SCIENCE SET
OPEN ACCESS PUBLISHERS

Research Article

Journal of Clinical and Biomedical Advances

The Effect of Trait Anger on Depression from the Perspective of Age and Gender—A Moderated Mediating Effect with Age as the Moderating Variable

Zhe Han

Northeast Normal University, Changchun

***Corresponding author:** Zhe Han, Northeast Normal University, Changchun.

Submitted: 12 March 2024 **Accepted:** 18 March 2024 **Published:** 25 March 2024

Citation: *Zhe Han (2024) The Effect of Trait Anger on Depression from the Perspective of Age and Gender—A Moderated Mediating Effect with Age as the Moderating Variable. J Clin Bio Med Adv 3(2), 01-14.*

Abstract

Background: *Previous studies have shown that there is a positive correlation between anger and depression, which are usually in the form of "comorbid". However, the complex relationship mechanism of the interaction between anger and depression is rarely involved. Inspired by the traditional Chinese medicine theory of "anger beyond depression", this paper explores the formation mechanism of endogenous factors of trait anger and reactive factors of life events on depression and the influence of age and gender on the mechanism of depression.*

Methods: *In this study, 89 normal healthy subjects were randomly selected as the reference group, and 115 outpatients with depression were selected as the control group. The above variables were analyzed using the statistical software SPSS and PROCESS.*

Results: *It is concluded that trait anger has a significant complete mediating effect on depression with life events as a mediator($R1^2$=0.05.F1=6.30; $R2^2$=0.13.F2=8.02) for the control group. On this basis, we will further explore the moderating effect of age on the above mediations and the gender differences in depression($R1^2$=0.09.F1=3.56; $R2^2$=0.13.F2=8.02) to obtain new depression diagnoses, intervention paths and secondary prevention measures.*

Conclusions: *This study is as follows: (1) There is a significant and complete mediating effect of trait anger on depression with life events as mediating variables in the control group. On the other hand, there is no significant mediating effect on this relationship in the reference group. (2) There was a significant gender difference in depression in the reference group, but the gender difference in depression in the control group was not significant. (3) Age has a significant moderating effect on the end of the above mediating effect (trait anger to life events) in the control group. When the age value is greater than the critical value, it can significantly positively predict that age moderates the mediating effect with depression, but there is no significant correlation between age and depression in the reference group.*

A Statement of Public Significance: *These conclusions explain the effects of gender on depression and age as a moderating variable in the mediating model, which provides a new perspective and thinking for the diagnosis and prevention of depression.*

Keywords: Trait Anger, Life Event, Depression, Age, Gender

Introduction

Major depression disorder (MDD) is also called depression. It is a mental disorder with a variety of clinical manifestations, such as persistent depression. The core symptoms are depression, lack of interest and pleasure, loss of will and behavior, which are called "three low" symptoms [1].

It has been shown that teenagers with depression express more anger than nondepressed teenagers through family interactions [2]. Grant et al. suggested that anger is related to depression and tends to occur at the same time [3]. Another study showed a moderate relativity between anger and depression [4]. Mohammad et al. found that there is a positive correlation between anger and depression in patients with clinical depression through a questionnaire survey of patients with MDD [5].

Previous studies have shown a positive correlation between anger and depression [6]. Many studies have found that there is a close relationship between anger and depression, so it is often in the form of "codisease" [3, 6, 5, 2]. However, depression is heterogeneous, so there may be multiple variables affecting it [7]. It is speculated that the components of anger may have a direct or indirect effect on depression with other variables. Some studies have shown that anger is related to sources of mental stress. (Amanda et al., 2015).

Beck & Alfird considered that depression can be divided into two categories, endogenous depression and reactive depression, according to different internal and external factors [8]. Endogenous depression is mainly caused by internal psychological factors (such as Strait anger), while reactive depression is caused by external stress. Stepanichev M. Y. et al. considered that severe acute stress events or mild chronic stress factors may induce depression [9]. In an animal experiment, male Wistar rats were exposed to two different chronic stress modes. The levels of cortisol and passive floating behavior in brain tissue and blood were measured, which further aggravated the depressive symptoms. Reactive life stress events are the direct factors leading to depression.

According to the epidemiological statistics of depression, age and gender also have a significant impact on depression [10]. Therefore, we explored the different effects of variables such as trait anger, life event stress, depression and gender with age as the moderator of depression. It is particularly important for the pathogenesis of depression, the choice of treatment path and the secondary prevention strategy of the disease.

The Mediating Effect of Trait Anger on Depression with Life Events as a Mediating Variable

Anger is a negative emotional state with changing intensity and persistence that is usually associated with emotional arousal and perception of the outside world [11]. Spielberger showed that state trait anger theory divides anger into state anger, trait anger and anger expression [12]. Trait anger is defined as the tendency of stable desituational anger within the individual, which is a lasting and stable personality trait in the frequency, duration and intensity of anger. Individuals with higher levels of trait anger are more likely to feel enraged in a variety of situations, so they are more likely to experience state anger. Trait anger plays a certain role in the clinical manifestation of depression [13].

Therefore, it is inferred that trait anger is an endogenous factor of depression. Rick E. I. et al. highlight possible ways in which anger leads to stress directly, with increasing anger and hostility leading to problematic interpersonal interactions [14]. Anger can activate the autonomic nervous system to increase psychological stress sensitivity and activate the adrenal cortex to secrete cortisol through the hypothalamus-pituitary-adrenal axis (HPA) (Mustafa A. U., Stephan B. & William R. L. 2020). The increase in cortisol reduces the recognition of anger in patients with depression and produces more anger than normal people in the field of endocrine research, in which corticosteroids are used as marker variables for the effects of mental stress on depression [15]. Higher anger is accompanied by an increase in cortisol with increased stress, and it is speculated that there is a positive correlation between the increase in mental stress and anger in depressed subjects. However, Amanda et al. (2015) found different results that showed that greater anger was associated with less cortisol output when they investigated the relationship between daily cortisol and anger in depression. Ellen et al. further found that the slowing of the daytime cortisol slope may have a protective effect on some forms of internalized psychopathology and relieve depressive symptoms in highly irritable cases, in which the effects of circadian rhythms of cortisol diurnal patterns on irritability lead to internalization and externalization symptoms in children 9 years later [16]. Joana S.C. P et al. explained that participants with trait anger showed the highest level of HPA activation in response to stressors, which activates hypothalamic paraventricular nucleus neurons (PVN) to secrete arginine vasopressin (AVP) and corticotropin releasing factor (CRF), which in turn promotes the production and secretion of adrenocorticotropin (ACTH) in the anterior pituitary [17]. Therefore, ACTH induces the production and secretion of corticosteroids and glucocorticoids (corticosterone). Cortisol enters the bloodstream from the adrenal cortex. Therefore, high levels of cortisol inhibit the further release of ACTH and CRF through a negative feedback mechanism with the binding of cortisol to glucocorticoid receptors (GRs) in the pituitary, hypothalamic paraventricular nucleus and hippocampus. This results in a return to the physiological state after acute activation of the system. In the case of depression, the negative feedback loop of the HPA axis is damaged, leading to a long-term increase in glucocorticoids [17]. It is precisely because the negative feedback mechanism of cortisol is impaired in patients with depression when they feel angry and the level of cortisol in the blood cannot return to a low level that life stress events lead to maladjustment. Maladjustment of stress can lead to disorders of the HPA axis and the immune system. Furthermore, it leads to cognitive and emotional disorders, thereby increasing the risk of depression [17].

Rick E.I. et al. considered that anger tends to increase sensitivity to life events, which is the core feature of depression [14]. Sensitization is also a necessary condition for depressive disorder (Scott B.P., 2008).

There was a significant positive correlation between stressful life experience and depression [18]. Therefore, mental stress is regarded as a major factor leading to depression [19]. Beck's model suggests that depression follows when stressful life events activate negative schemata, including dysfunctional attitudes [20]. Kai G. R. et al. showed that there was a negative correlation between stressful life events (SLEs) and gray matter volume (GMV) in the left medial prefrontal cortex, in which the relationship was determined between inquiring about the life event questionnaire and changes in GMV for 2 years [21]. That said, SLEs in adulthood are risk factors for diseases such as depression, and part of this risk is regulated by ways that change the physiology and structure of the brain. Davey et al. tried to clarify the relationship between stress and depression by studying twin experiments [22].

The etiology of depression is related to stress and HPA (Larrieu & Sandi, 2018). Trait anger affects depression by activating the HPA axis and sensitizing life stress events. Therefore, it is speculated that trait anger affects depression, as life stress events play a mediating role.

The Influence of Gender and Age on the Mediating Model of Trait Anger Affecting Depression

The Influence of Gender on Depression: Epidemiological studies have found that there are significant gender differences in the prevalence of MDD. The gender differences were mainly manifested in depression, with a lack of energy, psychomotor retardation and a pessimistic attitude toward the future [23]. Depressed women are approximately twice as likely as depressed men [24, 25]. The high risk of women suffering from MDD may be related to women's special physiological status, psychological status and sociopsychological factors [26]. Female depression is related to their special physiology, such as menstruation, pregnancy, delivery and menopause, in which abnormal estrogen secretion is an important factor leading to menstrual syndrome, with prenatal restlessness, postpartum depression and menopausal depression [27]. Women's depression is also related to the emotional expression of their psychological factors. Dong Lina, Chen Hong & Wang Yu have shown that feminized individuals are more likely than masculine individuals to suppress their anger [28]. They adopt contemplative coping styles and avoid conflict, resulting in lower life satisfaction, which leads to depression. Janet S. Hyde & Amy H., Mezulis analyzed vulnerability and susceptibility to depression in women based on the A (emotion), B (biology), and C (cognitive) models [25]. It also explains the reasons for the gender differences in depression. The study analyzed biological vulnerability (genetic, puberty, physiological hormone fluctuations, etc.), emotional vulnerability (temperament) and cognitive vulnerability (negative cognitive style, objective physical consciousness and meditation, etc.). Gender differences were in negative life events and sociocultural factors. Furthermore, the vulnerability-stress model is taken as the core theory, which provides a theoretical basis for the differential effects of depression in women under stress exposure. It can be seen that the influence of gender on the susceptibility to depression is affected by the subjects' physical, psychological and environmental factors, as well as by the micro and macro levels of society and culture [24].

Due to the complexity of the influencing factors of gender differences in depression, it is speculated that the influence of gender on depression is dynamic and unstable. Some studies also believe that the gender difference in depression caused by gender bias is related to social culture because men are less likely to report that some symptoms of depression are nonmasculine [29]. Motro D. &Ellis APJ. confirmed that in community samples that met the diagnostic criteria for MDD, 4 of the 26 symptoms were allowed or encouraged to be reported by female roles [30]. Gender differences in depression are affected by social development and regional cultural differences. Jonathan M. Platt et al. showed that the gender depression gap narrowed in the cohort of subjects born between 1955 and 1994, which was influenced by the trend of economic and social status equality between men and women in the same period [31]. Some studies also believe that there is no statistically significant difference in the prevalence of depression between genders [32]. E. Stordal et al. clarified that the difference may be due to differences in diagnostic methods, which was further explained by reported prevalence deviations due to different definitions of the concept of depression [32]. Women tend to complain about their depression, physical discomfort and other related depressive symptoms, which are easy to find and diagnose in the early stage of depression or mild to moderate dysfunction [26]. It is speculated that based on the measurement results of the depression questionnaire, there are some gender differences in the scores of the depression scale between healthy people and patients with depression.

A Moderating Mediator with Age as a Moderating Variable: Gin S., M. &J John Mann found that the first episode of depression occurs from mid-adolescence to 40 s, but almost 40% of people experience the first episode of depression before the age of 20, with an average age of approximately 25 years old [24]. MDD is a recurrent lifelong disease. Almost 80% of patients experience at least one attack in their lifetime. The probability of recurrence increases with the age of onset. E. Stordal et al. found that the relationship between age and depression was very different and that the average level and number of cases of depression increased almost linearly with age [32]. Alan Tuohy, Christina Knussen & Michael J. Wrennall believe that depression shows a U-shaped function with increasing age, and there is an upward trend in patients with depression after middle age [33]. Studies have found that older people tend to be associated with common risk factors for depression, such as somatic symptoms, bereavement, loneliness, singleness and other diseases [32]. With increasing age, degenerative diseases and a decline in overall vitality play an important role in the somatization symptoms of depression [34]. Eleven percent of older people in New York City suffer from mental, cognitive or emotional disorders that lead to learning, memory and concentration difficulties [35]. The incidence of insomnia also increases significantly with age and is usually chronic compared with adults. Insomnia and depression have a strong two-way relationship: insomnia tends to accelerate the onset of depression, and if not treated properly, insomnia will also increase the chance of depression recurrence [36]. A study found that the older the age, the higher the prevalence of central nervous system diseases such as Parkinson's disease [37]. However, Parkinson's disease is considered a sign of bipolar disorder in the future. Therefore, with the increase in age, the aging of the body and the increase or decrease in the risk of progressive diseases, elderly individuals are more likely to suffer from depression. Stress events are the main stressors leading to depressive disorders, such as unemployment, emotional changes, divorce, bereavement, other family members suffering from serious illness or death serious illness, among which the incidence of depressive disorder is the highest in divorced and widowed people, with concentrated in the 30-50 age group [27]. Therefore, with increasing age, the probability of subjects experiencing the above stress events will be greatly increased. It is speculated that age, as a moderating variable, moderates the mediating effect of trait anger on depression. The specific manifestation is that age can moderate the psychological stress caused by life events by interacting with trait anger and then have an indirect effect on depression. As a result, it can be concluded that age is used as a moderating variable to moderate the front part of the mediating effect of trait anger on depression mediated by life events.

The Present Study

According to the literature, with the relationship among trait anger, depression, life events, age and gender variables, the following questions are proposed: as endogenous factors, how do trait anger and life events affect depression? and what's its mechanism? Is this mechanism universal for people with depression or for people including healthy people? How to make use of the

influence mechanism of trait anger and life events on depression to develop a targeted intervention and regulation program will provide a basis for the prevention and treatment of depression. In view of the above problems, this paper proposes the following four hypotheses: hypothesis 1: In the control group, trait anger mediates depression with life events as mediating variables. Hypothesis 2: In the control group with depressive patients as subjects and the reference group with healthy people as subjects, there should be differences in the comparison of the mediating effect models between the two groups. Hypothesis 3: Age as a moderating variable interacts with trait anger in the control group, moderates the mediating effect on depression as a mediator of life events, and establishes a moderated mediating model. Hypothesis 4: The significant effect of gender on depression is different between the control group and the reference group.

Method

Participants

In the study, the reference group was set up, and the principles and conditions for the selection were as follows: in this study, 89 normal healthy people aged 16-80 years from April to September 2022 were randomly selected as the reference group. and conducted a questionnaire interview required clear consciousness, independent personality, no mental illness, alcoholism, chemical abuse, etc. In addition, they could finish the questionnaire adjustment voluntarily and independently. A total of 115 depression outpatients aged 16-80 years from April to September 2022 were randomly selected as the control group, and a questionnaire interview was conducted (the patients came from the depression clinic of two Grade III psychiatric hospitals of Beijing a Ding Hospital and Shenzhen Kang Ning Hospital). The patients were selected according to the following criteria: (1) patients with depressive and affective disorders met the criteria of the Diagnostic and Statistical Manual of Psychiatric Disorders (DSM-V). (2) The subjects did not take any depression drugs or mood stabilizers in the past 2 weeks; they did not take benzodiazepines for sleep in the last 3 days; they did not take fluoxetine in the last 1 month; and (3) they had no other mental disorders in the past, such as alcohol, drug dependence and clearly diagnosed cognitive impairment. (4) Patients without serious physical disorders, such as respiratory system, blood system, immune system, endocrine system, infectious diseases and tumors. In the reference group, 89 normal healthy people were randomly selected as subjects. The age, gender and distribution are shown in Table 1. The average age of the subjects was 39.37, and the SD was 14.96. The number of males was 34, accounting for 38.20%, and the number of females was 55, accounting for 61.8%. A total of 115 outpatients with depression were randomly selected as subjects in the control group. The age, gender and distribution are shown in Table 1. The average age of the subjects was 31.03, and the SD was 9.84; there were 38 males, accounting for 33.04%, and 77 females, accounting for 66.96%.

Table 1: Statistical Tables of Age and Sex Between the Control Group and Reference Group

Group	Variable1	Mean	SD	Variable2	Category	Quantity	Proportion
Reference (n=89)	Age	39.37	14.96	Gender	male	34	38.20%
					female	55	61.80%
Control (n=115)	Age	31.03	9.84	Gender	male	38	33.04%
					female	77	66.96%

Procedure

Before the formal start of the questionnaire, explain to the participants the purpose of this study and show the ethics committee's opinions on the examination and approval of this study. Then, the informed consent form was signed by the participants. The basic situation of the participants, including the age of the patients, the suffering of major diseases, the time of illness, and the experience of taking medicine, was investigated, and the participants who did not meet the requirements of the study were eliminated. Through the "questionnaire Star" app program, the participants were recommended to enter the questionnaire program to answer the questions of the questionnaire with their mobile phone. The operator in the study ensured that the participants could clearly and accurately understand each guiding word in the questionnaire. Once the participants finish the answers, thank them and ask if there is any discomfort in the process of answering the questions. Then, we explained the significance and function of the study in detail and answered the participants' doubts, which eliminated the potential adverse effects of the study. Finally, the questionnaire results were uploaded to "questionnaire Star" software and saved for verification. In this study, a total of 204 valid questionnaires were obtained, including 115 depression patients in the control group and 89 healthy subjects in the reference group, with an effective recovery rate of 96%.

Measures

Measuring Tools

(1) The life events scale (Selye, 1969) is used to measure stress. The scale is suitable for people over 16 years old and includes three dimensions. The first is family life (28), the second is work and study (13), and the third is social and other aspects (7). A total of 48 items were scored on a 5-point Likert scale, from no influence to extremely severe scores of 0, 1, 2, 3, and 4, that is, no influence = 0, mild = 1, moderate = 2, severe = 3, and extremely heavy = 4. The higher the total LES score, the greater the mental stress. The Cronbach's α coefficient is 0.947 in this study.

(2) STAXI-2, developed by Spielberger in 1999, provides a simple, easy and objective scoring measurement tool for the experience, expression and control of anger. The scale is divided into three dimensions, including state anger, trait anger and anger expression scale, with a total of 57 questions. Trait anger scale consists of two dimensions: temperament trait anger and reactive trait anger, with a total of ten items, with a score from 1 (not at all) to 4 (totally). The higher the score, the higher the level of trait anger. Anger expression consists of 31 topics in three dimensions, namely, outburst of anger (anger-out), inward outbreak of anger (anger-in) and anger control (anger-control). Outburst of anger means that after experiencing anger, individu-

als vent this emotion to each other or other environment; inward anger refers to suppressing anger into their own hearts; anger control means that individuals can consciously and effectively control and reduce the intensity of anger after experiencing anger. The scale adopts a 4-point Likert scoring method, and the numbers 1, 2, 3 and 4 stands for "almost never", "sometimes", "often" and "almost always", respectively. The subjects with higher scores on the scale will have strong feelings of anger, which can be suppressed or vented through extreme behavior, or both, which represents the full expression of anger. In this study, the Cronbach's α coefficient of internal consistency of the trait anger scale was 0.899, and the Cronbach's α coefficient of anger expression was 0.785.

(3) The Beck Depression Inventory (BDI), compiled by Beck in 1967, is a self-rating scale for investigating individual depression. The scale is divided into three dimensions: negative attitude and suicide, somatic symptoms and difficulty in operation. It contains 21 items on a 4-point scoring scale: asymptomatic (0), mild (1), moderate (2) and severe (3). The higher the score, the higher the degree of depression. The standard score was 0-10 points without depression, 10-15 points with mild mood disorders, more than 15 points with depression, and more than 25 points with severe depression. The scale has high reliability and validity, and the Cronbach's α coefficient is 0.919 in this study.

Statistical Analyses

The Mediating Effect of Trait Anger on Depression with Life Events as a Mediating Variable: Using IBM SPSS Statistics26.0 and SPSS macro program PROCESS.V3.5, the collected data of the reference group and the control group were collated and analyzed as follows:

1. Descriptive statistics of trait anger, life events, depression and age of the control group and the reference group, independent sample t test and Pearson product correlation analysis were used to test the data distribution and significance of each variable.
2. using the PROCESS.V3.5 macro program and bootstrap method to analyze and verify the mediating effect of variables in the control group and the reference group. Trait anger takes life events as mediating variables on depression: independent variable X has an effect on dependent variable Y, and if X affects Y through the influence variable M, then M is called the mediating variable. The following regression equations can be used to describe the relationship between variables:

$$Y = cX+e_1 \quad (1)$$
$$M = aX+e_2 \quad (2)$$
$$Y = c'X+ bM=e_3 \quad (3)$$

where the coefficient c of equation (1) is the total effect of independent variable X on dependent variable Y, the coefficient an of equation (2) is the effect of independent variable X on mediating variable M, and the coefficient b of equation (3) is the effect of mediating variable M on dependent variable Y after controlling the influence of independent variable X. The coefficient c^(,)is the direct effect of the independent variable X on the dependent variable Y after controlling the influence of the intermediary variable M, ande_1~e_3is the regression residual [38]. Wen Zhong lin et al. summarized the methods of directly testing the product of coefficients of mediating effect, including the Sobel test, multiplicative integral distribution method, bootstrap method and Bayesian method [39]. The nonparametric bootstrap method relies on sample data. By constantly taking bootstrap samples and calculating the estimates of an and b, the bootstrap interval estimation of ab is obtained. If the interval estimation does not contain 0, then the mediating effect is significant [39]. In this study, trait anger was taken as an independent variable, depression as a dependent variable, life events as a mediating variable, and gender and age as control variables. First, continuous variables were centralized, and then the nonparametric percentile bootstrap method (Model 4) of deviation correction by the SPSS macro program PROCESS.V3.5 was used to test the mediating effect model.

- On the basis of the mediating effect of trait anger on depression as the role of moderator with age was tested by the PROCESS macro program, and the interaction between age and trait anger effect on life events was analyzed by the Johnson-Neyman method. Wen Zhong, Lin and Ye Bao, Juan pointed out that the nonparametric percentile bootstrap method is used to test the product of coefficients [40]. If at least a pair of products is significant, the mediating effect is moderated. The mediating effect value is reported at the mean value of the moderating variable and the next standard deviation above the mean value.
- Common Method Bias Analysis: Because this study only uses questionnaires to collect data on five variables, namely, trait anger, age, sex, life events, and depression, to avoid affecting and interfering with the research results and to make the results more objective and effective, the subjects, samples and test process are controlled to a certain extent in the test process. In the process of data analysis, Harman single factor analysis is used to test the unrotated exploratory factor analysis. The results of exploratory factor analysis show that there are 35 factors with eigenvalues greater than 1, which can explain 75. 75% of the variation, and the first factor can explain 17.46% of the variation, which is much less than 40% of the critical value. Therefore, there is no serious common method deviation in this study.

Results

The variables in the reference group and the control group were tested by independent sample t test.

Independent sample t tests were performed on the four variables in the reference group and the control group, such as trait anger, depression, life events and age, and the results are shown in Table 2. First, we perform the variance homogeneity test of the data in the table, and then we perform the independent sample t test. The results showed that trait anger ($F=3.66$, $P=0.06>0.05$) indicated that the condition of homogeneity of variance was established, and trait anger was significantly different between the reference group and the control group ($t=-4.53$, $df=202$, $p<.0001$). There was a significant difference in depression ($F=14.28$, $P<0.001$) between the reference group and the control group, which indicated that the condition of homogeneity of variance was not acceptable. The reference group and the control group were not supposed to have equal variance data. Therefore, there was a

significant difference in depression between the reference group and the control group (t=-9.70, df=118.33, p<.0001). The life event item F=11.29, P < 0.001, reached a significant level, indicating that the condition of homogeneity of variance is not valid, and we chose tactile from the equal variance data, so there was a significant difference in life events between the reference group and the control group (t=-5.04, df=174.16, p<.0001). There was significant difference in age between the reference group and the control group (P < 0.001), indicating that the condition of homogeneity of variance was not tenable. The age item (F=14.21, P <. 0001) was selected from the equal variance data, so there was a significant difference between the reference group and the control group. Through the independent sample t test of the data of trait anger, depression, life events and age between the reference group and the control group, it was found that there were significant differences between the two groups (t=-4.55, df=144.28 p<.0001). As a result, it can be concluded that the subjects of the control group and the reference group are heterogeneous, which lays a foundation for the control conditions and design premise of this study.

Table 2: Independent Sample T Test of Trait Anger, Depression, Life Events and Age in The Reference Group and The Control Group

	Test for homogeneity of variance		T test				
	F	Sig.	t	df	p	MD	SD
Trait anger	3.66	0.06	-4.53***	202	0.000	-5.70	1.26
Depression	14.28	0.000	-9.70***	118.33	0.000	-22.38	2.31
Life events	11.29	0.001	-5.04***	174.16	0.000	-10.61	2.10
Age	14.21	0.000	5.55***	144.28	0.000	8.34	1.83

Note:* p<0.05; ** p<0.01; *** p<0.001, following table is the same as it.

Descriptive Statistics and Correlation Analysis

The correlation analysis of trait anger, depression, life events and age in the reference group is shown in Table 3. Pearson product correlation analysis showed that there was a significant positive correlation between life events (M=57.26, SD=9.67) and trait anger (M=17.76, SD=3.98), (r = 0.23*, p < 0.05) and between depression (M= 4.97, SD=2.98) and life events (r = 0.22*, p < 0.05). There was a significant negative correlation between age (M=39.37, SD=14.96) and trait anger (r=-0.39**, p<0.01).

The correlation analysis of trait anger, depression, life events and age in the control group is shown in Table 3. Pearson product correlation analysis showed that trait anger (M=23.46, SD=11.34) and depression (r = 0.84, p < 0.001) and trait anger and life events (r = 0.33, P < 0.001). There was a significant positive correlation between depression (M=27.35, SD=24.50) and life events (M=67.87, SD=19.71) (r=0.84***, p<0.001). There was a significant negative correlation between age (M=31.03, SD=9.84) and life events (M=67.87, SD=19.71) (r=-0.27*, p<0.0).

Table 3: Descriptive Statistics and Correlation Analysis of Variables Between the Control Group and The Reference Group.

Group	Variable	Mean	SD	Trait anger	Depression	Life events	Age
Reference (n=89)	Trait anger	17.76	3.98	1			
	Depression	4.97	2.98	0.18	1		
	Life events	57.26	9.67	0.23*	0.22*	1	
	Age	39.37	14.96	-0.39***	-0.1	0.01	1
Control (n=115)	Trait anger	23.46	11.34	1			
	Depression	27.35	24.5	0.84***	1		
	Life events	67.87	19.71	0.33***	0.36***	1	
		31.03	9.84	-0.28	-0.28	-0.39*	1

Analysis of Gender Differences in Depression Between the Reference Group and Control Group

Independent sample t tests were carried out for depression in the control group and the reference group according to gender, and the results are shown in Table 4. First, the variance homogeneity test is performed with the data in Table 4, and then the independent sample t test is performed. The results showed that the item of depression (F=0.03, P=0.87>0.05) in the reference group was not significantly higher than that in the control group, indicating that the condition of homogeneity of variance was established, and the effect of gender on depression in the reference group was significantly different (t=-2.98** df=87, p<.01). In the control group, there was no significant difference in depression (F=0.89, P=0.35>0.05), indicating that the condition of homogeneity of variance was established, and there was no significant difference between the sexes in the experimental group (t=0.95, df=113, p>.05).

To further determine the gender difference in depression in the reference group, univariate ANOVA was performed on the depressive variables in the control group.

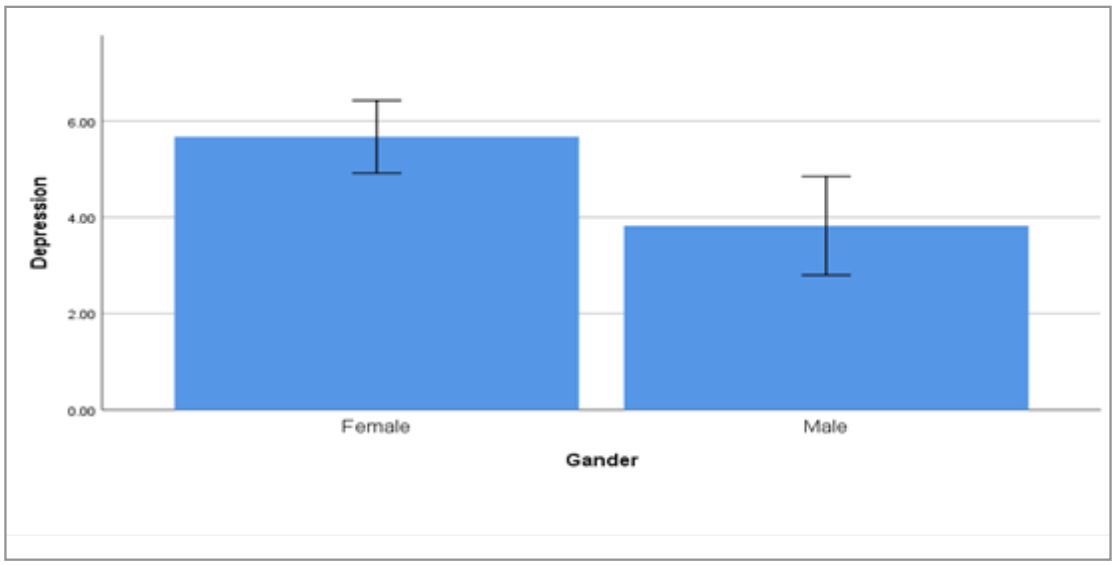
The results showed that F (1,87) =8.84** P<.01 η^2=0.09 in Table 4. There were significant differences in depression variables between the male and female groups in the control group. Figure 1 can directly reflect that the depression index of female subjects in the reference group is significantly higher than the depression index of male subjects.

Table 4: Independent Sample T Test of Depression Variables Grouped by Sex Between the Control Group and The Reference Group

Group		Test for homogeneity of variance		t	df	P	MD	SD
		F	P					
Reference (n=89)	Depression	0.03	0.87	-2.98**	87	0.004	-1.85	0.62
				-2.94**	67.38	0.005	-1.85	0.62
Control (n=115)		0.89	0.35	0.95	113	0.35	4.61	4.86
				1.23	107.67	0.22	4.61	3.74

Table 5: Anova Of Gender Effect Depression in The Reference Group.

Depression	Sum of Squares	df	Mean Square	F	P	n^2
Between Groups	71.85	1.00	71.85	8.84**	0.004	0.09
Within Groups	707.05	87.00	8.13			
Total	778.90	88.00				



Note 95% CL; ±1 SD

Figure 1: Effect of Gender Difference on Depression in The Reference Group

Analysis of Mediating Effect Between Control Group and Reference Group

Analysis of Mediating Effect in Reference Group: The results are shown in Table 6. It can be seen from the table that the influence of trait anger in the control group-trait anger in the path of life events on life events (Coeff = 0.23) because the bootstrap 95% confidence interval can be seen from the table (0.05 1.06) does not include 0, indicating that there is a significant existence of this path, which is a condition for the establishment of mediation. Then, we examined the influence of life events on depression from the path of life events to depression (coefficient = 0.18). The bootstrap 95% confidence interval (-0.01 0.12), including 0, the path model (P= 0.06 > 0.05), there is obviously no significant difference in this path. From this intermediary effect test, we can obtain the intermediary effect, which does not accord with the hypothesis of the model. In addition, the indirect effects of mediation are tested, as shown in Table 7. Effect=0.03 because its 95% confidence interval is (-0.01 0.09), the value of 0 is in this interval but not significant. As a result, it can be concluded that the mediating effect of trait anger on depression with life events as mediating variables in the reference group is not valid.

Analysis of Mediating Effect in Control Group: As table 6 shows, trait anger has a significant effect on life events in the control group (Coeff = 0.23 *), and the 95% confidence interval is (0.16 1.40), and the 0 value is not in this range; life events have a significant effect on depression (Coeff = 0.27), and the

95% confidence interval is (0.05 0.26), and the 0 value is not in this range. The effect of trait anger on depression was not significant (Coeff = 0.18) because its 95% confidence interval was (- 0.01 0.70), and the 0 value was in this range. From this, we can infer that the mediating effect of trait anger on depression with life events as mediating variables is established. Because the direct effect is not significant, it is concluded that the mediating effect is complete. The results of further analysis of the mediating effect are shown in Table 8. The confidence interval of total effect =0.46, 95% is (0.11 0.81), the 0 value is not in this range, so it is significant; direct effect =0.34, the proportion of relative mediating effect is 73%, its 95% confidence interval is (- 0.01 0.69), a value of 0 is in this range, so it is not significant. Indirect effect =0.12, the relative mediating effect accounts for 26.09%, and its 95% confidence interval is (0.04 0.29), the value of 0 is not in this range, so it is significant. It can be seen from the above that this model is a complete mediating effect of trait anger on depression with life events as mediating variables, and its mediating effect is shown in figure 2.

Table 6: Mediating Effect Analysis Between the Control Group and Reference Group

Group	Variable1	Variable2	*Coeff*	*SE*	*T*	95%CL		R^2	*F*	*P*
						LLCI	ULCI			
Reference (n=89)	Life events	Constant	0	1	0	-1.2	1.2	0.05	4.80*	0.01
		Trait anger	0.23	0.25	2.19	0.05	1.06			
	Depression	Constant	0	0.31	0	-0.61	0.61			
		Life events	0.18	0.03	1.71	-0.01	0.12	0.07	3.02	0.05
		Trait anger	0.14	0.08	1.33	-0.05	0.27			
Control (n=115)	Life events	Constant	0.00***	1.74	0	-3.44	3.44			
		Trait anger	0.23*	0.05	2.51*	0.16	1.40	0.05*	6.30*	0.01
	Depression	Constant	0.00***	0.95	0	-1.89	1.89			
		Life events	0.27**	0.05	2.97**	0.05	0.26			
		Trait anger	0.18	0.18	1.94	-0.01	0.70	0.13***	8.02***	0.0006

Table 7: Indirect Effect(S) Of X on Y of The Reference Group

Effect Boot SE	Boot LLCI	Boot ULCI
0.03 0.02	-0.01	0.09

Table 8: Analysis of The Mediating Effect of The Control Group

Group	effect	Boot SE	T	P	95%CL		Relative mediating effect
n=115					LLCI	ULCI	1.26
Total effect	0.46*	0.18	2.60*	0.01	0.11	0.81	100%
Direct effect	0.34	0.18	1.94	0.06	-0.01	0.69	73.91%
Indirect effect	0.12	0.06			0.04	0.29	26.09%

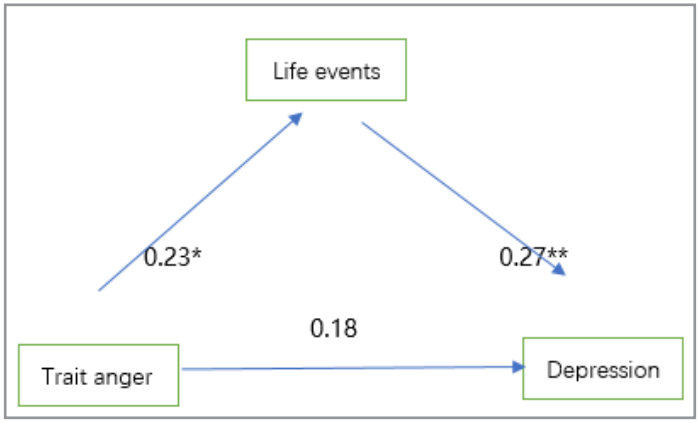


Figure 2: Mediating Effect of Trait Anger on Depression with Life Events as Mediating Variables in The Control Group

Analysis of Moderated Mediating Effects

The moderation test was carried out by using PROCESS software (model 7), and the results are shown in Table 9. Prior trait anger has a significant predictive effect on life events (β = 0.77 *), and its 95% confidence interval is (0.14 1.40), which is significant because the 0 value is not within this interval. Life events can significantly predict depression (β = 0.15***). The 95% confidence interval is (0.05 0.26), which is significant because the 0 value is not in this range. The prediction of trait anger to depression was not significant (β = 0.34), and its 95% confidence interval was (- 0.01 0.69), which was not significant because the 0 value was in this range. Trait anger has a complete mediating effect on depression with life events as mediating variables. The interaction item of age x trait anger (β = 0.07 *) has a significant effect on life events. The 95% confidence interval (0.003 0.13) is significant because the 0 value is not within this range, and the model fitting parameter As shown in the Table 9, it can be seen that the interaction between the age variable and trait anger has a moderating effect on the front of the mediating model in this study(R^2=0.13***.F=8.02***). To further explore the influence of the age of the moderating variable on the mediating effect, as shown in Table 11, the age of the moderating variable was analyzed by the significant interval analysis of the Johnson-Neyman diagram. When the age of the moderating variable is greater than the critical value of -1.93, as Figure 3 shows, the slope is positive and significant, indicating that the age of the moderating variable has a significant positive moderating effect on the mediating effect.

Table 9: Moderated Mediating Test with Age as A Moderating Variable

n=115	Life events			95%CL		Depression			95%CL	
Predictive variable	β	SE	T	LLUI	ULUI	β	SE	T	LLUI	ULUI
Trait anger	0.77*	0.32	2.43*	0.14	1.40	0.34	0.18	1.94	-0.01	0.69
Age	0.03	0.18	0.17	-0.33	0.39					
Age x Trait anger	0.07*	0.03	2.06*	0.003	0.13					
Life events						0.15***	0.05	2.97***	0.05	0.26
R^2	0.09*					0.13***				
F	3.56*					8.02***				

The results of moderated mediating effects tested by the bootstrap method are shown in Table 10. The moderated mediation index Index=0.01 has a confidence interval of 95% (0.003 0.03), which is significant because the value of 0 is not in this range. As figure 4 shows, a moderated mediating model with age as a moderating variable can be obtained.

Table 10: Index of Moderated Mediation

Index	BootSE	BootLLCI	BootULCI
0.01	0.01	0.003	0.03

Table 11: Moderator Value Defining Johnson-Neyman Significance Regions

Moderator	Value	% below	% above
Age	-1.93	46.09	53.91

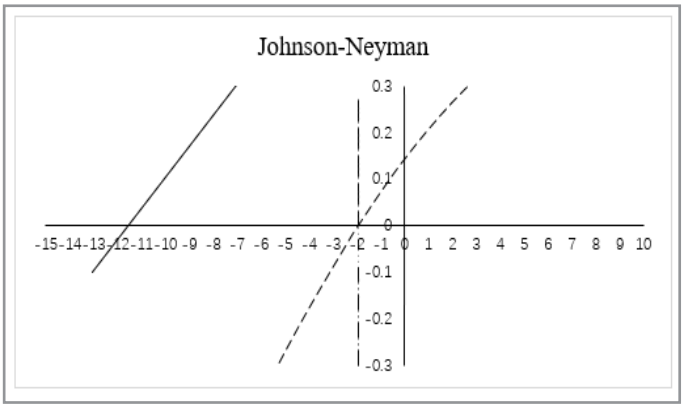


Figure 3: Significant Interval Distribution of Age as A Moderating Variable Johnson-Neyman

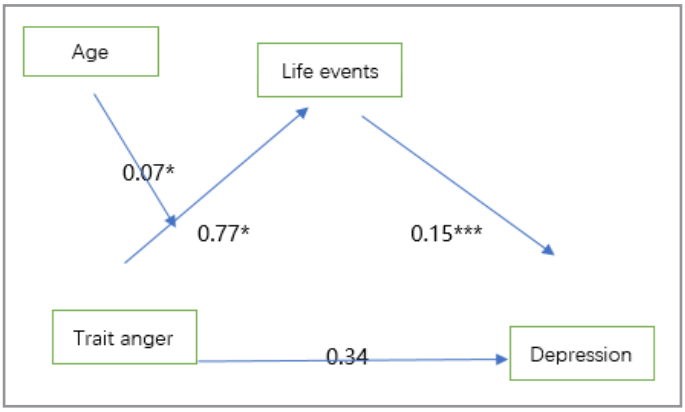


Figure 4: A Moderated Mediation Model with Age as A Moderating Variable

Discussion

Compares the Differences Between the Control Group and the Reference Group in Establishing the Mediating Effect of Trait Anger on Depression

According to the data in Table 6 of the results of this study, the reference group failed to construct the mediating effect model of trait anger with life events as mediating variables on depression, but the control group successfully constructed the above mediation model, as shown in Figure 2. The main reason for this difference is that compared with the general healthy population, the vulnerability of emotional regulation and cognition in patients with depression is related to the susceptibility to negative bias. Emotional disorder is the hallmark of depression [41]. Patients with depression use maladjustment strategies more frequently when regulating their emotions, and it is difficult to implement effective adaptation strategies (Jutta J. & Colin H.S., 2016). Depression is characterized by an increase in the description of negative information, difficulty in getting rid of negative materials, and cognitive control defects due to negative emotional states and negative life events when dealing with negative information [41]. Depression patients have lower activation in the prefrontal lobe and greater activation in the amygdala. This defect may make it difficult for depressed patients to detach or suppress their attention to negative stimuli, making them prone to depression when they encounter negative events in life [42]. Therefore, the bias in attention processing of negative information may be an important cognitive vulnerability factor in the occurrence and maintenance of depression [43]. The vulnerability-stress hypothesis explains that the occurrence of depressive disorders is due to the interaction between psychological vulnerability (for example, certain cognitive or specific information processing methods) and induced stressors (such as negative life events or other environmental factors) [41]. The results of the control group in this study show this process. That is, when subjects have depression, high trait angry individuals will experience more anger, and anger as a negative emotion makes it more difficult to separate the subjects' attention from the negative materials of psychological stress caused by life events. The defect in inhibition control leads to the further aggravation of depression. On the other hand, the normal healthy subjects in the reference group have the flexible ability of emotional regulation and the treatment mode of strong reaction and inhibition of negative materials that attract attention, which can make the behavior and emotional response constantly adapt to the psychological stress. to alleviate and relieve depression.

The results of this study show that the depression of normal people can also be measured in the form of a depression questionnaire, and sometimes there may be a high depression index, but it is a different concept from the depressive symptoms measured by patients with depression. The treatment and intervention of depression should be set according to the emotional regulation and cognitive characteristics of patients. According to Beck's depression theory, the influencing factors of depression can be divided into two categories: endogenous and reactive (exogenous). Trait anger in this study belongs to a relatively stable personality tendency and should belong to the endogenous influencing factors of depression; the psychological stress caused by life events to the subjects belongs to the source of stress and should belong to the reactive factors of depression. The results of this study show that in the control group, endogenous trait anger affects depression in the form of complete mediation through reactive life events. This process reflects the strong conduction effect of endogenous factors on depression through reactive factors. This proves a unique mechanism of endogenous and reactive factors affecting depression. According to this mechanism, the path of intervention for depression can be designed. As the result of this study is a complete mediation, we only need to consider intervening in the front part (trait anger to life event) of this mediation model to block the transmission of the above pathways to depression to alleviate depression.

Gender Differences in the Effects of Depression Between the Control Group and The Reference Group

There are inconsistent conclusions on the gender differences in depression in previous studies. Many studies have shown that there are significant gender differences in depression, and the prevalence of depression in women is significantly higher than that in men [23-25, 10]. However, some studies have come to the opposite conclusion that the gender difference in depression is not significant [32]. Other studies suggest that gender differences in depression are caused by gender bias in cultural differences [29, 30]. This kind of influence will have certain instability and dynamics with the development of the degree of social civilization and the trend of equal social and economic status of men and women [31]. The results of this study showed that there were significant gender differences in depression in the reference group, and the depression in females was significantly higher than that in males ($F(1,87) = 8.84^{**}$, $p = 0.004 < 0.01$ $\eta^2 = 0.09$), but there was no significant gender difference in depression in the control group ($t = 0.95$ $df = 113$ $p > .05$). Because the depression score comes from the self-rating questionnaire, it can be concluded that women of normal healthy people in the reference group are more inclined to self-evaluate depression than men due to the influence of their own physiological and cognitive characteristics and sociocultural factors. Even if the depression of women is significantly higher than that of men, it does not mean that the depression rate of women is higher than that of men. The complexity of the cause of depression lies in the affective disorder caused by many factors, such as endogenous and reactive factors. The results of depressed patients in the control group fully proved this point. Once depression is diagnosed, it occupies a strong position in common characteristics such as motivation, impairment of emotional regulation and negative cognitive bias, while gender characteristics have little influence on patients' motivation, emotion and cognitive function. Therefore, there is no significant difference in the gender variables of depression.

This suggests that the gender difference in depression is accurate in the epidemiological statistics of depression. First, our current criteria for the diagnosis of depression in the DSM-5 are mainly based on self-evaluation and other evaluation scales. Due to the current development level of psychological measurement technology and cognitive brain science, it is not possible to provide more direct and objective measurement indicators as a basis for diagnosis. In this way, there will be a certain overlap between the depression of normal people and the diagnosis of depressive symptoms of patients. It is possible to include the depression of normal people in the diagnostic criteria of depression, which expands the scope of diagnosis and leads to the trend of high prevalence and gender distribution. On the other hand, because

the social culture tends to define women as vulnerable groups, the hospital rate of depression and affective disorder is relatively higher than that of men. In addition, women are more likely to talk and communicate about their own depression than men, so they can obtain more social support to solve the problem (medical treatment or psychological counseling).

With the Moderator of Age on the Mediating Effect of Trait Anger on Depression

Previous studies on the relationship between age and depression have not been consistent. In some studies, depression scores increased with age, while in others, depression scores decreased with age [44-48]. Henderson A.S., Jorm A.F., Korten A.E., Jacomb P., Christensen H. &Roddgers, B, and in some studies, no significant difference was found between age and depression [49-51]. Similar conclusions are found in this study. As shown in Table 5, there was no significant correlation between age and depression in the reference group (r=-0.10, P>0.05) but only a significant negative correlation between age and trait anger (r = 0.39***, P < 0.001). From this, it can be inferred that with increasing age, life experience and personal experience, the personality of the reference group with healthy people as subjects will be improved, and personality tendencies such as trait anger will be weakened. There was no significant correlation between age and depression. However, the results of patients with depression in the control group were different. The results are shown in Tables 5 and 9. Although there is a significant negative correlation between age and life events (r = 0.39, p < 0.05), the main effect of age on life events is not significant (β = 0.03). The 95% confidence interval is (- 0.33 0.39). Because the 0 value is within this range, it is not significant. The interaction between age and trait anger significantly affects life events (β = 0.07 *). It can be concluded that the main moderating effect of age is that the interaction between age and trait anger positively affects life events and then has a positive effect on depression. As shown in figure 3, when the age value is greater than the critical value of -1.93 (the result of the negative factor centralization treatment), it has a significant positive moderating effect on the mediating effect. The critical value of age is only one, and it is lower than the average value of 1.93. It can be inferred that the moderating effect of age is significantly moderated in the course of the disease.

However, with increasing age, especially old age, people may experience more degenerative diseases, physical symptoms, physical decline and aging, as well as psychological stress events. However, this does not lead to an increased risk of depression. The results of the control group show that healthy people may effectively regulate and relieve depression through emotional regulation function, individual physical and mental growth and social support. The results of the control group may be related to the characteristics of depression. Depression is usually gradual, and severe depressive disorder is a recurrent lifelong disease. The probability of recurrence increases with increasing onset; the older the onset is, the less optimistic the outcome. A considerable proportion (up to 27%) of patients continue to develop chronic depression without recovery [24]. Patients with major depressive disorder (MDD) who have recovered or are in the process of recovery are still very likely to relapse again and are related to the biological vulnerability of patients with depression. N.L. Nixon et al. showed abnormalities in the default pattern network (DMN) related to the structure and function of brain regions during convalescence [52]. The control group showed significant task-based DMN connectivity with low retraction in the key areas of the DMN (bilateral wedge lobes). It can be seen that with the prolongation of the course of depression, there will be a trend of repeated attacks and procrastination. This suggests that we should pay attention to the course management of patients in the process of diagnosis and treatment of depression, optimize the best time point for diagnosis and treatment of the disease and control the development of the disease as soon as possible, which is not only beneficial to alleviating the pain of the patients. It is of great significance for patients to control the course of disease within as short a time as possible to prevent the disease from becoming serious or turning to chronic refractory disease. In addition, it is also very important for cured patients to pay attention to secondary prevention of the disease. It is necessary to strengthen daily health management and mental health education to prevent the recurrence of depression.

Study Limitations and Future Directions

From the perspective of psychological research on the effect of anger on depression, many theories appear alternately in their arguments, and there are many contradictions and conflicts. This is due to the complexity of the relationship between anger and depression. Due to the lack of empirical research evidence that different emotional titers influence each other, it is difficult to clarify the exact relationship between anger and depression. The above model of mediating the effects of trait anger on mental stress and age, which is proposed on the basis of literature research, needs to be verified in subsequent empirical studies. A series of problems, such as the deep regulation mechanism and the determination of the causality of age embodied in the model, are still worthy of further exploration in the future.

Individual Differences in the Effect of Anger on Depression

First, there are differences in the determination of the degree of anger in existing studies. For example, Gabrielle et al. explored the relationship between irritability and temper loss by taking community and clinical samples [53]. Research shows that there are differences in the understanding of the term's "irritability" and "frequent temper tantrum" in different sources and types of samples. It is not difficult to see that due to the lack of objective evaluation criteria for idiosyncratic anger and age, although this study takes into account the heterogeneity of the subjects, the subjects are divided into the reference group and the control group according to whether they suffer from depression. The inclusion and exclusion criteria of the subjects were properly controlled, but individual differences within the test group were inevitable. Therefore, it has a certain influence on the reliability and validity of the research results.

Second, there are individual differences in the effect of anger on depression. The emotional environment insensitivity (ECI) hypothesis holds that patients with depression have a weak response to both positive and negative stimuli. This is quite different from previous studies that believe that patients with depression are biased and susceptible to negative emotions [54-56]. ECL is based on an evolutionary model that holds that depression promotes a person's defensive disengagement from the environment and the interaction between depression and environmental adaptation. The environment can explain inconsistent findings about differences in anger responses among groups (Jennifer et al., 2011).

The Effects of Gender and Age on Anger and Depression

Finally, the gender difference in depression is also a topic worthy of attention in future studies. Women are twice as likely to suffer from depression as men [57]. Gender differences in hormone levels, cognitive styles, behavior habits and regional culture are bound to provide different perspectives for future research. Anna, Elisabeth & Paula reported that when 470 adolescents were selected as subjects, a structural equation model was used to explore the two longitudinal models [58]. The results showed gender-based differences, and internalized variables, such as emotional instability, were directly related to the prevention of depression in girls. Other studies have shown that gender differences in depression may be caused by differences in behavior in dealing with anger, at least to some extent. Renee found a link between gender-based activity-oriented behavior and the likelihood of reducing depression in a long-term study of the relationship between anger and depression [59]. Behaviors with masculine characteristics, such as cycling, may reduce depression through neurochemical changes. However, some studies believe that most of the gender differences in depression are based on the form of the questionnaire, which is not significant [32]. Due to cultural differences in the level of social development, there are gender differences in the expression of anger and the identification of depression. Therefore, this gender difference has a certain degree of instability and uncertainty in the dimension of time and social and cultural environment. In this study, different conclusions were drawn about the gender differences in depression between normal healthy people (reference group) and patients with depression (control group). It is suggested that it is more meaningful to explore the gender differences in depression in combination with the specific situation and the personality of the subjects. Future studies on gender differences in depression may put forward more conditional stratified studies under gender grouping to avoid the heterogeneity of the subjects but also make the research more practical [60-65]. Age was positively correlated with depression. This is related to the increase in age accompanied by degenerative diseases, functional aging and more psychological stress events. Depression is a lifelong disease with a high recurrence rate. With the extension of the course of the disease, the increased probability of recurrence will further increase the physical and mental damage of the patients, increasing the risk of disability and suicide. However, the long course and easy recurrence of depression lead to epidemic statistics about age and depression [66-73].

Transparency and Openness

We affirmed that the de-identified data on which the study conclusions are based are available and the link to access of information is provided. It is scripted in reference list.

IRB Statement

"Effect of mental stress on depression" was approved by Psychology school of NENU. Code:2021003.

References

1. Delgado PL (2004) Common pathways of depression and pain. The Journal of Clinical Psychiatry 65: 16-19.
2. Nadja B, Peter Kuppens, Nicholas B Allen, Lisa B Sheeber, Eva Ceulemans (2018) Affective family interactions and their associations with adolescent depression: a dynamic network approach. Dev Psychopathol 30: 1459-1473.
3. Grant LI, Douglas P Terry, Luz M, Zafonte R, McCrory P, et al. (2018) Anger and depression in middle-aged men: implications for a clinical diagnosis of chronic traumatic encephalopathy JNCN in Advance 31: 328-336.
4. Christine HO, Wendy AH (2018) Anger in the context of postnatal depression: An integrative review Wiley Birth Issues in Perinatal Care 45: 336-346.
5. Mohammad Ali Besharat, Mahin Etemadi Nia, Hojatollah Farahani (2013) Anger and major depressive disorder: The mediating role of emotion regulation and anger rumination. Asian Journal of Psychiatry 6: 35-41.
6. Liliana PC, Robert Chapman, Susannah E Murphy, Christopher-James Harvey, Anthony James, et al. (2019) A single dose of fluoxetine reduces neural limbic responses to anger in depressed Adolescents. Translational Psychiatry 30: 1-30.
7. Besharat MA, Nia ME, Farahani H (2013) Anger and major depressive disorder: the mediating role of emotion regulation and anger rumination. Asian Journal of Psychiatry 6: 35-41.
8. Baker, Alford (2021) Depression (Second Edition, translated by Yang Fang et al.). Machinery Industry Press 64-68.
9. Stepanichev MY, Tishkina AO, Novikova MR, Levshina IP, Gulyaeva NV (2016) Anhedonia but not passive floating is an indicator of depressive-like behavior in two chronic stress paradigms. Acta Neurobiologiae Experimentalis 76: 324-333.
10. Shan H, Duo S, Wei YJ (2020) The Association between Urbanization and Depression among the Middle-Aged and Elderly: A Longitudinal Study in China. The Journal of Health Care Organization Provision and Financing 24: 4574-4579.
11. Kassinove, Sukhodolsky (1995) Anger Disorders: Basic Science and Practice Issues. Issues in Comprehensive Pediatric Nursing 18: 173-205.
12. Spielberger C,Krasner S, Soloman E (1988) The experience expression and control of anger. In Janisse M(Ed.) Health Psychology: Individual Differences with Stress New York: Springer -Verlag 89 -108.
13. Wenze, Gunthert, Forand, Laurenceau (2009) The Influence of dysphoria on reactivity to naturalistic fluctuations in anger. Journal of Personality 77: 795-824.
14. Renee D Goodwin (2006) Association Between Coping with Anger and Feelings of Depression Among Youths. American Journal of Public Health 96: 664-669.
15. Katie MD, Richard JP (2012) Associations between hypothalamic–pituitary–adrenal axis function and facial emotion processing in depressed and control participants. Psychiatry and Clinical Neurosciences 66: 442-450.
16. Ellen M Kessel, Allison Frost, Brandon L Goldstein, Sarah R Black, Lea R Dougherty, et al. (2019) Developmental pathways from preschool irritability to multifinality in early adolescence: the role of diurnal cortisol. Psychological Medicine 51: 761-769.
17. Joana SCP, Kieran Rea, Yvonne M Nolan, Olivia FO'Leary, Timothy G Dinan, et al. (2020) Depression's Unholy Trinity: Dysregulated Stress Immunity and the Microbiome. Annual Review of Psychology 71: 49-78.
18. Rannveig, Bryndis, Sarah, Inga (2017) The Impact of sexual abuse family violence conflict spirituality and religion on Anger and depressed mood among adolescents. Journal of Interpersonal Violence 36: 557-597.

19. Maheen Nisar, Rubaab M Mohammad, Sani Fatima, Preet R Shaikh, Mehroze Rehman (2019) Perceptions pertaining to clinical depression in Karachi, Pakistan. Cureus 11: e5094.
20. Beck AT (1987) Cognitive models of depression. Journal of Cognitive Psychotherapy 1: 5-37.
21. Kai GR, Pfarr Julia-Katharina, Stein Frederike, Brosch Katharina, Meller Tina, et al. (2022) Association between stressful life events and gray matter volume in the medial prefrontal cortex: A 2-year longitudinal study. Hum Brain Mapp 43: 3577-3584.
22. Davey CG, López-Solà C, Bui M, Hopper JL, Pantelis C, et al. (2016) The effects of stress–tension on depression and anxiety symptoms: evidence from a novel twin modeling analysis. Psychological Medicine 46: 3213-3218.
23. Sabic D, Sabic A, Bacic BA (2021) Major Depressive Disorder and Difference between Genders. Materia sociomedical 33: 105-108.
24. Gin S Malhi, J John Mann (2018) Depression. The Lancet 5: 339-347.
25. Janet S Hyde, Amy H Mezulis (2020) Gender Differences in Depression: Biological Affective Cognitive, and Sociocultural Factors. Harvard Review of Psychiatry 28: 1-13.
26. Huang Xiu fang (2009) Gender differences in the prevalence of major depressive disorder. Chinese Journal of Health Psychology 17: 93-94.
27. Long Zhenzhao (2014) Analysis of the current status of epidemiological research on depressive disorders, Journal of Clinical and Experimental Medicine 13: 143-145.
28. Dong Lina, Chen Hong, Wang Yu (2014) Anger expression and depression in women of different genders. Chinese Journal of Mental Health 28: 234-238.
29. Sigmon ST, Pells JJ (2005) Gender differences in self-reports of depression: the response bias hypothesis revisited. Sex Roles 53: 01-11.
30. Motro D, Ellis APJ (2017) Boys don't cry: gender and reactions to negative performance feedback. J Appl Psychol 102: 27-35.
31. Jonathan M Platt, Lisa M Bates, Justin Jager, Katie A McLaughlin, Katherine M Keyes (2020) Changes in the depression gender gap from 1992 to 2014: Cohort effects and mediation by gendered social position. Social Science & Medicine 258: 1-9.
32. Stordal E, Bjartveit Krüger M, Dahl NH, Krüger Ø, Mykletun A, et al. (2001) Depression in relation to age and gender in the general population: the Nord-Trøndelag. Acta Psychiatrica Scandinavica 104: 210-216.
33. Alan Tuohy, Christina Knussen, Michael J Wrennall (2005) Effects of Age on Symptoms of Anxiety and Depression in a Sample of Retired Police Officers. Psychology and Aging 20: 202-210.
34. Guo Tong (2022) Clinical characteristics and related factors of patients with depression with somatization symptoms at different ages. Neurological Disorders and Mental Health 22: 539-546.
35. American Community Survey (2016) The population 65 years and older in the United States: 2016. https://www.census.gov/content/dam/Census/library/publications/2018/acs/ACS-38.pdf
36. Paul S, Suzanne ML, Britt K, Megan J (2018) Advancing cognitive behavior therapy for older adults with comorbid insomnia and depression. Cognitive Behavior therapy 47: 139-154.
37. Jääskeläinen, Juola T, Korpela H, Lehtiniemi H, Nietola M, et al. (2017) Epidemiology of psychoticdepression – systematic review and meta-analysis. Psychological Medicine 48: 905-918.
38. Wen Zhonglin, Ye Baojuan (2014) Mediating effect analysis: method and model development. Advances in Psychological Science 22: 731-745.
39. Wen Zhonglin, Ye Baojuan (2022) Methodological study on domestic mediating effects. Advances in Psychological Science 30: 1692-1702.
40. Wen Zhonglin, Ye Baojuan (2014) Moderated mediation model testing method: competition or substitution? Acta Psychologica Sinica 46: 714-726.
41. Ian HG, Jutta J (2010) Cognition and Depression: Current Status and Future Directions. Annu Rev Clin Psychol 6: 285-312.
42. Ming tian Zhong, Xiang Wang, Jing Xiao, Jinyao Yi, Xueling Zhu, et al. (2011) Amygdala hyperactivation and prefrontal hypoactivation in subjects with cognitive vulnerability to depression. Biological Psychology 80: 233-242.
43. Lemkeleyman, Rudideraedt, Rikschacht, Ernsth WK (2007) Attentional biases for angry faces in unipolar depression. Psychological Medicine 37: 393-402.
44. Beekman AT, D J Deeg, T van Tilburg, J H Smit, C Hooijer, et al. (1995) Major and minor depression in later life: a study of prevalence and risk factors. J Affect Disord 36: 65-75.
45. Blumenthal MD (1975) Measuring depressive symptomatology in a general population. Arch Gen Psychiatry 32: 971-978.
46. Murrell Sa, Himmelfarb S, Wright K (1983) Prevalence of depression and its correlates in older adults. Am J Epidemiol 117: 173-185.
47. Knight RG, Waal Manning HJ, Spears GF (1983) Some norms and reliability data for the State-Trait anxiety inventory and Zung self-rating depression scale. Br J Clin Psychol 22: 245-249.
48. Comstock GW, Helsing KJ (1976) Symptoms of depression in two communities. Psychol Med 6: 551-563.
49. Henderson AS, Jorm AF, Korten AE, Jacomb P, Christensen H, et al. (1998) Symptoms of depression and anxiety during adult life: evidence for a decline in prevalence with age. Psychol Med 28: 1321-1328.
50. Eaton WW, Kessler LG (1981) Rates of symptoms of depression in a national sample. Am J Epidemiol 114: 528-538.
51. Knight RG (1984) Some general population norms for the short form of Beck depression inventory. J Clin Psychol 40: 751-753.
52. Nixon NL, Liddle PF, Nixon E, Worwood G, Liotti M, et al. (2014) Biological vulnerability to depression: linked structural and functional brain network findings. The British Journal of Psychiatry 204: 283-289.
53. Gabrielle A Carlson, Allison P Danzig, Lea R Dougherty, Sara J Bufferd, Daniel N Klein (2016) Loss of Temper and Irritability: The Relationship to Tantrums in a Community and Clinical Sample. Journal of child and adolescent psychopharmacology 26: 114-122.
54. Rottenberg J, Gross JJ, Gotlib IH (2005) Emotion context insensitivity in major depressive disorder. Journal of Abnormal Psychology 114: 627-639.

55. Thompson RJ, Mata J, Jaeggi SM, Bushkuehl M, Jonides J, et al. (2012) The everyday emotional experience of adults with major depressive disorder: Examining emotional instability inertia and reactivity. Journal of Abnormal Psychology 121: 819-829.
56. Wingenfeld K, Terfehr K, Meyer B, Lowe B, Spitzer C (2013) Memory bias for emotional and illness-related words in patients with depression, anxiety and somatization disorders: An investigation with the directed forgetting task. Psychopathology 46: 22-27.
57. Edward, Tom (2016) Explaining the sex difference in depression with a unified bargaining model of anger and depression. Evolution Medicine and Public Health 2016: 117-132.
58. Anna Llorca, Elisabeth Malonda, Paula Samper (2016) The role of emotions in depression and aggression Emotions. Depression and Aggression 21: 59-64.
59. Renee D Goodwin (2006) Association Between Coping with Anger and Feelings of Depression Among Youths. American Journal of Public Health 96: 664-669.
60. American Psychiatric Association (2014) Diagnostic and Statistical Manual of Mental Disorders (5th edition, translated by Zhang Daolong et al.). Peking University Press 77-78.
61. Yuan Jiajin (2020) Cognitive neuroscience of emotional susceptibility. Science Press 230-231.
62. Gard DE, Gard MG, Kring AM (2006) Anticipatory and consummatory components of the experience of pleasure: A scale development study. Journal of Research in Personality 40: 1086-1102.
63. Jeanne Savage, Brad Verhulst, William Copeland, Robert R Althoff, Paul Lichtenstein, et al. (2016) A genetically informed study of the longitudinal relation between irritability and anxious/depressed symptoms. J Am Acad Child Adolesc Psychiatry 54: 377-384.
64. Klein DN, Neil J Santiago, Dina Vivian, Janice A Blalock, James H Kocsis, et al. (2004) Cognitive-behavioral analysis system of psychotherapy as a maintenance treatemt for chronic depression. J. Consult Clin Psych 72: 681-688.
65. Maya P, Marlene M, Moretti (2010) Ruminating on rumination: are rumination on anger and sadness differentially related to aggression and depressed mood? J Psychopathol Behav Assess 32: 108-117.
66. Nadja B, Peter Kuppens, Nicholas B Allen, Lisa B Sheeber, Eva Ceulemans (2018) Affective family interactions and their associations with adolescent depression: a dynamic network approach. Dev Psychopathol 30: 1459-1473.
67. Nicole Vliege, Patrick L (2008) The role of dependency and self-criticism in the relationship between postpartum depression and anger Personality and Individual Differences 45: 34-40.
68. Pablo Vidal-Ribas, Brenda Benson, Aria D Vitale, Hanna Keren, Anita Harrewijn, et al. (2020) Bidirectional associations between stress and reward processing in children and adolescents: a longitudinal neuroimaging study. Biol Psychiatry Cogn Neurosci Neuroimaging 4: 893-901.
69. Samuela T, Cristiana De Ranieri, Cecilia Dionisi, Monica Citti, Alessandro Capuano, et al. (2013) Clinical features, anger management and anxiety: a possible correlation in migraine children. The Journal of Headache and Pain 14: 39.
70. Simon MR, David Kealy, John S Ogrodniczuk, Zac E Seidler, Gabriela Montaner, et al. (2012) The anxiety depression pathway among men following a prostate cancer diagnosis: Cross-Sectional Interactions Between Anger Responses and Loneliness. American Journal of Men's Health 5: 1-9.
71. Spielberger C, Krasner S, Soloman E (1988) The experience expression and control of anger. In Janisse M(Ed.) Health Psychology: Individual Differences with Stress New York: Springer -Verlag 89 -108.
72. Stephanie S (2012) Expression of direct anger and hostility predict depression symptoms in formerly depressed women. Canadian Journal of Behavioral Science 44: 200-209.
73. Vanessa PB, Peter Kovalc D, Egon DD, Peter K (2018) Emotion regulation and mood brightening in daily life vary with depressive symptom levels. Cognition and emotion 22: 1-11.

Copyright: *©2024 Zhe Han. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.*