Neuropsychiatric Disease and Treatment

Dovepress
Taylor & Francis Group

Open Access Full Text Article

ORIGINAL RESEARCH

Effects of Parental Attitudes in Childhood on Depressive Symptoms Through Trait Anxiety and Negative Life Events in Adult Volunteers

Taito Hayashida[1,*], Masayuki Kikkawa[1,*], Jiro Masuya[1], Miki Ono[1], Shunichiro Ito[1], Rintaro Nibuya[1], Osamu Takashio[2], Yu Tamada[2], Naoki Hashimoto[3], Takeshi Inoue[1,4]

[1]Department of Psychiatry, Tokyo Medical University, Tokyo, Japan; [2]Department of Psychiatry, Tokyo Medical University Hachioji Medical Center, Tokyo, Japan; [3]Department of Psychiatry, Hokkaido University Graduate School of Medicine, Hokkaido, Japan; [4]Department of Psychiatry, Sapporo Hanazono Hospital, Hokkaido, Japan

*These authors contributed equally to this work

Correspondence: Takeshi Inoue, Department of Psychiatry, Tokyo Medical University, 6-7-1 Nishi-shinjuku, Shinjuku-ku, Tokyo, 160-0023, Japan, Tel +81-3-3342-6111, Email tinoue@tokyo-med.ac.jp

Background: The mechanism underlying how parental attitudes in childhood, trait anxiety (TA), and adult life events cause depression remains unclear. In this study, we investigated the associations among parental attitudes in childhood, TA, adult life events, and depressive symptoms to assess the mechanism of the development of depressive symptoms.

Participants and Methods: This study was conducted between January and August 2014. A total of 404 Japanese adult volunteers completed questionnaires, including Parental Bonding Instrument, State-Trait Anxiety Inventory Form Y, Life Experiences Survey, and Patient Health Questionnaire-9, to assess the parental attitudes of care and overprotection experienced in childhood, TA, life events, and depressive symptoms, respectively. The associations among these variables were analyzed by structural equation modeling.

Results: Parental attitude did not affect depressive symptoms directly, but affected TA directly. The influence of "care" on TA was negative, but that of "overprotection" on TA was positive. TA had a positive influence on negative life events (NLEs) and depressive symptoms in adulthood. Although NLEs in adulthood had a positive influence on depressive symptoms, positive life events in adulthood did not have any effect. Regarding indirect effects, parental attitude influenced NLEs and depressive symptoms through TA. TA worsened depressive symptoms through NLEs in adulthood. Regarding the parental attitude subscale of "care", this decreased depressive symptoms through TA and NLEs in adulthood. Care and overprotection showed opposite effects. The model fit was good or acceptable (Comparative Fit Index > 0.97 and Root Mean Square Error of Approximation < 0.08).

Conclusion: This study suggested that parental attitudes in childhood influence adult depressive symptoms through TA and NLEs. Improving the quality of child care may decrease TA, reduce NLEs in adulthood, and reduce depressive symptoms indirectly. Guidance for parents and interventions to reduce TA may be useful for the prevention of depression in adulthood.

Keywords: parental attitudes, trait anxiety, negative life events, depressive symptoms, structural equation modeling

Introduction

Depression is a common psychiatric disorder, with a lifetime prevalence in the Japanese population of 5.7%.[1] Depressive disorders were ranked 13th among the top 25 leading causes of disability-adjusted life-years in 2019, and second among the top 25 leading causes of years lived with disability.[2] Childhood abuse, inadequate parental bonding, genetic factors, neuroticism, physical illnesses, such as heart disease/cerebrovascular disease/cancer, and adult life events are associated with depression; particularly, childhood abuse is associated with increased suicide attempts.[3–12] In particular, large-scale prospective studies showed that depression is more likely to develop in the presence of genetic factors and neuroticism, together with stressful life events in adulthood.[4,6] Neuroticism reflects the tendency to experience negative effects, including irritability, anxiety, depression, anger, and embarrassment, and is the most well-known personality risk factor

Received: 3 November 2024
Accepted: 30 May 2025
Published: 15 June 2025

for depression.[13] Recently, among the several characteristics of neuroticism, trait anxiety (TA) has been identified as a major candidate for the risk of depression.[14–16]

Recently, our research group reported that TA mediates the effects of child abuse on depressive symptoms in adulthood.[17,18] Similar to TA, mediating effects on depressive symptoms were also observed for neuroticism, and hence both TA and neuroticism are commonly regarded as mediating factors in the association between child abuse and depression.[19] This similarity might be explained by the observation that neuroticism and TA share common characteristics and brain mechanisms.[20]

Similar to child abuse, inappropriate parental attitudes in childhood is a risk factor for depression and anxiety.[21–23] Alloy suggested that negative parenting practices and childhood abuse may be on a continuum; ie, low care may be similar to neglect.[8] However, regarding the concept of parental attitudes, high care is considered as positive parenting, and in this respect, it is quite different from childhood abuse, which does not include any positive effects. Therefore, childhood abuse and parental attitudes share some common characteristics, but are distinct concepts. Both positive and adverse childhood experiences play a major role in shaping mental health outcomes throughout a person's life, by decreasing and increasing mental health risks and are mediated by biological and social mechanisms.[24–26] Low care, overprotection, control, and authoritarianism from parents are associated with the development of several psychiatric disorders, including depressive and anxiety disorders.[22,23] Giakoumaki et al also reported that the experience of parental attitudes involving low care and high overprotection in childhood increases TA in adulthood.[27] Although low quality parental attitudes received from parents might facilitate depression by increasing TA, no study to date has investigated whether there are mediating effects in the associations among parental attitudes in childhood, TA, and depressive symptoms.

Depression can be triggered by life events experienced during adulthood.[5] Several stressors have been identified as risk factors for depression, including assault, financial difficulties, severe housing issues, serious illness or injury, legal trouble, loss of a close confidant, significant marital conflicts, robbery, and the death or serious illness of a person in the individual's social circle, etc.[5] A prospective study showed that more severe stressful life events are more likely to cause major depression.[6] Research has indicated that TA is associated with heightened stress sensitivity in adulthood, affecting both psychological and biological responses.[14,15,18] Therefore, a cascade from parental attitudes to TA, and then to stressful life events is plausible, and this link may induce or worsen depression. On the other hand, the subjective appraisal of life events is grouped into negative and positive, the former of which is designated as stressful life events.[28] The mediating role of negative life events (NLEs) comparing positive life events (PLEs) among parental attitudes, TA, and depression should be comparatively investigated.

Based on the above findings, in this study, we hypothesized that TA and NLEs in adulthood influence and link the effects of the quality of parental attitudes in childhood on adulthood depression. To test the hypothesis, we collected data regarding parental attitudes from parents, TA, life events, and depressive symptoms, from adult volunteers using a self-questionnaire survey, and analyzed their reciprocal associations by structural equation modeling.

Participants and Methods

Ethical Approval

This study was conducted in accordance with the Declaration of Helsinki (amended in Fortaleza 2013), with the approval of the Medical Ethics Review Boards of Tokyo Medical University and Hokkaido University (study approval numbers SH3308 and 013–0184, respectively). Written consent was obtained from all participants after informing them about the details of the study.

Participants

This study targeting Japanese adult volunteers was conducted from January to August 2014, as part of a larger study.[19,29] Volunteers were gathered through convenience sampling, by distributing flyers and spreading the information through the authors' personal networks around Hokkaido University Hospital. The inclusion criterion was being at least 20 years of age, and the exclusion criteria were having severe physical illnesses and severe psychiatric illnesses. Questionnaires were administered to adult volunteers, who consented to take part in this study, gave written consent, and completed valid

responses to the questionnaires. All questionnaires were administered on paper in 1 session, and were not repeated. The participants filled them out independently. Questionnaires were distributed to 853 Japanese adult volunteers, and 53.3% (455 volunteers) agreed to participate and submitted their responses in an anonymous fashion. It took 8 months to collect questionnaire answers from a sufficient sample size. The contents of the questionnaires were unlikely to be influenced by the season or any social events in Japan. As the responses of 51 participants were incomplete, 404 participants (88.8%; 220 men and 184 women; average age: 42.3 ± 11.9 years) were finally included in this study. As questionnaires, 1 questionnaire on demographic and clinical characteristics (sex, age, education years, current marital status, employment status, living alone or not, and current physical disease), and 4 questionnaires on parental attitude quality, TA, positive and negative change of life events, and depressive symptoms were distributed. Participants were additionally notified that their involvement in the study was completely voluntary, that they would not experience any disadvantages if they decided not to participate, and that the data would be coded in a nonidentifiable format to ensure that personal information remains confidential and not shared outside.

Questionnaires

Patient Health Questionnaire-9 (PHQ-9)

The PHQ-9 is a self-administered questionnaire for evaluating the severity of depressive symptoms.[30] It was developed by Spitzer et al in 1999.[30] Depressive symptoms during the previous 2 weeks are rated on a 4-point Likert scale (0 = not at all, 1 = several days, 2 = more than half the days, and 3 = nearly every day) for 9 items (eg, "Little interest or pleasure in doing things"). The Japanese version of the PHQ-9, which has been validated previously, was used.[31] In this study, the total score of the PHQ-9 was used for analysis. The Cronbach's α coefficient for the total score for the PHQ-9 was 0.849, indicating high internal consistency.

Parental Bonding Instrument (PBI)

The PBI is a self-administered questionnaire that evaluates the quality of parental attitudes that an individual experienced in childhood (until 16-years old), which is evaluated retrospectively from the participant's memory in adulthood.[32] It was developed by Parker et al in 1979.[32] The PBI consists of 25 questions (eg, "The parent was affectionate towards me" for care and "The parent tried to control everything I did" for overprotection), which are divided into 2 subscales (12 items about care and 13 items about overprotection), and asks about each of the mother and the father.[32] Each item is assessed using a 4-point Likert scale (very like, moderately like, moderately unlike, and very unlike). High care indicates that parents showed a lot of affection, emotional warmth, empathy, and intimacy, and were not indifferent or rejecting.[32] On the other hand, overprotection indicates that parents did not respect the independence of their children but had an imposing and disciplining attitude.[32] Depression is associated with low care and high overprotection.[11] Results of the PBI are stable over the long term.[33] In this study, the validated Japanese version of the PBI was used.[34] The Cronbach's α coefficients for the individual subscales of the PBI were found to be 0.909 for paternal care, 0.854 for paternal overprotection, 0.917 for maternal care, and 0.870 for maternal overprotection, and indicated high internal consistency.

State-Trait Anxiety Inventory form Y (STAI-Y)

The STAI-Y is a self-administered questionnaire that evaluates state anxiety and TA, and consists of 20 items each.[35] It was developed by Spielberger in 1983.[35] Each item for TA (eg, "I feel nervous and restless") was assessed using a 4-point Likert scale (1 = almost never, 2 = sometimes, 3 = often, and 4 = almost always). The present study focused on TA, which reflects a stable tendency to respond to anxiety-provoking experiences. The validated Japanese version of the STAI-Y was used in this study.[36] The Cronbach's α coefficient for the total score of the trait anxiety subscale was 0.919, indicating excellent internal consistency.

Life Experiences Survey (LES)

The LES is a self-administered questionnaire consisting of 57 items that ask about the experience of the participants in the previous 1 year, to assess life events in adulthood.[28] It was developed by Sarason et al in 1978.[28] Items (eg, "Death of a close family member", "Major change in social activities") are evaluated on a 7-point scale, from –3 (extremely negative) to +3 (extremely positive). The total positive score indicates the positive change score of life events, ie, the degree of positively perceived life events (PLEs), and the total negative score indicates the negative change score of life events, ie, the degree of negatively perceived life events (NLEs).[28] Our previous study confirmed the validity and

reliability of the Japanese version of the LES, as follows: the negative change score was significantly and positively correlated with depressive symptoms, state anxiety, and trait anxiety, but the positive change score was not significantly correlated with depressive symptoms, state anxiety, or trait anxiety.[37]

Data Analysis

Statistical analysis was conducted using SPSS Statistics version 28 software (IBM, Armonk, USA) and Mplus version 8.5 software (Muthén & Muthén, Los Angeles, USA). Pearson's correlation coefficient or the *t*-test was used to compare demographic characteristics and questionnaire data. Additionally, stepwise multiple regression analysis was performed with the PHQ-9 score as the dependent variable, using demographic information and questionnaire scores as independent variables (sex, age, education years, current marital status, current employment status, living alone, current physical disease, 4 PBI subscales [ie, maternal and paternal care; maternal and paternal overprotection], STAI-Y TA, and LES (positive change score and negative change score).

Robust maximum likelihood estimation was used to analyze the structural equation model, in which the latent variables, which consist of observed variables, were introduced. Structural equation modeling was considered to be an appropriate analyzing method because the association among the 4 factors is complex, and using only univariate or multiple regression analysis is not sufficient for analyzing the reciprocal associations of mediation or indirect effects. Furthermore, it was necessary to consider the influences of the father and mother in an integrated manner, using the latent variable combining paternal and maternal observed variables. Using the Comparative Fit Index (CFI) and Root Mean Square Error of Approximation (RMSEA), the goodness-of-fit of the model was evaluated comprehensively by combining multiple goodness-of-fit indices. An acceptable fit was defined as a CFI of more than 0.95, and an RMSEA of less than 0.08, and a good fit was defined as a CFI of more than 0.97 and an RMSEA less than 0.05.[38] All coefficients of the structural equation model were standardized (–1 to +1). Two structural equation models as described below were built using the PHQ-9 score, the 4 PBI subscale scores (maternal and paternal / care and overprotection), STAI-Y TA score, LES positive change score, and LES negative change score. Covariance structure analysis was performed using Mplus version 8.5 software, and complex associations and indirect effects among the 6 variables were analyzed.

Structural equation model 1 (see Figure 1): The hypothesis of this model is that care from parents in childhood influences TA, NLEs, PLEs, and depressive symptoms in adulthood, that TA influences NLEs, PLEs, and depressive symptoms in adulthood, and that NLEs and PLEs in adulthood influence depressive symptoms. Indirect effects of care on depressive symptoms through TA, NLEs, and PLEs were analyzed as well as the indirect effects of TA on depressive symptoms through NLEs and PLEs.

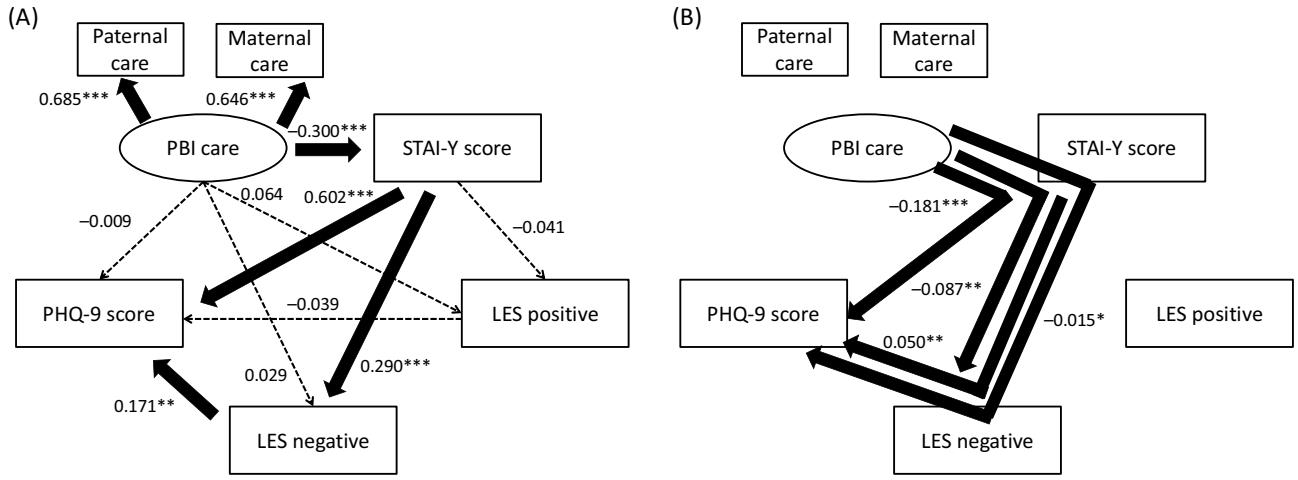

Figure 1 Results of the structural equation model with the parental attitude of "care" experienced in childhood as the latent variable, and trait anxiety (TA of STAI-Y), positive and negative life events (PLEs and NLEs of LES), and depression (PHQ-9) as the observed variables. The oval indicates the latent variable, and rectangles indicate the observed variables. The statistically significant paths are shown as arrows with solid lines, and the nonsignificant paths are shown as arrows with broken lines. Direct effects (**A**) and indirect effects (**B**) between the variables are shown. The numbers show the standardized path coefficients. *$p < 0.05$, **$p < 0.01$, and ***$p < 0.001$.

Structural equation model 2 (see Figure 2): The hypothesis of this model is that overprotection from parents in childhood influences TA, NLEs, PLEs, and depressive symptoms in adulthood, that TA influences NLEs, PLEs, and depressive symptoms in adulthood, and that NLEs and PLEs in adulthood influence depressive symptoms. Indirect effects of overprotection on depressive symptoms through TA, NLEs, and PLEs were analyzed as well as the indirect effects of TA on depressive symptoms through NLEs and PLEs.

This study was part of a larger study, in which the results of several questionnaires were analyzed. For this original large study, multivariable analysis of more than 20 independent variables was initially performed. In the present study, 404 participants were analyzed, excluding those with missing data. This is the reason for the sample size. A *p*-value of less than 0.05 was considered to indicate a statistically significant difference between groups.

Regarding the handling of missing values, as participants who left many questions unanswered on their questionnaires were excluded, the participants included in the analysis had only a few missing values, and no missing values for the path analysis. Imputation methods were not used for the analysis.

Results

Correlation or Association of Demographic Characteristics and Questionnaire Data with PHQ-9 Scores

Table 1 shows the correlation or association of demographic characteristics and questionnaire data of 404 adult volunteers with PHQ-9 score (the severity of depressive symptoms) at the time of the survey. Younger age, women, and unmarried participants were associated with high PHQ-9 scores. No association was found between other demographic factors and PHQ-9 score.

Maternal care and paternal care scores of the Parental Bonding Instrument (PBI, parental attitudes) were significantly negatively correlated with PHQ-9 score. PBI maternal overprotection score and PBI paternal overprotection score tended to correlate positively with PHQ-9 score, but their correlations were not statistically significant. State-Trait Anxiety Inventory Form Y (STAI-Y) trait anxiety (TA) score was significantly positively correlated with PHQ-9 score; the higher the TA, the more severe the depressive symptoms. LES positive change score (PLEs) did not correlate with PHQ-9 score, but LES negative change score (NLEs) correlated positively with PHQ-9 score.

Among the 404 participants, 74 participants (18.3%) had mild depression (5 ≤ PHQ-9 score ≤ 9), and 27 participants (6.7%) had major depression (PHQ-9 score ≥ 10).[39]

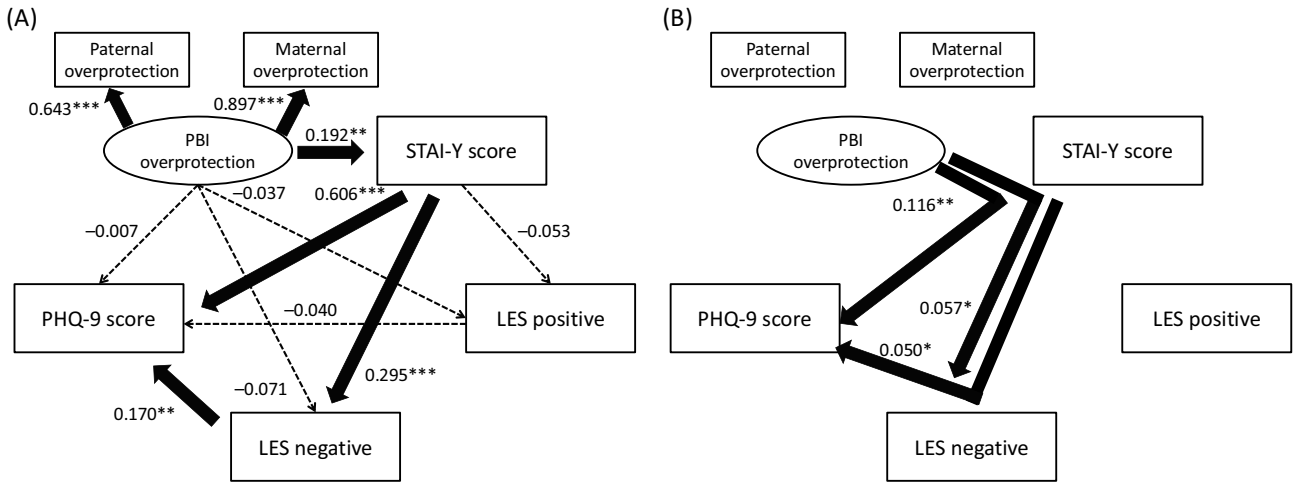

Figure 2 Results of the structural equation model with the parental attitude of "overprotection" experienced in childhood as the latent variable, and trait anxiety (TA of STAI-Y), positive and negative life events (PLEs and NLEs of LES), and depression (PHQ-9) as the observed variables. The oval indicates the latent variable, and rectangles indicate the observed variables. The statistically significant paths are shown as arrows with solid lines, and the nonsignificant paths are shown as arrows with broken lines. Direct effects (**A**) and indirect effects (**B**) between the variables are shown. The numbers show the standardized path coefficients. *$p < 0.05$, **$p < 0.01$, and ***$p < 0.001$.

https://doi.org/10.2147/NDT.S504446

Table 1 Demographic Characteristics, and PHQ-9, PBI, STAI-Y, and LES Scores and Their Correlation with PHQ-9 or Effects on PHQ-9 in 404 Adult Volunteers

Characteristic or Measure	Number or Mean ± SD	Correlation with PHQ-9 (*r*) or Effect on PHQ-9 Score (Mean ± SD of PHQ-9, *t*-Test)
Age	42.3 ± 11.9	$r = -0.138^{**}$
Sex (male: female)	220: 184	Male 2.9 ± 3.6 vs female 3.8 ± 4.1 (*t*-test)*
Education years	15.2 ± 2.0	$r = -0.049$, n.s.
Employment status (employed: nonemployed)	341: 56	Employed 3.3 ± 3.8 vs nonemployed 3.6 ± 4.4 (*t*-test), n.s.
Current marital status (married: unmarried)	286: 115	Married 3.0 ± 3.7 vs unmarried 4.0 ± 4.1 (*t*-test)*
Living alone (yes: no)	102: 294	Yes 3.6 ± 4.2 vs no 3.1 ± 3.7 (*t*-test), n.s.
Current physical disease (yes: no)	83: 317	Yes 3.9 ± 4.1 vs no 3.1 ± 3.7 (*t*-test), n.s.
PHQ-9	3.3 ± 3.8	
PBI		
Maternal care	27.7 ± 6.7	$r = -0.104^{*}$
Paternal care	23.9 ± 7.3	$r = -0.168^{**}$
Maternal overprotection	10.2 ± 6.8	$r = 0.096$, n.s.
Paternal overprotection	9.5 ± 6.1	$r = 0.085$, n.s.
STAI-Y trait anxiety score	41.7 ± 9.9	$r = 0.657^{**}$
LES		
Positive change	1.7 ± 3.0	$r = -0.056$, n.s.
Negative change	1.7 ± 3.1	$r = 0.335^{**}$

Notes: Data are presented as means ± standard deviations (SD) or numbers, *r* = Pearson correlation coefficient. $^{*}p < 0.05$, $^{**}p < 0.01$.
Abbreviations: n.s., not significant; PHQ-9, Patient Health Questionnaire-9; PBI, Parental Bonding Instrument; STAI-Y, State-Trait Anxiety Inventory-Form Y; LES, Life Experiences Survey.

Stepwise Multiple Regression Analysis of PHQ-9 Score

Table 2 shows the results of stepwise multiple regression analysis with PHQ-9 score as the dependent variable. The 14 independent variables listed in Table 1 were added to the analysis. The stepwise method demonstrated that the independent variables significantly predicting PHQ-9 scores are age, LES negative change score (NLEs), and STAI-Y TA score ($F = 61.204$; $p < 0.001$), and hence the other variables were excluded from the stepwise model. Multicollinearity was denied. The 4 subscales of the PBI, and LES positive change score were not significant independent variables in the multiple regression analysis of PHQ-9 score.

Analysis of Structural Equation Model 1

In structural equation model 1 of PBI parental care, "PBI care" was a latent variable consisting of 2 observed variables, namely, "maternal care" and "paternal care", as shown in Figure 1. Furthermore, PHQ-9, STAI-Y TA, and NLEs and

Table 2 Results of Stepwise Multiple Regression Analysis of PHQ-9

Independent Variable Selected By Stepwise Regression Analysis	Standardized Partial Regression Coefficient (Beta)	*p*-Value	VIF
Age	–0.101	0.009	0.992
LES negative change score	0.149	< 0.001	0.905
STAI-Y trait anxiety	0.597	< 0.001	0.904

Notes: Adjusted $R^2 = 0.323$, $F = 61.204$, $p < 0.001$. Dependent variable: PHQ-9 total score. Independent variables (14 in total): age, sex (male = 1, female = 2), education years, employment status (nonemployed = 1, employed = 2), current marital status (unmarried = 1, married = 2), living alone (no = 1, yes = 2), current physical disease (no = 1, yes = 2), LES (positive and negative change scores), PBI scores (maternal care, paternal care, maternal overprotection, and paternal overprotection), STAI-Y trait anxiety. Beta = standardized partial regression coefficient.
Abbreviations: VIF, Variance Inflation Factor; PHQ-9, Patient Health Questionnaire-9; PBI, Parental Bonding Instrument; LES, Life Experiences Survey; STAI-Y, State-Trait Anxiety Inventory Form Y.

PLEs of LES in adulthood were used as observed variables and were analyzed by a structural equation model. The goodness-of-fit of model 1 was a CFI of 0.977 and a RMSEA of 0.061, indicating an acceptable fit.

The standardized coefficients from the latent variable to the observed variables were 0.685 from "PBI care" to "paternal care", and 0.646 from "PBI care" to "maternal care", which were almost the same values. In this structural equation model expressing the association between variables, "PBI care" directly and significantly reduced STAI-Y TA score (–0.300, $p < 0.001$). However, the direct effects from "PBI care" to LES positive change score, LES negative change score, and PHQ-9 score were not significant (0.064, $p = 0.502$; 0.029, $p = 0.754$; and –0.009, $p = 0.867$, respectively). STAI-Y TA score directly and significantly increased LES negative change score and PHQ-9 score (0.290, $p < 0.001$; and 0.602, $p < 0.001$, respectively). STAI-Y TA score had no significant direct effect on LES positive change score (–0.041, $p = 0.519$). LES positive change score had no significant effect on PHQ-9 score, but LES negative change score directly and significantly increased PHQ-9 score (–0.039, $p = 0.242$; and 0.171, $p = 0.001$, respectively).

Regarding indirect effects, "PBI care" reduced the PHQ-9 score significantly through STAI-Y TA (–0.181, $p < 0.001$). "PBI care" also reduced LES negative change score significantly through STAI-Y TA (–0.087, $p = 0.001$). The indirect effects of "PBI care" on PHQ-9 score through both STAI-Y TA and LES negative change score were also significant (–0.015, $p = 0.030$). STAI-Y TA increased PHQ-9 score significantly through LES negative change score (0.050, $p = 0.007$). However, "PBI care" did not indirectly affect PHQ-9 score through LES positive change score or both STAI-Y TA and LES positive change score (–0.002, $p = 0.582$; and 0.000, $p = 0.555$, respectively).

The overall effect of "PBI care" on depressive symptoms was –0.203. The percentage mediation of STAI-Y TA on the overall association between "PBI care" and depressive symptoms was 96.6%.

Therefore, our results demonstrate that low parental care in childhood increases depressive symptoms indirectly through increasing STAI-Y TA and NLEs, supporting our hypotheses. Low parental care in childhood was also found to increase NLEs indirectly through increasing STAI-Y TA. The R^2 value was 0.458, indicating that this model explained 45.8% of the variability of depressive symptoms.

Analysis of Structural Equation Model 2

In structural equation model 2 of PBI parental overprotection, "PBI overprotection" was a latent variable consisting of 2 observed variables, namely, "maternal overprotection" and "paternal overprotection", as shown in Figure 2. PHQ-9, STAI-Y TA, and NLEs and PLEs of LES in adulthood were used as observed variables, and analyzed using a structural equation model (Figure 2). The goodness-of-fit of model 2 was a CFI of 0.989 and a RMSEA of 0.047, indicating a good fit.

The standardized coefficients from the latent variables to the observed variables were 0.643 from "PBI overprotection" to "paternal overprotection", and 0.897 from "PBI overprotection" to "maternal overprotection", indicating that overprotection from the mother has stronger effects than that of the father. In this model, "PBI overprotection" directly and significantly increased STAI-Y TA score (0.192, $p = 0.003$), but did not directly affect LES positive change score, LES negative change score, or PHQ-9 score (–0.037, $p = 0.500$; –0.071, $p = 0.225$; and –0.007, $p = 0.875$, respectively).

Regarding indirect effects, "PBI overprotection" increased PHQ-9 score significantly through its effects on the STAI-Y TA (0.116, $p = 0.004$). "PBI overprotection" indirectly and significantly increased LES negative change score through STAI-Y TA (0.057, $p = 0.012$). In addition, STAI-Y TA indirectly and significantly increased PHQ-9 score through LES negative change score (0.050, $p = 0.011$). "PBI overprotection" did not indirectly affect PHQ-9 score through both STAI-Y TA and LES negative change score (0.010, $p = 0.082$). Furthermore, "PBI overprotection" did not indirectly affect PHQ-9 score through LES positive change score, LES negative change score, or both STAI-Y TA and LES positive change score (0.001, $p = 0.597$; –0.012, $p = 0.220$; and 0.000, $p = 0.435$, respectively). "PBI overprotection" did not indirectly affect LES positive score through STAI-Y TA (–0.010, $p = 0.352$).

The overall effect of "PBI overprotection" on depressive symptoms was 0.108. The percentage mediation of STAI-Y TA on the overall association between "PBI overprotection" and depressive symptoms was 116.7%, because part of the mediation reduced depressive symptoms.

Therefore, our results demonstrate that high parental overprotection in childhood increases depressive symptoms indirectly through increasing STAI-Y TA, supporting our hypotheses. In addition, high parental overprotection was also

found to increase NLEs indirectly through increasing STAI-Y TA. The R^2 value was 0.458, indicating that this model explains 45.8% of the variability of depressive symptoms.

It may appear that the 2 above separate models (one focusing on "care", the other on "overprotection") can be combined into a single, more complex model. However, the model fit of this combined model was poor, and we hence could not adopt this model. The RMSEA was 0.168 (> 0.08), and the CFI was 0.823 (< 0.95).

Discussion

The primary finding of this study is that the quality of parental attitudes, which includes parental care and overprotection, experienced in childhood influences depressive symptoms in adulthood indirectly through affecting TA and negative (stressful) life events. TA and NLEs may hence be intermediate factors for the effect of parental attitudes on depressive symptoms. Furthermore, TA enhances depressive symptoms not only directly, but also indirectly through its effects on NLEs. To our knowledge, this is the first study to report on the associations among 4 factors, namely, parental attitudes in childhood, TA, life events in adulthood, and depressive symptoms in adult volunteers.

Low care from parents was reported to be a risk factor for depression in adulthood.[40] Several earlier studies have reported that personality traits, such as neuroticism and self-esteem, are mediators of the influences of parental attitudes in childhood on depression.[22,29,41] In the model used in this study, parental attitudes in childhood did not affect depressive symptoms directly, but did so only through pathways including TA. This suggests that TA is a crucial factor in the relationship between parental attitudes and depressive symptoms, and that other psychological factors are also involved in depressive symptoms only via their relationship with TA. Thus, we found that stress in adulthood influences the relationship between parental attitudes in childhood and adult depressive symptoms, but only in conjunction with TA.

NLEs contribute to the recurrence of major depression, and to intensify depressive symptoms.[5,6,16,37,42,43] Furthermore, NLEs affect depression indirectly, by influencing maladaptive cognitive emotion regulation strategies.[44] These previous findings are partially consistent with the adverse life events affecting depressive symptoms in the present study. However, it is worth noting that the results of the present study suggest that PLEs do not influence depressive symptoms, in contrast to NLEs. NLEs cause an immediate mobilization in terms of strong physiological, cognitive, emotional and social responses, which may lead to long-term distress if not dampened down, whereas PLEs help to anchor the person's life story and identity into the cultural norms of his or her society, which may act as a buffer against emotional distress.[45] Our study group has also documented that PLEs improve well-being (positive affect), whereas NLEs do not influence this, and that childhood stress is associated with NLEs but not with PLEs.[46] The present study showed that NLEs and PLEs are differently involved in the chain reaction among parental attitudes in childhood, TA, and depressive symptoms. This finding indicates that the intervention for PLEs may not be beneficial for decreasing depressive symptoms and that interventions for reducing NLEs may instead be beneficial.

Consistent with the findings of this study, previous studies have reported that TA is a full mediator of the association between stress exposure, including child abuse, major life events, and depression.[18,47] Weger and Sandi proposed a theoretical model in which high TA leads to the development of depression in combination with environmental factors, such as intense stress or long-term NLEs.[15] In their model, biological mechanisms, such as genetic factors, the hypothalamus-pituitary axis, mitochondrial function, and neurotransmitter signaling might contribute towards the high anxiety trait phenotype.[15] High TA creates a vulnerability to developing psychopathologies, including depression, but only when coupled with adverse environmental factors, such as NLEs. In addition, inappropriate parental attitudes in childhood may increase TA together with biological mechanisms, leading to vulnerability to stress.

The following are the clinically significant points of our present study. The background of TA that precedes depression may be associated with the parental attitudes in childhood. Accordingly, even for adult patients, it is necessary to pay close attention to the relationship between the patient and their parents. Cognitive vulnerability reportedly mediates the effect of the parental attitudes received from the parents on the patient's depression.[8] Therefore, in the treatment of such patients who have problems with the parental attitudes they received during their childhood, reducing TA and correcting cognitive distortion, such as cognitive-behavioral therapy, would be most desirable. As there has been a recent report that exercise reduces TA, more exercise is encouraged in such patients to reduce their TA.[48]

This study has some limitations. Because this study used self-administered questionnaires, which partly rely on the participant's memory of previous events, the existence of recall bias should be considered. Moreover, because the design of this study is cross-sectional, the causal association of the structural equation models cannot be concluded. There may be the possibility of reverse causality or reverse direction of the paths of our model. In addition, other psychological, cultural, or social factors should also be considered as confounding variables. In the future, by performing long-term prospective follow-up studies, the relationship between parental attitudes in childhood, TA, life events, and depression should be confirmed. Furthermore, as most of the participants in this study were healthy adult volunteers, the results may not completely apply to patients with depression. Finally, as the participants were gathered by convenience sampling via personal networks, a lack of diversity in the sample and cultural biases may have occurred.

Conclusions

This study showed that NLEs and TA are involved in the indirect effects of parental attitudes in childhood on depressive symptoms in adulthood. Our findings suggest that appropriate parental attitude qualities would lead to less TA in children, as well as reduce NLEs in their adulthood, and thereby indirectly reduce the number of patients with depression. Therefore, guidance for parents and interventions to reduce TA may be useful for the prevention of depression in adulthood.

Acknowledgments

We thank Dr. Helena Popiel of the Center for International Education and Research, Tokyo Medical University, for editorial review of the manuscript.

Author Contributions

All authors made a significant contribution to the work reported, whether 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.

Funding

This work was supported partly by a Grant-in-Aid for Scientific Research (no. 21K07510 to T. Inoue) from the Japan Society for the Promotion of Science, and by Japan Agency for Medical Research and Development (AMED) (grant no. JP23rea522113 to T. Inoue).

Disclosure

Jiro Masuya has received personal compensation from Otsuka Pharmaceutical, Eli Lilly, Astellas, and Meiji Yasuda Mental Health Foundation; grants from Pfizer. Yu Tamada has received personal compensation from Otsuka Pharmaceutical, Sumitomo Pharma, Eisai, MSD, and Meiji Seika Pharma. Osamu Takashio has received personal compensation from EA Pharma, Eisai, Janssen Pharmaceutical, Kyowa Pharmaceutical Industry, Meiji Seika Pharma, MSD, Otsuka Pharmaceutical, Sumitomo Pharma, Viatris Pharmaceuticals Japan Inc. and Takeda Pharmaceutical. Naoki Hashimoto has received personal compensation from Janssen Pharmaceutical K.K., Otsuka Pharmaceutical, Meiji Seika Pharma, Nippon Boehringer Ingelheim Co., Yoshitomiyakuhin, Sumitomo Pharma, Takeda Pharmaceutical; consulting fees from Nippon Boehringer Ingelheim Co. Takeshi Inoue has received personal compensation from Mochida Pharmaceutical, Takeda Pharmaceutical, Eli Lilly, Janssen Pharmaceutical, MSD, Yoshitomiyakuhin, Mitsubishi Tanabe Pharma, Meiji Seika Pharma, Lundbeck, Nippon Boehringer Ingelheim Co., and Viatris Pharmaceuticals Japan Inc.; grants and personal compensation from Shionogi, Eisai, Otsuka Pharmaceutical, Sumitomo Pharma, Daiichi Sankyo, and Kyowa Pharmaceutical Industry; and is a member of the advisory boards of Viatris Pharmaceuticals Japan Inc., Takeda Pharmaceutical, Nippon Boehringer Ingelheim Co., and Otsuka Pharmaceutical. All other authors declare that they have no actual or potential conflicts of interest associated with this study.

References

1. Ishikawa H, Tachimori H, Takeshima T, et al. Prevalence, treatment, and the correlates of common mental disorders in the mid 2010's in Japan: the results of the world mental health Japan 2nd survey. *J Affect Disord*. 2018;241:554–562. doi:10.1016/j.jad.2018.08.050
2. GBD Mental Disorders Collaborators. Global, regional, and national burden of 12 mental disorders in 204 countries and territories, 1990-2019: a systematic analysis for the Global Burden of Disease Study 2019. *Lancet Psychiatry*. 2022;9(2):137–150. doi:10.1016/S2215-0366(21)00395-3.
3. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders: DSM-5*. Washington, D.C: American Psychiatric Publication Inc.; 2013.
4. Caspi A, Sugden K, Moffitt TE, et al. Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. *Science*. 2003;301 (5631):386–389. doi:10.1126/science.1083968
5. Kendler KS, Karkowski LM, Prescott CA. Causal relationship between stressful life events and the onset of major depression. *Am J Psychiatry*. 1999;156(6):837–841. doi:10.1176/ajp.156.6.837
6. Kendler KS, Kuhn J, Prescott CA. The interrelationship of neuroticism, sex, and stressful life events in the prediction of episodes of major depression. *Am J Psychiatry*. 2004;161(4):631–636. doi:10.1176/appi.ajp.161.4.631
7. Sadock B, Sadock V, Ruiz P. *Kaplan & Sadock's Synopsis of Psychiatry: Behavioral Sciences/Clinical Psychiatry. Eleventh Edition* ed. Philadelphia: Lippincott Williams & Wilkins, Inc.; 2014.
8. Alloy LB, Abramson LY, Smith JM, Gibb BE, Neeren AM. Role of parenting and maltreatment histories in unipolar and bipolar mood disorders: mediation by cognitive vulnerability to depression. *Clin Child Fam Psych*. 2006;9(1):23–64. doi:10.1007/S10567-006-0002-4
9. Evans DL, Charney DS, Lewis L, et al. Mood disorders in the medically ill: scientific review and recommendations. *Biol Psychiatry*. 2005;58 (3):175–189. doi:10.1016/j.biopsych.2005.05.001
10. Anwar N, Kuppili PP, Balhara YPS. Depression and physical noncommunicable diseases: the need for an integrated approach. *WHO South East Asia J Public Health*. 2017;6(1):12–17. doi:10.4103/2224-3151.206158
11. Parker G. Parental 'affectionless control' as an antecedent to adult depression. A risk factor delineated. *Arch Gen Psychiatry*. 1983;40(9):956–960. doi:10.1001/archpsyc.1983.01790080038005
12. Ng QX, Yong BZJ, CYX H, Lim DY, Yeo WS. Early life sexual abuse is associated with increased suicide attempts: an update meta-analysis. *J Psychiatr Res*. 2018;99:129–141. doi:10.1016/j.jpsychires.2018.02.001
13. McCrae RR, Costa Jr PT. Validation of the five-factor model of personality across instruments and observers. *J Pers Soc Psychol*. 1987;52 (1):81–90. doi:10.1037//0022-3514.52.1.81
14. Sandi C, Richter-Levin G. From high anxiety trait to depression: a neurocognitive hypothesis. *Trends Neurosci*. 2009;32(6):312–320. doi:10.1016/j.tins.2009.02.004
15. Weger M, Sandi C. High anxiety trait: a vulnerable phenotype for stress-induced depression. *Neurosci Biobehav Rev*. 2018;87:27–37. doi:10.1016/j.neubiorev.2018.01.012
16. Yang S, Huang P, Li B, Gan T, Lin W, Liu Y. The relationship of negative life events, trait-anxiety and depression among Chinese university students: a moderated effect of self-esteem. *J Affect Disord*. 2023;339:384–391. doi:10.1016/j.jad.2023.07.010
17. Deguchi A, Masuya J, Naruse M, et al. Rumination mediates the effects of childhood maltreatment and trait anxiety on depression in non-clinical adult volunteers. *Neuropsychiatr Dis Treat*. 2021;17:3439–3445. doi:10.2147/NDT.S332603
18. Uchida Y, Takahashi T, Katayama S, et al. Influence of trait anxiety, child maltreatment, and adulthood life events on depressive symptoms. *Neuropsychiatr Dis Treat*. 2018;14:3279–3287. doi:10.2147/NDT.S182783
19. Ono K, Takaesu Y, Nakai Y, et al. Associations among depressive symptoms, childhood abuse, neuroticism, and adult stressful life events in the general adult population. *Neuropsychiatr Dis Treat*. 2017;13:477–482. doi:10.2147/NDT.S128557
20. Bishop S, Forster S. Trait anxiety, neuroticism, and the brain basis of vulnerability to affective disorder. In: Armony J, Vuilleumier P, editors. *The Cambridge Handbook of Human Affective Neuroscience*. Cambridge: Cambridge University Press; 2013:553–574.
21. Chubar V, Van Leeuwen K, Bijttebier P, et al. Gene-environment interaction: new insights into perceived parenting and social anxiety among adolescents. *Eur Psychiatry*. 2020;63(1):e64. doi:10.1192/j.eurpsy.2020.62
22. Enns MW, Cox BJ, Clara I. Parental bonding and adult psychopathology: results from the US National Comorbidity Survey. *Psychol Med*. 2002;32 (6):997–1008. doi:10.1017/s0033291702005937
23. Eun JD, Paksarian D, He JP, Merikangas KR. Parenting style and mental disorders in a nationally representative sample of US adolescents. *Soc Psychiatry Psychiatr Epidemiol*. 2018;53(1):11–20. doi:10.1007/s00127-017-1435-4
24. Fritz J, de Graaff AM, Caisley H, van Harmelen AL, Wilkinson PO. A systematic review of amenable resilience factors that moderate and/or mediate the relationship between childhood adversity and mental health in young people. *Front Psychiatry*. 2018;9:230. doi:10.3389/fpsyt.2018.00230
25. Felitti VJ, Anda RF, Nordenberg D, et al. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. The Adverse Childhood Experiences (ACE) Study. *Am J Prev Med*. 1998;14(4):245–258. doi:10.1016/s0749-3797(98)00017-8
26. Kuzminskaite E, Penninx BWJH, van Harmelen AL, Elzinga BM, Hovens JGFM, Vinkers CH. Childhood trauma in adult depressive and anxiety disorders: an integrated review on psychological and biological mechanisms in the NESDA cohort. *J Affect Disord*. 2021;283:179–191. doi:10.1016/j.jad.2021.01.054
27. Giakoumaki SG, Roussos P, Zouraraki C, et al. Sub-optimal parenting is associated with schizotypic and anxiety personality traits in adulthood. *Eur Psychiatry*. 2013;28(4):254–260. doi:10.1016/j.eurpsy.2012.07.002
28. Sarason IG, Johnson JH, Siegel JM. Assessing the impact of life changes: development of the Life Experiences Survey. *J Consult Clin Psychol*. 1978;46(5):932–946. doi:10.1037/0022-006X.46.5.932
29. Ono Y, Takaesu Y, Nakai Y, et al. The influence of parental care and overprotection, neuroticism and adult stressful life events on depressive symptoms in the general adult population. *J Affect Disord*. 2017;217:66–72. doi:10.1016/j.jad.2017.03.058
30. Spitzer RL, Kroenke K, Williams JB, the Patient Health Questionnaire Primary Care Study Group. Validation and Utility of a Self-report Version of PRIME-MD The PHQ Primary Care Study. *JAMA*. 1999;282(18):1737–1744. doi:10.1001/jama.282.18.1737
31. Muramatsu K, Miyaoka H, Kamijima K, et al. The patient health questionnaire, Japanese version: validity according to the mini-international neuropsychiatric interview-plus. *Psychol Rep*. 2007;101(3 Pt 1):952–960. doi:10.2466/pr0.101.3.952-960

32. Parker G, Tupling H, Brown LB. A parental bonding instrument. *Br J Med Psychol*. 1979;52(1):1–10. doi:10.1111/j.2044-8341.1979.tb02487
33. Wilhelm K, Niven H, Parker G, Hadzi-Pavlovic D. The stability of the Parental Bonding Instrument over a 20-year period. *Psychol Med*. 2005;35 (3):387–393. doi:10.1017/s0033291704003538
34. Kitamura T, Suzuki T. A validation study of the Parental Bonding Instrument in a Japanese population. *Jpn J Psychiatry Neurol*. 1993;47(1):29–36. doi:10.1111/j.1440-1819.1993.tb02026.x
35. Spielberger CD. *Manual for the State-Trait Anxiety Inventory STAI (Form Y)*. Palo Alto: Consulting Psychologists Press; 1983.
36. Hidano N, Fukuhara M, Iwawaki M, Soga S, Spielberger CD. *State-Trait Anxiety Inventory-Form JYZ*. Tokyo: Jitsumu Kyoiku Shuppan; 2000.
37. Nakai Y, Inoue T, Toda H, et al. The influence of childhood abuse, adult stressful life events and temperaments on depressive symptoms in the nonclinical general adult population. *J Affect Disord*. 2014;158:101–107. doi:10.1016/j.jad.2014.02.004
38. Schermelleh-Engel K, Moosbrugger H, Müller H. Evaluating the fit of structural equation models: tests of significance and descriptive goodness-of-fit measures. *MPR Online*. 2003;8:23–74.
39. Fukunishi T, Ono M, Kasuya K, et al. Investigation of risk factors associated with the development of depressive symptoms in healthy subjects exposed to long-term stress: a prospective study of the Japanese Antarctic research expedition wintering party. *Neuropsychopharmacol Rep*. 2024;44(4):821–828. doi:10.1002/npr2.12479
40. Parker G, Hadzi-Pavlovic D, Greenwald S, Weissman M. Low parental care as a risk factor to lifetime depression in a community sample. *J Affect Disord*. 1995;33(3):173–180. doi:10.1016/0165-0327(94)00086-o
41. Hayashida T, Higashiyama M, Sakuta K, et al. Subjective social status via mediation of childhood parenting is associated with adulthood depression in non-clinical adult volunteers. *Psychiatry Res*. 2019;274:352–357. doi:10.1016/j.psychres.2019.02.061
42. Nakai Y, Inoue T, Chen C, et al. The moderator effects of affective temperaments, childhood abuse and adult stressful life events on depressive symptoms in the nonclinical general adult population. *J Affect Disord*. 2015;187:203–210. doi:10.1016/j.jad.2015.08.011
43. Kendler KS, Thornton LM, Gardner CO. Stressful life events and previous episodes in the etiology of major depression in women: an evaluation of the "kindling" hypothesis. *Am J Psychiatry*. 2000;157(8):1243–1251. doi:10.1176/appi.ajp.157.8.1243
44. Stikkelbroek Y, Bodden DH, Kleinjan M, Reijnders M, van Baar AL. Adolescent depression and negative life events, the mediating role of cognitive emotion regulation. *PLoS One*. 2016;11:e0161062. doi:10.1371/journal.pone.0161062
45. Berntsen D, Rubin DC, Siegler IC. Two versions of life: emotionally negative and positive life events have different roles in the organization of life story and identity. *Emotion*. 2011;11(5):1190–1201. doi:10.1037/a0024940
46. Kanai Y, Takaesu Y, Nakai Y, et al. The influence of childhood abuse, adult life events, and affective temperaments on the well-being of the general, non-clinical adult population. *Neuropsychiatr Dis Treat*. 2016;12:823–832. doi:10.2147/NDT.S100474
47. Kok L, Sep MS, Veldhuijzen DS, et al. Trait anxiety mediates the effect of stress exposure on post-traumatic stress disorder and depression risk in cardiac surgery patients. *J Affect Disord*. 2016;206:216–223. doi:10.1016/j.jad.2016.07.020
48. Kikkawa M, Shimura A, Nakajima K, et al. Mediating effects of trait anxiety and state anxiety on the effects of physical activity on depressive symptoms. *Int J Environ Res Public Health*. 2023;20(7):5319. doi:10.3390/ijerph20075319