

ORIGINAL PAPER

G. C. Patton · C. Coffey · M. Posterino · J. B. Carlin · R. Wolfe

Parental 'affectionless control' in adolescent depressive disorder

Accepted: 21 December 2000

Abstract *Background:* Adults with depressive disorder report high rates of sub-optimal maternal care in childhood. Despite the greater salience of relationships with parents earlier in life, associations with parenting style have not yet been systematically studied in adolescent onset disorder. *Methods:* A six-wave, 3-year study of adolescent health in 2032 Australian secondary school students provided an opportunity to undertake a two-phase study of early onset depression. Between waves 2 to 6, a self-administered computerised form of the revised Clinical Interview Schedule (CIS-R) was used to generate a first phase diagnosis of ICD-10 depressive episode. Each subject with a CIS-R-defined depressive episode was selected for second phase assessment together with two subjects from the CIS-R non-cases in each school. Second phase assessment included a second diagnostic assessment using the depression and hypomania modules of the Composite International Diagnostic Interview (CIDI) and assessment of paternal and maternal style using the Parental Bonding Instrument. *Results:* A total of 1947 out of 2032 subjects in the sampling frame (95.8%) participated in the cohort study (phase 1) at least once; 406 (94%) of the 435 selected subjects completed second phase assessment. One hundred and nineteen subjects fulfilled criteria for depressive episode on the CIS-R at one or more waves. Over the 30-month study period, 69 subjects (10 male, 59 female) fulfilled both CIS-R and CIDI definitions of depression at the same wave and were classified as 'definite depressive disorder'. Low maternal and paternal care held independent associations with both definitions of depres-

sion, with the effects clearest in those in the lowest quartile of reported care. After adjusting for low parental care, the associations between high parental control and depression were small. *Conclusions:* Sub-optimal parenting is associated with depressive disorder in adolescents. Low maternal and paternal care are each associated with a two- to three-fold higher rate of depressive disorder. These findings are consistent with an effect of sub-optimal parenting on the onset rather than course of disorder. Whether sub-optimal parenting is associated with a risk for the onset of depression outside the adolescent years has yet to be clarified.

Introduction

Clinicians have long viewed adversity in childhood as a powerful determinant of later psychiatric disorder [1]. Even so, consistent associations with indices of childhood adversity, such as parental death or psychologically traumatic events, have been slow to emerge [2, 3]. One exception has been the link between sub-optimal parenting style during childhood and later affective disorder [4, 5]. Depressed adults, both in clinical and community settings, report lower levels of maternal and paternal care [2, 4, 6, 7]. Most studies have used the Parental Bonding Instrument (PBI), developed by Parker 20 years ago, to assess recollections of parental care and control (overprotection) – characteristics found in many other human relationships [8, 9].

Two hypotheses have been proposed to explain the association between sub-optimal parenting and depression [10]. Low parental care may give rise to a psychological style that brings interpersonal difficulties in adulthood, which in turn raise risks for depressive disorder [11–13]. A second hypothesis, that high levels of parental control might predispose to depression through interfering with processes leading to independent functioning during adolescence and young adulthood, has received less support [14]. Associations with depression have generally been found in patient series

G. Patton (✉)
Centre for Adolescent Health
2 Gatehouse St
Parkville 3052
Australia

G. C. Patton · C. Coffey · M. Posterino · J. B. Carlin · R. Wolfe
Department of Paediatrics
University of Melbourne
Melbourne, Victoria, Australia

[4, 10], but in non-clinical settings an association with parental control independent of low parental care has less consistently emerged [7, 14].

The relevance of these associations to causation has remained uncertain, partly because most of this research has taken place in older subjects well after the first onset of affective disorder. The passage of time, the experience of recurrent depression, ongoing difficulties in social relationships, subsequent course of the parent-offspring relationships and changing attitudes to parenting style with subsequent parenthood may all affect report of childhood parenting in adults. Additionally, it is uncertain to what extent associations between parenting style and adult depression might arise more from raising the likelihood of continuing disorder rather than influencing first onset [15].

For these reasons, the further study of associations between parenting style and depression early in the course of disorder is warranted. Depressive disorder appears to be uncommon before the age of 15 years, but incidence rises steeply thereafter, making the late teens a suitable point to study an association between parental style and early depressive disorder [16, 17].

Subjects and methods

Study sample

The study used a two-phase design, with the first phase set within the Victorian Adolescent Health Cohort Study (VAHCS). This six-wave study took place in the state of Victoria, Australia, between August 1992 and July 1995 (Fig. 1). This state has a population of 4.4 million, of whom 63% live in the capital city, Melbourne [18]. The first phase (cohort) sample was defined using a two-stage sampling procedure. Forty-five schools were selected from a stratified frame that included all secondary schools in the state (Government, Catholic and Independent strata). Within each stratum, schools were selected with a probability proportional to the number of Year 9 students in each stratum in the state (total number of students, 60,905; approximate age at study entry, 14.5 years). Two classes were selected from each school to constitute the phase 1 (cohort) sample. One class from each school entered in the latter part of the ninth school year (wave 1) and the second class entered 6 months later, early in the tenth school year (wave 2). Participants were reviewed at further four 6-monthly intervals, leading to a total of six waves for the first half of the cohort and five waves for the later-entering subcohort. One school from the

initial sampling frame participated at wave 1, but was unavailable for the cohort study, leaving a total sample of 88 classes from 44 schools, of which 24 were Government, 11 Catholic and 9 Independent Private. The second phase of the design took place between waves 2 and 6 of the VAHCS.

Procedure and measures

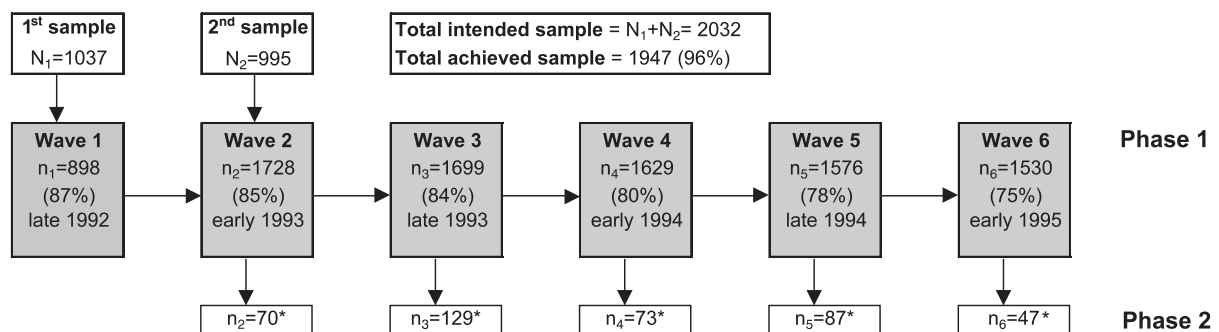
Phase 1: screening

In the first phase, the computerised version of the Revised Clinical Interview Schedule (CIS-R) was used [19, 20]. The CIS-R is a branched questionnaire, designed for assessing recent symptoms of depression and anxiety in non-clinical populations. Fourteen subscales determine the frequency, severity, persistence and intrusiveness of symptoms commonly found in depression and anxiety syndromes. These symptoms were used to delineate cases fulfilling criteria for ICD-10 depressive episode [21]. Each CIS-R case was selected for phase 2. For each CIS-R case, two subjects were concurrently selected at random from non-cases at that school, to provide a control group.

Second phase: assessment

Phase 2 was implemented between waves 2 and 5. All individuals selected for phase 2 were invited to attend for an interview that included further diagnostic assessment and more detailed measurement of putative risk factors for early onset depression. The depression and hypomania modules of the Composite International Diagnostic Interview (CIDI, Core Version 1.1) were used to generate lifetime ICD-10 diagnoses for affective disorder [22, 23]. The CIDI is a structured diagnostic interview devised for use by non-medical professionals after a standardised training [22]. A research psychologist, trained in the use of the CIDI and blind to first phase CIS-R scores or diagnostic status, conducted the second phase interviews. These took place at the school within 3 weeks of completion of at least 70% of first phase assessments in that school. Subjects diagnosed as CIDI cases on second phase interview were excluded from the selection procedure for phase 2 at subsequent study waves.

The Parental Bonding Instrument (PBI) [8] was administered to each subject immediately prior to their CIDI interview. The PBI is a 25-item questionnaire with a four-point fixed format response set ranging from '0 - very much like my parent' to '3 - very unlike my parent'. Two subscales provide indices of perception of parental care and parental control. Each phase 2 subject completed the instrument for mothers and fathers separately, with instructions to describe their relationship with each parent over the time they were growing up until the time of interview. Those not living with both natural parents were requested to describe the relationship with their main maternal and paternal figure over the previous 5 years.



* actual number of subjects interviewed

Fig. 1 Design and participation in a two-phase study of depressive disorder in Victorian secondary students

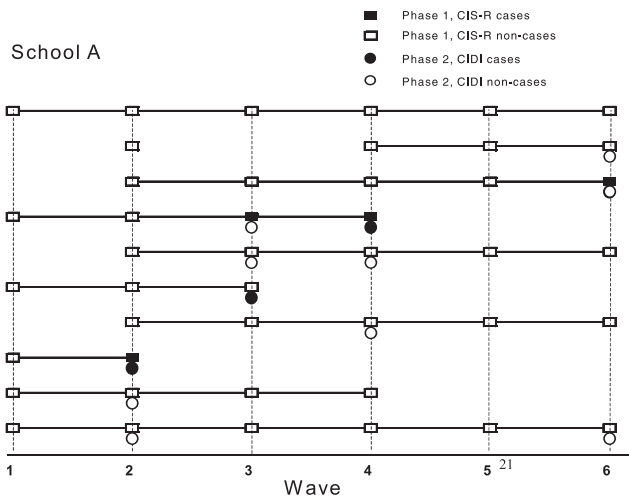


Fig. 2 Within-school sampling strategy for the two-phase study of adolescent on-set depressive disorder (DD), with each horizontal line representing a hypothetical first-phase participant. Interval censoring is indicated by a gap in the trace

Analysis

Data analysis was carried out using the Stata programme [24]. Stata's 'survey estimation' commands (svymean, svylogit) were used in the estimation, comparison of PBI scores and examination of associations between depressive disorder and covariates. Robust standard errors were calculated using the 'Information Sandwich' method [25]. Inverse probability weights, designed to approximate the inverse of the sampling fraction at the second phase assessment, were used in each of these analyses. Inverse probability weights adjusted for the potential bias that might arise from controls being more likely to be drawn from schools with a higher proportion of cases. For CIS-R cases, weights were the inverse of participation rates at phase 2. For CIS-R non-cases (controls), probabilities of selection were estimated for each school across all study waves. These were then used to group the 44 schools into tertiles corresponding to high, intermediate and low probabilities of control selection. The probability weight for all subjects within each tertile was obtained as the ratio of the number of screened subjects to the total number eligible within the tertile. The latter approach was used in order to smooth the estimated weights, as numbers within each school were too small to calculate meaningful weights.

Results

Phase 1 responses (the cohort study)

From a total sample of 2032 students on class registers, 1947 (95.8%) completed the first phase (cohort) questionnaire at least once in the course of the study. Based on the total sample of 2032, response rates across waves were as follows across waves 1 to 6: 87%; 84%; 82%; 79%; 76%; 74%. The gender ratio of the cohort (males 47%) was similar to that in Victorian schools at the time of sampling [26]. The mean age at wave 1 was 14.5 (SD 0.5) years and at wave 6, 17.4 (SD 0.4) years. There were 233 subjects (12% of participants) who completed only one or two waves of the study, and were designated 'low completers'. Low completers were compared with other participants in a multiple logistic regression analysis.

Males were over-represented in comparison to females [odds ratio (OR) 2.1, 95% confidence interval (CI) 1.6, 2.7], as were Australian-born subjects (OR 2.6, CI 1.5, 4.5), and those who had experienced parental divorce (OR 2.5, CI 1.5, 4.2). Depressive symptoms at entry (OR 1.6, CI 0.8, 3.2) and school location (metropolitan vs non-metropolitan) (OR 1.0, CI 0.6, 1.7) did not differ greatly between high and low completers.

Phase 2 responses

In all, 145 subjects across waves 2–6 fulfilled CIS-R criteria for depression and were selected for second phase interview. One hundred and thirty (90%) completed second phase assessment. Of these, 11 had been a CIS-R case at a previous wave and not confirmed at the time as a CIDI case, leaving a total of 119 first phase (CIS-R) cases. Two hundred and ninety subjects were selected from the CIS-R non-case stratum, of whom 276 (95%) participated at the second phase. Reasons for second phase non-participation were absenteeism or persistent interview non-attendance ($n=22$), parental refusal ($n=5$) and premature termination of interview ($n=2$).

Ninety-three subjects received a diagnosis of depressive disorder on the CIDI. Of these, 69 (74%) were also (first phase) CIS-R cases at the same wave and designated Definite Depressive Disorder. Fifty-nine (86%) of these cases were female (5.9% of first phase female participants) and ten (14%) were male (1.1% of first phase participants). Twenty-five (36%) reported one or more previous depressive episodes. The maximal episode severity according to ICD-10 rating was severe in 16 (23%), moderate in 32 (46%) and mild in 21 (30%).

Parental care and control

The PBI was completed for mothers by 396 (98%) of the second phase participants and for fathers by 390 (96%) of subjects. Reasons for non-completion were parental death (maternal PBI, $n=1$; paternal PBI, $n=3$), no contact with that parent in the previous 5 years (maternal PBI, $n=4$; paternal PBI, $n=7$) or incomplete forms (maternal PBI, $n=5$; paternal PBI, $n=6$).

Mean PBI scores for care and control were compared with each other and by gender (Table 1). Mean parental care was similar in males and females. Maternal care was 3.4 points higher than paternal care for both males and females. Little difference was found in levels of maternal and paternal control, but both were over two points higher in females than males. Positive associations were found between maternal and paternal care (Pearson correlation 0.49) and between maternal and paternal control (0.56). Negative associations between care and control were found for reports on mothers (-0.51) and fathers (-0.41).

Parental divorce or separation was associated with

Table 1 Mean scores^a (95 % confidence intervals) on the Parental Bonding Instrument (PBI) in participants in a two-phase study of adolescent depressive disorder

	Males (n=138 ^b)	Females (n=250 ^b)	Difference (male-female)
Maternal care	27.5 (26.4, 28.6)	27.1 (26.0, 28.2)	0.3 (-1.2, 1.8)
Paternal care	24.1 (22.7, 25.5)	23.7 (22.2, 25.2)	0.3 (-1.7, 2.3)
Difference (maternal-paternal)	3.4 (2.2, 4.5)	3.4 (1.9, 4.9)	
Maternal control	11.3 (9.8, 12.7)	13.2 (12.1, 14.6)	-2.1 (-4.0, -0.1)
Paternal control	10.1 (8.4, 11.8)	12.5 (11.2, 13.8)	-2.4 (-4.5, -0.3)
Difference (maternal-paternal)	1.1 (-0.2, 2.5)	0.8 (-0.3, 2.0)	

^a Calculated using the inverse probability weights designed to approximate the inverse of the sampling fraction at second-phase assessment

^b Nine observations (one male and eight female) were excluded from this analysis as they did not have complete sets of data on all variables

lower paternal care (weighted mean score difference 4.0; CI 0.8, 7.3) and lower maternal care (mean difference 2.6, CI 0.1, 5.1). There was also a suggestion of greater paternal control (mean difference 2.8, CI -0.2, 5.9) in the group with separated parents, but maternal control did not differ.

■ Parenting and depressive disorder

A series of multivariable analyses were undertaken to examine associations of maternal and paternal parental style with depressive disorder. Quartiles were used to categorise the parental style scales with the midpoints adjusted to incorporate the cutoffs recommended by Parker [8]. The highest quartile for the care scores and the lowest quartile for control were used as comparison categories. All analyses were weighted and adjusted for the potential confounders of sex, age, place of birth and parental separation or divorce.

Parental care and depression (Table 2)

CIS-R depressive episodes. Subjects reporting the lowest level of maternal care had a four-fold elevation in risk for depressive disorder. Low paternal care was associated with a more than two-fold elevation in risk.

Definite (CIS-R & CIDI) depressive disorder. Subjects in the lowest level (quartile) of maternal care had a more than four-fold increase in depressive disorder. Those in the lowest category of paternal care had an almost three-fold increase in risk. For both maternal and paternal care, risks for depressive disorder did not increase markedly until well below the cutoffs recommended by Parker and used in much of the previous literature. No second-order interactions were found.

Parental control and depression (Table 3)

CIS-R depressive episodes. The clearest indication of an association with parental control was found for the

Table 2 Associations with parental care in a two-phase study of ICD-10 depressive disorder in a population-based sample of older adolescents: odds ratios (OR) from multivariable logistic regression models

	First phase CIS-R disorder (n=391 ^a) OR (95 % CI)	Definite (CIS-R & CIDI) disorder (n=382 ^b) OR (95 % CI)
Age	1.5 (1.0, 2.3)	1.6 (1.0, 2.6)
Sex (female vs male)	3.8 (2.0, 7.2)	7.7 (3.2, 19)
Parental divorce	1.4 (0.69, 2.8)	1.9 (0.76, 4.6)
Australian birth	0.82 (0.35, 1.9)	1.8 (0.59, 5.4)
Maternal care		
Highest quartile 32–36	1	1
28–31	1.2 (0.46, 3.0)	1.5 (0.45, 5.1)
24–27	1.2 (0.42, 3.3)	0.88 (0.25, 3.1)
Lowest quartile 0–23	4.1 (1.6, 10)	4.8 (1.7, 14)
Paternal care		
Highest quartile 30–36	1	1
25–29	1.6 (0.63, 4.1)	0.73 (0.20, 2.6)
20–24	0.72 (0.26, 2.0)	0.70 (1.1, 7.3)
Lowest quartile 0–19	2.4 (1.0, 5.8)	2.9 (1.1, 7.3)

^a Fifteen CIS-R cases also appeared in subsequent waves and were not included in this analysis

^b Twenty-four CIDI cases who were not CIS-R cases were excluded from this analysis

Table 3 Associations with parental control in a two-phase study of ICD-10 depressive disorder in a population-based sample of older adolescents: odds ratios from multivariable logistic regression models

	First phase CIS-R disorder (n=391 ^a) OR (95 % CI)	Definite (CIS-R & CIDI) disorder (n=366 ^b) OR (95 % CI)
Age	1.2 (0.98, 1.3)	1.2 (0.78, 1.8)
Sex (female vs male)	3.0 (1.7, 5.4)	5.6 (2.5, 13)
Parental divorce	1.5 (0.79, 3.0)	2.2 (1.0, 5.1)
Australian birth	0.74 (0.35, 1.6)	1.6 (0.53, 4.9)
Maternal care		
Highest quartile 32–36	1	1
28–31	1.5 (0.65, 3.7)	1.2 (0.42, 3.6)
24–27	1.5 (0.58, 3.7)	0.94 (0.27, 3.3)
Lowest quartile 0–23	1.8 (0.70, 4.8)	2.2 (0.61, 7.9)
Paternal care		
Highest quartile 30–36	1	1
25–29	1.4 (0.62, 3.4)	2.9 (0.87, 9.4)
20–24	1.6 (0.63, 3.9)	2.9 (0.76, 11)
Lowest quartile 0–19	2.3 (0.90, 5.8)	4.9 (1.2, 20)

^a Fifteen CIS-R cases also appeared in subsequent waves and were not included in this analysis

^b Twenty-four CIDI cases who were not CIS-R cases were not included from this analysis

highest level of paternal control, where risks for disorder were more than two-fold higher.

Definite (CIS-R & CIDI) depressive disorder. Those with the highest level of maternal control had a two-fold higher risk for confirmed depressive disorder and those with the highest level of paternal control an almost five-fold elevation in risk.

Parental care, control and depressive disorder

The independent effects of parental care and control were examined in two further models examining CIS-R

Table 4 Associations between ICD-10 depressive episodes and low parental care and high parental control in a two-phase study of adolescent depressive disorder: odds ratios from multivariable logistic regression models

	First phase CIS-R disorder ($n=391^a$) OR (95 % CI)	Definite (CIS-R & CIDI) disorder ($n=366^b$) OR (95 % CI)
Age	1.5 (0.99, 2.2)	1.6 (1.0, 2.5)
Sex (female vs male)	3.8 (1.9, 7.3)	7.7 (3.1, 19)
Australian birth	0.7 (0.32, 1.6)	1.7 (0.58, 5.0)
Parental divorce	1.3 (0.6, 2.6)	2.1 (0.9, 4.7)
Low maternal care	3.2 (1.8, 5.8)	3.2 (1.4, 6.9)
Low paternal care	2.5 (1.4, 4.4)	3.4 (1.7, 7.0)
Maternal control	0.9 (0.4, 2.0)	1.3 (0.5, 3.5)
Paternal control	1.3 (0.6, 2.6)	1.5 (0.58, 3.9)

^a Fifteen CIS-R cases also appeared in subsequent waves and were not included in this analysis

^b Twenty-four CIDI cases who were not CIS-R cases were excluded from this analysis

depressive episodes and definite disorder (CIS-R & CIDI) respectively (Table 4). In the preceding analyses, the clearest associations were found with the lowest quartiles of care and highest quartiles of control. Therefore, binary variables were generated using these quartile cut-points.

CIS-R depressive episode. After adjustment for maternal and paternal care no independent association was found with either maternal or paternal control. The clearest association was with low maternal care, where rates of a depressive episode were three-fold higher.

Definite (CIS-R & CIDI) Depressive Disorder. Associations with both low paternal and maternal care were found in this model. Both low maternal and low paternal care were associated with three-fold higher rates of disorder. No independent association with either maternal or paternal control was found.

Discussion

On many counts, this study is consistent with previous work in older subjects. Associations with low parental care were found in those receiving a first phase diagnosis and those with Definite Depressive Disorder confirmed on the CIDI. High maternal and paternal control were also associated with depression on each definition. Also consistent with previous work, the independent effects of low parental care outweighed associations with high parental control. After adjustment for low care, the residual associations with parental control were small.

In other respects, this study breaks new ground. Associations with parental style held for both parents, with both low maternal and paternal care having similar independent risks for depression. The effects of parental care and control were most marked at the extremes, indicating that the threshold for sub-optimal parenting is below that previously used, at least in relation to risks for adolescent disorder [8]. Most importantly, it is clear that the associations with sub-optimal parenting hold early

in the course of depressive disorder – a finding consistent with an effect on onset rather than course of disorder alone.

‘Affectionless control’ has been inconsistently linked with depression in earlier studies in non-clinical samples, and some have failed to find an association with parental control independent of low care [7, 14]. Uncertainty about methodology (e.g. limited numbers of PBI items [7] or the study of less representative samples [14]) may have contributed to earlier negative findings, but it is also possible that parental control may have a clearer effect during adolescence, when conflicts around autonomy and independence are marked.

Few data have been reported previously on parental style and adolescent depression. Cubis et al. [27] found an association between low paternal care and high scores on the 12-item General Health Questionnaire in intact families in a community-based survey of adolescents. Rey [28] found low maternal care was associated with depression when depressed patients were compared with other patients in a small outpatients series. However, non-depressed psychiatric outpatients are a group where parenting is also likely to be sub-optimal, leading to uncertainty about the size of such an effect.

The main limitation of this current study is that parental bonding was not measured prospectively, and there is a possibility that depressed mood influenced reporting. Some reports have found that PBI scores are stable after recovery from an episode of depression [15, 29, 30], but others have found that subjects are more likely to report parents as less caring and more controlling when they are in a depressed state than when recovered [31]. This may be particularly so for an adolescent experiencing current depression. Therefore, it remains possible that the associations observed here are in part an artefact of the depressed state. A second potential limitation arises from differential participation. Although participation in both study phases was high, males and adolescents with divorced parents – both factors related to depressive disorder – had low phase 1 participation rates. The multiple waves of data collection should have done much to ensure the participation of these groups, but it remains possible that these characteristics were under-represented in the second phase.

An explanation for the associations between report of sub-optimal parenting and later adult depression has been that low care predisposes to insecure or remote attachment styles, and thence to both diminished interpersonal competence and unsatisfactory adult relationships [13, 32]. One study demonstrated no residual effect of early parenting on adult depressive symptoms after adjustment for adult social relationships – a pattern consistent with a mediating role for the latter [13, 33]. However, the current study raises a possibility that adolescent depression itself is the mediator. Poor adult social relationships may be a consequence of recurrent depression from an early age, as has been suggested in work on the effect of early onset psychiatric disorder on marital stability [34]. This has some similarity with the

suggestion of Bifulco et al. [35] that the development of adolescent depression is an important mediating step in the relationship between childhood risk factor of sexual abuse and later adult depressive disorder. This possibility has been acknowledged by Rodgers [12] and other researchers [36, 37], and the strength of associations found in the current study gives further weight to the consideration. Whether sub-optimal parenting is associated with depression with an onset outside the adolescent years has yet to be clarified.

■ **Acknowledgement** The authors acknowledge the support of the Victorian Health Promotion Foundation and the Australian Rotary Health Research Fund. They would also like to thank Professor Anthony Mann, Professor Gordon Parker, Dr Bryan Rodgers and Ms Celia Godfrey for their comments on an earlier draft of the paper.

References

- Granville-Grossman KL (1968) The early environment in affective disorders. In: Coppen A, Walk A (ed) *Recent advances in affective disorders*. London: Royal Medico-Psychological Association, pp 65–79
- Rodgers B (1996) Reported parental behaviour and adult affective symptoms. 1. Associations and moderating factors. *Psychol Med* 26: 51–61
- Rodgers B (1994) Pathways between parental divorce and adult depression. *J Child Psychol Psychiatry* 35: 1289–1308
- Parker G (1983) Parental 'affectionless control' as an antecedent to adult depression. *Arch Gen Psychiatry* 40: 956–960
- Bifulco AT, Brown GW, Harris TO (1987) Childhood loss of parent, lack of adequate parental care and adult depression: a replication. *J Affect Disord* 12: 115–128
- Parker G, Hadzi-Pavlovic D (1999) Parental representations of melancholic and non-melancholic depressives: examining for specificity to depressive type and for evidence of additive effects. *Psychol Med* 22: 657–665
- Parker G, Hadzi-Pavlovic D, Greenwald S, Weissman M (1995) Low parental care as a risk factor to lifetime depression in a community sample. *J Affect Disord* 33: 173–180
- Parker G, Tupling H, Brown LB (1979) A parental bonding instrument. *Br J Med Psychol* 51: 1–10
- Hinde RA (1979) *Towards understanding relationships*. 1st edn. London: Academic Press
- Parker G (1983) Parental overprotection. A risk factor in psychosocial development. 1st edn. New York: Grune & Stratton
- Parker G (1993) Parental rearing style: examining for links with personality vulnerability factors for depression. *Soc Psychiatry Psychiatr Epidemiol* 28: 97–100
- Rodgers B (1996) Reported parental behaviour and adult affective symptoms. 2. Mediating factors. *Psychol Med* 26: 63–77
- Parker G, Barrett EA, Hickie IB (1992) From nurture to network: examining links between perceptions of parenting received in childhood and social bonds in adulthood. *Am J Psychiatry* 149: 877–885
- Mackinnon A, Henderson AS, Andrews G (1993) Parental 'affectionless control' as an antecedent to adult depression: a risk factor refined. *Psychol Med* 23: 135–141
- Gotlib IH, Mount JH, Cordy NI, Whiffen VE (1988) Depression and perceptions of early parenting: a longitudinal investigation. *Br J Psychiatry* 152: 24–27
- Garrison CZ, Addy CL, Jackson KL, McKeown RE, Waller JL (1992) Major depressive disorder and dysthymia in young adolescents. *Am J Epidemiol* 135: 792–802
- Oldehinkel AJ, Wittchen HU, Schuster P (1999) Prevalence, 20-month incidence and outcome of unipolar disorders in a community sample of adolescents. *Psychol Med* 29: 655–668
- Australian Bureau of Statistics (1991) *Census of Population and Housing 6 August 1991, Australia in Profile*. Canberra: Commonwealth of Australia
- Mann AH, Wakeling A, Wood K, Monck E, Dibbs R, Szmukler G (1983) Screening for abnormal eating attitudes and psychiatric morbidity in an unselected population of 15-year-old school-girls. *Psychol Med* 13: 573–580
- Lewis G, Pelosi AJ (1992) *The manual of CIS-R*. London: Institute of Psychiatry, Ref Type: Report
- Patton GC, Coffey C, Posterino M, Carlin JB, Wolfe R, Bowes G (1999) A computerised screening instrument for adolescent depression: a population-based validation and application to a two-phase case control study. *Soc Psychiatry Psychiatr Epidemiol* 34: 166–172
- Robins L, Wing J, Wittchen HU, Helzer JE, Babor T, Burke J, et al. (1988) The Composite International Diagnostic Interview. *Arch Gen Psychiatry* 45: 1069–1077
- WHO (1993) *The ICD-10. Classification of Mental and Behavioural Disorders*. Geneva: World Health Organisation
- Stata (1997) *Texas: Stata Corporation*
- Carlin JB, Wolfe R, Coffey C, Patton GC (1999) Analysis of binary outcomes in longitudinal studies using weighted estimating equations and discrete-time survival methods: prevalence and incidence of smoking in an adolescent cohort. *Stat Med* 18: 2655–2679
- Australian Bureau of Statistics (1993) *Australia's Young People*. Canberra: Australian Government Publishing Service
- Cubis J, Lewin T, Dawes F (1989) Australian adolescents' perceptions of their parents. *Aust N Z J Psychiatry* 23: 35–47
- Rey JM (1999) Perceptions of poor maternal care are associated with adolescent depression. *J Affect Disord* 34: 95–100
- Parker G (1981) Parental reports of depressives. An investigation of several explanations. *J Affect Disord* 3: 131–140
- Duggan C, Sham P, Minne C, Lee A, Murray R (1998) Quality of parenting and vulnerability to depression: results from a family study. *Psychol Med* 28: 185–191
- Lewinsohn P, Rosenbaum M (1987) Recall of parental behaviour by acute depressives, remitted depressives, and non-depressives. *J Pers Soc Psychol* 52: 611–619
- Bowlby J (1977) The making and breaking of affectional bonds. *Br J Psychiatry* 130: 201–210
- Gittleman MG, Klein MH, Smider NA, Esses MJ (1998) Recollections of parental behaviour, adult attachment and mental health: mediating and moderating effects. *Psychol Med* 28: 1443–1455
- Kessler RC, Walters EE, Forthofer MS (1998) The Social consequences of psychiatric disorders. III. Probability of marital stability. *Am J Psychiatry* 155: 1092–1096
- Bifulco A, Brown GW, Moran P, Ball C, Campbell C (1998) Predicting depression in women: the role of past and present vulnerability. *Psychol Med* 28: 39–50
- Kessler RC, Magee WJ (1993) Childhood adversities and adult depression: basic patterns of association in a US national population. *Psychol Med* 23: 679–690
- Kendler KS, Kessler RC, Neale MC, Heath AC, Eaves LJ (1993) The prediction of major depression in women: toward and integrated etiologic model. *Am J Psychiatry* 150: 1139–1148