorders. Finally, the quality of parenting experienced by the participants was assessed retrospectively using a self-administered questionnaire, and, therefore, the influence of recall bias needs to be considered.

Conclusions

This study suggested that both resilience and neuroticism are mediators between parental rearing quality experienced in childhood and depressive symptoms in adulthood. Furthermore, our results also suggested that resilience acts against the influence of neuroticism on depressive symptoms in adulthood, although we cannot deny the alternative hypothesis that other background factors, such as social status, influenced the findings. Our findings suggest that providing support and education to parents to enhance their child(ren)'s resilience may lead to the prevention of the future onset of depressive symptoms in their child(ren). Further studies on these interventions are anticipated in the future.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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