International Journal of Biology and Life Sciences
ISSN: 2957-9511 | Vol. 12, No. 3, 2025

The Relative Impact of Maternal and Paternal Depression History on Externalizing Behaviors and Psychopathology Susceptibility in Children

Yansong Lu *

College of Sciences, Tianjin University of Commerce, Tianjin, China
*** Corresponding author Email:** yansongg51@163.com

Abstract: Adolescent depression is a major global public health issue, closely linked to familial risk. To elucidate the intergenerational transmission of depression, this study utilized baseline data from the Adolescent Brain Cognitive Development (ABCD) Study in the United States. Employing propensity score matching to control for confounders, we quantified the impact of parental depression history on behavioral problems and susceptibility to mental disorders (p-factor) in children aged 9-11. Results from the successfully matched sample showed that both maternal and paternal depression histories significantly increased adolescents' externalizing behavioral problems and general susceptibility to mental disorders. Notably, the average effect size for maternal depression history (Cohen's d = 35.56%) was significantly greater than that for paternal history (20.95%). This indicates that the influence of familial depression history is broad, extending beyond depressive symptoms to elevate overall vulnerability to various mental health problems. This study quantified the differential effects of maternal versus paternal depression history on adolescent mental health and specified their respective effect sizes on the general psychopathology factor (p-factor) in adolescents.

Keywords: Parental Depression; Intergenerational Transmission; Propensity Score Matching; P-factor.

1. Introduction

Depression is a widespread global health issue, affecting approximately 322 million individuals worldwide [1], with about 34% of adolescents being at risk of developing this disorder [2]. Adolescence represents a critical peak period for both the first incidence of depression and a marked increase in new cases [3], characterized by elevated incidence rates and significant vulnerability. Among the numerous risk factors for adolescent depression, family and psychological contexts are closely linked to its onset [4]. Notably, adolescents whose parents are affected by depressive symptoms face a substantially increased risk of developing depression themselves [5, 6].

To investigate the intergenerational transmission of depression, this study utilized data from the ABCD Study to precisely quantify the impact of a family history of depression on a range of behavioral outcomes and the general susceptibility to psychopathology (p-factor) in adolescents. The findings estimate the effect sizes of familial depression history on adolescent behavior and mental health, providing a reference for the early prevention and diagnosis of youth depression.

2. Method

2.1. Data Source

The Adolescent Brain Cognitive Development (ABCD) study is a longitudinal cohort study tracking brain development and health outcomes in 11,880 adolescents across the United States (https://data-archive.nimh. nih. gov/abcd). It integrates psychological, social, economic, health data, collected at multiple developmental stages from adolescence to early adulthood [7]. This paper utilizes baseline data from the ABCD Study Release 5.1. The study was approved by the Institutional Review Board at the University of California, San Diego, and local ethics committees at data collection sites. All parents provided written consent, and verbal assent was obtained from adolescents.

2.2. Adolescent Behavioral Data

The Child Behavior Checklist (CBCL) was employed in this study to screen for behavioral problems and social competencies in children and adolescents aged 4 to 16. This instrument demonstrates good validity, reliability, and measurement invariance [8]. Within the ABCD Study, the parent-report version was used, completed by parents based on their child's behavior over the past six months.

To quantify the influence of familial depression history on adolescent depressive symptoms and general susceptibility to psychopathology (p-factor), the following variables from the Child Behavior Checklist (CBCL) were selected: the total score, the DSM-5 Depressive Problems score, Anxious/Depressed, Withdrawn/Depressed, Somatic Complaints, Social Problems, Thought Problems, Attention Problems, Rule-Breaking Behavior, and Aggressive Behavior. All selected variables were standardized T-scores, which allow for comparison across groups with different ages, genders, and educational levels.

2.3. Demographic Variables

Demographic variables, including age, sex, race, the highest parental education level, and household income (six variables in total), were included as covariates. Due to the limited representation of other ethnic minorities, analysis was limited to African American and White participants.

2.4. Family History of Depression

This study examined the history of depression in six family

members: father, mother, paternal grandfather, paternal grandmother, maternal grandfather, and maternal grandmother. The ABCD dataset includes a composite variable for "any family history of depression" which is coded as 0 if no family member has a history and 1 if at least one member does. For subjects with a value of 0 on this composite variable, missing values in the six corresponding individual variables were recoded to 0.

2.5. Statistical Analysis

This study was conducted using R 4.4 and Python 3.12. All demographic, family history, and Child Behavior Checklist (CBCL) data were integrated. During data preprocessing, a strict inclusion criterion was applied: only participants with no more than one missing value across all variables were retained. For the limited missing values within this retained sample, imputation was performed using the Random Forest method via the mice package (v.3.16.0) in R [9].

(1) The p-factor in Adolescents

Following the approach of Elliot D Hill et al. [10], this study constructed a general psychopathology factor (p-factor) by performing a factor analysis on eight syndrome scales from the Child Behavior Checklist (CBCL). The scales assessed the following dimensions: anxious/depressed, withdrawn/depressed, somatic complaints, social problems, thought problems, attention problems, rule-breaking behavior, and aggressive behavior. These eight syndromes were reduced to a single latent factor representing general susceptibility to mental illness in adolescents.

(2) Propensity Score Matching

Six separate propensity score models were constructed. In each model, the treatment variable was the depression history of a specific family member: father, mother, paternal grandfather, paternal grandmother, maternal grandfather, or maternal grandmother. Demographic data served as covariates in all models. Additionally, to control for potential confounding and improve matching quality, the depression history variables of the other five family members were also included as covariates in each respective model. For each treatment variable, a value of 1 indicated the treatment group, and a value of 0 indicated the control group.

Greedy nearest-neighbor matching without replacement was employed. To control the matching scale while maximizing successful matches, the maximum number of matches was set to the floor value of the control-to-treatment group size ratio minus one, with an upper limit of a 1:4 ratio. Propensity scores were estimated via logistic regression using the psmpy library in Python, with a caliper width of 0.01 standard deviations of the propensity score.

Following the approach of Fan Nils Yang, covariate balance after matching was assessed using the standardized mean difference (SMD). An SMD of less than 0.10 for all covariates was achieved [11], indicating satisfactory balance and justifying the use of the matched sample for subsequent analysis.

(3) Cohen‘s d Effect Sizes

After obtaining propensity score-matched pairs with balanced covariates, Cohen's d effect sizes were calculated for 11 outcome variables to quantify the impact of a family history of depression on adolescent depression, behavioral issues, and p-factor. For each outcome variable, a t-test was performed to assess the difference between the treatment and control groups.

3. Results

3.1. Demographic

Table 1. Demographic

		Number	Percentage
Gender	Male	5471	52.2%
	Female	5004	47.8%
Race	Whites	7835	74.8%
	African Americans	2110	20.1%
Age	8 years old	20	0.2%
	9 years old	5437	51.9%
	10 years old	4992	47.7%
	11 years old	26	0.2%
Parental education status	< High school	701	6.7%
	High school diploma or GED	1098	10.5%
	Bachelor or Associate degree	6038	57.6%
	≥ Master's degree	2638	25.2%
Household income ($)	≤50000	3204	30.6%
	50000-100000	2913	27.8%
	>100000	4358	41.6%
Family History of Depression	mother	2113	20.2%
	father	1299	12.4%
	paternal grandfather	570	5.4%
	paternal grandmother	1144	10.9%
	maternal grandfather	860	8.2%
	maternal grandmother	1789	17.1%

For this analysis, samples with no more than one missing value across all demographic, adolescent behavior, and family

history variables were retained (N = 10475). Multiple imputation was performed on this sample using the MICE method. The sample comprised 5471 males and 5004 females; 7835 individuals were White and 2110 were Black.

3.2. Propensity Score Matching: Ratios and Success Rates

Based on the size of each treatment group, varying matching ratios were applied: a 1:4 ratio for paternal, paternal grandfather, paternal grandmother, and maternal grandfather depression history; a 1:2 ratio for maternal depression history; and a 1:3 ratio for maternal grandmother depression history. The match success rate across the different treatment groups ranged from 61.4% to 71.9%.

Table 2. Matching Parameters

Treatment Variable	Treatment Number	Matched Number	Matching Ratio
father	1299	798	1: 4
mother	2113	1498	1: 2
paternal grandfather	570	409	1: 4
paternal grandmother	1144	735	1: 4
maternal grandfather	860	618	1: 4
maternal grandmother	1789	1143	1: 3

Propensity score matching was performed separately for the six exposure variables. Balance was achieved only for paternal and maternal depression history. In these cases, all standardized mean differences (SMD) were below 0.10, indicating balanced characteristics between groups after matching.

For the remaining exposure variables, significant covariate imbalance persisted after matching, with standardized mean differences (SMD) exceeding 10% for some covariates. The treatment and control groups remained substantially different on these characteristics, which would confound effect estimation. Therefore, these matched samples were excluded from further analysis.

Table 3. Covariate Balance After Matching

	SMD Before → After Matching (%)					
covariates	father	mother	paternal grandfather	paternal grandmother	maternal grandfather	maternal grandmother
Gender	5.4→8.3	1.4→5.1	6.8→0.6	0.1→1.4	3.0→7.6	1.7→10.9
Whites	24.0→1.3	8.5→8.0	34.6→9.7	35.1→6.6	32.4→10.9	25.7→11.4
African Americans	9.6→3.8	3.2→4.8	31.4→6.1	26.1→0.9	23.2→7.9	9.8→9.5
Age	3.0→0.1	0.2→1.0	1.1→4.0	4.1→0.3	5.3→3.8	1.1→4.2
Parental education status	6.5→0.0	4.2→9.2	25.4→11.8	19.1→6.3	17.6→11.6	9.2→6.1
Household income	3.5→2.2	14.2→8.6	20.2→14.7	21.8→11.6	17.9→12.9	5.6→7.8
father		55.5→1.7	123.5→1.8	103.6→1.7	63.3→1.5	49.5→6.4
mother	67.5→6.0		65.4→4.1	59.0→2.4	95.8→0.5	95.5→7.0
paternal grandfather	85.0→2.6	37.0→2.6		78.3→0.2	70.6→5.4	41.1→1.5
paternal grandmother	98.0→0.8	45.8→4.6	107.6→0.7		68.2→3.0	60.2→4.7
maternal grandfather	52.7→0.4	65.5→1.1	85.5→2.9	60.0→0.5		51.8→5.5
maternal grandmother	56.6→2.9	89.6→0.7	68.2→4.1	72.6→3.4	71.0→4.7	

3.3. Cohen's d for Outcome Variables

Statistical analysis revealed significant differences ($p < 0.05$) for all 11 adolescent outcome variables when either paternal or maternal depression history was present. The Cohen's d effect sizes for paternal depression history ranged from 16.14% to 25.99%, averaging 20.95%. For maternal depression history, the effect sizes were larger, ranging from 28.21% to 47.97%, with an average of 35.56%.

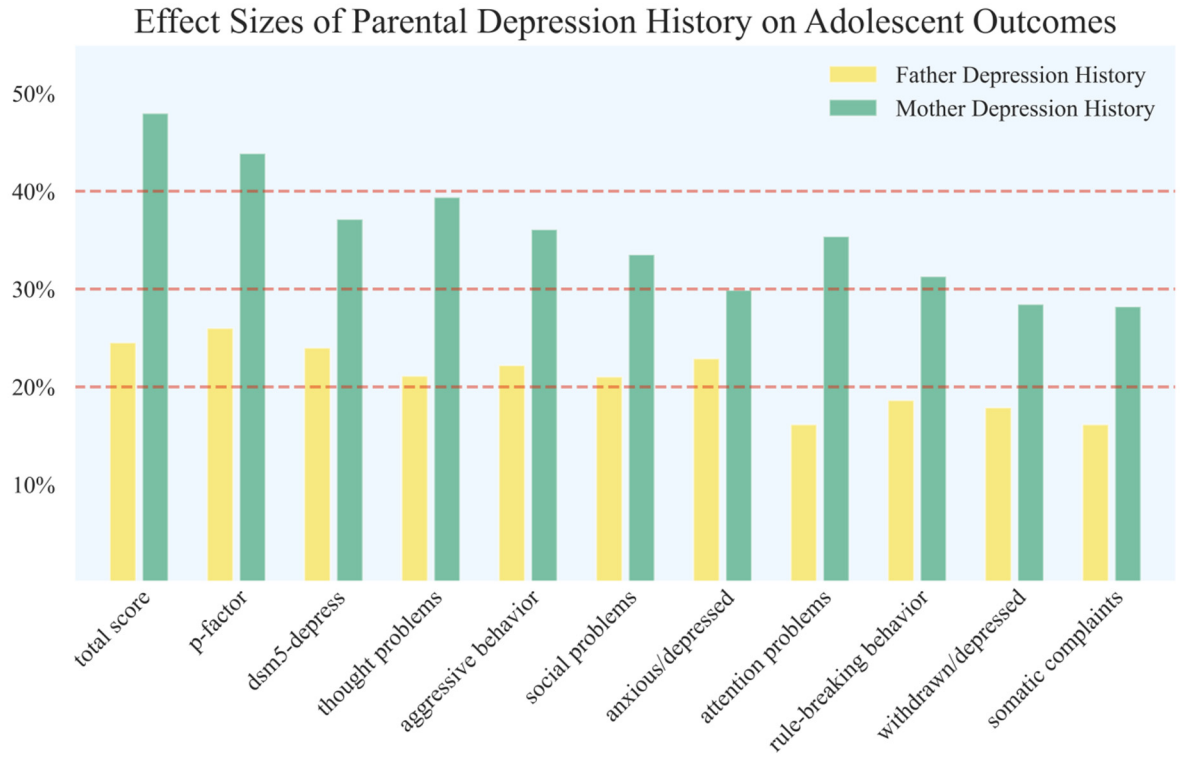


Figure 1. Effect Sizes (Cohen's d) of Parental Depression History

4. Discussion

This study employed propensity score matching to analyze the depression history of six family members as exposure variables. The results showed that only paternal and maternal depression histories satisfied the covariate balance criteria, thereby establishing comparable analytical samples. The Cohen's d effect size was used to quantify the impact of parental depression history on offspring mental health. Both paternal and maternal depression histories had significant effects on adolescent behavioral measures, CBCL total scores, depressive symptoms, and general psychopathological vulnerability (p-factor). The effect of maternal depression history was generally greater than that of paternal depression, with an average effect size approximately 15% higher, suggesting that mothers may play a more critical role in the intergenerational transmission of depression.

These findings suggest that the impact of a family history of depression on adolescent mental health is broad, extending beyond an increased risk for depression alone to elevate susceptibility to a range of psychiatric conditions. This provides an integrated perspective that transcends single diagnoses for understanding the familial aggregation of mental illness. The assessment of the p-factor aids in the earlier identification of individuals at high risk for mental health problems before the full manifestation of clinical symptoms, thereby offering a basis for implementing early prevention and intervention strategies.

This study's strength lies in its use of a large-scale dataset to quantify the impact of familial depression history on various adolescent externalizing behaviors, depressive symptom, CBCL total score, and the latent vulnerability to psychopathology (p-factor). Methodologically, the matching ratio was flexibly adjusted based on the sample size of the intervention group, which maximized data retention while ensuring quality. This approach provides more robust evidence for the intergenerational transmission of depression. However, further investigation into the underlying mechanisms of this transmission is still required.

First, our study employed a cross-sectional design, with all measurements taken at a single time point. This precludes examination of changes in adolescents' mental health over time. Second, the analysis relied solely on baseline data from the ABCD study, which includes only children aged 8 to 11. This age range represents a limitation, as children this young are not a high-risk population for depression.

Competing Interests

The authors declare no competing interests.

References

[1] Friedrich MJ. Depression is the leading cause of disability around the world. JAMA. 2017 Apr 18;317(15):1517.

[2] Shorey S, Ng ED, Wong CHJ. Global prevalence of depression and elevated depressive symptoms among adolescents: a systematic review and meta-analysis. Br J Clin Psychol. 2022 June;61(2):287–305.

[3] Hankin BL, Abramson LY, Moffitt TE, Silva PA, McGee R, Angell KE. Development of depression from preadolescence to

young adulthood: emerging gender differences in a 10-year longitudinal study. J Abnorm Psychol. 1998 Feb;107(1):128–40.

[4] Rao U, Chen LA. Characteristics, correlates, and outcomes of childhood and adolescent depressive disorders. Dialogues Clin Neurosci. 2009;11(1):45–62.

[5] van Dijk MT, Murphy E, Posner JE, Talati A, Weissman MM. Association of multigenerational family history of depression with lifetime depressive and other psychiatric disorders in children: results from the adolescent brain cognitive development (ABCD) study. JAMA psychiatry. 2021 July 1;78 (7):778–87.

[6] Weissman MM, Wickramaratne P, Gameroff MJ, Warner V, Pilowsky D, Kohad RG, et al. Offspring of depressed parents: 30 years later. Am J Psychiatry. 2016 Oct 1;173(10):1024–32.

[7] Casey BJ, Cannonier T, Conley MI, Cohen AO, Barch DM, Heitzeg MM, et al. The adolescent brain cognitive development (ABCD) study: imaging acquisition across 21 sites. Dev Cognit Neurosci. 2018 Aug; 32:43–54.

[8] Stewart LC, Asadi S, Rodriguez-Seijas C, Wilson S, Michelini G, Kotov R, et al. Measurement invariance of the child behavior checklist (CBCL) across race/ethnicity and sex in the adolescent brain and cognitive development (ABCD) study. Psychol Assess. 2024 Aug;36(8):441–51.

[9] Buuren S van, Groothuis-Oudshoorn K. mice: multivariate imputation by chained equations in R. J Stat Softw. 2011 Dec 12; 45:1–67.

[10] Hill ED, Kashyap P, Raffanello E, Wang Y, Moffitt TE, Caspi A, et al. Prediction of mental health risk in adolescents. Nat Med. 2025 June;31(6):1840–6.

[11] Yang FN, Xie W, Wang Z. Effects of sleep duration on neurocognitive development in U.S. early adolescents: a propensity score matched, longitudinal, observational study. Lancet, Child adolesc health. 2022 July 29;S2352-4642(22) 00188-2.