Parents' childhood experiences of bonding and parental psychopathology predict borderline personality disorder during adolescence in offspring

Maria Rita Infurna⁎, Anna Fuchs⁎, Gloria Fischer-Waldschmidt⁎, Corinna Reichl⁎, Birger Holz, Franz Resch, Romuald Brunner, Michael Kaess⁎

⁎ Section for "Translational Psychobiology in Child and Adolescent Psychiatry", Department of Child and Adolescent Psychiatry, University of Heidelberg, Heidelberg, Germany

Abstract

Previous studies on borderline personality disorder (BPD) development suggest a transgenerational transmission of parent-child relationship quality, which may also be influenced by parents' mental health status. The aim of this study was twofold. First, we aimed to investigate the transgenerational effect of parental bonding experiences on the development of BPD in their offspring. Second, we examined the association between parents’ mental health status and BPD in offspring. Ninety-one female adolescent psychiatric inpatients along with 87 mothers and 59 fathers were enrolled in the study. Adolescent BPD was assessed with the Structured Clinical Interview for DSM-IV-II, parental bonding with the Parental Bonding Instrument, and parents’ psychiatric symptoms with the Patient Health Questionnaire. We found that low parental care produced a transgenerational effect from mother to BPD in offspring. Further, significant associations were found between paternal psychiatric symptoms and adolescent BPD. High paternal stress levels mediated the association between maternal affect reported by fathers and BPD in daughters. There is evidence of a transgenerational effect of parental bonding specifically for female adolescents with BPD, compared with other clinical control subjects. Our findings highlight the importance of including both parents in future research and in early clinical treatment in adolescents with BPD.

1. Introduction

Borderline personality disorder (BPD) usually emerges during adolescence or early adulthood and is associated with severe impairment of psychosocial functioning and with several comorbid conditions, along with a suicide rate of about 8–10% (Chanen et al., 2008; Leichsenring et al., 2011). The core domains of BPD are marked by impulsivity and instability of relationships, affects, and self-image (American Psychiatric Association, 2013). BPD has also recently been confirmed as a valid and reliable diagnosis in patients during adolescence (Fonagy et al., 2015; Kaess et al., 2014; Miller et al., 2008).

In addition to genetic influences (Distel et al., 2008), findings from both clinical and community samples have widely highlighted the relevance of family environment as such and severe childhood maltreatment in particular for the development of BPD (Fruzzetti et al., 2005; Infurna et al., 2016; Lyons-Ruth et al., 2013; Winspace et al., 2012). According to attachment theory (Bowlby, 1983), caregivers play a key role in the development of internal models of the relational world. Therefore, parent-child bonding, representing quality of parent-child relationships and parenting, has been suggested to be an important influential factor in the pathogenesis of mental health problems (Long et al., 2015; Otowa et al., 2013; Wiggins et al., 2015). So far, several findings support the association between poor quality of parental bonding and BPD in offspring (Huang et al., 2014; Keinänen et al., 2012; Lyons-Ruth et al., 2011; Zanarini, 2000); however, in order to prevent these adverse outcomes it is important to understand the factors that may influence problematic or impaired parental bonding.

In line with the cycle of violence theory (Widom, 1989), parenting and parent-child relationships are influenced by processes of transgenerational transmission, as parents’ own experiences as a child affect their nurturing practices later. The attachment theory suggests that the parents’ experiences with their own parents may have led to an internal representation of their parents being responsive or, conversely, unresponsive to their needs, and this internal representation might influence the degree of responsiveness these parents are able to show toward their own children (Bowlby, 1988). Evidence from previous
research supports the hypothesis that grandparents’ harsh parenting predicts how parents function later in their own parent-child relationships (Brook et al., 2002; Conger et al., 2003; Möhler et al., 2001). Recently, this association was also confirmed for warm and supportive parenting (Belsky et al., 2005; Chen et al., 2008; Kerr et al., 2009). However, to date, research findings in this field are still controversial and incomplete; indeed, few studies have specifically examined transmission of parental bonding across generations (Miller et al., 1997; Wang et al., 2012). Additionally, to the best of our knowledge, no study has examined these associations in a sample of BPD patients or in particular in adolescent BPD patients, thus leaving the door open for further investigations.

Another important influential factor for BPD development is parents’ mental health status, an aspect also connected with the quality of parenting (Belsky and Jaffe, 2006). A large body of literature suggests that both BPD diagnoses and symptoms accumulate in families (e.g., Silverman et al., 1991; White et al., 2003; Zanarini et al., 2004). Previous studies on familial transmission of BPD have mainly focused on maternal psychopathology, suggesting an association between BPD (diagnosis or symptoms) in mothers and their offspring (Barnow et al., 2013; Reineit et al., 2014; Schuppert et al., 2015; Whalen et al., 2014). Further studies have assessed the mental health status of both parents or close relatives of BPD patients (Belsky et al., 2012; Bradley et al., 2005; Zanarini et al., 2004), revealing a higher incidence of BPD symptoms in parents of BPD patients. To date, few studies have directly examined the mental health status of both the mother and father of BPD patients or assessed their current psychiatric symptoms (e.g., White et al., 2003).

Moreover, previous studies on BPD’s antecedents were conducted mainly by comparing BPD patients with healthy controls (Lyons-Roth et al., 2011; Paris et al., 1994; White et al., 2003); however, a negative parent-child relationship can be considered an important risk factor for the majority of adult psychiatric disorders (Stenberg et al., 2014; Young et al., 2011). Thus, additional studies are needed to verify the specificity of perceived dysfunctional parental bonding as a risk factor for BPD.

The present study aimed to overcome these limitations by examining the association between perceived parental bonding in adolescents with BPD and their parents. Furthermore, we aimed to examine the associations between parents’ mental health status and BPD in offspring. We hypothesized that poor parental bonding reported by adolescents was related to adolescent BPD and that poor parental bonding reported by parents regarding the quality of their own upbringing was associated with BPD in their offspring. Thus, we hypothesized a transgenerational effect of impaired parental bonding affecting BPD in their offspring. Furthermore, we expected the association between parental bonding as reported by parents and BPD in offspring to be mediated by the offspring’s parental bonding as well as by parents’ psychiatric symptoms.

2. Methods

2.1. Procedure and participants

The ethics committee of the Medical Faculty, University of Heidelberg, approved the study protocol. Informed and written consent was obtained from both patients and their parents. Here, 138 female adolescent inpatients were approached at the Clinic of Child and Adolescent Psychiatry at the University Hospital of Heidelberg, Germany (for further information, see Infurna et al., 2016). Among them, 7 patients were excluded due to insufficient knowledge of the German language (n=2), the presence of acute psychotic symptoms (n=3), or an IQ lower than 75 (n=2). Then, patients and both their mothers and fathers were invited to participate in the study and they were included if the patient and at least one parent were willing to participate: 92.31% of mothers took part in our study, whereas 64.84% of fathers took part in the study. Estimation biases due to a potential systematic drop out for fathers were tested. No significant mean differences were found in regard to BPD symptoms between adolescents whose fathers participated in our study and those not having a father or whose fathers were not willing to participate. The final sample comprised 91 female adolescents (Mage=15.57 years; SD=1.36), 87 mothers (Mage=46.22 years; SD=5.50), and 59 fathers (Mage=49.15 years; SD=5.10). Overall, 83 (95.40%) biological mothers, 53 (89.83%) biological fathers, 2 (2.11%) stepfathers, 3 (3.45%) adoptive mothers, 4 (6.78%) adoptive fathers, and 1 foster mother (1.15%) participated in our study. Adolescents reported the parental bonding of the parent they conceived to be the main mother or father figure. At the time of the assessment, fifty-three adolescents (58.2%) were living with both their parents, 28 (30.8%) only with their mother, 3 (3.3%) only with their fathers, and 7 (7.7%) in the context of a residential child and youth service.

Diagnosis of BPD in inpatients was verified by experienced clinicians using structured interviews. Participants were included in the BPD group if they fulfilled at least five diagnostic criteria of BPD according to the Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV; APA, 1994). Forty-four patients (48.4%) met the BPD criteria (BPD group), while 47 patients (51.6%) did not meet DSM-IV criteria for BPD (clinical control group–CC group). The mean number of diagnoses in adolescents with BPD was significantly higher (t(89)=3.651, p<0.001) than in the CC group. The most prevalent comorbid psychiatric conditions diagnosed in the BPD group included: behavioral and emotional disorders with onset usually occurring in childhood and adolescence (P9; e.g. depressive conduct disorder); neurotic, stress-related and somatoform disorders (F4; e.g. adjustment disorders) and affective disorders (F3; e.g. moderate depressive episode). Predominant psychiatric diagnoses in the CC group were: neurotic, stress-related and somatoform disorders (F4) and affective disorders (F3). The CC group presented a significant higher number (p<0.05) of neurotic, stress-related and somatoform disorders diagnoses (F4) than the BPD group. As set by the inclusion criteria, no personality disorder was found in the CC group. Additional information about the diagnostic criteria for BPD and clinical Axis-I diagnoses in both groups are given elsewhere (Infurna et al., 2016).

2.2. Assessment measure

BPD was assessed by experienced clinical psychologists and child psychiatrists in the field of adolescent BPD, using the German version (Fydrich et al., 1997) of the Structured Clinical Interview for DSM-IV Personality Disorders (SCID-II; First et al., 1997). Assessors were rigorously trained and have previously demonstrated excellent inter-rater reliability of Cohen’s kappa of 1.00 (Kaess et al., 2013). Other clinical diagnoses according to the International Statistical Classification of Diseases and Related Health Problems, 10th revision (ICD-10), diagnostic criteria were ascertained by a well-established procedure of consensus between two child psychiatrists.

The German version (Richter-Appelt et al., 2004) of the Parental Bonding Instrument (PBI; Parker et al., 1979) was used to retrospectively assess two theoretically and empirically derived dimensions of parental bonding: parental affection and parental control. Each participant responded to the questionnaire twice on a 4-point Likert scale (0–3), once to describe the mother’s care and protection and again to describe that of the father. The 12 items on the care subscale allow for a maximum score of 36, indicating parental affection, understanding, closeness, and emotional support, and a minimum score of 0, indicating parental coldness, indifference, and emotional rejection. On the overprotection subscale, a maximum score of 39 is indicative of parental intrusiveness, control, and prevention of independent behavior, while a minimum score of 0 suggests encouragement of autonomy and independence. In this study, the PBI was administered to both patients and their parents.
The German version (Löwe et al., 2003) of the Patient Health Questionnaire (PHQ–Spitzer et al., 1999) was used to assess current psychiatric symptoms in the parents. For our purposes, we employed the full version of the PHQ, which contains 78 items divided in three main subscales: depression, somatic symptoms, and stress. The “depression” subscale assesses the presence and the severity of depressive symptoms. The “somatic symptoms” subscale includes 15 somatic symptoms, representing both the 15 most common physical symptoms of outpatients as well as the major DSM-IV criteria for somatization disorder. The “stress” subscale checks for psychosocial stress factors that may provide clues to triggering or to sustaining conditions of mental disorder. The PHQ has been extensively validated in various patient populations (Löwe et al., 2004, 2002).

According to a general procedure in our clinic, individuals with signs for potential mental retardation were examined using the German version of the fourth version of the Wechsler Intelligence Scale for Children (Petermann and Petermann, 2008).

### 2.3. Data analysis

Statistical analyses were completed using the Statistical Package for Social Sciences (SPSS; v.22). Descriptive statistics were calculated for both groups. Continuous variables were tested with an independent t-test; categorical variables were analyzed by using chi-squared tests. Univariate logistic regressions were tested for the PBI from both parents’ perception of their maternal bonding on BPD in offspring, using offspring experience of low care from mother or father (accordingly) and parents’ psychiatric symptoms as mediator variables. Bootstrapping estimated indirect point effects and associated 95% confidence intervals. A bias accelerated bootstrapping procedure was chosen as this is considered to be the most powerful approach for detecting statistical mediation (MacKinnon et al., 2007). Bonferroni correction for multiple testing was applied, resulting in a model’s p level of 0.008 (α/n) as indicator of significance.

### 3. Results

Significant differences were observed between BPD and CC groups for recalled parental bonding in all PBI subscales. Results showed that, compared to CC patients, BPD patients reported lower care from both mother (M=18.57 vs. M=28.11, t=5.15, p < 0.001) and father (M=15.93 vs. M=25.27, t=4.22, p < 0.001). Furthermore, BPD patients reported higher levels of overcontrol and intrusiveness from both mother (M=16.30 vs. M=10.28, t=−4.11, p < 0.001) and father (M=13.75 vs. M=9.71, t=−2.57, p=0.012) than CC patients.

When looking at parents’ recalled parental bonding, findings revealed that both mothers and fathers of BPD patients themselves reported inadequate levels of affection, warmth, empathy, and closeness from their parents. In particular, mothers of BPD patients reported a lower level of care from their mothers (M=18.48 vs. M=24.02, t=2.58, p=0.012) than mothers of CC patients did. Similarly, fathers of BPD patients reported a lower level of care by their mother (M=20.71 vs. M=27.19, t=2.94, p=0.005) than fathers of CC patients did. Univariate regression showed that a low level of motherly care reported by both mother and father of the patients was significantly associated with BPD outcome in offspring (see Table 1).

With respect to parents’ current mental health status, no significant differences were found between the mothers of adolescent BPD and CC patients. In contrast, fathers of BPD patients reported a higher level of psychiatric symptoms than fathers of CC patients. In particular, depression (M=7.67 vs. M=3.00, t=−4.15, p < 0.001) and psychosocial stress (M=7.70 vs. M=3.73, t=−3.85, p=0.001) were significantly more present in BPD patients’ fathers than in CC patients’ fathers. Furthermore, high levels of paternal depression and psychosocial stress were significantly associated with BPD in offspring (see ORs in Table 1).

Low maternal care in both parents predicted BPD in offspring. In order to analyze potential mediators of this transgenerational effect, six separated mediation models were explored. On the basis of the previous results, selected mediators were the offspring’s perception of their maternal bonding on BPD in offspring, using offspring experience of low care from mother or father (accordingly) and parents’ psychiatric symptoms as mediator variables. Bootstrapping estimated indirect point effects and associated 95% confidence intervals. A bias accelerated bootstrapping procedure was chosen as this is considered to be the most powerful approach for detecting statistical mediation (MacKinnon et al., 2007). Bonferroni correction for multiple testing was applied, resulting in a model’s p level of 0.008 (α/n) as indicator of significance.

### Table 1

<table>
<thead>
<tr>
<th>Variables</th>
<th>BPD (Mean ± SD)</th>
<th>CC (Mean ± SD)</th>
<th>OR (95% CI)</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fathers (BPD n=24; CC n=35) Care from mother</td>
<td>20.71 ± 10.21</td>
<td>27.19 ± 6.25</td>
<td>0.94 (0.90, 0.99)</td>
<td>2.94</td>
<td>0.005*</td>
</tr>
<tr>
<td>Care from father</td>
<td>19.09 ± 7.69</td>
<td>22.39 ± 7.76</td>
<td>0.97 (0.93, 1.02)</td>
<td>1.53</td>
<td>0.132</td>
</tr>
<tr>
<td>Mother overprotection</td>
<td>13.46 ± 7.49</td>
<td>11.34 ± 7.66</td>
<td>1.04 (0.97, 1.10)</td>
<td>1.03</td>
<td>0.307</td>
</tr>
<tr>
<td>Father overprotection</td>
<td>11.14 ± 5.46</td>
<td>8.94 ± 5.46</td>
<td>1.06 (1.00, 1.11)</td>
<td>−1.24</td>
<td>0.222</td>
</tr>
<tr>
<td>PHQ depression</td>
<td>7.67 ± 5.53</td>
<td>3.00 ± 2.86</td>
<td>1.33 (1.25, 1.41)</td>
<td>−4.15</td>
<td>&lt;0.001***</td>
</tr>
<tr>
<td>PHQ somatization</td>
<td>6.04 ± 3.87</td>
<td>4.06 ± 3.55</td>
<td>1.16 (1.06, 1.26)</td>
<td>−1.98</td>
<td>0.053</td>
</tr>
<tr>
<td>PHQ stress</td>
<td>7.70 ± 3.45</td>
<td>3.73 ± 3.37</td>
<td>1.31 (1.19, 1.45)</td>
<td>−3.85</td>
<td>&lt;0.001***</td>
</tr>
<tr>
<td>Mothers (BPD n=40; CC n=40) Care from mother</td>
<td>18.48 ± 10.28</td>
<td>24.02 ± 9.16</td>
<td>1.06 (1.02, 1.11)</td>
<td>2.58</td>
<td>0.012*</td>
</tr>
<tr>
<td>Care from father</td>
<td>19.25 ± 8.58</td>
<td>21.21 ± 9.35</td>
<td>1.02 (0.97, 1.07)</td>
<td>0.91</td>
<td>0.364</td>
</tr>
<tr>
<td>Mother overprotection</td>
<td>13.85 ± 8.82</td>
<td>11.21 ± 7.43</td>
<td>1.04 (1.00, 1.08)</td>
<td>−1.48</td>
<td>0.143</td>
</tr>
<tr>
<td>Father overprotection</td>
<td>11.79 ± 7.35</td>
<td>9.38 ± 6.69</td>
<td>1.03 (1.00, 1.06)</td>
<td>−1.29</td>
<td>0.200</td>
</tr>
<tr>
<td>PHQ depression</td>
<td>7.73 ± 5.57</td>
<td>6.26 ± 5.57</td>
<td>1.05 (1.00, 1.10)</td>
<td>−1.19</td>
<td>0.238</td>
</tr>
<tr>
<td>PHQ somatization</td>
<td>8.18 ± 5.22</td>
<td>6.79 ± 4.70</td>
<td>1.06 (1.01, 1.13)</td>
<td>−1.27</td>
<td>0.210</td>
</tr>
<tr>
<td>PHQ stress</td>
<td>6.36 ± 3.28</td>
<td>5.24 ± 3.69</td>
<td>1.10 (1.04, 1.16)</td>
<td>−1.43</td>
<td>0.158</td>
</tr>
</tbody>
</table>

Note: n = sample size; M = mean; SD = standard deviation; OR = odds ratio; p = probability; PHQ = Patient Health Questionnaire.

Odds ratios represent associations between the subscales of the Parental Bonding Instrument or the Patient Health Questionnaire with group affiliation (BPD vs. CC).

*p < 0.05.
**p < 0.01.
***p < 0.001.
4. Discussion

The present study examined potential transgenerational effects of parental bonding, assessing parent and offspring bonding quality within a clinical adolescent sample.

As a first important result, our data confirmed that adolescent BPD patients experience their parents, both mother and father, as lacking in affection and overcontrolling, and that both these dysfunctional parental bonding styles are highly associated with BPD outcome in offspring.

Similarly, both mothers and fathers of adolescents with BPD recalled their own mothers to show a non-affectionate bonding quality and this was associated with BPD outcome in offspring. This result indicates that parents’ bonding experiences with their own mothers might play a role in the development of BPD in their daughters, suggesting a transgenerational effect of parental bonding experiences to BPD in offspring.

The finding that BPD outcome in offspring was associated with parents’ dysfunctional bonding with their mothers and not with their fathers is in line with a result from a previous study (Madden et al., 2015) which assessed the intergenerational transmission of the quality

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Mediation model for the indirect effects of low maternal care in fathers on BPD diagnostic criteria through stress symptoms (Bootstrap (5000) confidence intervals).</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dependent variable: Stress</td>
<td>Low care from mother</td>
</tr>
<tr>
<td></td>
<td>BPD diagnostic criteria</td>
</tr>
<tr>
<td></td>
<td>Stress</td>
</tr>
</tbody>
</table>

* $p < 0.05$.
** $p < 0.008$. 

Fig. 1. Summary of mediator models for BPD criteria outcome and fathers’ low care from mother (n=59) across different transgenerational variables.

Fig. 2. Summary of mediator models for BPD criteria outcome and mothers’ low care from mother (n=87) across different transgenerational variables.
of parenting in both mothers and fathers in a UK birth cohort. This result potentially points out that the mother played a greater role as a primary caregiver. However, despite the strong similarity between parents’ and offspring’s perceived parental bonding, there were no associations between parents’ and offspring’s perceived parental bonding.

In regard to the mental health status of patients’ parents, findings revealed that although there were no differences between mothers of BPD and of CC patients, BPD patients’ fathers reported a higher level of psychiatric symptoms. Furthermore, the high levels of depression and psychosocial stress experienced by fathers were associated with BPD outcome in offspring. Since most previous studies to date have focused on the association between maternal psychopathology and BPD in offspring (e.g., Schuppert et al., 2015; Whalen et al., 2014), this finding should encourage a more detailed examination of maternal psychiatric symptoms in order to clarify potential associations between specific types of fathers’ mental suffering and BPD in offspring. Furthermore, since our sample included only daughters, one could theorize that there was a potential gender influence underlying the association between reported bonding quality of fathers and daughters. However, future studies are needed to confirm this hypothesis.

Finally, we found that level of stress perceived by fathers mediated the association between maternal bonding quality reported by fathers and BPD in daughters. This result suggests that a pathway of lower-quality bonding between fathers and their own mothers influences the father’s perceived stress level, which in turn seems to affect BPD pathology in daughters. The role of parenting stress in the context of BPD (Schuppert et al., 2015) and other psychiatric disorders (Epstein et al., 2008; Semke et al., 2010) has been examined before; however, those studies considered a global level of parental stress for mother and father taken together or maternal stress only. Our findings expand previous knowledge on parenting stress, exploring this dimension separately for mothers and fathers, and focusing on an adolescent sample of BPD patients.

From another point of view, it might also be possible that caregivers’ responses to the child provoking offspring reports of poor parental bonding could be linked to a parental trait of emotional dysregulation. Parental emotional dysregulation has been found to be associated with more frequent invalidation of adolescent’s emotional expression, which in turn seems to be related to higher levels of adolescent emotional dysregulation (Buckholdt, Parra, and Jobe-Shields, 2014). Additionally, parental emotional dysregulation could influence offspring regulatory capacities via genetic pathways (Buss and Plomin, 2014). Parental emotional instable behavior patterns could thus be partially transmitted to their offspring via genes and parenting behavior, increasing their risk of developing BPD.

4.1. Limitations and strengths

There are several limitations to the current study. First, all information about parental bonding was retrospective and was obtained via self-report questionnaires. Our assessment of parental bonding might therefore be confounded by specific personality traits and state-dependent factors (e.g., stress, mood, anxiety). In addition, we used a cross-sectional design; therefore, no causal inferences can be made. In addition, we need to point out that only female and only inpatients (and their parents) were included in this study. The composition of our sample is in accordance with the higher prevalence of BPD in women than in men (Lieb et al., 2004); however, reported results possibly cannot be generalized to male patients with BPD and nonclinical samples. In addition, we cannot rule out the possibility of a gender effect on parent-child bonding. Therefore, further studies are needed to confirm and extend these results to male and nonclinical populations. Furthermore, the modest size of our sample prevented us from conducting analyses that are more complex. Indeed, since dysfunctional bonding with one parent might be buffered by a nurturing and responsive bonding with the other (Jaffee et al., 2013), it would be important to consider potential interactions between maternal and paternal bonding on adolescent BPD development, which could be the subject of future research.

The main strength of our study involves the clinical characteristics of our sample, which includes the rigorous assessment of adolescent inpatients with full-syndrome BPD and the use of a clinical control group in order to detect the influence of etiological factors specifically on BPD. Furthermore, to the best of our knowledge, this is the first study that included both mothers and fathers of a well-defined adolescent BPD within a clinical group with mixed psychiatric diagnoses and that assessed parental bonding and parental mental health status.

4.2. Clinical and research implications

This study constitutes an initial contribution to examining the transgenerational effect of parental bonding on BPD, exploring both mothers’ and fathers’ bonding experiences. Our results might encourage other researchers to investigate this neglected topic by examining transgenerational effects in larger and mixed samples or by taking other aspects of parent–child relationships into account.

Furthermore, our results show the important contribution that paternal bonding and mental health status make to BPD development. Since examining fathers in this context has been neglected in this area of research, future studies are needed to confirm and expand these results and to offer a more comprehensive picture of BPD development.

From a clinical perspective, treatment programs should put a stronger focus on inquiring into mothers’ and fathers’ mental health status, offering clinical support if needed. Participation of family members is already part of some adolescent BPD treatment programs; our findings corroborate the importance of such approaches in order to improve the interpersonal relationship between adolescents and their parents.

Lastly, given that parental bonding impacts both parents and offspring, developing a parenting intervention specifically tailored for this high-risk population might be a useful step in breaking transgenerational cycles of poor parenting and inform prevention and intervention policies.

Conflict of interest

None of the authors had any conflict of interest.

References

Buckholdt, K.E., Parra, G.R., Jobe-Shields, L., 2014. Intergenerational transmission of