

Socio-economic status, family disruption and residential stability in childhood: relation to onset, recurrence and remission of major depression

S. E. GILMAN,¹ I. KAWACHI, G. M. FITZMAURICE AND S. L. BUKA

From the Departments of Maternal and Child Health, Health and Social Behavior, Biostatistics and Epidemiology, Harvard School of Public Health, Boston, MA; and the Centers for Behavioral and Preventive Medicine, Brown Medical School and The Miriam Hospital, Providence, RI, USA

ABSTRACT

Background. Childhood adversity significantly increases the risk of depression, but it is unclear whether this risk is most pronounced for depression occurring early in life. In the present study, we examine whether three aspects of childhood adversity – low socio-economic status (SES), family disruption, and residential instability – are related to increased risk of depression during specific stages of the life course. We also examine whether these aspects of childhood adversity are related to the severity of depression.

Method. A sample of 1089 of the 4140 births enrolled in the Providence, Rhode Island cohort of the National Collaborative Perinatal Project was interviewed between the ages of 18 and 39. Measures of parental SES, childhood family disruption and residential instability were obtained upon mother's enrolment and at age 7. Age at onset of major depressive episode, lifetime number of depressive episodes, and age at last episode were ascertained via structured diagnostic interviews. Survival analysis was used to identify risk factors for depression onset and remission and Poisson regression was used to model the recurrence rate of depressive episodes.

Results. Low parental SES, family disruption and a high level of residential instability, defined as three or more family moves, were related to elevated lifetime risks of depression; the effects of family disruption and residential instability were most pronounced on depression onset by age 14. Childhood adversity was also related to increased risk of recurrence and reduced likelihood of remission.

Conclusions. Childhood social disadvantage significantly influences risk of depression onset both in childhood and in adulthood. Early childhood adversity is also related to poor prognosis.

INTRODUCTION

Childhood adversity and depression onset

While long-term follow-up studies of children provide convincing evidence that early life experiences have substantial influence on the occurrence of depression (Power, 1991; Lundberg, 1993; Rodgers, 1994; Power *et al.* 1997; Sadowski *et al.* 1999; Gilman *et al.* 2002, 2003),

it remains unclear whether childhood conditions primarily influence early onset depression or whether the risk of depression associated with childhood adversity continues into adulthood. To our knowledge, only one study has addressed this question using a prospective design; Jaffee *et al.* (2002) analysed data from a 26-year birth cohort of individuals ascertained in Dunedin, New Zealand and observed significant differences in those childhood risk factors associated with adult *versus* child (≤ 14 years) onset depression. Based on a cross-sectional survey of adults, Kessler & Magee (1993) reported some

¹ Address for correspondence: Dr Stephen E. Gilman, Department of Maternal and Child Health, Harvard School of Public Health, 677 Huntington Avenue, Boston, MA 02115, USA.

types of childhood adversity were associated with depression onset throughout the life course (i.e. family drinking and parental marital problems), whereas other types of adversity were associated only with depression onset before age 20 (i.e. family violence and parental loss). Data from the National Comorbidity Survey suggest that effects of childhood adversity on subsequent psychopathology, including depression, peak in the years immediately following the adversity and diminish thereafter, also suggesting that childhood adversity is more strongly related to earlier rather than later onset disorders (Kessler *et al.* 1997). The link between childhood adversity and early onset depression is of particular concern because of the correspondence between early onset depression and increased recurrence risk (Giles *et al.* 1989; Keitner *et al.* 1992; Lewinsohn *et al.* 1994; Steffens *et al.* 1996; Klein *et al.* 1999), secondary psychiatric and substance use disorders (Christie *et al.* 1988; Rohde *et al.* 1991; Giaconia *et al.* 1994; Andrade *et al.* 1996; Kasch & Klein, 1996; Kovacs, 1996), and severe psychosocial consequences (Berndt *et al.* 2000).

Integrating the results of prior research on the timing of depression onset is complicated by varying definitions of 'early' onset depression across studies. Childhood *versus* adult onset depression has often been differentiated by using cutoffs in the late teens or early twenties (Kessler & Magee, 1993; Lewinsohn *et al.* 1993; Parker *et al.* 1997). However, there is also evidence to suggest that the aetiologies of pre-pubertal and post-pubertal depression are not entirely coincident. For example, results of various types of family studies indicate that the familial aggregation of depression, prevalence of familial risk factors for depression and rates of psychopathology in relatives of depressed probands vary by pre-pubertal *versus* post-pubertal age at onset (Weissman *et al.* 1988; Puig-Antich *et al.* 1989; Thapar & McGuffin, 1994; Harrington *et al.* 1997; Kovacs *et al.* 1997; Silberg *et al.* 1999; Warner *et al.* 1999). This evidence, although not consistent across all studies, suggests the importance of distinguishing between depression occurring before and after the early teens. Accordingly, in the present investigation we incorporated both of the distinctions made in prior research, and analysed risks for depression onset separately for the following three time

periods: ages ≤ 14 , between ages 15 and 20, and ages ≥ 21 .

Childhood adversity and the course of depression: recurrence and remission

A second issue of importance is whether childhood adversity is related to increased recurrence risk and decreased likelihood of remission among individuals with at least one depressive episode. If true, this may reflect a persisting psychological vulnerability imparted by the experience of adversity during a key developmental period (Bowlby, 1977; Abramson *et al.* 1978; Harris *et al.* 1990; Hertzman & Wiens, 1996). This possibility is likely according to several cross-sectional studies using population-based samples. For example, Kessler & Magee (1993) and Wainwright & Surtees (2002) observed a significant association between retrospectively reported parental divorce in childhood and elevated risk of recurrent episodes among individuals with a history of depression. However, using cross-sectional studies of adults to investigate childhood risks for recurrent depression is problematical owing to the general difficulty of recalling events that occurred many years in the past (Henry *et al.* 1994) as well as the unresolved issue of whether retrospective reports are altered by the depression itself (Kuyken & Brewin, 1995; Calev, 1996). An additional limitation of studies on recurrent depression is the tendency to define recurrence in a dichotomous fashion, for example, the presence or absence of a single depressive episode at some point in time following a first lifetime onset (Kessler & Magee, 1993; Flint & Rifat, 1999; Kendler *et al.* 2000). This obscures the degree of recurrence, which is a critical issue in depression given the highly recurrent nature of the disorder and the substantial variability in recurrence across individuals (Kessing *et al.* 2000).

In previous studies of depression risk with the Providence NCPP cohort, we observed an increased lifetime risk of depression among adult offspring of low socio-economic status (SES) parents (Gilman *et al.* 2002); independently of their SES, individuals who experienced family disruption during childhood were also at increased risk for depression at some time during their lives (Gilman *et al.* 2003). The present study extends the work of previous research in two ways. First, we examined the risk that early

childhood adversity confers on the development of major depression, and investigated whether this risk continues into adulthood or diminishes with age. Secondly, we analysed the effects of childhood adversity on the risk of recurrent episodes and likelihood of remission following onset of the disorder. We focused on three childhood disadvantages: SES, family disruption and residential instability. Low SES, defined here according to parental occupation during the child's first 7 years of life, is a consistent predictor of elevated depression risk, both in adults (Murphy *et al.* 1991; Muntaner *et al.* 1998) as well as in children (Johnson *et al.* 1999; Gilman *et al.* 2002). Family disruption is also known to increase the risk of psychopathology in the short term, although questions remain regarding the long-term psychiatric consequences of childhood family disruption (Hetherington & Stanley-Hagan, 1999). Finally, residential instability in childhood has been associated with behavioural and substance abuse outcomes among children in prior studies (Wood *et al.* 1993; DeWit, 1998), but the association between geographic relocations and depression has not been studied as extensively.

METHOD

Study sample and adult follow-up procedures

Subjects were offspring of participants in the Providence, Rhode Island cohort of the National Collaborative Perinatal Project (NCPPI) (Niswander & Gordon, 1972), a multi-site study that followed > 50 000 pregnancies through the first 7 years of life. Obstetrical intake occurred between 1959 and 1966, during which time a total of 4140 pregnancies were enrolled.

Selection of Providence NCPPI births for the adult follow-up studies occurred in two separate phases; in each, a stratified random sample was drawn from the entire cohort to investigate the association between several early life factors and adult psychiatric disorders. During phase one, initiated in 1984, 995 eligible subjects with and without pregnancy/delivery complications were selected (Buka *et al.* 1998). In the second phase, initiated in 1996, 1056 subjects with and without potential learning disabilities were selected (Buka *et al.* 1993). In total, 1267 were successfully located and interviewed, yielding an overall participation rate of 71.2%. Of these 1267

subjects, 1089 had complete data for the childhood and sociodemographic variables, and thus comprised the analysis sample for the present study.

Measures

Childhood SES was based on parental occupation at the time of enrolment and at the age 7 assessment. This was coded as either manual or non-manual according to the 1960 United States Census (US Bureau of the Census, 1963). Subjects in two-parent households were assigned to the higher occupation of both parents, whereas those from single-parent households were assigned to the occupation of the parent with whom they were living. Subjects whose parents were not in the paid labour force at either or both childhood assessments were categorized as 'not employed'. On the basis of previous analyses of the association between childhood SES and the lifetime risk of depression (Gilman *et al.* 2002), we dichotomized parental occupation into two categories: (1) manual or not employed at birth or age 7; (2) non-manual at birth and age 7. Family disruption was defined as either parental divorce or separation between the child's birth and seventh year. Lastly, residential instability was based on the number of moves since birth as reported by participants' mothers during the age 7 interview. On the basis of preliminary analyses in which the risk of depression increased sharply among children whose family moved 3 or more times by age 7, this variable was analysed dichotomously as either 3 or more *versus* 0, 1 or 2 moves.

Lifetime diagnosis of major depressive episode was determined with the Diagnostic Interview Schedule (DIS), administered to subjects by trained interviewers (Robins *et al.* 1981). The DIS has been used widely in community samples, and has demonstrated satisfactory psychometric properties for the diagnosis of depression (Oliver & Simmons, 1985; Erdman *et al.* 1987; Wittchen *et al.* 1989). During the first phase of the follow-up study, diagnoses were based on DSM-III criteria, whereas in the second phase, begun in 1996, a revised version of the DIS for DSM-IV was used. Age at depression onset was based on the DIS item that asked respondents the age at which they first had a period of ≥ 2 weeks of depressed mood contemporaneous with other depressive symptoms.

Among individuals with a lifetime diagnosis of depression, two measures of the subsequent course of the disorder were obtained. First, recurrence was defined as the self-reported number of lifetime depressive episodes. Secondly, current remission of depression was defined as a 6-month period without depressive episodes, and was operationalized using the DIS item asking respondents the age at which their most recent depressive episode occurred. Therefore, respondents whose most recent depressive episode occurred >6 months before interview were defined as in remission.

Statistical methods

Analyses of the onset and course of depression were conducted within a single analytical framework. Poisson regression was used to model the onset, recurrence and remission of major depressive episodes as a function of childhood and sociodemographic covariates. In the models for depression onset and remission, the Poisson model was equivalent to an exponential survival analysis (Wei, 1992) with piecewise constant hazards specified for each year at risk (Breslow, 1974). These models were based on the retrospectively reported ages at depression onset and most recent episode respectively. Accordingly, the model for depression onset included person-years from birth through age at onset for those with depression, and age at interview for all others. The model for remission was estimated in the subsample of respondents with depression, and included person-years from the age at depression onset through age at last episode (for those whose last episode was >6 months prior to interview) or age at interview.

To investigate risks for recurrent episodes, we estimated a Poisson regression model in which the dependent variable was the total number of lifetime depressive episodes (McCullagh & Nelder, 1989). Because of individual differences in the amount of time that elapsed between the age at depression onset and the age at interview, there was individual variability in the time at risk for recurrent episodes. We incorporated this variability into the analysis by dividing the total number of depressive episodes by the natural logarithm of the time at risk (McCullagh & Nelder, 1989). We also accounted for possible overdispersion in the analysis of recurrence (i.e. the departure of the observed variance in the

number of depressive episodes from the value expected under the Poisson distribution). Based on the method described by Fitzmaurice *et al.* (1997), we estimated the degree of overdispersion by fitting a 'maximal' model containing all of the covariates of interest plus all possible two-way interactions, and then incorporated this estimate into our analysis of recurrence as an adjustment factor in computing the standard errors of regression coefficients. Analyses were conducted using PROC GENMOD in SAS version 8 (SAS Institute, 1999).

In addition to indicators of childhood adversity, the following variables were included in the analyses. Family history of mental disorders prior to the birth of the child was controlled due to the increase in risk for depression among offspring of depressed parents (Billings & Moos, 1986; Beardslee *et al.* 1993). This was determined by the mother's report upon enrolment of whether she, the subject's father, or the subject's siblings had ever received treatment for emotional or behavioural problems. We also controlled for maternal age at the subjects' birth, the respondent's age at interview, sex, and race/ethnicity coded as White or non-White. To account for the stratified sampling of respondents selected for the study, we included a set of indicator variables representing the stratification factors. In the analyses of recurrence and remission, we controlled for age at depression onset; we also adjusted for number of depressive episodes in the analysis of remission.

RESULTS

Characteristics of the sample

The demographic characteristics of the sample are shown in the first column of Table 1. Slightly more than half of the sample was male (52.6%) and the majority was White (72.8%). Most respondents completed high school (76.6%), while 40.3% had at least some college-level education. The mean (s.d.) age of respondents at the time of the adult reinterview was 29.0 (5.7), and ranged from 18 to 39. In addition to demographic characteristics, measures of childhood adversity are also presented in the first column of Table 1. Approximately one-fifth of the sample had parents at the highest occupational level (non-manual) during their childhood, while the remainder was classified as either manual or

Table 1. Characteristics of the study sample (N = 1089) and the onset/course of major depression

	Distribution of sociodemographic characteristics		Onset*		Recurrence†		Remission‡	
			Lifetime prevalence of depression		Lifetime depressive episodes‡		Proportion of depressed sample with 6-month remission§	
	N	%	N	%	N	Mean (s.d.)	N	%
Childhood socio-economic circumstances								
Parental occupation at birth and age 7								
Manual or Not Employed at either age	880	80.8	236	26.8	192	6.1 (11.6)	118	61.5
Non-manual at both ages	209	19.2	36	17.2	33	3.5 (11.7)	26	78.8
Parents divorced or separated by age 7								
Yes	337	30.9	106	31.5	81	6.1 (11.8)	46	56.8
No	752	69.1	166	22.1	144	5.5 (11.7)	98	68.1
Number of moves between birth and age 7								
≥3	413	37.9	129	31.2	106	6.0 (11.8)	65	61.3
0, 1 or 2	676	62.1	143	21.2	119	5.5 (11.8)	79	66.4
Respondent sociodemographic characteristics								
Sex								
Female	516	47.4	150	29.1	129	5.9 (11.8)	81	62.8
Male	573	52.6	122	21.3	96	5.4 (11.8)	63	65.6
Ethnicity								
Non-White	296	27.2	59	19.9	42	3.2 (11.6)	24	57.1
White	793	72.8	213	26.9	183	6.3 (11.6)	120	65.6
Educational attainment, years								
0-9	99	9.1	37	37.4	24	9.1 (11.6)	12	50.0
10-11	156	14.3	38	24.4	28	5.6 (11.6)	15	53.6
12	395	36.3	90	22.8	73	6.5 (11.6)	42	57.5
≥13	439	40.3	107	24.4	100	4.3 (11.7)	75	75.0
Age at depression onset, years								
≤14			49	21.8	49	11.3 (15.4)	21	42.9
15-20			60	26.7	60	3.3 (11.6)	36	60.0
≥21			116	54.6	116	4.7 (14.0)	87	75.0

* Lifetime prevalence of major depressive episode in the entire sample, 25.0% (N = 272).

† Recurrence and remission estimated in the sample of respondents with depression with complete data on number of episodes and age at last episode (N = 225).

‡ Mean number of depressive episodes adjusted for number of years between age at onset of first episode and age at interview.

§ Proportion of respondents with depression with offset at least 6 months prior to age at interview, 64.0% (N = 144).

not employed at birth and/or age 7. Almost one-third (N = 337) of the sample experienced family disruption during early childhood (185 by parental divorce, 152 by parental separation). Lastly, 38% of respondents' families moved three or more times by the age of 7. Columns 2, 3, and 4 of Table 1 show the distribution of the childhood and demographic factors with respect to the onset, recurrence, and remission of depression, respectively.

Childhood adversity and risk for depression onset

The lifetime prevalence of major depression in the sample was 25.0% (N = 272). This is higher

than has been reported in the general population, e.g. 17.5% in the National Comorbidity Survey (Kessler *et al.* 1994). Results of survival analyses of depression onset are presented in Table 2 (Model I). Risk of depression onset was significantly higher among individuals from lower SES backgrounds (hazard ratio (HR) = 1.57; 95% confidence interval (CI), 1.08, 2.29). In addition, family disruption (HR, 1.41; CI, 1.09, 1.84) and residential instability (HR, 1.36; CI, 1.05, 1.78) by the age of 7 predicted an increased risk for the development of depression. These associations were independent of demographic factors, which themselves were related to depression; higher risks were observed for

Table 2. Risks for lifetime major depressive episode

	Model II†									
	Model I†		Effects varying by age at depression onset							
	Main effects only		Time-invariant effects		Onset age ≤14		Onset ages 15–20		Onset age ≥21	
	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI
Childhood socio-economic circumstances										
Parental occupation at birth and age 7										
Manual or Not Employed at either age	1.57*	1.08, 2.29	1.60*	1.10, 2.34						
Non-manual at both ages	1.00		1.00							
χ^2 (df = 1)		6.0*		6.4*						
Parents divorced or separated by age 7										
Yes	1.41**	1.09, 1.84			2.39***	1.46, 3.91	1.34	0.81, 2.21	1.05	0.72, 1.55
No	1.00				1.00		1.00		1.00	
χ^2 (df = 1)		6.6*				12.1***		1.3		0.1
Number of moves between birth and age 7										
≥3	1.36*	1.05, 1.78			2.62***	1.56, 4.39	1.08	0.66, 1.78	1.05	0.73, 1.53
0, 1 or 2	1.00				1.00		1.00		1.00	
χ^2 (df = 1)		5.2*				13.3***		0.1		0.1
Respondent sociodemographic characteristics										
Sex										
Female	1.75***	1.36, 2.23			0.91	0.57, 1.48	1.81*	1.12, 2.92	2.43***	1.71, 3.45
Male	1.00				1.00		1.00		1.00	
χ^2 (df = 1)		19.6***				0.1		5.9*		24.3***
Ethnicity										
Non-White	0.67**	0.50, 0.90	0.67**	0.50, 0.91						
White	1.00		1.00							
χ^2 (df = 1)		7.4**		7.3**						
Educational attainment										
0–9 years	1.82**	1.22, 2.71	1.84**	1.23, 2.75						
10–11 years	1.25	0.85, 1.85	1.22	0.83, 1.80						
12 years	1.03	0.77, 1.37	1.03	0.77, 1.37						
≥13 years	1.00		1.00							
χ^2 (df = 3)		9.1*		9.2*						

† Also controlling for family history of mental illness, maternal age, age at interview, and study selection factors.

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

females compared to males, Whites compared to non-Whites, and those with lower educational attainment.

A question of central interest is whether the effects of childhood adversity on depression diminish with time (Kessler *et al.* 1997). To address this question, we added interaction terms between the childhood and demographic risk factors and age, categorized as ≤ 14 , 15–20, or ≥ 21 . Three of these interactions were statistically significant at the 5% level: family disruption ($\chi^2=7.0$, $df=2$, $P=0.030$); residential instability ($\chi^2=9.6$, $df=2$, $P=0.008$); and sex ($\chi^2=10.6$, $df=2$, $P=0.005$). Results from the model containing these interactions are shown in the remaining columns of Table 2 (Model II). The time-invariant effects are shown first; these are effects that were constant across age categories. Of note, the effect of low SES in childhood on depression risk persisted into adulthood (HR, 1.60; CI, 1.10, 2.34). Conversely, the depressogenic effects of childhood family disruption and residential instability were specific to early onset depression. Hazard ratios for these factors were obtained by exponentiating linear combinations of the regression coefficients and are shown in the last 3 columns of Table 2. These indicate a doubling of risk for depression by age 14 and no increase in risk for depression thereafter. Finally, while females had an average lifetime risk of depression 75% higher than males, our results demonstrate the emergence of the female preponderance in depression in adolescence (HR, 1.81; CI, 1.12, 2.92) that increased in adulthood (HR, 2.43; CI, 1.71, 3.45), an observation consistent with expectation (Angold & Worthman, 1993; Kessler *et al.* 1993). We evaluated interaction terms between sex and the childhood disadvantages, but these were not statistically significant.

Childhood adversity and the course of depression

Of the 225 respondents with a lifetime diagnosis of depression, 59.1% reported at least 1 recurrent episode of depression; 15.6% reported ≥ 10 episodes. The mean (s.d.) number of depressive episodes reported by these respondents was 5.7 (12.4). We used Poisson regression to model the recurrence of depressive episodes as a function of childhood adversity and adult socio-demographic factors. As described above, we

estimated the overdispersion from a maximal model (Fitzmaurice *et al.* 1997) that contained all of the covariates plus their two-way interactions; the resulting estimate (2.37) was used to adjust the standard errors in the model for recurrence (Table 3, Model I). Recurrent episodes were more likely among individuals from lower SES backgrounds (rate ratio (RR), 1.61; CI, 1.11, 2.33). However, recurrence was not associated with family disruption or with residential instability in early childhood. Significant demographic correlates of elevated recurrence included White race/ethnicity and lower educational attainment. Of note, individuals with ≤ 9 years of education were at more than twice the risk for recurrent depression (RR, 2.56; CI, 1.93, 3.41) than individuals with ≥ 13 years. These effects were adjusted for age at depression onset, which itself was a significant predictor of recurrence. Relative to those with adult onset depression (≥ 21 years), respondents with childhood onset depression were at greater risk for recurrence, and respondents with adolescent onset depression were less likely to experience recurrent episodes.

We tested interaction terms between childhood risk factors and age at depression onset to evaluate possible differences in the predictors of recurrence depending on the age at onset. Only the interaction involving sex ($\chi^2=29.2$, $df=2$, $P<0.001$) revealed a pattern of recurrence risk that differed by age at onset. Following childhood onset depression, recurrent episodes were equally likely among males and females; however, recurrence risk in adolescent onset depression was significantly lower among females (RR, 0.53; CI, 0.35, 0.82), whereas in adult onset depression it was significantly higher among females (RR, 2.31; CI, 1.56, 3.43).

The final set of analyses concerns remission of depression, defined here as a 6-month period free of DSM threshold level symptomatology. Sixty-four per cent of respondents with a history of depression ($N=144$) were in current remission, while the remaining 81 respondents reported a depressive episode within 6 months of interview. Results of a survival analysis of remission are presented in the second column of Table 3 (Model II). Hazard ratios >1 indicate an increased likelihood of remission; hazard ratios <1 indicate reduced likelihood of remission, and therefore greater degree of

Table 3. *Childhood adversity and the course of major depression*

	Model I: Recurrence†‡ Poisson regression of the number of depressive episodes		Model II: Remission†‡ Survival analysis of the age at depression offset	
	RR	95% CI	HR	95% CI
Childhood socio-economic circumstances				
Parental occupation at birth and age 7				
Manual or Not Employed at either age	1.61**	1.11, 2.33	0.77	0.45, 1.32
Non-manual at both ages	1.00		1.00	
χ^2 (df=1)		6.9**		0.9
Parents divorced or separated by age 7				
Yes	0.97	0.79, 1.19	0.64*	0.41, 0.99
No	1.00		1.00	
χ^2 (df=1)		0.1		4.0*
Number of moves between birth and age 7				
≥ 3	1.02	0.83, 1.24	1.65*	1.10, 2.47
0, 1 or 2	1.00		1.00	
χ^2 (df=1)		0.0		5.8*
Respondent sociodemographic characteristics				
Sex				
Female	1.05	0.86, 1.28	0.90	0.62, 1.32
Male	1.00		1.00	
χ^2 (df=1)		0.2		0.3
Ethnicity				
Non-White	0.63***	0.48, 0.82	0.72	0.43, 1.18
White	1.00		1.00	
χ^2 (df=1)		12.4***		1.8
Educational attainment				
0-9 years	2.56***	1.93, 3.41	0.39**	0.20, 0.77
10-11 years	1.79***	1.31, 2.45	1.05	0.53, 2.10
12 years	1.59***	1.27, 1.99	0.54**	0.35, 0.85
≥ 13 years	1.00		1.00	
χ^2 (df=3)		43.9***		13.4**
Onset and course of depression				
Age at depression onset				
≤ 14 years	1.43**	1.12, 1.81	0.06***	0.03, 0.10
15-20 years	0.58***	0.44, 0.77	0.15***	0.09, 0.25
≥ 21 years	1.00		1.00	
χ^2 (df=2)		47.8***		113.2***
Number of lifetime depressive episodes				
χ^2 (df=1)			0.92***	0.87, 0.97
				19.4***

† Also controlling for family history of mental illness, maternal age, age at interview, and study selection factors.

‡ Estimated in the sample of respondents with depression ($N=225$).

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

persistence. As the number of lifetime depressive episodes was included as a covariate, the effects of childhood adversity on remission are independent of their effects on recurrence.

Of the three measures of childhood adversity, two were associated with remission of depression. Early childhood family disruption was related to a lower likelihood of remission (HR, 0.64; CI, 0.41, 0.99). In contrast, residential instability in childhood was related to a higher likelihood of remission (HR, 1.65; CI, 1.10, 2.47). To explore this unexpected finding further,

we estimated several additional models. Alone, residential instability in childhood predicted a prolonged course of depression (HR, 0.67; CI, 0.48, 0.93). The association between childhood residential instability and the course of depression was marginally attenuated after adjustment for childhood SES, family disruption and the sociodemographic variables (HR, 0.72; CI, 0.50, 1.03), and moved further towards the null value of 1 once number of depressive episodes was controlled for (HR, 0.83; CI, 0.80, 1.20). It was not until age at depression onset

was introduced into the equation that the effect of residential instability switched directions. This may be interpreted to suggest the presence of multiple pathways linking childhood residential instability to adult depression. In one pathway, residential instability increases risk for childhood onset depression, which in turn predicts increased persistence. On the basis of our results, there exists an additional pathway linking residential instability in childhood to a shorter course of depression subsequent to the onset episode, possibly reflecting positive changes in the child's environment and/or tolerance of change.

The analysis of remission also revealed significant effects of lower educational attainment and younger age at depression onset on the reduced likelihood of remission. Finally, each additional episode of depression was associated with a longer time to remission.

DISCUSSION

Limitations

There are several limitations to this study. Our analyses rely on the accuracy of subjects' recollection of the age at which their depressive symptoms first occurred. The ability of instruments such as the DIS to reliably assess the chronology of symptom onset is constrained by human memory (Prusoff *et al.* 1988; Rogler *et al.* 1992). However, research indicates that the reliability of the DIS age of onset questions for depression lies within an acceptable range (Farrer *et al.* 1989; Wittchen *et al.* 1989). For example, Wittchen *et al.* conducted a test-retest study of the DIS onset questions for depression and found that the intraclass correlation (ICC) between assessments performed approximately 2 days apart was between 0.49 and 0.77 (Wittchen *et al.* 1989). In an analysis of the one-year reinterview component of the Epidemiologic Catchment Area Survey, Gilman & Abraham (2001) reported that the correlation between two assessments of the onset of depressive symptomatology was 0.87. These results suggest that the DIS records the timing of depression onset with acceptable reliability. Presumably, this also applies to retrospective reports of respondent's age at most recent episode, especially since events closer in time are reported with a higher degree of reliability

(Farrer *et al.* 1989); the study by Wittchen and colleagues (1989) provided moderate support for this. In their study, the mean percentage agreement of the recency items for depression was 69.5%; the median kappa values, correcting for agreement by chance, was statistically significant but lower (0.34). To examine the potential impact of the retrospective age at onset reports on the findings of the present study, we re-estimated the survival models of depression onset and remission by fitting logistic regression models to binary indicators of lifetime major depressive episode and current remission respectively. We observed a similar pattern of associations between childhood disadvantages and adult depression in these logistic regression analyses as in the survival analyses presented in Tables 2 and 3. While this lends support to the finding that childhood disadvantages are associated with an increased risk of depression onset as well as a prolonged course of the disorder, the finding that some childhood disadvantages – i.e. family disruption and residential instability – predict childhood onset depression rather than depression with adolescent or adult onset needs to be replicated by studies in which the occurrence of depressive episodes is recorded prospectively.

Our analyses of remission were also limited by the lack of data on the timing of individual episodes of depression. This meant that we could not investigate the pattern and duration of depressive episodes over the life course.

Less is known regarding the reliability of reports of lifetime depressive episodes; however, in the study by Wittchen *et al.* described above (1989), the ICC was nearly perfect for the number of depressive episodes, but the level of agreement on the exact number was considerably lower (18.3%). There is also concern regarding the validity of such reports in light of the small number of respondents indicating a very high number of episodes. As noted above, 15.6% ($N=35$) of respondents with depression reported ≥ 10 episodes; however, 4.9% ($N=11$) reported ≥ 20 . We chose not to truncate the number of episodes in our analyses because any ceiling we would apply to these data would be arbitrary; ultimately, research is needed to validate self-reported episodes against either longitudinal or externally-derived information.

It should be noted that our analyses of recurrence and remission were based on the subset of respondents ($N=225$) meeting diagnostic criteria for major depression and with complete data for all study variables. Therefore, we had reduced statistical power to detect significant associations between childhood factors and the course of depression. These analyses should be replicated in larger samples of depressed individuals. In particular, replication is needed to further evaluate sex-differences in recurrence.

Other limitations of our study concern the generalizability of our results in two respects. First, the Providence NCPP sample was not designed to be fully representative of the general population in Providence, or of the general United States population. Secondly, our findings may not generalize to time periods other than that which was studied. Childhood adversity occurring during the 1960s might have more severe consequences for children than it does presently; however, reports based on contemporary samples of children suggests that this is not the case (Aseltine, 1996; McLeod & Shanahan, 1996; Amato, 2001). It should also be noted that the maximum age of subjects in our study was 39, meaning that we could not examine the role of childhood factors on depression beyond this age; this is important in light of evidence that the age at depression onset is related to its subsequent course well into adulthood (Steffens *et al.* 1996; Philibert *et al.* 1997; Reynolds *et al.* 1998).

Comment

Our results shed insight into the complex relations between environmental risk factors for depression – particularly in childhood – age at depression onset and the subsequent course of the disorder. We discuss each of these dimensions in turn.

Childhood risks for depression onset

Our analyses revealed that low SES, family disruption, and residential instability in childhood predict an increased risk of major depression. These results are consistent with prior research on the childhood origins of depressive disorder in showing the significant influence of childhood adversity on subsequent depression (Power, 1991; Lundberg, 1993; Wood *et al.* 1993;

Rodgers, 1994; Simpson & Fowler, 1994; Sadowski *et al.* 1999; Stoneman *et al.* 1999). We found that the three aspects of childhood adversity studied were independently related to depression risk. As childhood adversities frequently cluster together (Kessler *et al.* 1997; Sadowski *et al.* 1999), the finding that each adversity confers added risk for depression is of substantial public health concern. Finally, our analyses of sex differences in depression onset replicate the results from numerous other studies demonstrating a female preponderance in depression emerging in adolescence and continuing into adulthood (Weissman & Klerman, 1977; Angold & Worthman, 1993; Kessler *et al.* 1993).

We previously reported that low SES and family disruption during childhood were associated with elevated lifetime risks of depression (Gilman *et al.* 2002, 2003). This investigation distinguished the risks for depression across three time periods: early childhood, adolescence and adulthood. By doing so, we found that family disruption and residential instability were related to depression risk through age 14 but not thereafter. In contrast, childhood SES was associated with depression onset well into adulthood. The specificity of the association between family disruption and residential instability and childhood onset depression highlights the utility of decomposing lifetime risk according to developmental stage. Additional knowledge of risk factors that differentially predict childhood *versus* adult depression will be particularly important for reducing the heterogeneity of the depression phenotype (Kaufman *et al.* 2001; Jaffee *et al.* 2002). Therefore, we suggest that data on the timing of depressive symptoms be routinely incorporated into aetiological studies of depression.

There is considerable evidence that risk for childhood depression increases in the years following family disruption and frequent moves (Fergusson *et al.* 1994; Aseltine, 1996; Hetherington & Stanley-Hagan, 1999; Amato, 2001; Jaffee *et al.* 2002). Family disruption portends significant change in children's lives, often involving adaptation to new roles (Hetherington *et al.* 1989). Although family socio-economic status is also frequently impacted by parental divorce (Eggebeen & Lichter, 1991), the increased risk of depression was independent of SES.

The nature of the long-term consequences of childhood family disruption is less clear. There is evidence that adults who experienced family disruption during their childhood are more likely to report depressive symptoms than their counterparts from non-disrupted families (Amato, 1991; Rodgers, 1994; Cherlin *et al.* 1998). In part, this may be due to a history of childhood depression, which tends to be highly recurrent (Lewinsohn *et al.* 1994). In the present study, we distinguished between risks for first onset and those for recurrent depression, and investigated the risks for first onset depression over the lifespan. By doing so, we found that childhood family disruption predicted childhood onset depression but not adult onset depression, and that it was significantly related to an extended time to remission. This pattern of results suggests that for some individuals, childhood family disruption increases risk for depressive episodes throughout the life-course (Ross & Mirowsky, 1999). Most children who experience family disruption, however, do not manifest psychiatric symptoms in adulthood (Hetherington & Stanley-Hagan, 1999).

The increased risk of depression among individuals from lower SES backgrounds persisted in adulthood. The long-term association between childhood SES and depression likely exists because of the influence of SES on psychological processes that are more proximal to etiology of depression; moreover, these processes likely vary over time. In childhood, low SES has been shown to directly impact psychological well-being (McLeod & Shanahan, 1993); it may also interrupt important aspects of psychological development, which in turn increase adult vulnerability to depression. For example, socio-economic adversity may reduce children's sense of control over their environments, thereby resulting in long-term vulnerability to depression (Abramson *et al.* 1978; Chorpita & Barlow, 1998). Although in our study the association between childhood SES and adult depression was independent of adult SES, the persistence of depression risk among individuals from lower SES backgrounds also may reflect the continuity of socioeconomic conditions from childhood to adulthood. For example, Power & Matthews (1997) demonstrated the significant correspondence between economic deprivation in childhood and adult socio-economic outcomes.

Childhood factors and the course of depression

As in prior studies (Lewinsohn *et al.* 1994; Reynolds *et al.* 1998), we found that depression onset in early childhood had a higher recurrence rate and a lower probability of remission than adult onset depression. The substantial influence of age at onset on the course of depression supports the argument that this factor distinguishes aetiological subtypes of the disorder (Kaufman *et al.* 2001; Buka & Gilman, 2002). Age-at-onset differences in the familial aggregation of major depression lend further support to this assertion (Cadoret *et al.* 1977; Weissman *et al.* 1984, 1987, 1988; Puig-Antich *et al.* 1989; Orvaschel, 1990; Kovacs *et al.* 1997; Klein *et al.* 1999).

Apart from younger age at depression onset and a history of depressive episodes, the determinants of recurrent depressive episodes vary considerably across studies. In our analyses, we found that low SES in childhood, but not family disruption or residential instability, predicted an increased risk for recurrent episodes. In prior studies, significant childhood risks for recurrent depression included parental divorce (Wainwright & Surtees, 2002), family violence (Kessler & Magee, 1994) and parental psychopathology (Lewinsohn *et al.* 2000). Evidence for sex differences in the recurrence of depression is also inconsistent, with increases in recurrence risk among females reported in some (Bracke, 1998; Lewinsohn *et al.* 2000), but not all (Kessing *et al.* 2000; Wainwright & Surtees, 2002) studies. We observed an increased risk of recurrence among females that was limited to individuals with adult onset depression.

One difficulty in identifying risks for recurrence is that, by definition, these studies are restricted to samples of individuals with a history of depression, and are often limited by small sample sizes. Also, differences in how recurrence is operationalized across studies doubtless contributes to inconsistencies. Due to the highly recurrent nature of major depression (Solomon *et al.* 2000), identification of risks for recurrent episodes remains a pressing concern for the secondary prevention of depression.

A second dimension of the course of major depression that we analysed was remission, defined here as a 6-month period free of depressive episodes. Factors that were associated with a

reduced likelihood of remission – and therefore a prolonged course of the disorder – included early childhood family disruption and lower educational attainment. These effects were independent of their associations with younger age at onset and increased recurrence, which themselves predicted the persistence of depression. Results of prior studies on the remission of depression are mixed. McLeod *et al.* (1992) found modest support for an association between lower educational attainment and reduced odds of remission from depression, but failed to find any association between age at onset and number of prior depressive episodes and the speed of remission.

In our sample, childhood residential instability predicted a shorter course of depression net of its relation with younger age at onset. This suggests that for some individuals, geographic relocation may reflect positive changes, whereas for others, it has adverse psychological consequences including early onset depression. Residential instability has also been associated with conduct disorder (Cohen *et al.* 1990), behavioural problems (Humke & Schaefer, 1995), difficulties in school (Haveman *et al.* 1991; Wood *et al.* 1993; Simpson & Fowler, 1994) and substance abuse (DeWit, 1998). Interpretation of this study's findings pertaining to residential instability is constrained by the fact that a simple count of the number of moves is not an adequate representation of the context in which the moves occurred or the resources available to children throughout these transitions (Hendershott, 1989; Humke & Schaefer, 1995). What is therefore needed is a greater degree of precision in future studies of the childhood environment, perhaps by integrating qualitative and quantitative methodologies, to determine whether the long-term effects of environmental 'risk' factors may be protective in certain contexts. This has previously been hypothesized with respect to parental divorce regarding situations where the divorce leads to the termination of family violence (Amato, 1993).

Conclusions

Childhood social conditions have significant consequences for the onset and course of major depression. In the present investigation, we found a strong association between family disruption and frequent geographic relocation by

age 7 and the onset of depression by age 14. Programmes and policies that attempt to buffer the effects of changes in family structure on children may be one avenue for reducing the prevalence of early onset depression. We also found that childhood SES was associated with an increased risk of depression in both childhood and adulthood. Further research is needed to identify the pathways between childhood SES and depression and to determine whether these pathways change with age (Power & Hertzman, 1997). Identifying risk factors for depression onset at different ages may lead to more refined prevention strategies for depression. It may also enhance our understanding of the role that age of onset plays in the clinical course of depression.

Low childhood SES conferred increased risk for recurrent episodes of depression following onset. In addition, family disruption by age 7 was related to a reduced likelihood of remission. These results suggest that both primary and secondary prevention are needed to mitigate the long-term effects of childhood adversity on depression. While risks for the onset of depression may not be entirely coincident with risks for recurrent depressive episodes, convincing evidence is emerging that various aspects of the childhood environment have profound consequences for adult mental health, both in terms of the initial onset and subsequent course of depression.

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