Mediating role of borderline personality disorder traits in the effects of childhood maltreatment on suicidal behaviour among mood disorder patients

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Original article

1. Introduction

Childhood experiences correlate with adult physical and mental well-being [1,2]. The most adverse outcomes include associations of childhood maltreatment (CM) with premature death [3] and suicidal behaviour [4]. While research on suicide deaths remains scarce [5], considerable evidence shows an association between CM and suicidal ideation and attempts [6]. Although several previous studies have examined this relationship by estimating the effect of covariates in analyses, few studies have investigated potential mediating mechanisms [6–10].

Borderline personality disorder (BPD) ranks among the most prevalent personality disorder comorbidities among mood disorder patients [11,12]. Subthreshold BPD traits are even more common [13]. Within mood disorder samples, CM is associated with comorbid BPD diagnosis [14] and traits [15]. Aetiology of BPD is multifactorial, including both inherited vulnerabilities and developmental factors [16]. Family and twin studies of BPD demonstrate familial transmission and moderate heritability [17]. Psychological theories of development of BPD remark CM and dysfunctional parenting [18–20] as environmental factors.
Although CM is neither sufficient nor necessary for the development of BPD [16], prospective studies show that children exposed to CM are at higher risk of developing BPD [21,22]. In cross-sectional studies, adult BPD patients report ample CM and dysfunctional parenting [23–25], and a dose-response relationship exists between CM and BPD symptoms [15,26,27].

Epidemiological and clinical cohort studies affirm connections between depression, hopelessness, and suicidal ideation. Progression to suicide attempts, by contrast, appears to be more associated with severity of depression, anxiety or agitation, and impaired self-control [28–31]. Dysregulation of emotions and impaired self-control characterize the core phenotype of BPD, together with unstable relationships and cognitive distortions/identity disturbance [32]. BPD, in turn, is a substantial risk factor for suicidal behaviour [33,34].

Suicidal ideation and acts are common among patients with mood disorders [28,30,31], and these patients constitute a high-risk group for suicide [35,36]. Our previous work [28] showed that among patients with mood disorder, risk factors for suicidal ideation and suicide attempts likely differ. Several putative remote risk factors may also influence this risk through more proximate clinical characteristics [28]. CM could explain the mutual association as a common denominator for both suicidal behaviour and BPD traits. In addition to dysregulation of emotions, however, affective lability is frequently present in mood disorders. The only previous study of which we are aware that investigated direct mediators between CM and suicidal behaviour in a clinical mood disorder sample showed mediation through affective lability in patients with bipolar disorder [37]. Other explanatory factors may include heritability of psychiatric disorders and impulsive-aggressive traits; tentatively interacting with familial recurrence of CM [38].

We investigated mediators between CM and suicidal behaviour within a psychiatric mood disorder patient cohort. We also modelled as confounders the effects of parental mental health and substance use. We hypothesized (a) BPD traits to be significant mediators between CM and suicidal behaviour, and due to impaired self-control, (b) BPD traits to demonstrate stronger mediating effects on suicide attempts than on suicidal ideation. Finally, we explored possible differences in mediating roles of specific forms of CM.

2. Methods

2.1. Setting

This study was executed within the mood disorder arm of the Helsinki University Psychiatric Consortium (H UPC) Study, a joint research project between the Faculty of Medicine, University of Helsinki; the Department of Psychiatry, Helsinki University Central Hospital; the Department of Health and the Mental Health Unit of the National Institute of Health and Welfare; and the Department of Social Services and Health Care, Psychiatric Services, City of Helsinki, Finland. The Ethics Committee of Helsinki University Central Hospital and the appropriate research committees approved the study design. A complete description of the H UPC study methodology is available elsewhere [28,39] and is briefly outlined below.

Participating regional units consisted of all 10 communal mental health centres, 24 of the 35 psychiatric inpatient wards, and one of the 8 day-care hospitals. The sampling was executed from 12th January 2011 to 20th December 2012. Patients were randomly drawn by stratified sampling method from regional units to generate representativeness. Every ≥18-year-old patient was considered eligible, excluding patients suffering from mental retardation or neurodegenerative disorders or possessing insufficient Finnish language skills.

2.2. Sampling

From the mood disorder units, a total of 904 patients were drawn and 784 were reached and invited to participate in the study; 375 declined participation and 336 completed the study (response rate 43%, 336/784). Excluding missing surveys and other principal lifetime diagnoses resulted in a final sample of 287 patients with either depressive disorder (n = 188) or bipolar disorder (BD) (n = 99). No significant differences emerged in age or gender when stratified by principal diagnosis and regional sampling relative to the patient population in the respective psychiatric services. For the description of the sample, see Table 1.

2.3. Lifetime principal diagnosis

The study diagnoses were formed by the clinical diagnoses assigned by the attending physicians and according to the International Statistical Classification of Diseases and Health Problems, 10th Revision (ICD-10); Diagnostic Criteria for Research. The authors (K.A., I.B., B.K., M.K.), however, carefully weighed the validity of the diagnoses by re-examining all available information from patient records and specified the diagnosis when needed. A lifetime principal diagnosis was hierarchically established by giving precedence to severe depressive, bipolar affective, and psychotic disorders. The BD diagnosis was subtyped according to the Finnish national treatment guidelines [40] into type I and II disorders.

2.4. Trauma and Distress Scale (TADS)

The TADS is a self-report questionnaire of childhood maltreatment and distressing experiences [41,42]. The 25 items of the scale measure five subdomains of CM, including physical, sexual, and emotional abuse, and emotional and physical neglect. The items of the scale inquire about gradually more severe experiences and rate the frequency of occurrence of each by a five-point Likert scale from 0 to 4 (0 = never, 1 = rarely, 2 = sometimes, 3 = often, 4 = nearly always). The TADS provides both subscales for each type of CM and, by adding the subscales, a sum score. The reliability between self-reported and interviewed TADS, and internal consistencies of the subscales indicate good psychometric properties in a Finnish community sample [43]. In our sample, Cronbach’s alphas for the subscales ranged from 0.675 to 0.908, and for the sum score 0.924.

2.5. McLean Screening Instrument for Borderline Personality Disorder (MSI-BPD)

The MSI-BPD is a 10-item self-report screening instrument for borderline personality disorder (BPD), where each item rates true/false screening for the presence of BPD symptoms. The MSI-BPD shows good sensitivity (0.81) and specificity (0.85) with a clinical cut-off score of 7 or more, as well as good internal consistency (Cronbach’s alpha = 0.74) [44], confirmed in a Finnish validation study [45]. In our sample, Cronbach’s alpha was 0.753 and analyses were conducted by omitting the suicidality item to avoid content overlap.

2.6. Other assessments

Beck Depression Inventory (BDI) is a self-report instrument (21 items) for depressive symptoms [46]. In our sample, Cronbach’s alpha for the scale was 0.923. The suicidality item in the analyses was omitted to avoid circularity.

The survey inquired about family history of mental health and substance abuse that had required treatment or caused significant
impaired. Following a positive answer, details of family relationship, quality of mental health disorder, and primary substance used were inquired with open-ended questions, providing also a list of examples. For this study, we formed composite variables for parental history of any mental health disturbance or substance abuse.

### 2.7. Suicidal behaviours

Lifetime suicidal behaviours were evaluated by two questions adopted from the National Comorbidity Survey [47] inquiring about: (1) lifetime suicidal ideation (“Have you ever seriously thought about committing suicide?”) and (2) lifetime suicide attempts (“Have you ever attempted suicide?”). In addition, the survey inquired about the number of possible lifetime suicide attempts. The self-reported information on lifetime suicidal behaviour was complemented by extracting the number of lifetime suicide attempts from medical records (defined as involving at least some degree of intention to die). A composite variable on lifetime suicide attempts was then generated by aggregation of information from self-reports and medical records on any lifetime suicide attempt.

### 2.8. Study design

In this sample, we examined the extent to which the effects of CM on suicidal behaviour are mediated through borderline personality disorder traits (MSI-BPD), and whether this mediation effect differs between suicidal ideation and attempts. We modelled three different outcomes: (i) lifetime suicidal ideation without a lifetime suicide attempt, (ii) lifetime suicidal ideation and/or suicide attempt, and (iii) lifetime suicide attempt.
2.9. Statistical methods

The hypothesized mediation of the effect of TADS score on lifetime suicidal behaviour by MSI score was tested using the “mediation” package version 4.4.5 for R software version 3.2.2 (2015-08-14) [48–50]. Lifetime suicidal behaviour outcomes were modelled using logistic regression models [49,51]. Linear regression model (age, sex, and TADS score as predictors) was used for MSI (the mediating variable). Possible differences in mediation statistics between the models for outcomes (i) and (iii) were tested using a random permutation tests with 1000 random permutations. A non-parametric inference (bootstrap) version of the mediation analysis was used with 2000 simulations [48,49]. The Total Effect was defined as a difference in outcome between the modelled “exposure” and “control” conditions, respectively, set to the maximal and minimal TADS mean scores. The estimated Total Effect of TADS on suicidal behaviour was further divided into the Average Causal Mediation Effect (ACME) via MSI and the Average Direct Effect (ADE) reflecting the direct effect of TADS on outcome (i.e., the part of the Total Effect that is unrelated to MSI). We also explored, if the mediation effect through BPD traits involved separate psychopathological BPD subcomponents by comparing the mediation models using MSI total score to models using three subscores reflecting the affective, interpersonal or cognitive/behavioral components of BPD as mediators. Lastly, to detect potential heterogeneous effects across different types of maltreatment, we conducted an omnibus test for differences among TADS subscales in predicting lifetime suicidal behaviour (alternatively MSI) by comparing Akaike’s Information Criteria (AIC) [52] for models using subscales versus the main scale as independent variables. Lower AIC values indicate a better model. Further methodological details are provided in the Supplementary materials.

3. Results

Of the 287 patients with a mood disorder, 62 (21.6%) showed no lifetime suicidal ideation or attempts, 97 (33.8%) showed lifetime suicidal ideation without attempts, and 128 (44.6%) had a lifetime suicide attempt.

The effect of TADS on suicide attempt was strongly mediated by MSI (99.7% of the total effect). However, the effect of TADS on the ideation outcome was much less mediated by MSI (21.2% mediated for ideation only, and 43.3% for ideation and/or attempt; Fig. 1, Table 2). To test statistical significance of these differences, we conducted formal random permutation tests (see the Supplementary materials). Comparing the estimates between the two outcomes, “lifetime ideation only” versus “lifetime suicide attempt(s)”, there was no significant difference in Total Effect of TADS on suicide attempt ($P = 0.379$), but ACME was clearly stronger for attempts than for ideation ($P = 0.002$) and ADE was weaker for attempts than for ideation ($P = 0.003$). This suffices to verify that the effect of TADS on suicide attempts was indeed more strongly mediated by MSI than its effect on suicide ideation (because the proportion-mediated estimates combine uncertainty in both ACME and ADE, they showed only a trend; $P = 0.195$ (Fig. 1). Adjusting for BDI had practically no effect on the TADS-MSI-attempt mediation estimate (100% versus 99.7%; cf. adjusted ACME 0.24 and ADE 0.00 to Table 2). The results of mediation models using MSI total score versus subcomponents were similar (Fig. 2). A permutation test for differences in mediation effects for suicide attempt outcome was non-significant between affective and behavioral-cognitive components, but significant between interpersonal and behavioral-cognitive components ($P = 0.006$ for equal ACME and $P > 0.978$ for ADE). Similarly to the total sample, MSI mediated considerably more strongly the effect of TADS on suicide attempts than on suicidal ideation both in depressive and in bipolar subsamples. The analyses showed no interaction between bipolar disorder status and the variables in the mediation model. In models adjusted for mother’s and father’s mental health, and their joint mental health (interaction), the estimates remained virtually unchanged relative to the model without these adjustments (Table 2).

We examined possible differences in prediction of lifetime suicidal behaviour between types of CM by a comparing model

![Fig. 1. Mediation of the trauma and distress scale (TADS) effects on lifetime suicidal behaviour by borderline personality disorder traits (MSI). ACME: Average Causal Mediation Effect; ADE: Average Direct Effect; Total Effect = ACME + ADE. ACME and ADE are shown both for maximal TADS score (dots; answer 4 on all items) and minimum TADS score (circles; no trauma or distress). The whiskers indicate 95% confidence intervals, and the different panels are for the different outcomes indicated in the panel titles.](image-url)
Table 2
Mediation analyses of the effect of childhood maltreatment on lifetime suicidal behaviour through borderline personality disorder traits with adjusted sensitivity analyses.

<table>
<thead>
<tr>
<th>Adjusting variables</th>
<th>Percent mediated Estimate</th>
<th>ACME Estimate</th>
<th>ADE Estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lifetime suicidal ideation without suicide attempts</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex and age (baseline)</td>
<td>21</td>
<td>0.120</td>
<td>0.441</td>
</tr>
<tr>
<td>Sex, age, familial mental disorder</td>
<td>21</td>
<td>0.123</td>
<td>0.467</td>
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<tr>
<td>Sex, age, familial substance abuse</td>
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<td>0.452</td>
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<tr>
<td><strong>Lifetime suicidal ideation or suicide attempt</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex and age</td>
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<td>0.149</td>
<td>0.195</td>
</tr>
<tr>
<td>Sex, age, familial mental disorder</td>
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<td>0.147</td>
<td>0.208</td>
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<tr>
<td>Sex, age, familial substance abuse</td>
<td>42</td>
<td>0.158</td>
<td>0.217</td>
</tr>
<tr>
<td><strong>Lifetime suicide attempt</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex and age</td>
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<td>0.001</td>
</tr>
<tr>
<td>Sex, age, familial mental disorder</td>
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<td>0.011</td>
</tr>
<tr>
<td>Sex, age, familial substance abuse</td>
<td>90</td>
<td>0.331</td>
<td>0.036</td>
</tr>
</tbody>
</table>

ACME: Average Causal Mediation Effect; ADE: Average Direct Effect.

Fig. 2. Mediation of the trauma and distress scale (TADS) effects on lifetime suicidal behaviour by separate components of borderline personality disorder traits (MSI). ACME: Average Causal Mediation Effect; ADE: Average Direct Effect; Total Effect = ACME + ADE. ACME and ADE are shown both for maximal TADS score (dots; answer 4 on all items) and minimum TADS score (circles; no trauma or distress). The whiskers indicate 95% confidence intervals. The columns are shown for different outcomes and panels on the rows for separate components of borderline personality disorder traits as indicated in the titles. Affective component includes MSI items “increased anger”, “mood instability”, and “feeling of emptiness”. Interpersonal component includes MSI items “troubled relationships”, and “avoidance of abandonment”. Behavioral-cognitive component includes MSI items “impulsivity”, “dissociative symptoms”, “distrustfulness”, and “identity disturbance”.

K.I. Aaltonen et al. / European Psychiatry 44 (2017) 53–60
using the overall assessment of CM to a model using all the different subtypes. Regarding such an omnibus comparison of total TADS versus TADS subscales as predictors of lifetime suicidal behaviour, only the model predicting “suicidal ideation or attempt” was slightly better with the subscales than with the total scale (ΔAICsub = 238.7, ΔAICmain = 239.3, ΔAIC = -0.6), whereas the same did not hold for ideation only (ΔAICsub = 176.9, ΔAICmain = 157.6, ΔAIC = 1.3) or for suicide attempts (ΔAICsub = 341.2, ΔAICmain = 336.9, ΔAIC = 4.7), nor for MSI (ΔAICsub = 11.7, ΔAICmain = 11.0, ΔAIC = 0.8). Thus, we found negligible evidence for using the subscales separately over summing them to the main scale; however, in all of the above models, the subscale Physical Abuse was always a significant independent predictor, or nearly so (P = 0.008–0.062), whereas the other subscales rarely were. In the case of MSI, also the subscale Emotional Neglect was a significant independent predictor (P = 0.005).

4. Discussion

We investigated mechanisms mediating the effects of CM on different types of suicidal behaviour among psychotic mood disorder patients. The total magnitude of the effects of CM on either lifetime suicidal ideation or lifetime suicide attempts appeared comparable. However, the mediating factors differed between these behaviours. Concordant with the study hypotheses, our data showed minor mediation effects via BPD traits on lifetime suicidal ideation, whereas the effect on lifetime suicide attempts appeared substantial. This pattern was found in both depressive and bipolar disorder subsamples. These results support our previous findings that risk factors for suicidal ideation and attempts differ [28]. Temporally remote vulnerability factors, such as childhood maltreatment, may influence suicidal behaviour through proximate clinical characteristics, like BPD traits.

The majority of mood disorder patients engage in suicidal ideation or attempts during their lifetime [30,31]. Moreover, patients diagnosed with mood disorders and those diagnosed with BPD represent high-risk patient groups for suicide [34]. Our study is among the first to investigate potential mechanisms mediating CM and suicidal behaviour among psychiatric patients with mood disorders. Therefore, the findings may be more generalizable to clinical practice. We executed stratified, random sampling in the corresponding psychiatric care organizations of the Helsinki metropolitan area to obtain a representative cohort of patients with mood disorders [28]. Four in five patients of the study sample reported lifetime suicidal behaviour. The patients also reported frequent CM, one in five having been physically abused and over one in ten sexually abused at least sometimes [15]. The Trauma and Distress Scale (TADS) evaluates both progressively more severe experiences and frequency of exposure to CM. The scale provides a summary score for severity of CM overall and for each subsdomain. The study design permits clinically and theoretically important examination of difference of mediation effects between suicidal ideation and attempts. We thoroughly assessed lifetime suicide attempts by complementing survey data with all available information from medical records. Furthermore, analyses modelled BPD traits as a continuous variable, thereby increasing statistical sensitivity relative to a binary diagnostic classification. The applied general approach to causal mediation served to handle the non-continuous outcome data [48].

The results should be interpreted in light of the study limitations. First, an observational, cross-sectional, and retrospective study design restricts inferences on direction of causation, and is susceptible to unobserved confounding factors. Second, possible alterations in patients’ self-reflection may predispose to imprecisions in self-reports [53] and the validity of retrospective reports of childhood experiences remain disputed [54–56]. Retrospective reports likely recognize the most severe CM histories, and these cases may have more comorbidities [57]. Concerns exist about retrospective reports yielding false negatives due to more resilient subjects underreporting adversity [58], and current depressive symptoms influencing whether or not an individual reports adverse childhood experiences [59]. Similarly, depressive symptoms may inflate estimates of concurrent BPD symptoms [60]. In our study, controlling for current depressive symptoms attenuated slightly the total effect, but altered negligibly the mediation estimates. Although we controlled for current age, exact timing of CM remained unavailable and there may be sensitive periods for CM [61,62]. Third, study response rate (43%) remains lower than anticipated. However, the survey was demanding and time-consuming, and sampling was implemented among busy clinical practice. Whether the sample is subjected to selection bias remains partly unknown, although in terms of age and gender the sample is comparable with the entire treated patient population. Additionally, the socio-demographics of the sample are in accord with other screening-based, representative MDD and BD cohorts from the area [11,63]. Fourth, we lack information on impulsive-aggressive traits, which often correlate and partly overlap with cluster B personality traits. Fifth, MSI may correlate not only with BPD emotional dysregulation, but also with BD mood instability. However, our findings were similar among unipolar and bipolar patients. Sixth, diagnoses are based on nationally employed ICD-10-DCR criteria evaluated in psychiatric secondary care and confirmed by the authors. We are confident that all of the participants suffered from a mood disorder, but some inaccuracies may remain in the specific diagnosis. Seventh, possible effects of treatment remain unknown. Finally, analyses do not directly provide information on suicide deaths.

Our main findings indicate that the mediating role of BPD traits in the effects of CM on suicidal ideation or suicide attempts differs. This mediation effect of BPD traits appeared pertinent to BPD mainly as a single construct. However, of the components of BPD psychopathology, the affective or cognitive/behavioural traits may be somewhat more important mediators than the interpersonal. Similarly, when we compared overall CM exposure against the subdomains of CM as predictors of suicidal behaviour we found mainly similarities in effects, in line with previous findings [64]. Childhood physical abuse, however, was the most robust and independent predictor among the maltreatment types. Overall, CM exposures show sub-additive effects and a stronger association with mental disorders or suicidal behaviour among younger age groups [65–67]. The World Mental Health Surveys reported that the overall effects of CM on suicidal ideation persist after controlling for a wide range of axis I disorders [66]. In population surveys, however, the influence of severity of individual disorders remains unclear. In general, depressive symptoms and hopelessness appear as major risk factors for suicidal ideation [28,30,31]. One previous study reported hopelessness to mediate the effect of childhood sexual abuse on suicidal ideation among suicide attempters [8]. The detailed mechanisms of the interaction between CM and suicidal ideation warrant further research, although we found little evidence for notable mediation effects via BPD traits.

We studied the mediating mechanisms between CM and suicide attempts among psychiatric mood disorder patients. Our main findings are concordant with a considerable mediation effect of CM on suicide attempts through BPD traits. Previous literature shows association between CM and BPD; cluster B personality disorders or BPD traits in turn constitute major risk factors for suicide deaths [68] or attempts [28]. In our sample, BPD traits mediated entirely the effect of CM on suicide attempts. This
contrasts with an earlier study of a depressive sample with CM effects persisting after controlling for comorbid BPD diagnosis \[14\]. We modelled, however, BPD traits as a continuous variable. Although cross-sectional study design is a limitation, the reported childhood experiences preceded or coincided with evolving BPD traits, thus conforming with the implied temporal order for mediation analyses. Causation still remains uncertain due to possible confounding by familial mental disorders or substance abuse, and other adversities commonly associated with CM \[65,67\]. In our data, when controlling for parental psychiatric or substance use disorders, the mediation effect of CM on lifetime suicide attempts through BPD traits remained unaltered.

Besides direct genetic influence in development of BPD, hereditary factors may correlate or interact with environmental factors \[17\]. In recent twin studies of BPD, practically all of the association between CM and BPD traits was accounted for common familial or hereditary factors \[69,70\]. However, interpretation of the results of the co-twin control regression design remain difficult \[71\]. Impulsive-aggressive traits show the highest heritability of all BPD symptom domains \[72\], and are risk factors for suicide deaths \[73\]. Childhood maltreatment predicts both impulsive-aggressive traits and suicide attempts, and may constitute either an environmental risk factor for both, or inherited impulsive-aggressive traits may influence exposure to CM and risk for suicidal behaviour \[14\]. Family studies of suicidal behaviour show intergenerational transmission of impulsive-aggressive traits and possible familial transmission of CM \[38\]. Investigating familial transmission of CM, however, remains intricate \[74\].

Our findings are consistent with the presumed influence of CM on suicidal behaviour via BPD traits. While the magnitude of the total effect of CM is comparable over the suicidal behaviour continuum, the mechanisms for suicidal ideation and attempts apparently diverge. In our data, BPD traits substantially mediated the effects of CM on risk for lifetime suicide attempts. In addition to having implications for future research on CM and suicidal behaviour, if reinforced in future prospective studies, our findings may be clinically informative. If BPD traits are on the causal path from CM to suicide attempts, treatment of BPD traits could reduce the risk of suicide attempts in mood disorder patients subjected to CM.

**Disclosure of interest**

The authors have not supplied their declaration of competing interest.

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**Appendix A. Supplementary data**

Supplementary data associated with this article can be found, in the online version, at [http://dx.doi.org/10.1016/j.eurpsy.2017.03.011](http://dx.doi.org/10.1016/j.eurpsy.2017.03.011).

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