Psychological Medicine, 1998, **28**, 737–742. Printed in the United Kingdom

BRIEF COMMUNICATION

Dysfunctional parenting as a risk factor to lifetime depression in a sample of employed Japanese adults: evidence for the 'affectionless control' hypothesis

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ABSTRACT

Background. Although many case–control studies have replicated an association between dysfunctional parenting and a lifetime diagnosis of depression, few epidemiological studies have explored the association. In addition, little is known about the association in non-western countries.

Methods. Using logistic regression analyses, additive and interactive contributions of parental child-rearing behaviours, as measured by the Parental Bonding Instrument (PBI), toward the risk for having a lifetime diagnosis of major depressive disorder were explored in 418 employed Japanese adults. The diagnosis was provided by using the Inventory to Diagnose Depression, lifetime version. The analyses were conducted for male and female subjects separately.

Results. Parental care rather than parental protection was primary in predicting lifetime depression in both male and female subjects. An interactive combination of low care and high protection ('affectionless control') was a significant risk factor for lifetime depression in male respondents' reporting child-rearing behaviours of both parents and female respondents' reporting paternal child-rearing behaviours. Model improvements when entering the PBI scores were larger in male subjects than in female subjects.

Conclusions. The results suggested that a combination of low care and over-protection increases a risk to lifetime depression even in a non-clinical sample; that an association between dysfunctional parenting (particularly low care) and the development of depression is independent of culture; and that Japanese boys are more sensitive than Japanese girls to dysfunctional parenting as regards the development of depression.

INTRODUCTION

Since the first report by Parker (1979), a large body of literature has grown to support the view that dysfunctional parenting, as measured by the Parental Bonding Instrument (PBI: Parker *et al*. 1979), is a risk factor for the development of depressive disorders in adulthood. Case–control studies (Parker, 1983; Parker *et al*. 1987; Plantes *et al*. 1988; Parker & Hadzi-Pavlovic, 1992; Rodriguez *et al*. 1993; Rey, 1995; Sato *et al*. 1997) repeatedly show that subjects with depressive disorders, in particular, those with non-melancholic or reactive depression, reported their parents as both less caring and more over-protective on the PBI, while a lower care score was more critical than a higher protection score in discriminating depressive subjects from controls. The findings appeared independent of depression level (Parker, 1981) and a trait characteristic such as neuroticism (Parker, 1979),

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suggesting that the association is not due to response biases and may reflect a causal linkage. Several researchers (Parker, 1979; 1983; Parker *et al.* 1987; 1992; Plantes *et al.* 1988; Sato *et al.* 1997) were interested in whether a combination of care and protection increased a risk for depressive disorders, dichotomizing the PBI care and protection scales at their cut-off points and assigning parents to one of four PBI quadrants: 'affectionless control' (low care–high protection), 'neglectful parenting' (low care–low protection), 'affectionate constraint' (high care–high protection) and 'optimal bonding' ('high care–low protection). Among the four quadrants, 'affectionless control' presented the highest risk for non-melancholic depression and 'optimal bonding' the lowest.

Findings from the case–control studies have been replicated in part in non-clinical subjects (Mackinnon *et al.* 1989; 1993; Kerver *et al.* 1992; Oakley-Browne *et al.* 1995; Parker *et al.* 1995; Rodgers, 1996*a*, *b*). These studies have evaluated depression in two different manners: measuring a current level of depression and diagnosing a lifetime history of depression. As pointed out in Mackinnon *et al.*'s study (1993), diagnosing a lifetime history of depression is more appropriate for searching the association. To our knowledge, only two studies have so far explored an association between the PBI scorings and a lifetime history of depression in a large non-clinical sample. Parker *et al.* (1995) found that low parental care is a significant risk factor for a lifetime history of major depressive disorder. Mackinnon *et al.* (1993) showed that parental low care alone was primary in the risk for having lifetime depression and that effects of combining low care and high protection was not significant.

The present study investigates dysfunctional parenting, as measured by the PBI, as a risk factor for a lifetime diagnosis of depression in a non-clinical sample of 418 employed Japanese adults. Additive and interaction effects of care and protection are explored for male and female subjects separately. The study provides some evidence for the 'affectionless' hypothesis in non-clinical subjects reared in non-western culture and some differences in the effects of dysfunctional parenting between male and female subjects.

METHOD

Instruments

The Japanese version of the PBI, which was developed and validated by Kitamura & Suzuki, (1993), was used. The Inventory to Diagnose Depression, lifetime version (IDDL: Zimmerman & Coryell, 1987) provided lifetime diagnoses of depression in this study. The IDDL is a highly structured self-report questionnaire that enables researchers to diagnose a lifetime history of major depressive disorder. It contains 22 questions about broad depressive symptoms that respondents answer as they consider their most profound depression; they indicate if they experienced these depressive symptoms and if the duration was more than 2 weeks. The IDDL was originally developed for diagnosing DSM-III (American Psychiatric Association (APA), 1980) major depressive disorder; however, the broad depressive symptoms included in the instrument make it possible to utilize it to diagnose DSM-III-R (APA, 1987), and DSM-IV (APA, 1995) major depressive disorder. Zimmerman & Coryell (1987) reported that the lifetime diagnosis of DSM-III major depressive disorder by the IDDL has validity (kappa = 0·60) when compared to the Diagnostic Interview Schedule (DIS). This kappa was higher than that for the inter-rater reliability of the DIS in diagnosing a lifetime history of DSM-III major depressive disorder (kappa = 0·50), suggesting that lifetime diagnoses of major depressive disorder provided by the IDDL are not less reliable or less valid than those provided by a structured interview method. The Japanese version of the IDDL was developed and validated by us (Uehara *et al.* 1995; Sakado *et al.* 1996). In the present study, we used the IDDL to diagnose DSM-IV major depressive disorder.

Sample

Five hundred and sixty-two individuals, all workers except those at managerial levels in three medium-scale companies in Niigata City area were asked to fill out the PBI and the IDDL anonymously together with some questions on their age, education, and income. Explanation of the hypothesis that dysfunctional parenting may cause adult depression was not given to the respondents. Their income ranged from 2 500 000

to 6 500 000 yen, placing them in the middle class in Japan. Four hundred and twenty-five (76%) participated and completed all instruments. Since mean PBI scores of male and female subjects were not significantly different among the three companies in univariate analyses separating out effects of age and education (analyses of variance), all data were combined and treated together. Of the 425 subjects, seven (two men and five women) rated only one parent on the PBI due to the death of or lack of contact with the other parent. These subjects were excluded from the analysis, leaving 418 subjects. The final sample consisted of 197 men (47%) and 221 women (53%) with a mean age of 39·2 (S.D. = 10·8; range = 18–66) years. Mean education was 13·7 (S.D. = 2·0; range = 9–20) years.

Forty-six (11%) workers were diagnosed by the IDDL as having had a lifetime history of DSM-IV major depressive episode. These subjects were 12 men (26%) and 34 women (74%), and their mean age and mean education were 41·7 (S.D. = 11·3; range = 19–65) and 13·1 (S.D. = 2·0; range 9–16) years, respectively. There were significant differences in sex ratio and education between subjects with and without a history of depression (percentage of females, 74 *v.* 50%, $\chi^2 = 9{\cdot}2$, df = 1, $P = 0{\cdot}002$, two-tailed; education, 13·1 (S.D. = 2·0) *v.* 13·8 (S.D. = 2·0), $t = 2{\cdot}1$, df = 416, $P = 0{\cdot}035$, two-tailed). Age was not significantly different between the two groups (41·7 (S.D. = 11·3) *v.* 38·9 (S.D. = 10·8), $t = 1{\cdot}7$, $P = 0{\cdot}09$, two-tailed). Depressive episodes as reported in the IDDL had occurred when the subjects were 20–55 years old.

Analysis

Logistic regression analyses were principally used to estimate contributions of dysfunctional parenting toward a risk for having a lifetime depressive episode. Additive and interaction effects of care and protection were examined for male and female respondents separately with partialling out effects of age and education. We used a computer package, SPSS for MACINTOSH (SPSS Japan, 1995). The significance of odds ratios was estimated by confirming that their confidence intervals did not include 1·0.

RESULTS

Mean PBI scores (S.D.) in the whole sample were 24·5 (7·1) for paternal care; 9·8 (5·8) for paternal protection; 28·3 (6·3) for maternal care; and 9·3 (5·3) for maternal protection. Care and protection were highly correlated: −0·489 between paternal care and paternal protection; −0·529 between maternal care and maternal protection.

Dimensional scores on care and protection were entered sequentially into logistic regression analyses in this order and then in the reverse order. For estimating interaction effects, a variable was created by multiplying protection with lack of care. This variable has the highest value for a combination of low care and high protection ('affectionless control') and the lowest for a combination of high care and low protection ('optimal bonding'). Table 1 shows the results of the analyses. Odds ratios were calculated for a 5-point lower care score and a 5-point higher protection score.

At first, parental care was entered (step '1A'), showing a significant improvement in the fit. Analyses for male subjects demonstrated larger model improvements than those for female subjects, resulting in larger odds ratios of care in male respondents (range: 2·49–2·85 in male subjects; and 1·31–1·39 in female subjects). Into the model already including care, parental protection was then entered (step '1B'), but no significant model improvement was observed. No odds ratio for protection was significant in the models including both care and protection.

Next, parental protection was entered prior to care (step '2A'). A significant improvement in the fit was observed only in cases when entering maternal protection in male subjects and paternal protection in female subjects. After protection was already included, parental care was then added (step '2B'). This step always produced a significant model improvement. In the models including both protection and care, all odds ratios for care were significant.

These results can be summarized as follows: entering parental care always produced a significant improvement in the fit, whether or not parental protection had already been included. Parental protection did not significantly improve the model fit when care was already included, and significant model improvements were ob-

Table 1. *Improvements in the fit of logistic models associated with adding PBI scores sequentially*

Step	Variables added	Male respondents χ^2 (df)	Male respondents Odds ratio	Female respondents χ^2 (df)	Female respondents Odds ratio
Paternal PBI Scores					
1A	PC	16·3 (1)***	2·49***	6·3 (1)*	1·39*
1B	PO after PC	1·2 (1)	0·75	1·6 (1)	1·27
2A	PO	0·5 (1)	1·18	3·9 (1)*	1·49*
2B	PC after PO	17·0 (1)***	2·76***	4·0 (1)*	1·46*
3A	PC × PO	3·9 (1)*	1·09*	4·7 (1)*	1·07*
Maternal PBI Scores					
1A	MC	16·8 (1)***	2·85***	4·3 (1)*	1·31*
1B	MO after MC	0·4 (1)	1·14	0·4 (1)	0·88
2A	MO	4·0 (1)*	1·54*	0·4 (1)	1·11
2B	MC after MO	13·2 (1)***	2·74***	4·3 (1)*	1·39*
3A	MC × MO	7·6 (1)**	1·18**	2·0 (1)	1·05

PC , Paternal Care; PO, Paternal Over-protection; MC, Maternal Care; MO, Maternal Over-protection.

× in the column indicating logistic models means an interaction effect.

Model improvements were estimated by the reduction in $-2 \times$ (log likelihood), which is distributed as χ^2.

Odds ratios were calculated as a risk for having a lifetime history of depression. The calculations were made for a 5-point lower care score or a 5-point higher protection score.

All analyses were conducted after separating out effects of age and education.

*, $P < 0{\cdot}05$; **, $P < 0{\cdot}01$, ***, $P < 0{\cdot}001$.

served only occasionally when protection was first entered, suggesting that parental care played a more critical role than did parental protection in increasing a risk for lifetime depression. Given high correlation coefficients between care and protection scores, the significance seen when entering protection first did not appear to reflect a unique contribution of protection, but was suggested to be simply produced by the primary role of care in predicting lifetime depression. The models including both care and protection (models at '1B' and '2B') fit data significantly better than did the models with protection alone, but not better than did the models with care alone, suggesting that there were no significant additive effects of care and protection.

Table 1 also shows the results of estimating interaction effects of care and protection in each parent. Significant effects were detected in both parents' PBI scores of male subjects and in paternal PBI scores of female subjects. Odds ratios of the significant interaction effects were larger than 1·0, indicating that 'affectionless control' increased a risk for lifetime depression when either parent of male subjects or a father of female subjects was allocated into the PBI quadrant.

DISCUSSION

This study examined the role of dysfunctional parenting, as measured by the PBI, in a lifetime history of major depressive disorder in a non-clinical sample of employed Japanese adults. The results showed that parental care rather than parental protection was primary in increasing a risk for lifetime depression in male and female subjects, and that an interactive combination of low care and over-protection ('affectionless control') increased such a risk when male respondents reported child-rearing behaviours of both parents and female respondents reported paternal child-rearing behaviours.

Since Parker (1979) originally proposed the 'affectionless control' hypothesis, many case–control studies (Parker, 1983; Parker & Hadzi-Pavlovic, 1992; Parker *et al.* 1987; Plantes *et al.* 1988) have supported the hypothesis, while no epidemiological study has found evidence of 'affectionless control' being a significance risk factor for a lifetime history of depression in a non-clinical sample (Mackinnon *et al.* 1993; Parker *et al.* 1995). Some researchers (Mackinnon *et al.* 1989; Parker *et al.* 1995) have suggested a possibility that depressive subjects' help-seeking behaviours in the case–control studies may account for the significance of 'affectionless control' and that individuals with depressive disorders in general do not necessarily report their parents as being 'affectionless control'. The results in the present study indicate that evidence supporting 'affectionless control' can also be drawn from a non-clinical sample and that help-seeking behaviours of depressive subjects may not be the only explanation of the significance of 'affectionless control' provided by previous case–control studies.

The case–control studies related 'affectionless control' to non-melancholic depression but not melancholic depression, and the subdivision of depressive disorders into melancholic and non-melancholic depression was not adopted in previous non-clinical studies. This may be one more possible explanation of the lack of evidence supporting 'affectionless control' in the non-

https://doi.org/10.1017/S0033291797006430 Published online by Cambridge University Press

clinical studies: that is, exposure to 'affectionless control' from a parent may increase a risk for non-melancholic or reactive depression even in a non-clinical sample, but a methodology treating depressive disorders as an unity may account for no evidence for this in the non-clinical studies. Mackinnon *et al*. (1993) presented some evidence of a significant contribution of high protection toward predicting lifetime dysthymia in a non-clinical sample, somewhat supporting this explanation. Further non-clinical study subdividing depressive disorders into melancholic and non-melancholic depression is needed, and such a study would help us see more clearly whether and how parental 'affectionless control' is related to a risk for depressive disorders in a non-clinical sample.

The weak power of protection in predicting a lifetime history of depression found in both this study and previous studies may be due to the protection dimension of the PBI being flawed. Recent studies (Kendler, 1996; Murphy *et al*. 1997) indicated that the dimension contained two factors. This suggests that an association between dysfunctional parenting and depression ought to be re-evaluated on the basis of the three dimensions constructing the PBI. Such a re-evaluation study may detect a more important role of an element of parental protection in the risk to depressive disorders.

This study associated dysfunctional parenting, as measured by the PBI, to a higher risk for lifetime depression in a Japanese non-clinical sample. The results were similar to those of previous studies – all were conducted in Western culture – showing parental care rather than parental protection as primary in predicting lifetime depression. The results, when considered with those of a case–control study conducted in Japan (Sato *et al*. 1997) together, suggest that an association between dysfunctional parenting (particularly parental low care) and the development of depression may be independent of culture.

In this study, much larger model improvements were observed in male subjects than in female subjects, suggesting that Japanese boys are more sensitive than Japanese girls to dysfunctional parenting as regards the development of depression. In this respect, the results of this study appear significantly different from those of previous Western studies, which found no such sex-dependent difference in respondents (Mackinnon *et al*. 1993). The difference in the results would be clarified by shedding light on how boys and girls are reared in Japan and how parental child-rearing behaviours differ in Japan and Western countries.

Some cautions in interpreting the results in this study should be noted: first, the sample was workers in three companies and only 76% of approached subjects participated in the study, which may have caused a selection bias. Secondly, all instruments used in the study were self-reported. Although the IDDL has enough validity for the epidemiological use, a question may be raised as to whether lifetime diagnoses of depression and assessments of parental child-rearing behaviours were really independent of each other. The results in this study need to be replicated by a study of subjects drawn from a general population in which a lifetime depression is, independent of assessments of parental child-rearing behaviours, diagnosed by using a structured interview method. Thirdly, this study did not explore lifetime diagnoses of mental disorders other than major depressive disorder. Parker *et al*. (1995) demonstrated that an association between dysfunctional parenting and a lifetime diagnosis of major depressive disorder was weakened when influences of mental disorders coexisting with major depressive disorder were removed. The results in this study may have been strengthened by co-morbid mental disorders.

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