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Family depression profiles among adolescents and their parents: A group-based multi-trajectory

modeling

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Author Note: Part of the current paper was presented at the poster session of the 2020 National Council on Family Relations annual conference.

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Abstract

Early onset of depression predicts unfavorable psychosocial and health outcomes, and depression often co-occurs for children and their parents, yet family profiles of depression trajectories are not fully examined. This population-based longitudinal prospective cohort study included 2,111 families drawn from the Chinese Family Panel Study (CFPS) with biannual assessments from 2010 to 2018. Group-based multi-trajectory modeling was used to identify depression trajectories of children, fathers, and mothers. Six distinct profiles of depression symptoms were identified. Based upon multi-trajectory findings of family depression profiles, when adolescents are at risk for depression, there is likely at least one parent concurrently at risk for depression, but not vice versa. Families with social disadvantages and children of delayed developmental milestones are at elevated risk for depression. Even when children are at low risk for depression, depression in parents can spill over to impact other psychosocial and health outcomes. These findings suggest examining depression and its associating psychosocial factors could help identify trajectories of varying onset and continuity, which can inform early prevention and intervention from a family system perspective.

Keywords: adolescent depression; parental depression; maternal; paternal; trajectory modeling

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Depression is a leading cause of disability worldwide and is predicted to be the first disease burden by 2030 (Malhi & Mann, 2018). While depression is relatively rare during childhood, adolescence marks the age with the highest risk for depression onset (Kessler et al., 2001). Adolescent depression has been shown to associate with a range of unfavorable outcomes, including, but not limited to, academic difficulties, parent-teen conflicts, and physical health problems (Fröjd et al., 2008; Goodman, 2020; Marmorstein & Iacono, 2004; Myrtveit et al., 2014). Importantly, adolescent depression often occurs in a joint family system.

Parental Depression and Adolescent Depression

A substantial body of research suggest high co-occurrence of adolescents' and parents' depression (Goodman, 2020; Gotlib et al., 2020; Sweeney & MacBeth, 2016; Wilkinson et al., 2013). Children of depressed parents are three to six times more likely to have depression, compared to children of non-depressed parents (Gotlib et al., 2020; Sweeney & MacBeth, 2016), with the rates of depression being estimated to be 20% to 41% among school-aged and adolescent children of depressed mothers (Goodman, 2007). Similarly, major depressive disorder (MDD) is significantly more prevalent among parents of children with MDD (34.4%) than in parents of children without (16.1%; Kendler et al., 1997).

The relationship between maternal and adolescents' depression has been well documented, with the role of paternal depression being increasingly recognized in adolescent depression (Connell & Goodman, 2002). Both maternal and paternal depression are correlated with adolescents' depression (Jacobs et al., 2015; Kane & Garber, 2004), with meta-analysis findings indicating a stronger association between maternal and offspring depression (Connell &

Goodman, 2002). Recent research also suggested more complex interrelationships such as an additive effect (Gutierrez-Galve et al., 2019; Reeb et al., 2010) or differential impact of paternal and maternal depression depending on offspring symptom severity (Klein et al., 2005).

While providing important information regarding the parent-child associations of depressive symptoms, most studies mentioned above are limited to individual-level (e.g., adolescents only) or dyadic-level (e.g., cross-lagged panel models of adolescents' and maternal depression) analyses. Such methodology overlooked the complexities and nuances of depressive symptoms' unfolding processes among all members within a family unit. Group-based trajectory modeling techniques (Nagin et al., 2018) can potentially remedy this gap in our knowledge. Univariate group-based trajectory modeling (also known as growth mixture modeling and latent class growth analysis) is increasingly employed to explore the heterogeneity of youth depression by determining subgroups of symptom trajectories (Ellis et al., 2017; Schubert et al., 2017). Findings from these univariate trajectory studies have indicated 20-40% youth of varying symptom levels at different time points during adolescence, with common subgroups including consistent high/moderate symptoms, increasing/decreasing, adolescent-limited, or resurging depressive symptoms (Ellis et al., 2017; Schubert et al., 2018).

Although the parallel trajectories of depressive symptoms between youth and their parents had not be explicitly explored in the literature, treatment studies for adolescents (i.e., no parental involvement) reported significant associations among child's, mother', and father's depressive symptoms at baseline, as well as changes in symptoms over time (e.g., Eckshtain et al., 2018; Wilkinson et al., 2013). Conversely, successful treatment and improvements in parents' depression have also been shown to associate with corresponding improvements in offspring depressive symptoms (Beardslee et al., 2007; Gunlicks & Weissman, 2008). These

findings supported the dynamic changes of depressive symptoms within a family system. Nevertheless, these treatment efforts tend to focus on adolescents or their parents only. In fact, while there had been proliferation of prevention and intervention efforts targeting adolescents, only an estimated 32% of treatments on youth's depression include parents in any capacity (Sander & McCarty, 2005). One of the barriers to effective care is the lack of understanding how depressive symptoms jointly unfold within a family system.

Associative Factors and Outcomes with Depression Trajectories

A range of risk factors and psychosocial outcomes have been identified to associate with adolescent depression trajectories (see Schubert et al., 2017 and Shore et al., 2018 for a review). Salient demographic factors including gender, minority group membership, and family socioeconomic status have been shown to associate with adolescent depression (Goodman, 2020; Lemstra et al., 2008). For instance, comparing to boys, girls have been reported as experiencing not only a higher level of depression (Goodman, 2020), but also potentially different depression trajectories (Ellis et al., 2017; Fernandez Castelao & Kröner-Herwig, 2013). Parent-level factors, in particular parents' psychological distress (Ensminger et al., 2003) and parent-child relationship (Goodman et al., 2020) were closely related to offspring depression. In addition, longitudinal studies have identified certain early developmental factors as risk factors for later depressive symptoms and/or disorder in adolescence, such as premature birth (Patton et al., 2004) and developmental delay (Colman et al., 2007). Last but not least, researchers have suggested the association between adolescent depression with a range of impaired outcomes, including physical health and other mental health disorders (Johnson et al., 2018). Lower academic achievement as a result of depression with family is of particular concern among adolescence, given that academic performance likely reflects the downstream effects of

depression on their broader cognitive functioning (Letourneau et al., 2013; Li et al., 2021). As a substantial portion of the within-family correlation of depression may be accounted for by confounding factors (Pelham et al., 2021), a comprehensive examination of these associative factors within a large-scale group-based modeling study is needed and critical for the identification of risk and modifiable factors in order to design targeted prevention and intervention.

Cultural Context of the Current Study

With the associations between parental and offspring depression being well-established in the developed countries, this link is limitedly explored among low- and middle- income nations. The rapid increase in the prevalence of depression in China, accompanying limited resources for diagnosis and treatment and high medical cost, raises a significant public health concern (Qin et al., 2018; Que et al., 2019; Ryder et al., 2012). Persistent mental health stigma may further restricted the detection and treatment of depression (Ryder et al., 2012; Z. Xu et al., 2017). In addition, adolescent depression within the family system in China warrants special attention on two grounds: First, from a developmental psychopathology perspective, Chinese adolescents may be differently vulnerable to stressors (e.g., academic performance) that precede their presentations of depressive symptoms (Ryder et al., 2012). Second, family cohesiveness and harmony are particularly valued in the Chinese cultural context (Kavikondala et al., 2016). Thus, the occurrence of depressive symptoms among Chinese adolescents and their parents may present a more concordant pattern compared to those from countries where independence and individualism during adolescence are more valued. The family structure under the one-child policy may also amplify the mutual influence between Chinese adolescents and their parents

(Settles et al., 2013). These contextual factors set important basis for understanding the dynamic changes of depressive symptoms within the Chinese family system.

The Current Study

The primary objective of the current study was to investigate the joint prevalence and fluctuations of depression among a large sample of youth and parents in a prospective longitudinal study. Utilizing data from the Chinese Families Panel Study (CFPS), the current study aimed to estimate the depression trajectories among adolescents and their parents jointly. Based upon previous adolescents-only depression trajectory studies (Ellis et al., 2017; Schubert et al., 2017; Shore et al., 2018), we hypothesized that the largest subgroup would be low risk in depression among all family members. Given the explorative nature of the current multi-trajectory analysis, no other prior hypotheses were made about the number and shape of the other subgroups. Similar to prior adolescent-only depression trajectory studies (Ellis et al., 2017), a range of variables were examined to identify risk and other associated factors amongst the subgroups. These auxiliary variables contained commonly assessed factors (Ellis et al., 2017; Schubert et al., 2017), including demographics, perinatal and postnatal factors, pre/early adolescence factors, and late adolescence/early adulthood factors.

Method

Participants and Procedure

Data were drawn from the Chinese Families Panel Study (CFPS), an ongoing longitudinal cohort study that recruited Chinese families with population-based sampling from 2010 to date. The initial cohort was collected from 25 most populous province-level regions of China, consisting of 14,960 families with 42,590 participants. Ethical approval was obtained from the local research ethics committee. The open-source data website contains detailed information about the sampling methods, survey questions, and coding manuals (Institute of Social Science Survey, 2019).

The current analysis was based upon the cohort of families with a child between ages 10 and 15 interviewed in 2012 (N = 2,573). Families that have a deceased parent (n = 116), and families in which depression data were not obtained due to divorce (n = 72), migration for work (n = 246; known as left-behind children (Xu & Xie, 2015)), or other reasons at times of data collection (n = 28) were further excluded. The final sample included 2,111 families with 2,406 youth. See Table 1 for more detailed sample descriptions. For families with two or more children between 10 and 15 in 2012 (n = 266, 13%), only one child was randomly selected to reduce duplicated/nested estimates of depression trajectories.

Measures

Depression. Adolescents', fathers' and mothers' depression symptoms were measured in 2012, 2016, and 2018 by the Center for Epidemiologic Studies Depression Scale-8 (CESD-8; Van de Velde et al., 2009). CESD-8 is a widely used and validated tool in international research (Radloff, 1977; Van de Velde et al., 2009) to assess the presence of depressive symptoms over the last two weeks. Each item (e.g., "I felt that everything I did was an effort") is rated on a four-point Likert-type scale from 0 (= rarely or none of the time, less than one day) to 3 (= most or all of the time, 5 to 7 days). Mean scores were computed, with internal consistency reliabilities ranged from .64 to .79 for adolescents (STable 1), .72 to .84 for fathers (STable 2), and .74 to .81 for mothers (STable 3).

Family characteristics and demographics. Adolescent sex, residency, and ethnicity were collected in 2010 with female (vs. male), rural (vs. urban) residency, and Han Chinese (vs. 55 ethnic minority statuses including Zhuang, Hui, Man, etc.) as the reference group. The annual

family income in 2012 was coded into quintile, adjusted for urban and rural status (see Table 1). Father's and mother's education level were coded as illiterate/semi-literate (=1), primary school (=2), junior high school (=3), senior high school (=4), and college or above (=5). Data were missing for child sex (0%), urban/rural residency (1%), ethnic minority status (9%), per capital annual household income (3%), father's education (23%), and mother's education (14%).

Perinatal and postnatal factors. One primary caregiver reported in 2010 on behalf of the child of their premature birth status (less than nine months), frequency of illness before age one (ranging from 0 to 50, Median = 1, Mean = 3.01, SD = 5.06), age (in months) of walking independently, speaking the first full sentence, and counting up to ten, and whether the child has been separated from parents over one month before age three (1 = yes, 0 = no). Data were missing for premature birth (6%), frequency of illness before age one (17%), age of walking independently (6%), speaking the first full sentence (6%), and counting up to ten (9%), and separation from parents before age three (9%).

Pre/Early adolescent factors. Child grades in verbal (Mandarin Chinese) and math during the last semester were reported by one primary caregiver in 2010, each using a 4-point Likert-type item: excellent (= 1), good (= 2), average (= 3), poor (= 4). Adolescents reported in 2012 how many times they had quarrels with parents (i.e., parent-child conflicts) and how many times they witnessed parents quarrel with each other (i.e., marital conflicts) in the last month. Due to the positive skewness of parent-child conflicts (Mean = .95, SD = 2.51, Range = 0 to 30, Skewness = 6.70) and martial conflicts (Mean = .59, SD = 1.66, Range = 0 to 30, Skewness = 7.00), logarithm transformation – log(x+1) – was applied. Data were missing for child's verbal grades (3%), child's math grades (3%), parent-child conflict last month (10%), and martial conflict last month (14%).

The Kessler Psychological Distress Scale (K-6; Kessler et al., 2010) was administered in 2010 and 2014 to all individuals over ten years old (i.e., adolescents and parents). The K-6 is a self-report measure of psychological distress, drawn from both depression and anxiety symptomology, to assess risk for mental illness in the general population. Respondents indicated how often they had six different feelings or experiences during the past 30 days using a 5-point Likert scale from 0 (None of the time) to 4 (All of the time). As data were scaled by linking the cohorts together on the basis of child's age in a cohort-sequential design, only one K-6 score was retained to capture adolescents' and parents' psychological stress during the early adolescence phase (10 to 13 years old). The utilization of K-6 with Chinese populations have been validated (Kang et al., 2015). Cronbach α s were .80, .86, and .84 for adolescents, mothers, and fathers respectively in the final sample. Data were missing for child's K-6 (8%), father's K-6 (13%), and mother's K-6 (9%),

Late adolescent/early adult factors. The acute illness was self-reported by children, fathers, and mothers in 2018 with a binary item (1 = yes, 0 = no) on "was there any physical discomfort over the last two weeks." Chronic illness was self-reported by fathers and mothers with a binary item (1 = yes, 0 = no) on "any diagnosed chronic illness in the last six months." Current subjective health status was self-reported by children, fathers, and mothers with a 5point Likert scale item: 1 (=Healthy), 2 (=Fair), 3 (=Relatively unhealthy), 4 (=Unhealthy), and 5 (= Very unhealthy).

Children's cognitive ability in verbal (Mandarin Chinese) and math were measured in 2018 using a standardized test battery developed by the CFPS study (Huang et al., 2015). For the verbal test, children were presented with 34 Chinese characters sorted in ascending order of difficulty and were asked to read the character aloud (score range 0-34). For the math test,

children were presented with 24 mathematical problems sorted in order of increasing difficulty (score range 0-24). Entry and termination rules were applied for efficiency purposes (refer to Huang and colleagues (2015) for more details describing the testing approach). Both the verbal and math tests were standardized within their age group with Mean = 100 and SD = 15 to control for the effect of age on cognitive development. Data were missing for child's verbal performance (34%), child's math performance (53%), child's acute illness (23%), child's self-rated health (19%), father's chronic illness (12%), father's acute illness (12%), father's self-rated health (10%), mother's chronic illness (13%), mother's acute illness (13%), and mother's self-rated health (10%).

Attrition Analysis and Longitudinal Model

The CESD-8 data were missing at T1-2012 (3%-17%), T2-2016 (15%-30%), and T3-2018 (21%-40%). As the missingness in longitudinal, multi-informant data is not linear, we hereby reported that 50% adolescents (n = 1,045) completed all three waves of CESD-8 in 2012, 2016, and 2018, 33% adolescents (n = 694) completed two waves, and 18% (n = 372) completed only one wave of CESD-8. Around 59% fathers (n = 1,248) completed all three waves, 25% (n =538) completed two waves, and 15% (n = 325) completed only one wave of CESD-8. Around 64% mothers (n = 1,358) completed all three waves, 23% (n = 478) completed two waves, and 13% (n = 265) completed only one wave of CESD-8. Altogether, around 33% families have all three members each provided data from all three waves. We further rescaled the child, father, and mother data (see STables 1-3) from three waves (i.e., 2012, 2016, 2018) to 12 waves spanning from late childhood (age 10) to young adulthood (age 21) by linking the cohorts together on the basis of child's age in a cohort-sequential design (Miyazaki & Raudenbush, 2000). The Little's MCAR test was conducted with depressive symptoms from all 12 waves and

child gender to evaluate the likelihood that missing data were completely at random for trajectories analyses. The test supported a missing completely at random pattern, $\chi^2(175) = 195.58$, p = .14. Therefore, we employed maximum likelihood estimation to retain all participants in trajectory analyses (Graham, 2009).

In addition, as reported in the measures section, missingness for the auxiliary variables ranged from 0% to 53% with the majority of the auxiliary variables missing below 15%. We performed multiple imputation analysis based upon the 2,111 families to account for missing values in the auxiliary variables in order to reduce parameter bias due to longitudinal attrition (Asendorpf et al., 2014). Following Asendorpf et al.'s (2014) guideline, we conducted analysis on the anxuillary variables using 40 imputed dataset. Results from the multiple imputations were similar as results without multiple imputation (i.e., pairwise deletion), thus only results from the multiple imputation were presented.

Statistical Analysis

Group-based multi-trajectory modeling was conducted with SAS 9.1 PROC TRAJ (Jones et al., 2001; Nagin et al., 2018) to jointly estimate trajectories of adolescents', fathers', and mothers' depressive symptoms. Multi-trajectory modeling is an extension of the univariate group-based modeling technique and allows the identification of different profiles by the joint development of depressive symptoms within one family unit. Parameters were estimated using maximum likelihood estimation through a Newton-Raphson optimization algorithm and censored-normal models.

A two-stage procedure was used to identify the best fitting. As a first step, only linear slope factors were included with class numbers ranging from one to eight. As indicated in Table 2, models were selected based on low Bayesian information criteria (BIC) and sample size

adjusted BIC (SABIC) values (Nagin et al., 2018). Both six and seven group models provided the best BIC fit. In the second stage, quadratic terms were fitted in the six and seven group models. Based on these two criteria, a six group multi-trajectory model provided the best fit to the data and was selected. Further, criteria for judging the adequacy of the selected model (Nagin et al., 2018), including the average posterior probabilities of assignment (APPA > 70%), odds of correct classification (OCC > 5), and sufficient class sizes (minimum 1 % of sample size per class) were presented in Table 3. Overall model fit criteria supported the adequacy of the sixgroup model, with the exception of some APPA indices. Each family was assigned to the multitrajectory group having the highest posterior probability.

As explorative analyses, after the selection of the final model, auxiliary variables were examined for differences across the trajectory groups by analysis of variance and chi-square tests. For continuous variables, the mean parameters were estimated for each group. For categorical variables, the probabilities for each category were estimated for each group. Pairwise group comparisons after establishing the overall significance of ANOVA and Chi-Square tests were then computed. The Benjamin-Hochberg procedure (Benjamini & Hochberg, 1995) was used to adjust for the false discovery rate (FDR) from multiple comparison rate, which controls the expected proportion of false discoveries (q) amongst the rejected null hypotheses. The falsepositive rate was set at q = 0.05.

Results

Preliminary Analysis

The CESD-8 mean scores across ranged from .50 to .67 for adolescents, .61 to .69 for fathers, and .74 to .88 for mothers (Supplemental Table S1-S3). The bivariate correlations were .02 to .31 between adolescent's and fathers' CESD-8 scores, .03 to .29 between

adolescent's and mother's CESD-8 scores, and .17 to .43 between parents' CESD-8 scores at each age across 12 years (Supplemental Table S4). In addition, paired *t* tests indicated that mothers' CESD-8 scores were significantly higher than fathers' CESD-8 scores at all measurement points, ps < .007, with small effect sizes [.24-.30].

Family Depression Profiles

As described under Statistical Analysis, six family profiles emerged, displaying distinct developmental patterns of concomitant depression among adolescents, fathers, and mothers. We characterized depressive symptoms into low (0-.8), moderate (.8-1.25), high (more than 1.25) based upon prior studies for labeling and interpretation purposes, although optimal cut-off scores for CESD-8 remain unclear depending on the sensitivity and specificity for targeted populations (e.g., Briggs et al., 2018; Steffick, 2000).

The first and the largest group, which was estimated to comprise 35.6% of the sampled population, endorsed the low, non-clinically significant depressive symptoms among all family members. This group was relatively stable in their depressive symptoms across time, albeit with slight fluctuations of mothers' CESD-8 scores. The other five groups, compared to the first group, were presented with varying levels of risk for depression. The second group, estimated to account for 13.1% of the sampled population, indicated low but increasing depressive symptoms among adolescents, and moderate and stable depressive symptoms among parents. The third and smallest group accounted for 3.2% of the sampled population. In this group, adolescent started with low depressive symptoms at age 10 and gradually increased to moderate symptoms at age 21. Mothers started with moderate depressive symptoms and increased to high symptoms. Whereas fathers started with moderate depressive symptoms, which decreased to low around child age 16 and then resurged back to moderate depressive symptoms. In the fourth group, with

an estimated 26.8% of the sampled population, depressive symptoms remained low and stable for both adolescents and fathers, whereas mothers' depressive symptoms were moderate with slight increase. In contrast, group five, with an estimated 12.5% of the sampled population, indicated low and stable depressive symptoms for both adolescents and mothers, whereas fathers' depressive symptoms increased over time. The sixth and the final group, which was estimated to comprise 8.8% of the sampled population, indicated high depressive symptoms for both fathers and mothers. Whereas adolescents' depressive symptoms started at the moderate level, which decreased until around age 16, and increased back to moderate level. Multitrajectory groups are displayed in Figure 1.

Auxiliary Variables

The family characteristics of each profile and FDR-adjusted bivariate analysis with multiple imputation (Asendorpf et al., 2014) were presented in Table 4. These exploratory analyses indicated that these six distinct family profiles of depression were not significantly different in terms of child sex, premature birth rate, and frequency of illnesses before age one. For the 26 significant effects, the effect sizes ranged from small to moderate [.08-.31]. Rural residency, lower household income, ethnic minority status, lower parental education, delayed developmental milestones, and separation from parents were identified as demographic and early childhood risk factors associated with elevated depression symptom profiles (i.e., Groups 2 to 6). Concurrently, parent-child conflict, marital conflict, lower academic achievement and cognitive performance, and deterioration in physical health status were associated with elevated depression symptom profiles. The level of psychological distress measured by K-6 were similar to the level of depressive symptoms across six family profiles.

Discussion

This study identified six distinct family profiles of depression trajectories from children's late childhood to early adulthood. Specifically, there were varying elevated depressive symptoms for adolescents in Group 2, 3, and 6, for fathers in Group 3, 5, and 6, and for mothers in Group 2, 3, 4, and 6. In other words, when adolescents presented with elevated depressive symptoms, at least one parent (i.e., mother) was jointly in an elevated symptom trajectory group, but not vice versa (i.e., Groups 4 and 5). These findings provide unique empirical evidence to understand the longitudinal and dynamic associations of depression among children and their parents.

The profiles of adolescent trajectories are consistent with prior findings (Ellis et al., 2017; Schubert et al., 2017; Shore et al., 2018), which typically demonstrated a three- to five-profile solution. In contrast to some prior studies (e.g., Ellis et al., 2017; Schubert et al., 2017; Shore et al., 2018), however, no evidence of a decreasing or "recovery" adolescent trajectory was detected in the current study. Possible explanations may include the varying measurement intervals and frequencies employed across these trajectory studies (Timmons & Preacher, 2015). Moreover, the treatment rates for depression in China was particularly low in comparison to those of developed countries (Que et al., 2019), which might contribute to the lack of a decreasing or "recovery" trajectory emerging from our data, further emphasizing the needs to increase treatment access for those experiencing depression in China.

To the best of our knowledge, this is the first study that jointly estimated trajectories for both mothers and fathers during the same developmental period as their children. The current study extends from the well-established findings on intergenerational transmission of depression (Goodman, 2020; Gotlib et al., 2020) and identifies distinctive depression trajectories from early adolescent to early adulthood. Subgroups of fathers (Group 5) and mothers (Group 3) with increasing symptoms from their children's late childhood to early adulthood were identified,

supporting the notion that adolescence may be a risk period for both children *and* their parents (Weymouth et al., 2016). Prior studies have suggested earlier onset of depression can result in a worse prognosis for adolescents (e.g., Kessler et al., 2001) and their mothers (Kouros & Garber, 2010). Taken together with our findings, there are reasons to speculate all family members may be at higher risks for depression when adolescents have early onset depression (Group 6).

As explorative analyses and in accordance with the literature, these trajectories were associated with a wide range of demographic, early childhood risk factors, and psychosocial and health outcomes (Schubert et al., 2017). The unique combination of these factors at different times set not only children but also parents on divergent trajectories (George & Engel, 1980). The largest emerged profile (Group 1; 35.6%) was associated with the least early childhood risk factors and the best psychosocial and health outcomes, which is not surprising given all family members endorsed no or low depressive symptoms in this profile. Presenting with an early onset of adolescent depression and high depressive symptoms among all family members, Group 6 was associated with the most early childhood risk factors and the worst psychosocial and health outcome. In contrast to Group 6, Group 2 presented with a late onset of adolescent depression and moderate depressive symptoms for parents. Group 2 was also associated with numerous unfavorable outcomes, but only for adolescents and not for parents. Overall, individual family member's psychosocial and health outcomes are congruent with their respective depressive symptom levels. In other words, higher depressive symptoms in adolescents, fathers, and mothers were associated with more unfavorable psychosocial and health outcomes in adolescents, fathers, and mothers, respectively. However, it is interesting to note the potential spillover phenomena in these findings—children who were at low risk for depression but with a father (Group 5) or a mother (Group 4) who endorsed elevated depressive symptoms also

indicated with more unfavorable outcomes (e.g., developmental milestones, academic outcomes, and psychological distress)—compared to the all low-risk profile (Group 1). Although the current study is limited in scope in concluding the causation between these auxiliary variables and the trajectory groups, in line with that being recommended by Pelham and colleagues (2020), these explorative findings provide important insights for future directions that go beyond the intergenerational association of depression and inform the examination of potential causative pathways to depression in a family system.

Limitations and Strengths

Several cautions need to be considered in interpreting the current findings. The current analysis used depressive symptoms measured at three time points to estimate the trajectories spanning 12 years based on child's age in a cohort-sequential design using maximum likelihood estimation (Miyazaki & Raudenbush, 2000; Nagin et al., 2018). Additional measurement occasions in future studies are encouraged to increase the model fit and offer more nuanced changes in the unfolding process of depressive symptoms (Timmons & Preacher, 2015). As with other longitudinal studies, patterns of attrition can also potentially bias the results. In addition, the biopsychosocial factors of depression and the interrelations among children and their parents are highly complex, thereby making causal inferences from longitudinal associations—as in the current analytical approach—difficult to formulate. Nonetheless, these findings still provide potentially useful information to design population-based or targeted interventions to address depression within a family system. Lastly, due to the nature of a secondary data analysis, some other important auxiliary variables not available in the dataset such as parenting practices (Goodman et al., 2020) were not explored.

Despite the above-mentioned limitations, the current study presents with several strengths. This study was conducted with a nationally representation, population-based cohort of Chinese children, fathers, and mothers using multi-informant reports and a prospective longitudinal design. This work contributes to the growing knowledge for early identification and prevention as well as understanding the debilitating effects of depression, especially given the lack of knowledge in low- and middle- income countries (Schubert et al., 2017). Importantly, the lack of identification of a decreasing/recovery adolescent depression trajectory underscores the importance for increasing prevention and intervention efforts for depression among Chinese adolescents. The identification of differing family profiles of depression trajectories and their associated factors is consistent with the biopsychosocial approach to understanding developmental psychopathology (George & Engel, 1980), encouraging future studies to continue exploring factors beyond the direct association between parental and adolescent depression. Taken together, the findings from this study highlight the need to integrate parents as prevention and treatment for adolescent depression from a family systems approach (Sander & McCarty, 2005).

Conclusions

To the best of our knowledge, this is the first study that jointly estimated the depression trajectories of a family unit spanning from adolescents' late childhood to early adulthood. Six distinct family depression profiles were identified in this population-based cohort study. Compared to the low-risk profile (35.6%), other trajectories profiles were associated with varying demographic and early childhood risk factors and less desirable psychosocial and health outcomes, especially in profiles when multiple family members had elevated depressive symptoms. Overall, these findings imply that the etiology of depression symptoms is

multifaceted and needs to be further integrated into a family systems perspective. These findings can also be potentially translated into interventions and treatments for families with elevated risk, which could in turn, prevent or reduce depression and other detriments in life.

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Characteristics	Study sample (2 111 families	Full sample (2 111 families
Characteristics	with 2 111 youth)	with 2 406 youth)
Boys (vs. girls) (%)	1 126 (53%)	1 269 (53%)
Ethnic minority (vs. Han) (%)	224 (12%)	1,209 (0070)
Urban (vg. mral) familias (%)	224(1270) 012(280/)	
Upper la constant de	For when formilies	
Household annual income per	For urban families $(1-)$ 164 families $(M-1001)$	SD = 1.245
capita (in yuan) by quintile	(1=) 164 families (M = 1,991, S	SD = 1,243
	(2=) 163 families (M = 5,/36, S	SD = 990)
	(3=) 163 families (M = 9,424, S	SD = 1,181
	(4=) 163 families (M = 14,6/6,	SD = 1,847
	(5=) 163 families (M = 35,513,	SD = 37,675)
	For rural families	
	(1=) 246 families (M = 1,084, S	SD = 666)
	(2=) 246 families (M = 3,459, S	SD = 721)
	(3=) 245 families (M = 6,081, S	SD = 811)
	(4=) 245 families (M = 9,538, S	SD = 1,263)
	(5=) 245 families (M = 18,855,	SD =9,278)
Father's age	M = 38.28, $SD = 5.01$, Median	= 37
Father's education	277 (17%) illiterate/semi-literate	te
	448 (27%) primary school	
	575 (35%) junior high school	
	217 (13%) senior high school	
	116 (7%) college or above	
Mother's age	M = 36.45, $SD = 4.77$, Median	= 36
Mother's education	533 (29%) illiterate/semi-literat	te
	497 (27%) primary school	
	526 (29%) junior high school	
	160 (9%) senior high school	
	99 (5%) college or above	

Table 1Participant Demographic Characteristics

Note. The income quintile was divided by urban and rural residency in accordance to the 2012 Chinese census data (National Bureau of Statistics of China, 2012). College or above education included 2- to 3- year college, 4- year college/bachelor's degree, master's and doctoral degree. Father's and mother's ages when their children were 10 years old were calculated.

Κ	BIC	SABIC
1	12052.31	12043.38
2	11458.88	11443.00
3	11381.85	11359.02
4	11292.55	11262.78
5	11274.55	11237.83
6	11255.72	11212.05
7	11258.66	11208.05
8	11279.78	11222.22

Table 2.Model Fit for Growth Mixture Models

Table 3Model fit criterion of the multi-trajectories of adolescents', fathers', and mothers'depression

ucpression			
Trajectory Group	Sample	Average Posterior Probability of	Odds of Correct
	Size %	Group Membership	Classification (OCC)
1	35.6%	79.8%	7.1
2	13.1%	64.4%	12.0
3	3.2%	78.3%	108.9
4	26.8%	65.0%	5.1
5	12.5%	67.8%	14.7
6	8.8%	76.7%	34.3
5 6	12.5% 8.8%	67.8% 76.7%	34.3

Table 4.

Proportions and Mean Level of Family Characteristics for the Six Multi-trajectory Groups: FDR-Adjusted Bivariate Analysis

	Group 1	Group 2	Group 3	Group 4	Group 5	Group 6	adj. p	Effect	Pairwise				
	(n = 808)	(n = 238)	(n = 56)	(n = 576)	(n = 246)	(n = 187)		size	comparisons				
Family characteristics and demographics													
Boys (vs. girls) (%)	52%	53%	54%	54%	55%	55%	.954						
Urban (vs. rural) (%)	47%	32%	38%	37%	40%	24%	<.001	.15	1>4,2,6				
Ethnic minority (vs. Han) (%)	9%	18%	12%	10%	11%	20%	<.001	.12	1<2,6 4<6				
Per capital annual household income	3.21 (1.40)	3.10 (1.45)	2.80 (1.37)	2.96 (1.38)	2.70 (1.44)	2.53 (1.34)	<.001	.16	1>4,5,6 2>5,6				
Father's education	2.85	2.52	2.32	2.64 (1.14)	2.64 (1.02)	2.17	<.001	.18	4>6 1>4,2,3,6 5,4,2>6				
Mother's education	2.60 (1.15)	2.11 (1.08)	(1.00) 1.94 (0.95)	2.30 (1.17)	2.29 (1.07)	(1.07) 1.77 (0.96)	<.001	.23	1>4,5,2,3,6 4,5,2>6				
Perinatal and postnatal factor	ſS												
Premature mature birth (%)	2.4%	3.2%	1.9%	2.6%	1.8%	2.9%	.954						
Frequency of illness before	2.73	3.23	3.16	3.32	3.08 (4.69)	3.01 (5.41)	.167						
Age of walking (months)	(4.74) 13.95 (4.35)	(5.51) 14.56 (4.14)	(4.63)	14.69 (5.33)	15.25 (5.82)	16.20 (5.89)	<.001	.14	1<5,6 2,4<6				
Age of speaking (months)	20.03 (8.07)	19.65 (8.01)	20.75 (8.91)	20.56 (8.00)	21.77 (9.14)	23.15 (9.54)	<.001	.12	1<5,6 2,4<6				
Age of counting (months)	32.87 (14.22)	35.80 (16.75)	38.73 (14.51)	36.85 (15.70)	36.20 (16.22)	42.82 (15.85)	<.001	.19	1<5,4,6 2,5,4<6				
Separation from parents over one month prior to age 3 (%)	13%	17%	22%	15%	17%	24%	.005	.10	1<6				
Pre/Early adolescence factors													
Child grades – verbal	2.11 (0.91)	2.43 (0.98)	2.02 (0.96)	2.27 (0.97)	2.30 (0.94)	2.62 (0.93)	<.001	.17	1<4,5,2,6 3<2,6				

									4,5<6
Child grades – math	2.09	2.42	2.24	2.27	2.29	2.60	< 001	16	1<4,2,6
China grades – matri	(0.95)	(0.99)	(0.99)	(1.05)	(1.05)	(0.97)	001	.10	4,5<6
Child psychological distress	1.38	1.59	1.52	1.48	1.40	1.59	<.001	.14	1,5<2,6
enna psychological albaess	(0.54)	(0.65)	(0.75)	(0.59)	(0.54)	(0.69)	.001	•1 •	1<4
Father psychological distress	1.27	1.52	1.56	1.43	1.71	1.90	<.001	.37	1<4,2,3,5<6
	(0.40)	(0.59)	(0.57)	(0.54)	(0.70)	(0.75)	1001	,	4,2<5
	1.35	1.56	1.98	1.58	1.56	1.92	. 001	•	1,5,2,4<6,3
Mother psychological distress	(0.51)	(0.62)	(0.85)	(0.65)	(0.65)	(0.80)	<.001	.29	1<2,4
T C	22	51	20	40	20	12			5<4
Log frequency of parent-child	.33	(70)	.32	.40	.38	.43	<.001	.08	1<2
L og fraguenev of marital	(.39)	(.70)	(.55)	(.04)	(.03)	(.07)			
conflict last month	.22	.57	.29	.29	.23	.33	.005	.10	1<2
I ate adolescence/early adulth	(.43)	(.04)	(.40)	(.33)	(.31)	(.38)			
Late aublescence/carry adultity	101 32	08 76	08 76	100.60	00.52	95.60			
Child verbal performance	(14.34)	(15, 10)	(16.33)	(14.64)	(15.43)	(16.49)	.004	.11	1,4>6
	101 22	96.68	100.69	100.60	(15. 4 5) 99 5 4	(10.4 <i>)</i>) 97 52			
Child math performance	(14.48)	$(14\ 51)$	(15,76)	(14.48)	(1531)	(18.16)	.046	.11	1>2
	(11.10)	(11.51)	(15.70)	(11.10)	(15.51)	(10.10)			1<6.3.2
Child acute illness	11%	30%	27%	16%	16%	26%	<.001	.17	5.4<2
	2.07	2.60	2.34	2.22	2.21	2.44	1	10	1<6.2
Child self-rated health	(0.89)	(0.99)	(0.98)	(0.82)	(0.95)	(0.99)	<.001	.19	5,4<2
F (1 1 1 11)	00/	100/	100/	110/	170/	200/	< 0.01	11	1<5,6
Father chronic illness	9%	12%	19%	11%	1/%	20%	<.001	.11	4<6
Father acute illness	14%	28%	29%	18%	36%	42%	<.001	.23	1,4<2,3,5,6
	262	2 00	2 25	2 95	2 26	2 56			1<4,2,3,5,6
Father self-rated health	(1, 10)	2.98	5.55 (0.88)	(1.10)	(1 13)	(1.08)	<.001	.29	4< 3,5,6
	(1.10)	(1.09)	(0.88)	(1.10)	(1.13)	(1.00)			2<5,6
Mother chronic illness	8%	1/1%	36%	17%	17%	2/1%	< 001	18	1<5,4,6,3
Wother enrome miless	070	17/0	5070	1//0	1//0	27/0	<.001	.10	2,5,4 <3
Mother acute illness	20%	35%	62%	39%	30%	48%	< 001	23	1<5,2,4,6,3
momer acute miless	2070	5570	0270	5770	5070	-1070	.001	.23	5<6,3

									2, 4<3
Mother self rated health	2.74	3.23	4.13	3.39	2.97	3.58	< 001	31	1<5<2,4,6<3
Woller sen-rated health	(1.09)	(1.19)	(1.13)	(1.21)	(1.24)	(1.16)	<.001	.31	2<6

Note. FDR = False Discovery Rate. Effect size was calculated only when p value was significant.*Cramer's V*was calculated for categorical variables and chi-square tests.*Cohen's f*was calculated for ordinal/continuous variables and ANOVA tests. To account for the non-normality of the raw data for the frequency of illness before one, log transformation was conducted prior to the ANOVA test



Figure 1. Family Profiles of Depression Symptoms from Group-Based Multi-Trajectory Modeling

Supplemental Materials

Final Syntax for Multi-Trajectory Modeling in Stata:

traj, multgroups(6) var1(t10-t21) indep1(time10-time21) order1(2 2 2 2 2 2) model1(cnorm) min1(0) max1(3) var2(f10-f21) indep2(time10-time21) order2(2 2 2 2 2 2) model2(cnorm) min2(0) max2(3) var3(m10-m21) indep3(time10-time21) order3(2 2 2 2 2 2) model3(cnorm) min3(0) max3(3)

multtrajplot, xtitle(Age) ytitle1(Adolescent Depression) ytitle2(Father Depression) ytitle3(Mother Depression) ylabel1(0.3(0.3)1.5) ylabel2(0.3(0.3)1.5) ylabel3(0.3(0.3)1.5)

Table S1

		1	· · ·	0	0	*		~ ~		~	0		
Cal	out					А	dolescent	Age (year	:s)				
Cor	iori –	10	11	12	13	14	15	16	17	18	19	20	21
	Ν	283	-	-	-	285	-	256	-	-	-	-	-
1	%	16%	-	-	-	16%	-	24%	-	-	-	-	-
	α	.69	-	-	-	.74	-	.74	-	-	-	-	-
	Ν	-	280	-	-	-	234	-	211	-	-	-	-
2	%	-	10%	-	-	-	25%	-	32%	-	-	-	-
	α	-	.66	-	-	-	.66	-	.76	-	-	-	-
3	Ν	-	-	336	-	-	-	280	-	260	-	-	-
	%	-	-	8%	-	-	-	24%	-	29%	-	-	-
	α	-	-	.68	-	-	-	.75	-	.77	-	-	-
4	Ν	-	-	-	354	-	-	-	270	-	232	-	-
	%	-	-	-	7%	-	-	-	29%	-	39%	-	-
	α	-	-	-	.71	-	-	-	.72	-	.79	-	-
5	Ν	-	-	-	-	332	-	-	-	256	-	226	-
	%	-	-	-	-	6%	-	-	-	28%	-	36%	-
	α	-	-	-	-	.64	-	-	-	.73	-	.74	-
6	Ν	-	-	-	-	-	330	-	-	-	253	-	217
	%	-	-	-	-	-	8%	-	-	-	30%	-	40%
	α	-	-	-	-	-	.71	-	-	-	0.74	-	.75
Μ		.56	.58	.50	.57	.57	.57	.62	.62	.64	.61	.67	.64
SD		43	40	39	40	40	40	43	45	42	43	44	42

Adolescent Sample Size, Missingness Percentage, and Internal Consistency of the CES-D Scale by Child Age and Cohort

 $\frac{\text{SD}}{\text{Note. CES-D} = \text{The Center for Epidemiologic Studies Depression Scale. Dashes indicate that CED-D data are not available at that age of cohort. After rescaling the full sample ($ *n*= 2,111) into six cohorts linked by child's age, each cohort's maximum possible sample sizes are: 338 (Cohort 1, Child Age = 10 in 2012), 311 (Cohort 2, Child Age = 11 in 2012), 367 (Cohort 3, Child Age = 12 in 2012), 381 (Cohort 4, Child Age = 13 in 2012), 354 (Cohort 5, Child Age = 14 in 2012), and 360 (Cohort 6, Child Age = 15 in 2012). '-' indicates data were not collected at the time.

Ta	ble	S2

Cal	out		Adolescent Age (years)													
Cor	iori –	10	11	12	13	14	15	16	17	18	19	20	21			
	Ν	292	-	-	-	284	-	257	-	-	-	-	-			
1	%	14%	-	-	-	16%	-	24%	-	-	-	-	-			
	α	0.72	-	-	-	0.79	-	0.78	-	-	-	-	-			
	Ν	-	264	-	-	-	253	-	228	-	-	-	-			
2	%	-	15%	-	-	-	19%	-	27%	-	-	-	-			
	α	-	0.74	-	-	-	0.84	-	0.75	-	-	-	-			
	Ν	-	-	303	-	-	-	309	-	290	-	-	-			
3	%	-	-	17%	-	-	-	16%	-	21%	-	-	-			
	α	-	-	0.74	-	-	-	0.76	-	0.76	-	-	-			
	Ν	-	-	-	326	-	-	-	308	-	272	-	-			
4	%	-	-	-	14%	-	-	-	19%	-	29%	-	-			
	α	-	-	-	0.72	-	-	-	0.74	-	0.76	-	-			
	Ν	-	-	-	-	307	-	-	-	299	-	273	-			
5	%	-	-	-	-	13%	-	-	-	16%	-	23%	-			
	α	-	-	-	-	0.81	-	-	-	0.79	-	0.82	-			
	Ν	-	-	-	-	-	310	-	-	-	293	-	277			
6	%	-	-	-	-	-	14%	-	-	-	19%	-	23%			
	α	-	-	-	-	-	0.72	-	-	-	0.79	-	0.73			
Μ		0.63	0.61	0.64	0.61	0.62	0.62	0.67	0.68	0.69	0.68	0.67	0.68			
SD		0.44	0.44	0.47	0.45	0.49	0.50	0.48	0.47	0.51	0.49	0.53	0.48			

Father Sample Size, Missingness Percentage, and Internal Consistency of the CES-D Scale by Child Age and Cohort

Note. CES-D = The Center for Epidemiologic Studies Depression Scale. Dashes indicate that CED-D data are not available at that age of cohort. After rescaling the full sample (n = 2,111) into six cohorts linked by child's age, each cohort's maximum possible sample sizes are: 338 (Cohort 1, Child Age = 10 in 2012), 311 (Cohort 2, Child Age = 11 in 2012), 367 (Cohort 3, Child Age = 12 in 2012), 381 (Cohort 4, Child Age = 13 in 2012), 354 (Cohort 5, Child Age = 14 in 2012), and 360 (Cohort 6, Child Age = 15 in 2012). '-' indicates data were not collected at the time.

Т	ab	le	S3

Cal	ant					А	dolescent	Age (year	:s)	-			
Con	iori –	10	11	12	13	14	15	16	17	18	19	20	21
	Ν	313	-	-	-	286	-	259	-	-	-	-	-
1	%	7%	-	-	-	15%	-	23%	-	-	-	-	-
	α	0.74	-	-	-	0.79	-	0.8	-	-	-	-	-
	Ν	-	281	-	-	-	259	-	239	-	-	-	-
2	%	-	10%	-	-	-	17%	-	23%	-	-	-	-
	α	-	0.75	-	-	-	0.74	-	0.74	-	-	-	-
	Ν	-	-	340	-	-	-	303	-	291	-	-	-
3	%	-	-	7%	-	-	-	17%	-	21%	-	-	-
	α	-	-	0.77	-	-	-	0.81	-	0.78	-	-	-
	Ν	-	-	-	353	-	-	-	313	-	293	-	-
4	%	-	-	-	7%	-	-	-	18%	-	23%	-	-
	α	-	-	-	0.76	-	-	-	0.79	-	0.77	-	-
	Ν	-	-	-	-	323	-	-	-	289	-	272	-
5	%	-	-	-	-	9%	-	-	-	18%	-	23%	-
	α	-	-	-	-	0.75	-	-	-	0.79	-	0.79	-
	Ν	-	-	-	-	-	348	-	-	-	288	-	275
6	%	-	-	-	-	-	3%	-	-	-	20%	-	24%
	α	-	-	-	-	-	0.8	-	-	-	0.81	-	0.79
Μ		0.74	0.75	0.79	0.74	0.74	0.76	0.81	0.77	0.82	0.78	0.88	0.81
SD		0.46	0.48	0.50	0.49	0.48	0.53	0.54	0.51	0.54	0.53	0.55	0.53

Mother Sample Size, Missingness Percentage, and Internal Consistency of the CES-D Scale by Child Age and Cohort

Note. CES-D = The Center for Epidemiologic Studies Depression Scale. Dashes indicate that CED-D data are not available at that age of cohort. After rescaling the full sample (n = 2,111) into six cohorts linked by child's age, each cohort's maximum possible sample sizes are: 338 (Cohort 1, Child Age = 10 in 2012), 311 (Cohort 2, Child Age = 11 in 2012), 367 (Cohort 3, Child Age = 12 in 2012), 381 (Cohort 4, Child Age = 13 in 2012), 354 (Cohort 5, Child Age = 14 in 2012), and 360 (Cohort 6, Child Age = 15 in 2012). '-' indicates data were not collected at the time.

Table S4

Group		Constant	p	Linear	р	Quadratic	
1	Adolescent	.393	.187	011	.785	<.001	
	Father	.694	.015	055	.142	.002	
	Mother	.985	<.001	073	.045	.002	
2	Adolescent	-1.861	.008	.322	<.001	008	
	Father	066	.908	.088	.250	002	
	Mother	.369	.528	.058	.458	002	
3	Adolescent	765	.466	.155	.023	004	
	Father	4.494	.001	494	.007	.015	
	Mother	.198	.871	.106	.026	001	
4	Adolescent	1.255	.009	088	.154	.003	
	Father	.785	.063	026	.639	.001	
	Mother	1.102	.009	041	.054	.002	
5	Adolescent	.109	.876	.039	.673	001	
	Father	.430	.492	.036	.009	<.001	
	Mother	.903	.119	044	.567	.002	
6	Adolescent	2.466	<.001	230	.006	.008	
	Father	071	.909	.176	.035	005	
	Mother	.219	.725	.151	.071	005	

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<u>p</u> .524

.098 .043

.002 .334 .481 .492 .008 .886 .171 .673 .282 .776 .913 .510 .005 .044 .071

Bivariate Correlations ar	nong Ad	olescents	, Fathers	, and Mo	others CI	ESD-8 SC	cores at E	ach Age				
	Adolescent Age (years)											
	10	11	12	13	14	15	16	17	18	19	20	21
Adolescent & Father	.31**	.27**	.14*	.16**	.22**	.13**	.18**	.04	.18**	.19**	.22**	.02
Adolescent & Mother	.29**	.14*	.26**	.28**	.23**	.17**	.18**	.14**	.16**	.20**	.22**	.03
Father & Mother	.43**	.24**	.23**	.32**	.33**	.35**	.34**	.23**	.29**	.25**	.17**	.24**

Table S5 Bivariate Correlations among Adolescents', Fathers', and Mothers' CESD-8 Scores at Each Ag

Note. * p < .05; ** p < .01.