Early-onset and recurrent depression in parents increases risk of intergenerational transmission to adolescent offspring

Sara R. Jaffee,[1] Judith L. Sligo,[2] Helena M. McAnally,[2] Aroha E. Bolton,[2] Joanne M. Baxter,[3] and Robert J. Hancox[2]

[1]Department of Psychology, University of Pennsylvania, Philadelphia, PA, USA; [2]Department of Preventative and Social Medicine, University of Otago, Dunedin, New Zealand; [3]Kōhatu Centre for Hauora Māori, Health Sciences Division, University of Otago, Dunedin, New Zealand

Background: To assess whether the age-of-onset or the recurrence of parents' major depressive disorder (MDD), measured prospectively in a longitudinal birth cohort study, predicted offspring depression at age 15. **Methods:** A two-generation study of New Zealanders, with prospective, longitudinal data in the parents' generation ($n = 375$) and cross-sectional data from their adolescent offspring ($n = 612$). Parent and offspring depression was measured with structured clinical interviews. Parent depression was measured at six time points from age 11 to 38 years. Adolescent offspring depression was measured at age 15. **Results:** Compared to adolescents whose parents were never depressed, those whose parents met criteria for MDD more than once and those whose parents first met criteria before adulthood had more symptoms of depression. The combination of early-onset and recurrent depression in parents made adolescents particularly vulnerable; their odds of meeting criteria for MDD were 4.21 times greater (95% CI = 1.57–11.26) than adolescents whose parents were never depressed. The strength of the intergenerational effect did not vary as a function of parent or offspring sex. The prevalence of adolescent depression was 2.5 times higher in the offspring than at age 15 in the parents' generation. **Conclusions:** Recurrent depression in both fathers and mothers increases offspring risk for depression, particularly when it starts in childhood or adolescence, but a single lifetime episode does not. Health practitioners should be aware of age-of-onset and course of depression in both parents when assessing their children's risk for depression. **Keywords:** Depression; family history; developmental psychopathology; longitudinal studies.

Introduction

Adolescent-onset depression often follows a chronic course and is associated with psychosocial impairment in adulthood (Lewinsohn, Rohde, Seeley, Klein, & Gotlib, 2003; Weissman et al., 1999). One of the strongest predictors of adolescent-onset depression is family history: The risk of becoming depressed is at least three times greater for adolescents whose parents have had major depressive disorder (MDD) compared to those whose parents have never been depressed (Hammen & Brennan, 2003; Josefsson, Vikström, Bladh, & Sydsjö, 2019; Klein, Lewinsohn, Rohde, Seeley, & Olino, 2005; Lieb, Isensee, Hofler, Pfister, & Wittchen, 2002; Weissman et al., 2016).

Two aspects of family history may be especially informative for predicting adolescent risk. First, the chronicity of depression may be relevant: Adolescents whose mothers have persistent depression are particularly vulnerable to depression (Campbell, Morgan-Lopez, Cox, & McLoyd, 2009) and, when their mother's depression is persistent and severe, they are at risk of suicidal ideation (Hammerton, Zammit, Thapar, & Collishaw, 2016). Thus, it may be important to differentiate parents who have had a

single episode of depression from those who have experienced chronic or recurring depression.

Second, the timing of depression may be important. Intergenerational effects may be particularly strong when a parent's depression emerges in childhood or adolescence because of the risk that it will follow a chronic course (Dunn & Goodyer, 2006; McLeod, Horwood, & Fergusson, 2016; Weissman et al., 1999) and because, as adults, individuals with early-onset depression are at risk for interpersonal problems, health risk behaviors, and antisocial behaviors that increase children's risk for depression (Wilson, Hicks, Foster, McGue, & Iacono, 2015). Moreover, these risks associated with early-onset depression may increase the likelihood that parents will engage in high levels of harsh parenting and low levels of warm, sensitive parenting which also increase offspring risk of depression. Finally, because individuals tend to be less accurate at remembering when they *first* experienced symptoms than whether they *ever* experienced symptoms (Prusoff, Merikangas, & Weissman, 1988; Simon & VonKorff, 1995), it is important to measure age-at-onset with prospective, longitudinal data. It is also important to determine whether early-onset depression is more than a marker of recurrence (Wilson et al., 2015). If early-onset depression increases offspring risk for depression simply because it is

highly recurrent, then we would expect that recurrent depression that first emerges in adulthood would be equally strongly associated with adolescent risk for depression as recurrent depression that first emerges in childhood or adolescence.

Intergenerational transmission effects as a function of parent or child sex

Although depression is more common in women than men, an estimated 21% of fathers experience an episode of depression by the time their children are 12 years old (Davé, Petersen, Sherr, & Nazareth, 2010). Some studies find that adolescent risk for MDD is elevated if either parent has a history of MDD (Jacobs, Talati, Wickramaratne, & Warner, 2015; Lieb et al., 2002; Mikkonen, Moustgaard, Remes, & Martikainen, 2016; Pearson et al., 2013). Others show that paternal depression is less strongly associated with adolescent MDD than maternal depression (Brennan, Hammen, Katz, & Le Brocque, 2002; Klein et al., 2005; Low et al., 2012). Finally, a number of studies, including a meta-analysis of 193 studies, have shown that girls are more vulnerable than boys to parents' depression (Goodman et al., 2011).

The current study

We used a unique, two-generation, prospective, longitudinal study involving children born to members of the Dunedin Study birth cohort (Poulton, Moffitt, & Silva, 2015) to test whether intergenerational transmission of depression varied by when parents first became depressed and how often they met criteria for depression. We also tested whether intergenerational transmission effects varied by parent or offspring sex and both parents' lifetime history of depression. Given that maternal depression is nonspecifically associated with offspring psychopathology (Goodman et al., 2011), we tested whether parental depression was associated with other forms of adolescent disorder. Finally, our study provides a rare opportunity to examine generational differences in depression among New Zealand adolescents by comparing parents and their children at the same age, assessed with the same instrument, at different points in time. Age 15 marks the start of a period in adolescence when rates of depression start to rise (Hankin et al., 1998). The current study provides an opportunity to identify features of parent disorder that are associated with adolescent vulnerability to depression at the beginning of that developmental period.

Methods

The Dunedin Multidisciplinary Health and Development Study is a longitudinal investigation of health and behavior in a population-based birth cohort (Poulton et al., 2015). Study members were born in Dunedin, New Zealand, between April 1972 and March 1973. One thousand and thirty-seven children (91% of eligible births, 52% male) participated in the first follow-up at 3 years, which constituted the base sample for the remainder of the study. Further assessments occurred at ages 5, 7, 9, 11, 13, 15, 18, 21, 26, 32, 38, and 45 years, with retention rates above 90%. The cohort is mostly of New Zealand European ethnicity and represented the full range of socioeconomic status in this South Island city at the time of their birth.

Since 2007, the Next Generation Study has assessed the 15- to 17-year-old biological and nonbiological children of the Dunedin Study members (Poulton et al., 2015; Sligo, Rothstein, Salter, & Hancox, 2009). The Next Generation Study invites adolescents and their primary caregiver (who may or may not be the Dunedin Study member) to a one-day multidisciplinary assessment, which largely replicates the Dunedin Study assessment in which their parent participated when they were 15 years old (Sligo et al., 2009). The nonprimary caregiver participates in the Next Generation Study via a telephone interview. Ethical approval was obtained from the Lower South Regional Ethics Committee (LRS/06/10/048/AM01). Participants provided written informed consent. The Next Generation participants included in the current analysis were seen between 2007 and 2019.

At the time of analysis, 375 of the Dunedin Study members had at least one child who consented to participate in the Next Generation Study (88% of those eligible; see Figure 1). Dunedin Study members ranged in age from 18 to 32 years when their first Next Generation participant child was born (*M* = 25.4, *SD* = 3.7). Among the Next Generation participants, 91% identified their ethnicity as New Zealand European, 16% as Māori, 5% as Pacific ethnicities, and 13% identified as other ethnicities (23% of participants reported more than one ethnicity). Next Generation participants ranged in age from 14.9 to 16.9 years (*M* = 15.8, *SD* = 0.6).

Dunedin Study members' depression

At age 11, 13, and 15 years, Dunedin Study members were administered the Diagnostic Interview Schedule for Children (DISC) (Costello, Edelbrock, Kalas, Kessler, & Klaric, 1982). Research diagnoses of MDD were made according to DSM-III criteria. At age 18, 21, 26, 32, and 38 years, study members were administered the Diagnostic Interview Schedule (DIS) (Robins, Cottler, Bucholz, & Compton, 1995). Research diagnoses of MDD were made according to DSM-III-R criteria at ages 18 and 21 and DSM-IV criteria at ages 26, 32, and 38 years. Both the DISC and DIS demonstrate good interrater reliability ($\kappa > 0.85$) and validity in this cohort (McGee et al., 1990; Newman et al., 1996). For both the DISC and the DIS, the reporting period was twelve months prior to the interview. Interviewers were blind to the study members' psychiatric history. We derived two measures of study members' depression (Table 1). *Number of episodes* comprised the number of study waves in which a Dunedin Study member met criteria for MDD. Because relatively few study members met criteria for MDD at three or more time points, this variable was defined as '0' *never depressed;* '1' *depressed at one time point;* and '2' *depressed at two or more time points. Age-at-onset* was defined as childhood/adolescence (age 11, 13, 15, or 18 years) or adulthood (age 21, 26, 32, or 38 years). Age-at-onset and number of episodes were related; 61% of those who first met criteria for depression in childhood or adolescence had two or more episodes of depression versus 38% of those who first met criteria for depression as adults, χ^2 (1) = 9.98, p = .002.

Next generation mental health

Data on major depressive disorder (MDD), generalized anxiety disorder (GAD), conduct disorder (CD), and attention deficit/
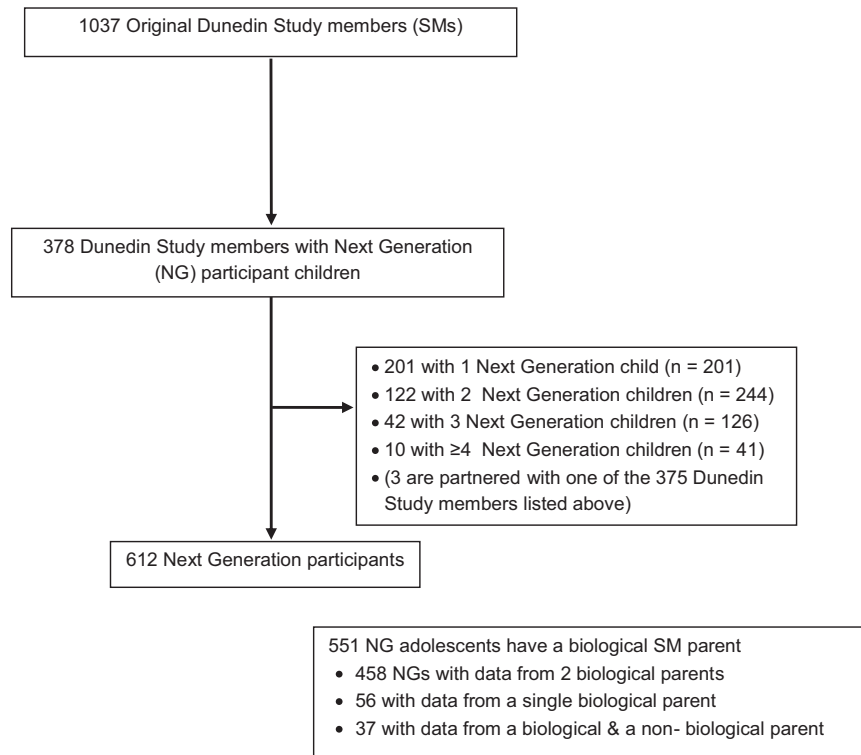
Figure 1 Flow chart representing sample organization

hyperactivity disorder (ADHD) were collected via a computerized version of the DISC-IV (Shaffer, Fisher, Lucas, & Comer, 2007). A trained researcher read each question aloud and recorded the participant's responses digitally. Diagnoses for past year presence of disorder were made according to DSM-IV criteria (Table 1).

Non-Dunedin Study member's depression

Caregivers who were not Dunedin Study members ($n = 394$; 89% of whom were biological parents) were asked selected questions from the DIS (Robins et al., 1995) to establish if they had ever been diagnosed with depression. In nine additional cases, both caregivers were members of the Dunedin Study. The member of the pair who identified as the 'nonprimary caregiver' was treated as the 'nonstudy member' parent, and their DISC and DIS scores were used to establish a lifetime depression diagnosis. See Table 1 for prevalence of lifetime history of depression among the non-Dunedin study members who were the biological parents of the adolescents ($n = 360$).

Comorbidity with study member's depression

Comorbidity with study member's depression was measured with a variable that counted the number of mental disorders (apart from MDD) for which study members met depression between the ages of 11 and 38 years (ranging from 0 to 8). For example, if a study member ever met criteria for GAD, that was counted as one 'other' disorder, regardless of how many times (between age 11 and 38) the study member met criteria for GAD. Among study members with any lifetime history of MDD, 13% did not ever meet criteria for another disorder and the mean number of other disorders was 2.96 ($SD = 2.18$). Among study members with no lifetime history of MDD, 30% did not ever meet criteria for another disorder and the mean number of other disorders was 1.36 ($SD = 1.45$).

Socioeconomic status (SES)

Socioeconomic status (SES) was based on the non-Dunedin Study members' current (or most recent) occupation reported in the telephone or in-person interview and on the Dunedin Study members' current (or most recent) occupation reported at the age 38 assessment of the Dunedin Study. SES was measured with the New Zealand Socioeconomic Index (NZSEI-06) which codes each occupation based on its associated education level and income in the New Zealand census following the NZSEI-06 algorithm (score range, 10 [low socioeconomic status] to 90 [high socioeconomic status]) (Davis, Jenkin, & Cooke, 2003). The higher of the two parents'/caregivers' scores was included as a covariate in the analyses ($M = 53.56$, $SD = 16.12$).

Statistical analysis

To account for the clustered nature of the data (multiple children within families), we estimated random intercept models in Stata 14 (StataCorp, 2015). We first estimated the unadjusted associations between the two measures of the Dunedin Study parents' depression (number of episodes, age-at-onset) and symptoms of depression in their children. The age-at-onset variable was dummy coded to make the never-depressed group the reference category. We then estimated these associations adjusting for parent and offspring sex, parent and offspring age, parent and offspring biological relatedness, and SES. We also tested whether the effect of number of episodes or age-at-onset was moderated by parent sex, offspring sex, or the second parent's lifetime history of depression. In addition, we tested whether the two measures of the Dunedin Study parents' depression were associated with offspring symptoms of conduct, ADHD, or generalized anxiety disorder, additionally controlling for the number of non-MDD disorders for which parents met criteria in their lifetimes. Finally, we tested whether there were generational changes in

Table 1 Prevalence of depression for parents and children

	Total	Females	Males
	% (N)		
Parent (Dunedin study member, $n = 375$[a])			
Number of MDD Episodes[b]			
0	50% (187)	40% (85)	64% (102)
1	25% (96)	29% (63)	21% (33)
2+	25% (92)	31% (67)	16% (25)
First Onset of MDD			
Never	51% (187)	40% (85)	64% (102)
Child/Adolescent (age 11, 13, 15, or 18)	22% (83)	28% (60)	14% (23)
Adult (age 21, 26, 32, or 38)	27% (100)	31% (66)	21% (34)
Parent (Nonstudy Member, $n = 338$)[c]			
Never depressed	59% (201)	40% (69)	79% (132)
Lifetime history depression	41% (137)	60% (103)	21% (34)
Child (Next Generation, $n = 612$)			
MDD past 12 months	9.6% (59)	15% (44)	5% (15)
Depression Symptoms (Median, IQR)	0 (0)	0 (0)	0 (0)

IQR, interquartile range.
[a]Three Dunedin study parents are not included as they were partners of other Dunedin study parents.
[b]Number of episodes ranged from 0 to 6 (Median = 1.00, M = 0.92, SD = 1.16), but was collapsed to three categories because only 10% of Dunedin Study members met criteria for MDD at more than 2 waves of the Study.
[c]Data on lifetime depression were missing for 22 biological parents because they did not complete phone interviews or did not answer questions.

the prevalence of depression. Hypothesis tests were two-sided with significance levels set to $p < .05$.

Results

Is there an association between parents' and adolescent offspring depression?

As shown in Table 2, adolescents whose parents had two or more episodes of depression had significantly more symptoms of depression than adolescents whose parents never met criteria for MDD. Adjusting for all covariates, their odds of meeting criteria for a diagnosis of depression were 2.57 times greater than for those whose parents were never depressed (95% CI = 1.09–6.06). In a fully adjusted model in which all independent and dependent variables were standardized, their symptoms of depression were a third of a standard deviation higher than the offspring of parents who were never depressed. In contrast, those whose parents met criteria for MDD at only one time point were not at elevated risk for depression.

The fully adjusted model in Table 2 also shows that adolescents whose parents were first depressed in childhood or adolescence had significantly more

Table 2 Fixed effects from random intercept model of the association between parent MDD (number of episodes and onset period) and offspring symptoms of depression

	Unadjusted Coefficient (95% CI)	Adjusted[a] Coefficient (95% CI)
Number of Parental MDD Episodes		
0	REF	REF
1	0.29 (−0.14; 0.71)	0.33 (−0.10; 0.76)
2+	**0.84 (0.41; 1.26)**	**0.74 (0.29; 1.18)**
Parental Onset of MDD		
Never	REF	REF
Childhood/Adolescence	**0.68 (0.23; 1.12)**	**0.73 (0.27; 1.18)**
Adulthood	**0.45 (0.04; 0.86)**	0.36 (−0.05; 0.78)
Onset × Number Episodes		
Never		
Childhood/Adol: 1 episode	0.26 (−0.37; 0.90)	0.38 (−0.25; 1.02)
Childhood/Adol: 2+ episode	**0.95 (0.41; 1.49)**	**0.96 (0.41; 1.51)**
Adulthood: 1 episode	0.32 (−0.17; 0.82)	0.34 (−0.15; 0.83)
Adulthood: 2 + episode	**0.63 (0.06; 1.20)**	0.41 (−0.17; 1.00)

Bolded coefficients are statistically significant at $p < .05$.
CI, Confidence Interval.
[a]Adjusted for parent sex, offspring sex, biological relatedness of parent, SES, offspring age in years, and parent age in years. Daughters had significantly more symptoms of depression than sons, and higher SES was associated with fewer symptoms of depression. Analyses accounted for clustering within families.

symptoms of depression than adolescents whose parents were never depressed. They did not, however, have significantly more symptoms of depression than adolescents whose parents first met criteria for depression in adulthood ($b = 0.36$, 95% CI = -0.13 to 0.86, $p = .15$).

To isolate effects of depression onset and persistence, we created four groups including adolescents whose parents: (a) were first diagnosed in childhood/adolescence and had a single episode ($n = 52$); (b) were first diagnosed in childhood/adolescence and had two or more episodes ($n = 79$); (c) were first diagnosed in adulthood and had a single episode ($n = 100$); and (d) were first diagnosed in adulthood and had two or more episodes ($n = 72$). These groups were compared with adolescents whose parents were never depressed ($n = 302$).

In the unadjusted model (Table 2), adolescents whose parents had two or more episodes of depression (regardless of when they were first depressed) had significantly more symptoms of depression than adolescents whose parents were never depressed. In the fully adjusted model, however, only those whose parents first met criteria for depression in childhood or adolescence and had two or more episodes were at elevated risk. The odds of having a diagnosis of depression were 4.21 times greater (95% CI = 1.57–11.26) in this group compared with adolescents whose parents were never depressed. Figure 2 shows mean MDD symptom scores across the five groups.

As a sensitivity check, all models were rerun, retaining only parents and children who were biologically related ($n = 551$; 90% of parent–offspring pairs). Results were unchanged.

### Does the intergenerational transmission effect vary as a function of key covariates?

The intergenerational effect did not generally vary as a function of parent or offspring sex (Table S1).

There was a nonsignificant trend for the offspring sex x age-of-onset interaction (χ^2 (2) = 5.57, $p = .06$), such that female adolescents whose parents were first depressed in childhood or adolescence had more symptoms of depression than girls whose parents were never depressed, but differences in boys' levels of depression as a function of parent age-at-onset were smaller.

We also tested whether having two depressed parents moderated the strength of intergenerational transmission. These analyses were restricted to adolescents who were the biological children of both the Dunedin and the non-Dunedin study members ($n = 468$). Adjusting for all covariates, adolescents had more symptoms of depression if their second parent had a lifetime history of depression, both in the model that included number of episodes ($b = 0.61$, 95% CI = 0.19–1.03) and in the model that included age-of-onset ($b = 0.60$, 95% CI = 0.07–1.07). Having a second depressed parent did not strengthen the effect of the first parent's recurrent or child- or adolescent-onset depression on offspring risk for depression, however (all $p > .40$).

Is the intergenerational transmission effect specific to adolescent depression?

As shown in Table 3, adolescent CD, ADHD, and GAD were associated with some or all features of parents' MDD in unadjusted models. Adjusting for covariates (including the lifetime presence of disorders other than MDD), many of these associations were reduced to nonsignificance. However, adolescents whose parents had a single episode of MDD in childhood or adolescence had significantly higher CD and ADHD scores than adolescents whose parents were never depressed, even in fully adjusted models. When models were restricted to parents and adolescents who were biologically related, results were unchanged.
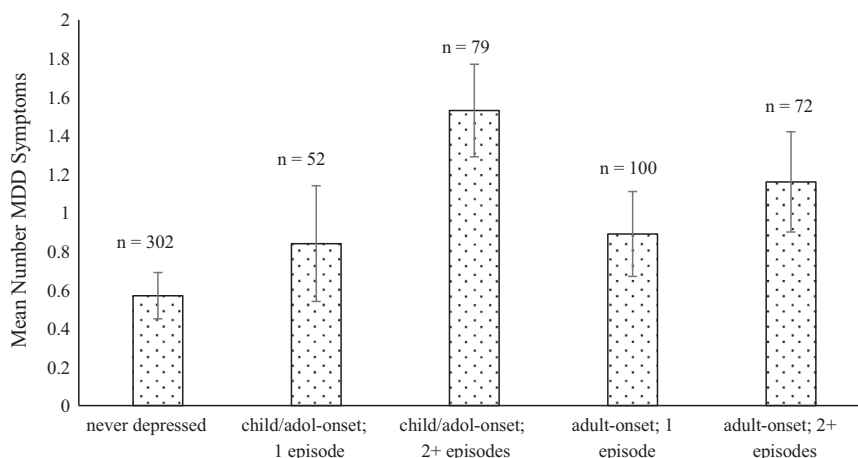
Figure 2 Mean adolescent depression symptom scores as a function of their parents' history of major depressive disorder. Note: Bars are standard errors

Table 3 Fixed effects from random intercept models testing the association between parents' depression and offspring symptoms of conduct disorder, ADHD, and generalized anxiety disorder

	Conduct Disorder		ADHD		Generalized Anxiety Disorder	
	Coefficient (95% CI)		Coefficient (95% CI)		Coefficient (95% CI)	
	Unadj	Adj	Unadj	Adj	Unadj	Adj
Number of MDD Episodes (Parent)						
0	REF	REF	REF	REF	REF	REF
1	**0.29 (0.04 to 0.54)**	0.18 (0.00 to 0.50)	0.32 (−0.36 to 1.00)	0.08 (−0.62 to 0.79)	0.07 (−0.23 to 0.36)	0.07 (−0.23 to 0.36)
2+	0.16 (−0.09 to 0.41)	−0.02 (−0.14 to 0.37)	0.54 (−0.15 to 1.22)	0.08 (−0.72 to 0.88)	0.16 (−0.13 to 0.45)	−0.01 (−0.35 to 0.33)
First Onset of MDD (Parent)						
Never	REF	REF	REF	REF	REF	REF
Childhood/Adolescence	**0.50 (0.24 to 0.76)**	**0.38 (0.08 to 0.68)**	**1.01 (0.29 to 1.73)**	0.79 (−0.05 to 1.64)	−0.05 (−0.36 to 0.26)	−0.11 (−0.47 to 0.25)
Adulthood	0.01 (−0.23 to 0.25)	−0.01 (−0.26 to 0.24)	−0.01 (−0.67 to 0.65)	−0.22 (−0.91 to 0.47)	0.25 (−0.04 to 0.53)	0.13 (−0.16 to 0.42)
Onset × Number Episodes						
Child/Adol: 1 episode	**0.77 (0.40 to 1.15)**	**0.66 (0.28 to 1.04)**	**1.38 (0.35 to 2.41)**	**1.21 (0.14 to 2.28)**	0.07 (−0.37 to 0.51)	0.08 (−0.37 to 0.54)
Child/Adol: 2 + episodes	**0.32 (0.01 to 0.06)**	0.12 (−0.25 to 0.49)	0.77 (−0.10 to 1.64)	0.45 (−0.57 to 1.48)	−0.13 (−0.50 to 0.24)	−0.27 (−0.70 to 0.17)
Adulthood: 1 episode	0.06 (−0.23 to 0.35)	0.01 (−0.27 to 0.30)	−0.18 (−0.98 to 0.61)	−0.35 (−1.15 to 0.45)	0.08 (−0.26 to 0.42)	0.05 (−0.29 to 0.39)
Adulthood: 2 + episodes	−0.06 (−0.39 to 0.27)	−0.10 (−0.45 to 0.25)	0.24 (−0.67 to 1.14)	−0.07 (−1.04 to 0.90)	**0.48 (0.09 to 0.87)**	0.23 (−0.18 to 0.64)

Bolded coefficients are statistically significant at $p < .05$. CI, confidence interval. Adjusted for parent sex, offspring sex, biological relatedness of parent, SES, offspring age in years, parent age in years, and number of non-MDD disorders in the parent's lifetime. Analyses account for clustering within families.

Has Depression Prevalence Changed Across Generations?

Among the Dunedin Study members who participated in the Next Generation Study (as parents), 3.9% met past-year DSM-III criteria for a major depressive episode when they were 15 years old. In contrast, 9.6% of their children met DSM-IV criteria for MDD at the same age. Cross-generational differences in prevalence rates were bigger for girls (3.9% in the parent generation vs. 15% in the offspring generation) than for boys (3.9% in the parent generation vs. 5.0% in the offspring generation).

Discussion

We used a unique, two-generation study with prospective, longitudinal data in the parents' generation and cross-sectional data from their teenage children to estimate the magnitude of the association between mothers' and fathers' lifetime history and course of depression on their children's mental health. Adolescents whose parents first became depressed in childhood or adolescence and adolescents whose parents met criteria for depression two or more times by age 38 had more symptoms of depression than adolescents whose parents were never depressed. These intergenerational transmission effects did not generally vary as a function of the sex of the adolescent or the parent.

Adolescents whose parents' depression was both early emerging and persistent were a particularly vulnerable group, with a fourfold increase in risk for depression themselves. It is noteworthy that early emerging depression was not merely a marker for recurrent depression; recurrent depression was only associated with offspring risk for depression if it started in childhood or adolescence and not in adulthood. One possibility is that depression that emerges in childhood or adolescence disrupts the formation of supportive interpersonal relationships and hinders academic success. The lack of these supports may not only increase the risk of subsequent depression, but may also provide challenges in making a successful transition to adulthood in relationship, work, and family domains (Copeland, Wolke, Shanahan, & Costello, 2015). As a result, the offspring of parents with early emerging and recurrent depression may experience higher levels of family conflict and poverty, potentially causing difficulties for parents with a history of depression in their parenting of the next generation.

Consistent with a literature showing that the intergenerational transmission of risk for psychopathology tends to be nonspecific (Connell & Goodman, 2002; Swales et al., 2020; Thapa, Selya, & Jonk, 2017), recurrent parental depression and early emerging parental depression were associated with adolescent offspring conduct problems and

symptoms of ADHD in unadjusted models. Some of these effects were reduced to nonsignificance in models that adjusted for risk factors that were more characteristic of parents with (vs. without) a lifetime history of MDD, such as an earlier transition to parenthood or a lifetime history of other mental disorders.

Even adjusting for covariates, however, adolescents whose parents had a single episode of depression in childhood or adolescence had significantly more symptoms of CD or ADHD than adolescents whose parents were never depressed. Although these models controlled for the lifetime presence of other (non-MDD) disorder, it is possible that elevated rates of externalizing problems were explained by the persistence of parent externalizing disorders specifically. In future research, we will explore whether a general psychopathology ('p') factor that captures the covariation among disorders over the life course and is highly correlated with recurrence and early age-at-onset (Caspi et al., 2020) is a more efficient way of identifying offspring at greatest risk of psychopathology.

Secular changes in the prevalence of adolescent depression

We found that the prevalence of adolescent depression has more than doubled from the 1980s. This increase is unlikely to be the result of changes in the diagnostic criteria for depression, since the number of symptoms (and the symptoms themselves) required to meet criteria for the disorder effectively stayed the same from DSM-III to DSM-IV. Rates of clinically significant levels of depression (as measured with depression screeners) are as high as 24% among 15-year-olds in New Zealand and have risen since 2012 (Fleming et al., 2020). This rise in the prevalence of depression is also consistent with the rise in depression globally (World Health Organization, 2017) and among adolescents in the UK (Collishaw, 2015). Our findings suggest that this rise in adolescent depression may not only have lifelong consequences for the current generation, but could adversely affect the mental health of their children and subsequent generations.

Implications for practice

Although prevention and treatment of maternal depression are a public health priority in many countries (Bauer, Parsonage, Knapp, Iemmi, & Adelaja, 2014; Curry et al., 2019; World Health Organization, 2015), paternal depression has received relatively little attention. Our findings highlight that paternal depression *and/or* maternal depression that emerges before adulthood and recurs over time puts adolescents at greater risk for depression and conduct problems. Effective treatment of depression

– particularly depression that emerges before adulthood – would have personal positive effects and may plausibly contribute to reductions in the transmission of risk for depression across generations. Finally, our findings suggest that support services would benefit from understanding the lifetime course of a person's depression in terms of assessing offspring risk for externalizing problems as well as depression.

Strengths and limitations

The study overcomes several limitations of previous studies – particularly those involving fathers – that rely on symptom scales or infer depression from prescription records (Mikkonen et al., 2016). The focus on adolescent psychopathology adds to a literature that typically covers early childhood, particularly in relation to fathers' depression (Sweeney & MacBeth, 2016). We used prospectively collected information on mental health disorders using standard DSM diagnostic criteria with very high rates of participation among two generations. However, the Next Generation Study participants are not representative of all children born to the Dunedin Study members because many of their children are not yet old enough to participate. Nevertheless, rates of lifetime depression were identical for Dunedin Study members whose children have participated in the Next Generation Study and those who are not parents of Next Generation participants (50% in each group), and differences in socioeconomic status were small (Cohen's $d = .20$, with parents of Next Generation participants having lower SES). Moreover, Study members whose children participated in the Next Generation Study and Study members who were not parents of Next Generation participants were diagnosed with MDD at equally high rates when they were 15 years old (3.9% in both groups), although gender differences in depression favoring females were more pronounced for the Study members who were not parents of Next Generation participants compared with those who were.

Other limitations concern the measures of depression. The parents who were not part of the Dunedin Study were only administered a brief questionnaire about lifetime depression symptoms. Such retrospective, lifetime questionnaires are not optimal for testing hypotheses about additive and nonadditive effects of parental depression on offspring outcomes. We do not know whether adolescents who reported clinically significant levels of depression at age 15 were also depressed at an earlier point in their lives and we did not measure adolescent depression throughout the full range of risk (e.g., through age 17). Finally, we did not have information about the severity of the parents'

depression or its timing in relation to the adolescent's mental health assessment.

Conclusions

Our research contributes new information about the intergenerational effects of parents' mental health on the mental health of their children. When working with family history data, health practitioners should recognize the importance of clarifying details around whether a parent's history of disorder was persistent or recurrent and when in the life course symptoms emerged. Effective support and treatment of those with depression would reduce the burden of suffering experienced by the individuals and also support the well-being of future generations, a finding that highlights the importance of accessible primary care and specialized mental health services. Although there is a substantial evidence base around the treatment of mothers' depression and interventions to change parenting among depressed mothers, more research is needed to understand how depressed fathers transmit risk for depression to their children.

Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article:

Table S1. Fixed effects from random intercept models testing whether parent or offspring sex moderate the intergenerational transmission effect of parental depression on offspring symptoms of depression.

Correspondence

Sara R. Jaffee, Department of Psychology, University of Pennsylvania, 425 S. University Ave., Philadelphia, PA 19104, USA; Email: srjaffee@psych.upenn.edu

Key Points

- A parent's history of depression increases an adolescent's risk of depression by approximately three-fold, but some adolescents may be at greater risk than others depending on the chronicity and timing of their parent's depression. Prospective, longitudinal data on parents' mental health would determine this.
- Adolescents whose parents had early-onset, recurrent depression had a four-fold risk of major depressive disorder compared with adolescents whose parents were never depressed. Adolescents whose parents had a single lifetime depression diagnosis were not at elevated risk.
- There is meaningful heterogeneity in parents' lifetime histories of depression, suggesting that youth who would benefit most from preventive interventions are those whose parents' depression has an early onset and recurrent course.

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