The association between paternal and adolescent depressive symptoms: evidence from two population-based cohorts

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Summary

Background Incidence of depression increases markedly around age 13 years, and nearly three-quarters of adults report that their mental health problems started in adolescence. Although maternal depression is a risk factor for adolescent depression, evidence about the association between paternal and adolescent depression is inconclusive, and many studies have methodological limitations. We aimed to assess the association between paternal and adolescent depressive symptoms in two large population-based cohort studies.

Methods We used data for two-parent families from two representative prospective cohorts in Ireland (Growing up in Ireland [GUI]) and the UK (Millennium Cohort Study [MCS]). Parental depressive symptoms were measured with the Centre for Epidemiological Studies Depression Scale in the GUI cohort when children were 9 years old, and the Kessler six-item psychological distress scale in the MCS cohort when children were 7 years old. Adolescent depressive symptoms were measured with the Short Mood and Feelings Questionnaire (SMFQ) at age 13 years in the GUI cohort and age 14 years in the MCS cohort. We analysed data using linear regression models, before and after adjustment for confounders, in both multiply imputed and complete case samples.

Findings There were 6070 families in GUI and 7768 in MCS. After all adjustments, a 1 SD (three-point) increase in paternal depressive symptoms was associated with an increase of 0·24 SMFQ points (95% CI 0·03–0·45; p=0·023) in the GUI cohort and 0·18 SMFQ points (0·01–0·36; p=0·041) in the MCS cohort. This association was independent of, and not different in magnitude to, the association between maternal and adolescent depressive symptoms (Wald test p=0·435 in the GUI cohort and 0·470 in the MCS cohort).

Interpretation Our results show an association between depressive symptoms in fathers and depressive symptoms in their adolescent offspring. These findings support the involvement of fathers as well as mothers in early interventions to reduce the prevalence of adolescent depression, and highlight the importance of treating depression in both parents.

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Introduction

Incidence of depression increases markedly around age 13 years, and almost 75% of adults with depression report mental health problems that started in adolescence.[1,2] Early adolescence is therefore important for the primary prevention of adult depression—a leading public health problem worldwide. One of the most important risk factors for adolescent depression is maternal depression;[3] however, there are fewer studies of the association between paternal and adolescent depression. This association is important given that paternal depression is fairly common,[4] and that fathers are increasingly involved in the care of their children in many countries.[5]

Few studies of paternal and adolescent depression take account of maternal depression, an important possible confounder.[6] Studies that do account for maternal depression have various limitations. Many are small, containing few fathers, and their findings are inconsistent.[7–12] Some larger studies exist, but they either assess depression retrospectively[13] or use routine clinical data,[14] so are susceptible to recall and ascertainment bias. The only large prospective study that adjusted for maternal depression and did not rely on clinical data found no association between antenatal depression in fathers and adolescent depression at age 18 years.[15] However, antenatal maternal depression might act via the intrauterine environment,[3] in which case one would not expect an association between paternal antenatal depression and adolescent depression. Fathers might also take on a larger role in childcare, and become more influential, later in childhood.[16]

In this study, we used two large prospective population-based cohorts to investigate the association between paternal depression in childhood and offspring depression in adolescence, and whether it was independent of maternal depression.

Methods

Study design and participants

Growing up in Ireland (GUI)[17] is an ongoing representative study of two cohorts of children living in Ireland in 2006: a child cohort recruited at 9 years old, and an infant cohort recruited at 9 months old. The present study is a secondary analysis of the child cohort

Research in context

Evidence before this study

Studies of the influence of paternal depression on offspring have focused on outcomes in early childhood. There are fewer studies of the link between paternal and adolescent depression—a major public health problem. We searched PubMed for studies published in English before May 31, 2017, that investigated paternal and adolescent depression. We used the search terms ("paternal" or "father") and ("depress*"). We then manually searched reference lists from these studies. Evidence of the association between paternal and adolescent depression was inconsistent and few studies had taken account of maternal depression, an important possible confounder. Studies that had taken account of maternal depression had various limitations, including small samples that contained few fathers. Some large studies had accounted for maternal depression, but they either reported no evidence of an association, assessed depression retrospectively, or used clinical data, and were therefore susceptible to recall or ascertainment bias.

Added value of this study

We found evidence of an association between paternal and adolescent depressive symptoms and the effect size was independent of, and not different in magnitude to, maternal depression. To our knowledge, this is the first time this association has been reported in prospective population-based cohorts that do not rely on clinical data.

Implications of all the available evidence

Current interventions for preventing adolescent depression focus largely on mothers. Our results suggest that when the mother is depressed, clinicians should also consider the associated yet independent influence of depression in the father, particularly since men are less likely to seek treatment for depression. Our results are inconsistent with the idea that mothers are responsible, or even to blame, for children's mental health, whereas paternal influences are negligible. They suggest that the mental health of both fathers and mothers is important for the mental health of their children. Interventions to improve adolescent mental health should therefore target both parents, irrespective of their sex.

(n=8568). 1105 primary schools were invited to participate, and 910 (82%) agreed, from which children were recruited. 17 054 families (households) were then randomly selected and invited to participate, 9645 (57%) consented, and 8568 (50%) provided usable data. Two waves of data collection have taken place in the child cohort. Wave one occurred between August, 2007, and May, 2008 (n=8568), when children were a mean age of 9 years (SD 0·13), and wave two occurred between August, 2011, and March, 2012 (n=7525), when children were a mean age of 13 years (SD 0·13; 88% response rate).

One adult was asked to identify as the primary caregiver, providing most day-to-day care. Resident partners were automatically nominated as secondary caregivers. Caregivers did not have to be biologically related to the child and did not include non-resident fathers (attempts were made to contact non-resident secondary caregivers, but response rates were low). Of primary caregivers (n=8568), 8465 (99%) were mothers (female primary caregivers), 8358 (99%) were biological parents, and 107 (1%) were another parent or guardian. Of families with a primary caregiver, 7118 (83%) were two-parent families with data from a secondary caregiver. Of the 7118 secondary caregivers, 7072 (99%) were fathers (male secondary caregivers). Of these fathers, 6775 (96%) were a biological parent, 216 (3%) a step-parent, and 81 (1%) another parent or guardian.

The Millennium Cohort Study (MCS)[18] is an ongoing representative study of 18 552 families and 18 818 children born in England and Wales between Sept 1, 2000, and Aug 31, 2001, and in Scotland and Northern Ireland between Nov 24, 2000, and Jan 11, 2002. Children were identified through government child benefit records and recruited when they were 9–11 months old. Socially deprived areas and ethnic minority groups were oversampled to increase representation. Six waves of data collection have taken place: when children were aged about 9 months (MCS1), 3 years (MCS2), 5 years (MCS3), 7 years (MCS4), 11 years (MCS5), and 14 years (MCS6). We used data from MCS4 and MCS6 to provide the closest possible replication of the timepoints used in the GUI cohort. At MCS4, 13 857 (72%) of 19 244 families participated and at MCS6, 11 726 (76%) of 15 415 families participated. At MCS4, data were provided by 13410 mothers (female main respondents identifying themselves as the parent). Of these mothers, 13 392 (99%) were biological parents. Interviews were done with 9429 partners, 9036 of whom were fathers (male partners identifying as parents). Of these fathers, 8477 (94%) were biological parents. Because of the household survey structure of MCS, there were 246 sets of twins and ten sets of triplets. We included only singleton children (n=13 681 for MCS, n=8568 for GUI).

In each sample we included two-parent families only because of our focus on the independence of paternal and maternal depressive symptoms. All fathers lived with the child. Because of evidence that the association between parent and offspring depression does not differ according to genetic relatedness,[19] we included biological and non-biological mothers and fathers, but adjusted for whether fathers were biologically related. We did not

adjust for whether mothers were biologically related because of the very small numbers who were not. Participants were not involved in the design of either cohort study.

All families provided written informed consent. GUI data were obtained from the Irish Social Sciences Data Archive, upon receipt of a signed application. Original data collection methods for GUI were approved by the Health Research Board's Standing Research Ethics Committee. MCS data were obtained from the UK Data Archive. Ethics approval for MCS was obtained from a National Health Service Research Ethics Committee.

Procedures

In both samples, adolescents completed the Short Mood and Feelings Questionnaire (SMFQ) at follow-up timepoints only (wave two for GUI and MCS6 for MCS). The SMFQ is a 13-item self-report measure of DSM-IV depressive symptom severity in the past 2 weeks.[20] Possible scores range from 0 to 26, with higher scores indicating more severe depressive symptoms. Reliability was high (Cronbach's α was 0·87 for the GUI cohort and 0·97 for MCS6). We analysed a continuous score to maximise statistical power.

In GUI, parents completed the short eight-item version of the Centre for Epidemiological Studies Depression Scale (CES-D) at waves one and two.[21] The CES-D assesses DSM-IV depressive symptoms in the past week using a four-point Likert scale, which ranges from "none or almost none of the time" to "all or almost all of the time". Possible scores range from 0 to 24, with higher scores indicating more severe depressive symptoms. At wave one, reliability was high for mothers (Cronbach's α 0·87) and fathers (0·80). We analysed a continuous score to maximise statistical power.

In MCS, parents completed the Kessler six-item psychological distress scale (K6) at waves two to six.[22] The K6 assesses general distress in the past month, using items such as "How often did you feel so depressed that nothing could cheer you up?" and "How often did you feel hopeless?". Participants respond using a five-point Likert scale, ranging from "none" to "all of the time". Possible scores range from 0 to 24, with higher scores indicating more severe symptoms. The scale has good psychometric properties and an estimated area under the curve of 0·83 (range 0·76–0·89, IQR 0·81–0·85) against a standard diagnostic assessment of depression (the World Mental Health Composite International Diagnostic Interview module for major depression).[22] At MCS4, K6 had high reliability for mothers (Cronbach's α 0·97) and fathers (0·99). We analysed continuous scores to maximise statistical power.

We identified variables previously associated with exposure and outcome that could be common causes of depression: family income, parent's education, parent's and child's ages, ethnicity, parental substance abuse, child's emotional symptoms, and interparental conflict. Measures were similar in each sample. Family income was the total income adjusted for household size and composition, in quintiles. Parental education ranged from one (none or compulsory education) to six (postgraduate), classified into compulsory and non-compulsory. We adjusted for the child's ethnicity in MCS and father's ethnicity in GUI (either white or ethnic minority, because of small numbers). Childrens' ethnicities were unavailable in GUI. We also adjusted for the sex of the child and whether the father was the biological parent. In both cohorts, we adjusted for paternal and maternal frequency of alcohol use, ranging from one (never) to seven (every day), classified into tertiles (at MCS4 and baseline in GUI). We adjusted for parent's reports on the emotional symptoms scale of the Strengths and Difficulties Questionnaire, a widely used measure of internalising and externalising symptoms (at MCS4 and at baseline in GUI).[23] In GUI, we adjusted for baseline paternal and maternal reports on the short seven-item version of the Dyadic Adjustment Scale, a widely used measure of relationship conflict and satisfaction.[24] In MCS, we adjusted for paternal and maternal reports on a short four-item version of the Golombok Rust Inventory of Marital State, which were available at MCS3.[25]

Statistical analysis

All analyses were done with Stata (version 14), and were weighted to account for sampling design. Our primary analysis was of the multiply imputed sample with complete data for paternal and maternal depressive symptoms.

First, we tested univariable associations between paternal and adolescent depressive symptoms using linear regressions. We also tested the correlation between paternal and maternal depressive symptoms. Second, we adjusted for maternal depressive symptoms in a bivariable model to test independent associations. Finally, we adjusted for potential confounders in a set of multivariable models: we adjusted the bivariable model for family income; paternal and maternal education; paternal, maternal, and child age at time of exposure; sex of the child; ethnicity; whether the father was a biological parent; and paternal and maternal alcohol use at time of exposure. We then adjusted this model for child emotional symptoms at the time of the exposure. We assumed that parental depressive symptoms were a potential cause of interparental conflict, which could be on the causal pathway. However, interparental conflict could also be a cause of both parental depressive symptoms and later adolescent depressive symptoms. We therefore present our multivariable model before and after further adjustment for interparental conflict.

We tested whether the magnitude of the association between paternal and adolescent depressive symptoms differed from maternal symptoms using the test command in Stata, which performs a Wald test to

	GUI cohort (N=6070)		MCS cohort (N=7768)	
	No (n=5900)	Yes (n=170)	No (n=7572)	Yes (n=196)
Lowest family income quintile	456 (12%)	18 (14%)	591 (8%)	62 (32%)
Compulsory education only				
Maternal	2681 (63%)	79 (68%)	4105 (52%)	144 (71%)
Paternal	3089 (60%)	101 (68%)	3824 (55%)	129 (75%)
Sex of the child				
Female	2836 (46%)	73 (43%)	3844 (51%)	101 (51%)
Male	2855 (54%)	90 (57%)	3728 (49%)	95 (48%)
White ethnicity*	5359 (91%)	155 (94%)	6938 (94%)	170 (91%)
Biological father	5710 (97%)	164 (97%)	7111 (94%)	181 (91%)
Frequency of alcohol use (>5 days per week)				
Maternal	139 (2%)	4 (2%)	607 (10%)	12 (7%)
Paternal	250 (4%)	6 (11%)	1016 (16%)	21 (14%)
Depressive symptoms (CES-D or K6 score)†				
Mother's depressive symptoms (score)	1·9 (4·4)	3·3 (5·8)	2·6 (4·2)	4·8 (5·7)
Father's depressive symptoms (score)	1·1 (2·1)	11·1 (5·3)	2·6 (3·2)	15·3 (3·1)
Age of parent (years)				
Mother	39·8 (7·9)	39·3 (5·9)	37·5 (10·3)	35·8 (6·9)
Father	41·7 (7·6)	41·6 (7·3)	39·9 (10·9)	39·0 (7·9)
Child's emotional symptoms (Strengths and Difficulties Questionnaire score)	2·0 (2·8)	2·8 (3·2)	1·3 (2·2)	1·7 (2·2)

Data are n (%) or mean (SD). Ns are unweighted, and means and percentages are weighted to be representative of the samples overall. Data are for the sample with complete exposure data (maternal and paternal depressive symptoms) only. Other variables therefore have missing data. For paternal depression, the cutoff score for yes was 8 on the CES-D and 13 on the K6. GUI=Growing up in Ireland. MCS=Millennium Cohort Study. CES-D=Centre for Epidemiological Studies Depression Scale. K6=Kessler six-item psychological distress scale. *Children's ethnicity was unavailable in GUI. †CES-D was used for the GUI cohort and K6 for the MCS cohort.

Table 1: **Characteristics of samples with complete exposure data at baseline, according to presence of paternal depression**

compare coefficients. We also tested an interaction between paternal depressive symptoms and sex of offspring to investigate whether associations differed between female and male adolescents. Finally, we tested an interaction between paternal depressive symptoms and whether the father was biologically related to the child, to investigate whether associations differed according to biological relatedness. Given that we used additive rather than multiplicative models, and because these interaction tests could be underpowered, all interactions were exploratory.[26]

We used multiple imputation by chained equations to account for missing data because complete-case analyses can introduce bias when data are not missing completely at random.[27] We assumed that missingness was dependent on observed data (missing at random) and imputed 50 datasets. To predict missing data, we used all variables selected for analysis models and several auxiliary variables. We imputed up to the sample with complete data for paternal and maternal depressive symptoms. As sensitivity analyses, we also report results based on the complete-case samples.

Role of the funding source

The funder of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report. The corresponding author had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Results

In the GUI cohort, data for depressive symptoms were available for 7789 mothers and 6400 fathers, and data for both were available for 6070 participants (table 1). In this sample, the mean CES-D score for fathers was 1·4 (SD 3·1) and for mothers was 1·9 (4·4). Paternal and maternal CES-D scores were correlated (r=0·15, 95% CI 0·11–0·19). Data for adolescent depressive symptoms were available for 5424 of these families. The mean offspring SMFQ score was 3·7 (SD 5·3). When all confounders were included, data for a complete-case sample were available for 4397 families.

In the MCS cohort, data for depressive symptoms were available for 12 270 mothers and 8169 fathers, and data for both were available for 7768 participants (table 1). In this sample, the mean K6 score for fathers was 2·9 (SD 3·8) and for mothers was 2·6 (4·3). Paternal and maternal K6 scores were correlated (r=0·18, 95% CI 0·15–0·21). Data for adolescent depressive symptoms were available for 6087 of these families. The mean offspring SMFQ score was 5·4 (SD 8·3). When all confounders were included, data for a complete case sample were available for 4864 families.

In GUI, 4397 participants were in the complete case sample and 4171 were not (N=8568). In MCS, 4864 singleton children were in the complete case sample and 8817 were not (N=13681).

Families not included in complete-case samples had higher parental depressive symptom scores. In GUI, the mean difference for mothers was 0·71 (95% CI 0·57–0·86; p<0·0001) and for fathers it was 0·12 (0·00–0·24; p=0·049). In MCS, the mean difference for mothers was 1·10 (0·97–1·24; p<0·0001) and for fathers was 0·34 (0·19–0·49; p<0·0001). Adolescent depressive symptom scores were higher in participants with missing data in GUI (mean difference 0·16, 95% CI 0·04–0·36; p=0·11) and in MCS (0·37, 0·14–0·60; p=0·0016). Families with missing data were also more likely to be in lower family income categories. In GUI, 747 (unweighted 21%) families with missing data were in the lowest income category versus 308 (unweighted 7%) with complete data. Of the participants with missing data in MCS, 2537 (unweighted 29%) were in the lowest income category compared with 283 (unweighted 6%) with complete data. Families with missing data were also more likely to have compulsory education only. Of the participants with missing data in GUI, 2308 (unweighted 55%) were in lower maternal education categories versus 1900 (unweighted 43%) of those with complete data. A similar pattern emerged for level of

	GUI cohort (n=6070)		MCS cohort (n=7768)	
	Paternal depression	Maternal depression	Paternal depression	Maternal depression
Univariable	0·35 (0·14 to 0·56; p=0·0012)	0·26 (0·08 to 0·44; p=0·0044)	0·28 (0·11 to 0·46; p=0·0019)	0·48 (0·34 to 0·63; p<0·0001)
Bivariable*	0·30 (0·09 to 0·50; p=0·0047)	0·23 (0·05 to 0·41; p=0·012)	0·20 (0·02 to 0·38; p=0·028)	0·45 (0·30 to 0·59; p<0·0001)
Adjusted for confounders†	0·30 (0·10 to 0·51; p=0·0036)	0·22 (0·05 to 0·40; p=0·014)	0·23 (0·05 to 0·40; p=0·010)	0·42 (0·27 to 0·56; p<0·0001)
Adjusted for emotional symptoms‡	0·26 (0·05 to 0·46; p=0·014)	0·15 (–0·03 to 0·32; p=0·103)	0·22 (0·05 to 0·39; p=0·012)	0·34 (0·20 to 0·49; p<0·0001)
Adjusted for parental conflict§	0·24 (0·03 to 0·45; p=0·023)	0·14 (–0·04 to 0·33; p=0·130)	0·18 (0·01 to 0·36; p=0·041)	0·32 (0·17 to 0·47; p<0·0001)

Data are change in adolescent Short Mood and Feelings questionnaire points for a three-point increase in parent depressive symptoms (95% CI; p value). A three-point increase is about 1 SD. GUI=Growing up in Ireland. MCS=Millennium Cohort Study. *In the bivariable model, we adjusted for maternal and paternal depressive symptoms to test independent associations. †Bivariable model (*) adjusted for family income; paternal and maternal education; paternal, maternal, and child's age at time of exposure; sex of child; ethnicity; whether father is biological parent; and paternal and maternal alcohol use at time of exposure. ‡Adjusted bivariable model (†) further adjusted for child emotional symptoms at time of exposure. §Adjusted bivariable model (‡) further adjusted for paternal and maternal reports of interparental relationship conflict.

Table 2: **Associations between paternal and adolescent depressive symptoms in multiply imputed samples**

paternal education (1712 [unweighted 63%] participants with missing data were in lower education categories versus 2225 [51%] with complete data). For maternal education in the MCS cohort, 6479 (unweighted 74%) participants with missing data were in lower education categories versus 2330 (unweighted 48%) of those with complete data. For paternal education, 4060 (unweighted 72%) with missing data were in lower education categories versus 2605 (unweighted 54%) with complete data.

In univariable models, we found evidence of a positive association between paternal and adolescent depressive symptoms (table 2). In GUI, for each three-point (1 SD) increase in paternal depressive symptoms, adolescent depressive symptoms increased by 0·35 of an SMFQ point, and in MCS by 0·28 of a point (table 2). Associations between paternal and adolescent depressive symptoms remained after adjustment for maternal depressive symptoms and all other confounders (table 2). Results from the complete-case sample were similar (table 3).

We found no evidence that regression coefficients for maternal and paternal depressive symptoms differed (Wald test p=0·435 for the GUI cohort and 0·470 for the MCS cohort). In the GUI cohort, the association between paternal and adolescent depressive symptoms was stronger for female than for male adolescents ($p_{interaction}$=0·016). In the MCS cohort, we found no evidence that associations differed according to the sex of the child ($p_{interaction}$=0·769). There was no evidence of an interaction between paternal depressive symptoms and whether the father was biologically related ($p_{interaction}$=0·720 for the GUI cohort; $p_{interaction}$=0·660 for the MCS cohort).

Discussion

In two large, contemporary, population-based cohorts from the UK and Ireland, we found evidence that paternal depressive symptoms during childhood were associated with offspring depressive symptoms in adolescence. This association was independent of, and not different in magnitude to, the association between maternal and adolescent depressive symptoms.

Our study has several strengths. Our cohorts were representative and measures and age ranges were similar, allowing for replication in independent settings. These population-based samples also provided information about the many parents with depression who do not present to clinical services. Missing data are a potential weakness of all cohort studies and attrition was high in both samples. However, we used multiple imputation to address any bias that might have arisen through attrition.

Our study also has several limitations. We used brief self-administered assessments of depressive symptoms instead of clinical interviews. This approach might have produced less precise estimates, but these errors are probably random with respect to our hypothesis, and such random measurement error could not have led to our results. There are also advantages of symptom measures. Depressive symptoms exist as a continuum in the general population and symptom measures capture this variation in severity, as well as increasing statistical power. Furthermore, our measures show high sensitivity and specificity with diagnoses of depression in similar community samples. The K6 scale, used to assess parent depressive symptoms in the MCS cohort, was designed as a general measure of psychopathology. However, this measure has also shown excellent specificity and sensitivity for depressive diagnoses. Therefore, our findings should be relevant to depression diagnoses.

We adjusted for children's emotional symptoms rather than for depressive symptoms. The emotional symptoms subscale of the Strengths and Difficulties Questionnaire was reported by parents rather than offspring, and describes a concept broader than depression, although it does include items such as "I am often unhappy, down-hearted or tearful". Depression is relatively uncommon before puberty, so adjustment for a wider range of emotional symptoms at this age could be a better way to account for differences in future depression risk, though residual confounding by childhood depression is still possible.[28] Our study did not include single-parent families because we investigated the potential independence of paternal and maternal depression. Research into the role of fathers in

	GUI cohort (n=4397)		MCS cohort (n=4864)	
	Paternal depression	Maternal depression	Paternal depression	Maternal depression
Univariable	0·27 (0·05 to 0·49; p=0·015)	0·20 (0·03 to 0·37; p=0·019)	0·34 (0·14 to 0·53; p=0·0008)	0·52 (0·34 to 0·69; p<0·0001)
Bivariable*	0·24 (0·02 to 0·46; p=0·030)	0·18 (0·01 to 0·34; p=0·035)	0·26 (0·06 to 0·46; p=0·010)	0·48 (0·30 to 0·65; p<0·0001)
Adjusted for confounders†	0·25 (0·03 to 0·47; p=0·023)	0·17 (0·01 to 0·34; p=0·037)	0·28 (0·08 to 0·48; p=0·0054)	0·45 (0·28 to 0·63; p<0·0001)
Adjusted for emotional symptoms‡	0·21 (–0·00 to 0·43; p=0·055)	0·09 (–0·07 to 0·25; p=0·25)	0·29 (0·09 to 0·48; p=0·0046)	0·37 (0·18 to 0·55; p<0·0001)
Adjusted for parental conflict§	0·21 (–0·01 to 0·43; p=0·060)	0·10 (–0·07 to 0·26; p=0·25)	0·25 (0·04 to 0·45; p=0·017)	0·36 (0·17 to 0·54; p<0·0001)

Data are change in adolescent Short Mood and Feelings questionnaire points for a three-point increase in parent depressive symptoms (95% CI; p value). A three-point increase is about 1 SD. GUI=Growing up in Ireland. MCS=Millennium Cohort Study. *In the bivariable model, we adjusted for maternal and paternal depressive symptoms to test independent associations. †Adjusted bivariable model (*) further adjusted for family income; paternal and maternal education; paternal, maternal, and child's age at time of exposure; sex of child; ethnicity; whether father is biological parent; and paternal and maternal alcohol use at time of exposure. ‡Adjusted bivariable model (†) adjusted for child emotional symptoms at time of exposure. §Adjusted bivariable model (‡) further adjusted for paternal and maternal reports of interparental relationship conflict.

Table 3: **Associations between paternal and adolescent depressive symptoms in complete case samples**

single-parent families is scarce. Of course, residual confounding is always a possibility in observational studies—eg, we did not have detailed information about comorbid health problems in parents. Further research that acknowledges the complexity of these associations in both parents, and their implications for offspring, would be beneficial.

Finally, for both maternal and paternal depressive symptoms, it is difficult to judge the potential clinical importance of the observed associations. An increase of 1 SD in paternal depressive symptoms was associated with an increase in adolescent depressive symptoms of 0·04 of an SD in GUI and 0·03 in MCS. Though small, these findings were observed after follow-up of 4 years for GUI and 7 years for MCS, and several factors could have led to an underestimation of the association, including error in the paternal depression measure and outcome. We also used brief measures of depressive symptoms before the main period of depression incidence in offspring. There is good evidence that treatment of maternal depression in clinical populations leads to meaningful improvements in offspring outcomes.[29] Our evidence suggests that similar improvements in offspring outcomes would be expected if paternal depression were treated, although future research to test this possibility is required.

Several studies of adolescent depression report no influence of paternal depression, or that the influence of maternal depression is stronger.[12,30–32] However, many of these studies were small, contained few fathers, or did not examine adolescent depression as an outcome. In studies from previous decades, fathers possibly had less involvement with children than in our more contemporary samples. In the MCS cohort, the magnitude of the maternal depression association appeared stronger than that of paternal depression, but there was no statistical evidence to support a difference. In the GUI cohort, there was no evidence of any difference. Our results also suggest that a child with two parents with depression is at greater risk than a a child with one parent with depression.

There is evidence that the intergenerational transmission of depression occurs predominantly through environmental mechanisms, although genetic influences are also important.[19] Environmental mechanisms could include social modelling of depressive thinking styles.[33] There is also good evidence that mothers and fathers with depression experience difficulties in parenting and parent–child relationships, which partly account for the influence of depression on their children.[34] Most of the work on mechanisms has been done with mothers, and less is known about possible mechanisms in relation to fathers.[35]

Our exposure variables were measured before puberty, when the prevalence of depression is low, and our outcomes in early adolescence, when incidence is only just beginning to rise. Adolescent depressive symptom scores were higher in MCS than in GUI, possibly because adolescents were, on average, 14 years old in MCS and 13 years old in GUI. This is an important difference in age for the adolescent increase in depressive symptoms, which only begins at around the age of 13 years.[1] Our findings, if they reflect a causal relationship, are therefore important for the primary prevention of depressive disorder.

Current interventions for preventing adolescent depression focus largely on mothers. Depressive symptoms in parents are associated, and depression in one parent is a risk factor for depression in the other.[36] When the mother is depressed, clinicians should therefore also consider the associated yet independent influence of depression in the father, especially since men are less likely to seek treatment for depression.[37] This is particularly important given that children are at even higher risk when both parents have depressive

symptoms. Our findings, if they reflect a causal relationship, suggest that the priority should be treatment of depression in both parents. Our results are inconsistent with the idea that mothers are responsible, or even to blame for children's mental health, whereas paternal influences are negligible. Rather, they suggest that the mental health of both parents is important for the mental health of their children. Interventions to improve adolescent mental health should therefore target both parents, irrespective of their sex.

Contributors

GeL conceptualised the manuscript, with input from EP, MN, and GlL. All authors assisted with the design of the study and the development of the analysis plan. GeL analysed the data and drafted the manuscript. EP and MN provided the GUI data and advised on the GUI analyses. EF provided advice on acquisition and analysis of the MCS data. GlL provided senior supervision. All authors read, drafted, and revised the whole report. All authors act as guarantors for the manuscript.

Declaration of interests

We declare no competing interests.

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