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Antecedents and risk factors for borderline personality disorder: Etiopathogenic models based on a multi-level meta-analysis☆

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ABSTRACT

Background: Empirically-based developmental psychopathology approach identified three domains involved in the emergence of borderline personality disorder (BPD): i) underlying liabilities to develop psychopathology (i.e., early patterns of internalizing and externalizing manifestations); ii) invalidating relational experiences (e.g., childhood traumatic experiences, maladaptive parenting, problematic peer relationships); iii) regulatory mechanisms of emotions and behaviors. Nevertheless, no studies have quantitatively summarized empirical findings concerning how and to what extent these domains might be temporally associated to the emergence of BPD features from adolescence to adulthood.

Methods: The current multi-level meta-analysis included 106 studies ($N = 86,871$ participants) assessing the role of previously mentioned antecedents and risk factors for BPD.

Results: The analysis showed moderate effect sizes capturing temporal associations between early internalizing/externalizing psychopathological manifestations, different invalidating relational experiences, emotion/behavior regulation processes with later BPD features. The effect sizes of these domains were not statistically different from each other.

Conclusion: This evidence supports a transactional developmental model of BPD. Consistently, the emergence of BPD could be viewed in the light of dynamic interplays between an underlying liability to psychopathology and invalidating relational experiences across different stages of development, which are progressively reinforced through increasing alterations of emotion and behavior regulation mechanisms.

1. Introduction

Borderline Personality Disorder (BPD) is a severe mental disorder characterized by a pervasive pattern of instability in affect regulation, impulse control, interpersonal relationships, and self-image (APA, 2013). Historically, four main clinical theories of BPD have proposed different etiopathogenic models of this condition. Kenberg's (1967) theory posits that BPD is characterized by an excessive aggressiveness, which could be genetically determined or due to excessive frustration in childhood. Linehan's biosocial model (1993) of BPD hypothesizes that the core feature of the disorder is a pervasive alteration of emotion regulation processes, which emerge from continuous transactions between a biological emotional vulnerability and invalidating environments. Starting from the attachment perspective, Bateman and Fonagy (2004) conceptualize BPD as a deficit in mentalization (i.e., inability to identify mental states in oneself or in other and to recognize how these mental states are mutually influenced), which begins in early stages of development due to parental failures to help children recognize their

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feelings or those they evoke. Ultimately, Gunderson and Lyons-Ruth (2008) have developed the interpersonal hypersensitivity theory of BPD that postulates a genetic predisposition to hypersensitivity and hyperreactivity to interpersonal cues.

These etiopathogenic models posit that both genetic and environmental factors are involved in the emergence of BPD. However, the impact of these determinants vary in each of these theoretical frameworks (Gunderson et al., 2018). Specifically, the excessive aggression (Kernberg, 1967) and interpersonal hypersensitivity (Gunderson and Lyons-Ruth, 2008) theories hypothesize a predominance of a genetic base; whereas, the failed mentalization theory of BPD (Bateman and Fonagy, 2004) assumes a key role of relational experiences, especially those in early infancy. Finally, the emotional dysregulation theory of BPD (Linehan, 1993) posits concurrent effects of genetic predispositions and invalidating environments in the emergence of the disorder, assuming a transactional view of development (Crowell et al., 2009; Fruzzetti et al., 2005). Alternatively, developmental psychopathology could be a useful empirically-based theoretical framework in the understanding of BPD etiopathogenesis.

1.1. Developmental psychopathology and BPD

Developmental psychopathology is an integrative discipline that has the goal of understanding psychopathology and its relation to normative adaptation referring to a developmental life-span framework (Cicchetti and Rogosch, 2002). The transactional approach at the base of development psychopathology assumes that intrinsic/individual and external/environmental together with proximal and distal factors dynamically interact with each other over time determining adaptive and maladaptive developmental outcomes (Cicchetti and Lynch, 1993; Sroufe, 2009). These complex dynamic interactions involved in modeling individual differences of developmental pathways have been conceptualized referring to two key concepts, namely equifinality and multifinality (Cicchetti and Rogosch, 1996). On the one hand, the equifinality describes a well-known scenario related to multiple pathways for a specific developmental outcome. On the other hand, the multifinality captures the evidence concerning that multiple developmental outcomes are observed departing from a same set of initial conditions. In this scenario, the complex interactions between risk and protective factors over time play a key role in explaining different developmental trajectories and outcomes (Harvey et al., 2004; Lynch et al., 2021).

Empirically, one of the most widely used approaches for studying underlying liabilities to psychopathological conditions is the evaluation of covariation patterns across disorders during life-span, which have identified a hierarchical structure of latent dimensions (e.g., general p-factor, internalizing, externalizing) (Achenbach et al., 2016; Blanco et al., 2023; Caspi et al., 2014; Kim and Eaton, 2015) acting as predisposing conditions, to increase the probability to develop a specific disorder within a given spectrum in relation to the exposure of specific risk factors (e.g., adverse childhood experiences, positive family history for specific disorders) (Blanco et al., 2016).

Following these notions, the empirical investigation of risk factors for the emergence of BPD has taken into account different aspects. Looking at underlying liabilities, several studies explored how patterns of early psychopathological manifestations were longitudinally associated with later BPD features (Chanen and Kaess, 2012). Whereas, the most relevant environmental factors have been represented by different forms of invalidating interpersonal experiences (e.g., childhood traumatic experience, maladaptive parenting style, adverse social and intimate relationships) (Fitzpatrick et al., 2021). Furthermore, a strong interest has been focused on developmental trajectories of the emergence of BPD features in connection with adaptive and maladaptive regulatory mechanisms of emotions and behaviors, which represent core dimensions for understanding psychopathological manifestations of the disorder (Chapman, 2019; Unoka and Richman, 2016). Particularly, these regulatory processes are widely considered as a result of transactions between latent psychopathological liabilities and protective/risk environmental factors, especially relational ones (Cavicchioli et al., 2023a, 2023b; Duprey et al., 2023; Kim and Cicchetti, 2010; Kim et al., 2009; Tottenham et al., 2010).

1.2. Antecedents and risk factors for BPD: state of the art and unresolved issues

Looking at underlying liabilities captured by patterns of early onset of different psychopathological manifestations, several empirical studies have been conducted evaluating prospective associations between internalizing (e.g., anxious, depressive, somatic symptoms) and externalizing (e.g., aggressive and rule-breaking behaviors, attention deficits) problems with emerging BPD features across the life-span (e.g., Geselowitz et al., 2021; Haltiganm, & Vaillancourt, 2016; O'Grady and Hinshaw, 2023), suggesting that both these psychopathological manifestations could be relevant antecedents and moderating factors of the disorder (Skabeikyte and Barkauskiene, 2021). However, there is an ongoing debate concerning which domain of developmental psychopathology (i.e., internalizing or externalizing) could be the most representative for the emergence and maintenance of BPD across the life-span, and in turn providing support for specific core psychopathological mechanisms of the disorder (Wolf et al., 2023).

The most investigated environmental factors in BPD are childhood traumatic experiences (i.e., sex/emotional/physical abuse, emotional/physical neglect) (for a review: Yuan et al., 2023). Referring to quantitative meta-analytic results of retrospective studies (Porter et al., 2020), the associations between sex and physical abuse with BPD was small-to-moderate considering clinical and non-clinical samples. Whereas, there was found a robust correlation between childhood emotional abuse and neglect with BPD psychopathology among clinical samples, but modest associations between these interpersonal traumatic experiences and BPD features in non-clinical subjects. When considering maladaptive parenting styles (e.g., authoritarian, permissive) and peer relationships (e.g., bullying, victimization, social isolation), in line with the invalidating contexts of Linehan's model of BPD, some qualitative reviews (Boucher et al., 2017; Musser et al., 2018; Runions et al., 2021) and meta-analysis of cross-sectional data (Lee et al., 2022) highlighted moderate associations between such experiences and the BPD features. Nevertheless, no studies have quantitatively summarized the longitudinal impact of these forms of interpersonal invalidation on the emergence of BPD, nor compared their effects with traumatic ones.

According to the hypothesis of a key role of altered emotion regulation (ER) in BPD, this domain has been widely explored with cross-sectional studies in both clinical and non-clinical samples pointing out moderate-to-large associations with a rigid use of maladaptive (e.g., experiential avoidance, rumination, dissociation) and low levels of adaptive ER strategies (e.g., mindfulness, acceptance and tolerance of emotions) (for reviews and meta-analysis see: Bud et al., 2023; Cavicchioli and Maffei, 2022; Cavicchioli et al., 2015; Daros and Williams, 2019; Scalabrini et al., 2017; Sorgi-Wilson and McCloskey, 2022).

Prospective (e.g., Beeney et al., 2021; McQuade, 2022) and retrospective studies (e.g., Goodman et al., 2010, 2013) further confirmed the associations between altered ER mechanisms and the lifetime emergence of BPD features. However, no study quantitatively summarized to what extent ER processes are longitudinally associated to BPD features. Similar considerations could be extended to the relevance of behavioral regulation (BR) mechanisms (e.g., response inhibition, decision-making) for BPD psychopathology. Indeed, several cross-sectional and case-control studies among adult individuals with BPD supported significant deficits with BR (for a meta-analysis see: Unoka and Richman, 2016). Prospective and retrospective studies (e.g., Brière et al., 2015; Homan et al., 2017) were also conducted in order to clarify the impact of this dimension on the emergence and maintenance of BPD features. Nevertheless, no quantitative studies have summarized the extent of

temporal associations between BR and later BPD features. Ultimately, suicide attempts and non-suicide self-injury (NSSI) behaviors, especially during childhood and adolescence (e.g., Auerbach et al., 2021; Kaess et al., 2021), have been widely considered overt forms of maladaptive mechanisms of ER and difficulties with BR (Hamza et al., 2015; McKenzie and Gross, 2014) rather than psychopathological phenomena themselves (Oppenheimer et al., 2022). According to BPD clinical presentation, it has been suggested that early suicide attempts and NSSI behaviors might represent "*useful marker for the detection of individuals at risk of development of BPD*" (Reichl and Kaess, 2021; p. 140) due to their robust connections with alterations of different self-regulation processes, especially those related to emotions and behaviors within relational contexts (Reichl and Kaess, 2021). Despite this evidence, the implications of these behaviors as proxies of emotion and behavior dysregulation for the emergence of BPD are still unclear (Reichl and Kaess, 2021; Stead et al., 2019) (Fig. 1).

1.3. The present study

The current study aims at conducting a quantitative meta-analysis to clarify the implications of underlying liabilities to psychopathology (i.e., early onset of psychopathological manifestations), invalidating interpersonal experiences (i.e., childhood traumatic experiences, maladaptive parenting styles and peer relationships) together with ER and BR mechanisms for the emergence of BPD features from early adolescence. Adolescence was considered a critical period for the emergence of key psychopathological manifestations linked to BPD (Sharp and Fonagy, 2015).

Specifically, this meta-analysis aims at clarifying:

i) which domain of developmental psychopathology (i.e., internalizing and externalizing) is the most relevant for the emergence of BPD clinical characteristics across-life span;
ii) whether traumatic and the other forms of invalidating relational experiences can be commonly involved in the development of BPD features;
iii) whether alterations in ER and BR could highlight robust temporal associations with BPD psychopathology in order to effectively corroborated the key role of these dimensions for the emergence of this disorder, as hypothesized by well-established cross-sectional evidence.

Departing from these aims, the current meta-analysis included longitudinal studies, both prospective and retrospective, that empirically assessed associations between the previously mentioned antecedents and risk factors with BPD features among clinical and non-clinical populations.

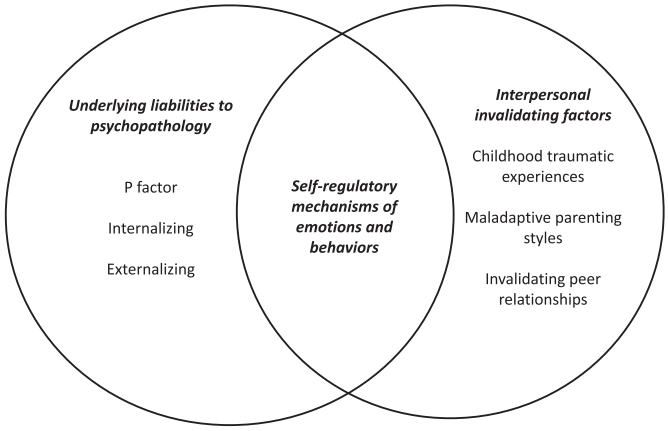

Fig. 1. The system of antecedents and risk factors for BPD.

The current quantitative approach also allowed to lay the foundations for a provisional evidence-based etiopathogenic model of BPD. Accordingly, three different scenarios could be hypothesized:

i) *intrinsic psychopathological liability model*: pooled effect sizes of underlying liabilities to psychopathology as possible proxies of prevalent genetic determinants (Allegrini et al., 2020; Murray et al., 2016) might be significantly larger than those reflecting relational and regulatory risk factors.
ii) *environmental-related model*: invalidating interpersonal experiences could highlight larger effect sizes than the other antecedents and risk factors.
iii) *transactional model*: no significant differences among pooled effect sizes of different antecedent and risk factors might be detected. This should suggest a concurrence of underlying liabilities and environmental factors together with their interplay (i.e., regulatory mechanisms) in the emergence of BPD.

2. Methods

2.1. Criteria for selecting studies

The current meta-analytic review was conducted in line with the PRISMA guidelines (Page et al., 2021). In order to consider studies of comparable quality, the analysis included only those that were published in scientific journals. PsychINFo, Pubmed, ISI Web of Knowledge and Scopus online databases were used to generate potentially relevant articles. The keywords used for the online search are included as supplementary materials. The starting point was 1980 because this was the year when reliable criteria for BPD were introduced (APA, 1980).

M.C. and I.P. conducted the online research. The screening process was double-checked in order to produce a reliable initial sample of articles to consider for the inclusion in the meta-analysis. From the initial online research, M.C. and I.L. considered for the screening process all articles that showed, within the abstract section, at least an assessment of BPD features together with risk factors of interest (i.e., developmental psychopathology, invalidating relational experiences, ER/BR processes) which were retrospectively and prospectively measured. Cohen *k* was estimated for inte-rrater reliability of studies selection (Cohen, 1960).

In order to be included in the current meta-analytic review, the studies had to meet the following inclusion criteria to test the hypotheses of study and to support both the validity and the reliability of results:

i) studies should report prospective associations between developmental psychopathology symptoms, invalidating interpersonal experiences and regulatory mechanisms (t_0) with later BPD features (t_1);
ii) retrospective studies should explicitly assess each antecedent and risk factor within a specific developmental stage (i.e., infancy, childhood, adolescence) temporally preceding the evaluation of BPD features;
iii) BPD features, developmental psychopathology manifestations, invalidating relational experiences and regulatory mechanisms were assessed using valid and reliable instruments (i.e., self-report, interview, multi-informant) (see Table 1s for a detailed description);

BPD features, developmental psychopathology manifestations, invalidating relational experiences and regulatory mechanisms were assessed using valid and reliable instruments (i.e., self-report, interview, multi-informant) (see Table 1s for a detailed description);

iv) suicide attempts and NSSI behaviors were included as antecedents for the emergence of BPD (Reichl and Kaess, 2021). These factors were considered as maladaptive overt forms of ER/BR according to a huge amount of empirical data demonstrating

robust associations between these behaviors and maladaptive regulatory processes (e.g., Hamza et al., 2015; McKenzie and Gross, 2014).

Disagreements on the inclusion of full-text articles were resolved through consensus between screeners.

2.2. Data analyses

The current meta-analysis was based on the *r* coefficient as an effect size measure. Values of *r* greater than or equal to 0.10, 0.30, and 0.50 were interpreted as small, moderate, and large effect sizes, respectively (Cohen, 1992). The correlations of each study were converted to Fisher's *z* scale, which was used to perform all the analyses in order to control for bias in estimating standard error (SE) of effect sizes (Alexander et al., 1989). The summary effect (r_{pooled}) and its 95 % confidence interval (CI) were then converted back into correlations for presenting pooled effect sizes. We conducted a multi-level random-effect meta-analysis using the {metafor} *R* package in order to adequately estimate pooled effect sizes taking into account multiple correlations among effect sizes reported within each study (for a detailed description of statistical procedures see: Viechtbauer, 2010). The estimation of model parameters was based on the restricted maximum likelihood method (Harrer et al., 2021). We conducted a 3-level meta-analysis assuming that effect sizes (level 2) were nested within each study (level 3).

Heterogeneity in effect sizes was computed through *Q* statistic (Hedges and Olkin, 1985) and a multi-level version of I^2 index (Cheung, 2014). The advantage of conducting a 3-level model was statistically demonstrated by comparing the Akaike (AIC) and Bayesian Information Criterion (BIC) indexes of a 3-level model with a reduced 2-level model together with the application of a likelihood ratio test (LRT) between models. Three-level mixed-effect models were estimated in order to test the impact of several variables on effect sizes of each risk factor domain. Specifically, it was estimated the impact of: i) year of publication; ii) research design (prospective vs retrospective) iii) sample size; iv) gender (men + females vs only females vs only men); v) sample characteristics (clinical vs non-clinical); vi) age at the moment of BPD evaluation; vii) years between risk factors assessment and BPD evaluation; viii) assessment procedures (self-report vs interview vs external sources). According to the hypotheses of study, we also conducted a meta-regression exploring the effects of internalizing and externalizing psychopathological manifestations on effect sizes reflecting the underlying liabilities. Similarly, we investigated the moderating effects of specific invalidating relational traumatic experiences (i.e., traumatic vs maladaptive parenting vs maladaptive peer relationships). The *Z*-test method (Borenstein et al., 2011) using an adequate Bonferroni correction was applied for subgroup comparisons. Egger's regression (Egger et al., 1997) was estimated to detect publication bias. Bootstrap methodology (i.e., bias corrected and accelerated; Davison and Hinkley, 1997) was applied in computing the significance of the previous parameter. These meta-analytic procedures were also applied within each domain of interest separately considered prospective and retrospective studies in order to further control possible confounding effects due to systematic memory bias related to retrospective evaluations found among BPD patients (Ebner-Priemer et al., 2006; Mneimne et al., 2021).

3. Results

3.1. Descriptive statistics

Fig. 2 graphically summarizes the inclusion process of studies. A detailed description of characteristics of studies are reported as supplementary materials (see Table 1s). One hundred six studies were included for a total of 86,871 participants. Fifty (47.2 %) studies evaluated associations between developmental psychopathology symptoms and later BPD features. Sixty (56.7 %) studies assessed invalidating interpersonal experiences as antecedents of BPD clinical characteristics. Eighteen (17.0 %) studies provided temporal associations between altered regulatory mechanisms and BPD features. Sixty-two (58.4 %) studies prospectively investigated relationships between risk factors and later BPD characteristics. Whereas, 44 (41.5 %) studies retrospectively evaluated antecedents of BPD. Fifty-eight (54.7 %) studies included participants from clinical settings; whereas, 48 studies (45.3 %) recruited individuals from the general population. The mean age of participants was 22.86 (SD = 8.38) years old at the moment of BPD assessment. The mean of time frame between risk factors evaluation and BPD assessment was 12.17 (SD = 8.33) years. Table 1 provides descriptive statistics of studies included.

3.2. Intrinsic factors: underlying liabilities to psychopathology

Considering together prospective and retrospective studies, we found small-to-moderate and significant association (r_{pooled} = 0.24 [0.20–0.28]; $p < .001$) between developmental psychopathology manifestations and later BPD features (for details see: Table 2s-3s). The heterogeneity ($Q_{(164)} = 3393.31$; $p < .001$) within ($I^2 = 32.58$ %) and between ($I^2 = 62.87$ %) studies was significant. Meta-regression analyses showed 3 significant moderators of effect sizes:

i) Larger effect sizes (Z = 2.68; $p < .01$) were observed in retrospective studies (r_{pooled} = 0.33 [0.25–0.41] $p < .001$) than in prospective ones (r_{pooled} = 0.21 [0.17–0.25] $p < .001$);
ii) Sample size was negatively related to effect sizes ($b = -0.00001$; $p < .01$);
iii) The age of BPD assessment was positively related to the effect sizes (older age at diagnostic assessment, higher effect size; b = 0.009 [0.004–0.01]; $p < .001$). This represented the best fit model

Interestingly, the domain of developmental psychopathology (i.e., internalizing vs externalizing) did not represent a significant moderator. Egger's regression showed a bias of publication (b = 2.92; 95 % bootstrap CI [1.76–4.04]; $p < .001$).

Table 3s reports detailed results of meta-analytic procedures separately conducted for prospective and retrospective studies. Moderation analysis of prospective studies confirmed a significant positive relationship between the age of BPD assessment and effect sizes reflecting temporal association between underlying liabilities to psychopathology and the later emergence of BPD features. This finding was not replicated for retrospective studies. The heterogeneity of findings remained large and significant for both prospective and retrospective studies. Bias of publication was found in both types of research design.

3.3. Environmental factors: invalidating relational experiences

Overall, invalidating relational experiences highlighted a small-to-moderate and significant associations with later BPD features (r_{pooled} = 0.28 [0.24–0.32] $p < .001$) (for details see: Table 4s-5s). A significant heterogeneity ($Q_{(249)} = 4219.40$; $p < .001$) of results was detected within ($I^2 = 23.02$ %) and between ($I^2 = 73.07$ %) studies. Meta-regression analysis found 2 significant moderators:

i) Childhood traumatic experiences (r_{pooled} = 0.30 [0.26–0.34]; $p < .001$) showed larger effect sizes (Z = 2.47; $p < .01$) than maladaptive parenting styles (r_{pooled} = 0.23 [0.18–0.28]; $p < .001$). No significant differences were observed between the effect of maladaptive peer relationships (r_{pooled} = 0.26 [0.16–0.34]; $p < .001$) and of the other invaliding relational experiences;
ii) Clinical samples highlighted larger (Z = 2.49; $p < .01$) associations between invaliding relational experiences and later BPD features (r_{pooled} = 0.32 [0.27–0.37]; $p < .001$) than samples recruited from general population (r_{pooled} = 0.23 [0.15–0.43]; $p < .001$). This represented the best fit model.

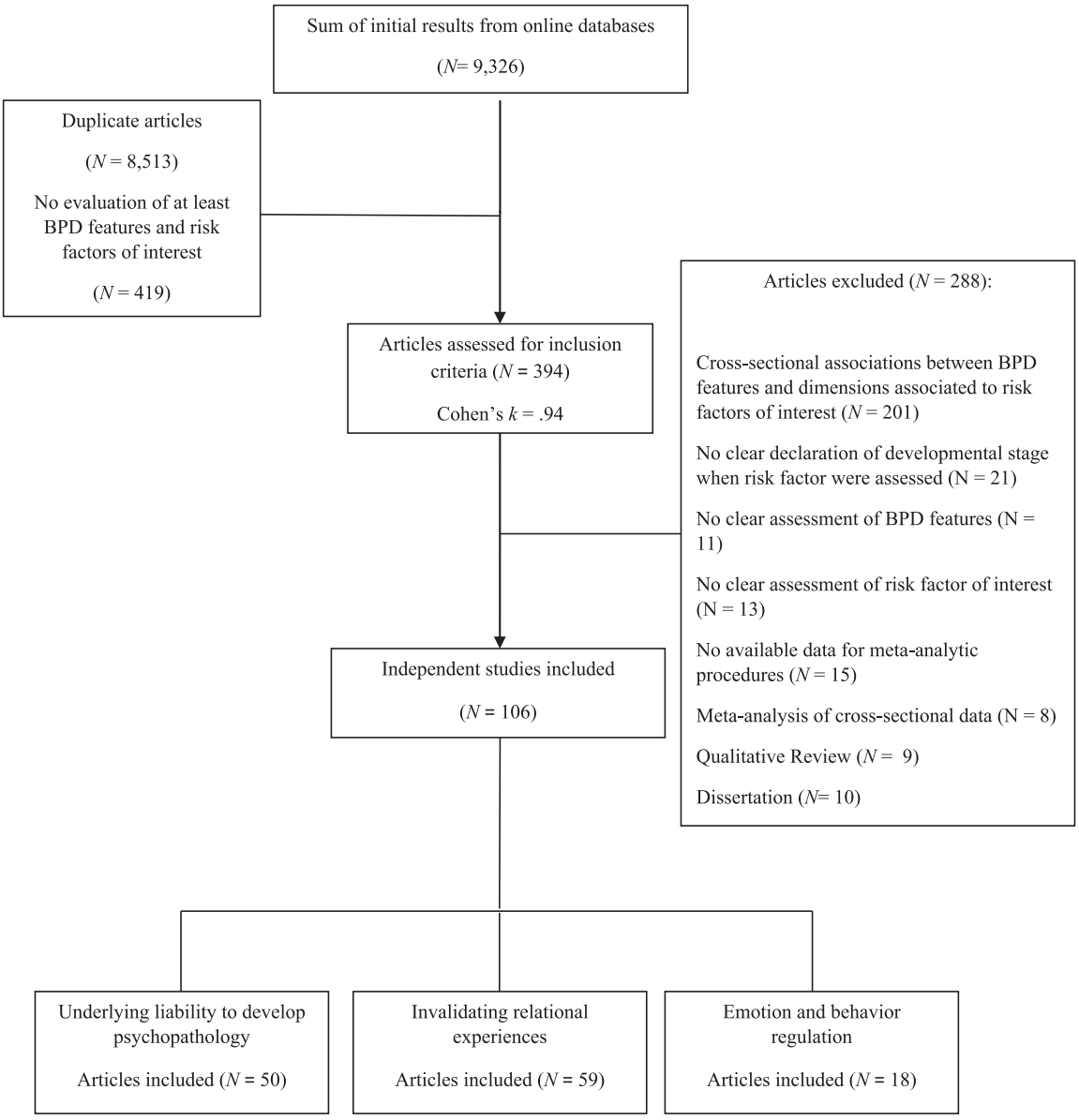

Fig. 2. CONSORT flow chart of studies inclusion process.

Bias of publication was not detected (Egger's coefficient: $b = 0.46$; 95 % bootstrap CI [−0.60–1.62]; *ns*).

Table 5s summaries findings of meta-analytic procedures separately conducted for prospective and retrospective studies. First, the estimation of overall pooled effect sizes found that prospective studies ($r_{pooled} = 0.20$ [0.16–0.23]; $p < .001$) showed significant smaller associations ($Z = -2.77$; $p < .01$) between invalidating relational experiences and later BPD features than retrospective ones ($r_{pooled} = 0.30$ [0.27–0.32]; $p < .001$). The heterogeneity of results remained large and significant for both types of research design. The moderating effect of specific invalidating relational experiences was replicated for retrospective studies, but not for prospective ones. The absence of bias of publication was replicated for both types of research design.

3.4. Regulatory factors: ER and BR mechanisms

Exploring alterations of regulatory processes (ER and BR) as antecedents of BPD features, we detected a significant and small-to-moderate association ($r_{pooled} = 0.28$ [0.19–0.36]; $p < .001$) (for details see: Table 6s-7s). However, there was significant variability ($Q_{(36)} = 1353.81$; $p < .001$) of findings within ($I^2 = 50.79$ %) and between ($I^2 = 46.63$ %) studies. The meta-regression analysis found 4 main moderators:

i) the age of BPD was positively related to effect sizes ($b = 0.02$ [0.002–0.03]; $p < .05$);
ii) the time frame between regulatory mechanisms and BPD features assessment was significantly and negatively related to effect sizes ($b = -0.01$ [−0.02 - -0.005]; $p < .01$);
iii) clinical samples showed larger ($Z = 1.70$; $p < .05$) effect sizes ($r_{pooled} = 0.38$ [0.26–0.51]; $p < .001$) than samples recruited from general population ($r_{pooled} = 0.21$ [0.05–0.37] $p < .01$).
iv) sample size was negatively associated to the extent of effect sizes ($b = -0.0001$ [−0.0001 - -0.0000]; $p < .01$). This was the best fit model.

The analysis detected significant bias of publication (Egger's coefficient: $b = 6.27$; 95 % bootstrap CI [4.08–8.30]; $p < .001$).

Table 7s reports meta-analytic results for prospective and retrospective studies. No significant differences ($Z = 0.63$; *ns*) were observed

Table 1
Descriptive statistics of studies included ($N = 106$).

	N	%	M (SD) [min – max]
Overall			
Total subjects	86,871		706.70 (1278.16) [28–7155]
Subjects from clinical samples	15,784		272.56 (418.42) [28–2450]
Subjects from general population	71,087		1182.52 (1692.66) [56–7155]
Studies from clinical samples	58	54.7	
Studies from general population	48	45.3	
Age of BPD assessment			22.86 (8.38) [10–51.80]
Time frame between risk factors assessment and BPD evaluation			12.17 (8.33) [1–40]
Prospective studies	62	58.4 %	
Retrospective studies	44	41.5 %	
Self-report assessment of BPD features	74	69.8 %	
Hetero-administered assessment of BPD features	32	30.2 %	
Self-report assessment of risk factors	58	54.7 %	
Hetero-administered assessment of risk factors	48	45.3 %	
Men + women	85	80.1 %	
Only women	18	17.0 %	
Only men	3	2.8 %	
USA	57	58.3 %	
Canada	4	3.8 %	
Brazil	1	0.9 %	
Germany	12	11.3 %	
United Kingdom	6	5.7 %	
Netherlands	5	4.7 %	
Italy	4	3.8 %	
Norway	3	2.8 %	
Denmark	2	1.9 %	
Spain	2	1.9 %	
Turkey	2	1.9 %	
Sweden	1	0.9 %	
Switzerland	1	0.9 %	
Japan	2	1.9 %	
China	1	0.9 %	
Korea	1	0.9 %	
Underlying liabilities to develop psychopathology			
Developmental psychopathology symptoms	50	–	
Internalizing psychopathology	27	54.0 %	
Externalizing psychopathology	41	82.0 %	
Total subjects	52,337		806.80 (1333.13) [50–7155]
Subjects from clinical samples	4128		187.63 (123.31) [50–524]
Subjects from general population	48,209		1293.28 (1629.64) [100–7155]
Studies from clinical samples	22	44.0 %	
Studies from general population	28	56.0 %	
Age of BPD assessment			21.94 (7.55) [10.11–43.20]
Time frame between risk factors assessment and BPD evaluation			9.92 (7.20) [0.50–30]
Prospective studies	39	78.0 %	
Retrospective studies	11	22.0 %	
Self-report assessment of BPD features	25	50.0 %	
Hetero-administered assessment of BPD features	25	50.0 %	
Self-report assessment of developmental psychopathology symptoms	38	76.0 %	
Hetero-administered assessment of developmental psychopathology	12	34.0 %	
Men + women	39	78.0 %	
Only women	9	18.0 %	
Only men	2	4.0 %	
Invalidating relational experiences			
Invalidating relational experiences	60	–	
Traumatic (i.e., sex/physical/emotional abuse, physical/emotional neglect)	46	76.7 %	
Maladaptive parenting styles (i.e., authoritarian, permissive, rejection, harsh punishment, psychological control, intrusiveness, low warmth)	8	13.3 %	
Maladaptive peer relationships (i.e., victim of bullying, conflicts with peer and friends, distressing romantic relationships)	6	10.0 %	
Total subjects	39,556		659.26 (1096.14) [24–6050]
Subjects from clinical samples	8821		252.02 (247.17) [24–986]
Subjects from general population	30,735		1229.40 (1512.20) [155–6050]
Studies from clinical samples	35	58.3 %	
Studies from general population	25	41.7 %	
Age of BPD assessment			24.58 (8.49) [11–51.80]
Time frame between risk factors assessment and BPD evaluation			16.82 (9.81) [0.50–46]
Prospective studies	25	41.7 %	
Retrospective studies	35	58.3 %	
Self-report assessment of BPD features	33	55.0 %	
Hetero-administered assessment of BPD features	27	45.0 %	
Self-report assessment of invalidating relational experiences	47	78.3 %	
Hetero-administered assessment of invalidating relational experiences	13	21.7 %	
Men + women	50	83.3 %	
Only women	9	15.0 %	
Only men	1	1.7 %	
Altered emotion and behavior regulation			
Altered emotion and behavior regulation	18	–	
Total subjects	21,667		1203.77 (1627.76) [52–5315]
Subjects from clinical samples	897		149.50 (73.77) [52–234]
Subjects from general population	20,771		1730.91 (1784.08) [100–5315]
Studies from clinical samples	6	33.3 %	

(*continued on next page*)

Table 1 (*continued*)

	N	%	M (SD) [min – max]
Studies from general population	12	66.7 %	
Age of BPD assessment			18.94 (5.41) [12–30]
Time frame between risk factors assessment and BPD evaluation			6.97 (6.13) [0.50–23]
Prospective studies	14	77.8 %	
Retrospective studies	4	22.2 %	
Self-report assessment of BPD features	5	27.8 %	
Hetero-administered assessment of BPD features	13	72.2 %	
Self-report assessment of altered ER/BR	12	66.7 %	
Hetero-administered assessment of altered ER/BR	6	33.3 %	
Men + women	14	77.8 %	
Only women	3	16.7 %	
Only men	1	5.6 %	

between pooled effect sizes of prospective ($r_{pooled} = 0.27$ [0.17–0.27]; $p < .001$) and retrospective ($r_{pooled} = 0.33$ [0.15–0.50]; $p < .001$) studies. The heterogeneity of findings was large and significant for both types of research design. There was replicated a significant negative relationship between sample size and effect sizes capturing the impact of altered regulatory mechanisms on the emergence of BPD for both prospective and retrospective studies. Similarly, bias of publication was detected in both types of research design.

3.5. Comparisons among domains of antecedents and risks factors for BPD

No significant differences emerged among pooled effect sizes related to underlying liabilities to psychopathology, invalidating relational experiences and regulatory mechanisms. This evidence was confirmed when there were compared pooled effect sizes of these domains separately considered results from prospective and retrospective studies.

4. Discussion

The current meta-analysis sought to clarify unresolved clinical and theoretical issues related to antecedents and risk factors for the emergence of BPD. In particular, we focused our attention on associations between i) early patterns of psychopathological manifestations, ii) different invalidating relational experiences, iii) alterations of ER/BR processes and the emergence of BPD in later stages of life.

Our meta-analytic results showed four main findings:

i) developmental psychopathology manifestations were moderately associated to later BPD features, especially when they were assessed among older individuals;
ii) independently of their nature, antecedent invalidating relational experiences showed a moderate association with BPD clinical characteristics, especially among clinical samples;
iii) regulatory mechanisms were moderately related to BPD features, especially considering small clinical samples;
iv) no significant differences among pooled effect sizes of different antecedents and risk factors for the emergence of BPD.

One of the most relevant findings is that larger associations between developmental psychopathology symptoms and later BPD features were detected when BPD was assessed among older individuals. This finding is in line with the mutualism theory of BPD development (Choate et al., 2021), which considers it as a result of evolving transactions across antecedent symptoms preceding the emergence of BPD (Sharp and Wall, 2018).

Moreover, looking at early psychopathology manifestations and their implications for the emergence of BPD, our results suggested that both internalizing and externalizing conditions are moderately involved in the development of BPD features. This finding might suggest two main conclusions. First, this could support evidence-based frameworks that have hypothesized how BPD may reflect a general predisposition to develop psychopathology ascribed to the well-established *p*-factor (Choate et al., 2023; Gluschkoff et al., 2021; Watts et al., 2020; Wilson and Olino, 2021), which has been associated to robust genetic determinants (for a review see: Smith et al., 2020). Additionally, the implications of internalizing and externalizing developmental psychopathology might be associated to the large clinical heterogeneity of BPD and its course over time (Shah and Zanarini, 2018), especially considering co-occurring disorders characterized by high levels of internalized negative affectivity (e.g., depressive disorders, anxiety disorders, obsessive-compulsive disorder, eating disorder) and externalized behavioral manifestations (e.g., substance use disorder, antisocial personality disorder, attention-deficit and hyperactivity disorder) (Ringwald et al., 2023). Nevertheless, the small-to-moderate pooled effect size of underlying liabilities to psychopathology suggested that other factors should be considered in order to support a comprehensive view of the emergence of BPD across life-span.

According to the previous consideration and clinical models of BPD (Bateman and Fonagy, 2004; Linehan, 1993), the current meta-analysis showed that invalidating relational experiences represented relevant antecedents of the disorder, replicating and extending previous literature that highlighted moderate relationships between childhood traumatic experiences and BPD in adulthood (Porter et al., 2020). Indeed, we also showed moderate associations between maladaptive parenting styles (e.g., authoritarian, permissive, rejection, harsh punishment, psychological control, intrusiveness, low warmth) and peer relationships (e.g., victim of bullying, conflicts with peer and friends, distressing romantic relationships). These results are in line with theoretical frameworks (e.g., Fruzzetti et al., 2005) and empirical evidence (e.g., Beeney et al., 2018; Lazarus and Cheavens, 2017; Stepp et al., 2009; Wolke et al., 2012) that have supported how core BPD manifestations are reinforced by detrimental interactions with significant others (e.g., parents, friends, teachers, partner) across different stages of development. Notably, childhood traumatic experiences showed larger effect sizes than maladaptive parenting styles, while no significant differences were detected comparing childhood traumatic experiences with invalidating peer relationships during adolescence. This latter result might provide a support for theoretical considerations (Sharp and Wall, 2018) that view adolescence as a sensitive period for the development of BPD taking into account the impacts of stressful relational events on pre-existing psychopathological liabilities and iterative processes of integration and organization of knowledge about self and others into a coherent whole, which represent key challenges during this specific developmental stage (Sebastian et al., 2009). Moreover, this observation is in agreement with current perspectives on biological effects of early stress on the developing brain, which define a sensitive time window for factors affecting both, brain structural connectivity, and subsequent emotional and behavioral discontrol, in a period encompassing late childhood and adolescence, also stressing a major role for peer relationships (e.g., Teicher et al., 2010), which affect the same structures detrimentally influences by parental abuse (Choi et al., 2009). These effects persist in the adult life of patients with mood disorders, detrimentally influencing psychopathology, brain structure and function, and outcomes (Benedetti et al., 2014; Poletti et al., 2022), and could then well jointly shape the cortico-limbic control of emotions and cognitive generation of affects, as observed in BPD (Vai et al., 2018). Interestingly, the model with the best goodness of fit to data showed that

invalidating relational experiences, independently of their quality, highlighted larger associations with BPD features among clinical samples than non-clinical ones. This finding might suggest that repeated relational invalidating experiences across different stages of development should represent relevant environmental factors involved in explaining the severity of general personality functioning (e.g., d'Huart et al., 2022; Ernst et al., 2022; Gander et al., 2020) and poor psychosocial adjustment (e.g., Fitzgerald et al., 2008; Kamsner and McCabe, 2000; Strøm et al., 2018).

Taking these findings, underlying liabilities to psychopathology and invalidating relational experiences should be simultaneously considered in the emergence of BPD features across life-span. Specifically, developmental psychopathology manifestations represent a substrate progressively reinforcing the emergence of BPD through repetitive transactions with invalidating relational contexts, which are involved in supporting the severity of personality functioning and maladjustment.

Meta-analytic results concerning alterations of ER/BR viewed as key processes at the base of transactions between underlying liabilities to psychopathology and invalidating environments (Kiff et al., 2011; Sameroff, 2009) confirmed a moderate effect of these dimensions in the emergence of BPD features. This finding might provide a partial support to a huge amount of cross-sectional data that highlighted large associations between maladaptive ER/BR and BPD (Bud et al., 2023; Daros and Williams, 2019; Sorgi-Wilson and McCloskey, 2022; Unoka and Richman, 2016), especially when clinical samples were considered. This evidence is fully in line with a biosocial theoretical framework (Linehan, 1993) and DSM-5 alternative model of BPD (APA, 2013) that viewed emotion dysregulation and behavioral dyscontrol as core features of the disorder, respectively. The meta-regression also detected a significant effect of time frame between regulatory mechanisms and BPD features: the larger is the time frame between regulatory mechanisms and BPD assessment, the lower are the associations between them. Whereas, we also found that the temporal associations between dysfunctional ER/BR and BPD features were larger when BPD was assessed among older subjects. These findings might suggest a dynamic nature of regulatory mechanisms across different stages of development (Constantinidis and Luna, 2019; Riediger and Bellingtier, 2022), which progressively reinforce themselves (especially maladaptive mechanisms) from childhood to late adolescence and adulthood (Cole et al., 2019) in emerging psychopathological conditions (Thompson, 2019), including BPD (Chapman, 2019; Hughes et al., 2012; Putnam and Silk, 2005).

The lack of significant differences among pooled effect sizes of antecedents and risk factors might provisionally suggest that the etiopathogenic theory of BPD with the best goodness of fit to data should be the transactional biosocial model (Linehan, 1993; Crowell et al., 2009), if it is compared to the other theoretical frameworks (i.e., excessive aggression, interpersonal hypersensitivity, failed mentalization), which have hypothesized a predominance of genetic or relational determinants, respectively.

Notably, the previously discussed findings were also confirmed when prospective and retrospective studies were separately analyzed. Nevertheless, prospective studies showed smaller, albeit significant, effect sizes than retrospective ones. This evidence is in line with empirical data that have demonstrated a substantial overlap between prospective and retrospective evaluations among BPD patients, even though the latter are characterized by a systematic, but modest, overestimation bias of psychopathological phenomena severity (Mneimne et al., 2021). Our meta-analytic results confirmed this systematic difference (~ 0.10) between prospective and retrospective effect sizes across all antecedents and risk factors for the emergence of BPD.

Despite this evidence, some limitations must be discussed. First of all, we detected a large heterogeneity of results for each domain of antecedents and risk factors, which remained unexplained even after controlling for the effect of several possible sources. This unexplained heterogeneity could be related to either the different assessment tools used in original studies (see Table 1s), or to the wide evidence-based heterogeneity of BPD manifestations themselves, especially referring to classical DSM criteria (Samuel and Griffin, 2015).

Secondly, the analysis found bias of publication for results concerning developmental psychopathology manifestations and alterations of regulatory processes. One possible source of this bias of publication might refer to the fact that studies with statistically significant results are more likely to be published than studies that report not statistically significant results (Dickersin, 2005). Furthermore, it could be possible to hypothesize a culture bias due to the fact that roughly 60 % of studies were conducted in North America (i.e., USA and Canada) and, up to 30 % of studies were conducted in European countries (e.g., Germany, Italy, Denmark, the Netherlands). Finally, there is a lack of a sufficient number of original studies to conduct a cross-lagged meta-analysis (Kuiper and Ryan, 2020) to effectively summarize longitudinal transactional effects between early developmental psychopathological manifestations, invalidating relational experiences and altered ER/BR in the emergence of BPD across life-span.

On the one hand, developmental psychopathology approach posits a key role of different protective processes (e.g., resilience) in explaining individual differences of adaptive and maladaptive developmental pathways (Masten et al., 2021; Masten and Tellegen, 2012). On the other hand, there is a lack of available empirical literature that have explored the impact of several protective factors on the complex transactions among investigated domains of antecedents and risk factors involved in the emergence of BPD. This represents an additional limitation in order to provide a comprehensive evidence-based etiopathogenic model of BPD. Therefore, future longitudinal studies should systematically evaluate how risk and protective factors dynamically interact to each other across life-span in order to explain the heterogeneity of developmental pathways of BPD.

This is the first meta-analysis that quantitatively evaluated the temporal impacts of the most relevant antecedents and risk factors for the emergence of BPD, taking into account different etiopathogenic theories together with developmental psychopathology principles. Results support a transactional developmental model of BPD. Accordingly, the emergence of BPD could be viewed in the light of dynamic interplays between an underlying liability to psychopathology and invalidating relational experiences across different stages of development, which are progressively reinforced through increasing alterations of emotion and behavior regulation mechanisms (see Fig. 3 for a graphical summary). This evidence might support an alternative conceptualization of BPD diagnosis that should be focused on the evaluation of developmental pathways of the previously mentioned domains together with the effects of protective factors (e.g., resilience) and their dynamic interactions across life-span, especially referring to equifinality and multifinality principles at the base of developmental psychopathology approach. This should be adopted in order to surpass current categorical and trait-based diagnostic systems that do not allow to fully understand the large heterogeneity of developmental outcomes and clinical course of BPD across life-span (Álvarez-Tomás et al., 2019; Winsper, 2021). Furthermore, current meta-analytic results, especially those related to a moderate impact of invalidating relational experiences on the emergence of BPD, support the ongoing and not definitive debate concerning the differential diagnosis between BPD and complex post-traumatic stress disorder (Cavicchioli et al., 2023a, 2023b; Cloitre et al., 2014; Ford and Courtois, 2021), which should be differentiated to each other on the base of different developmental histories and presence of repetitive interpersonal traumatic experiences (e.g., childhood sex abuse, physical abuse and neglect) predominantly characterizing complex post-traumatic stress disorder rather than BPD (Frost et al., 2020; Jowett et al., 2020; Scalabrini et al., 2024).

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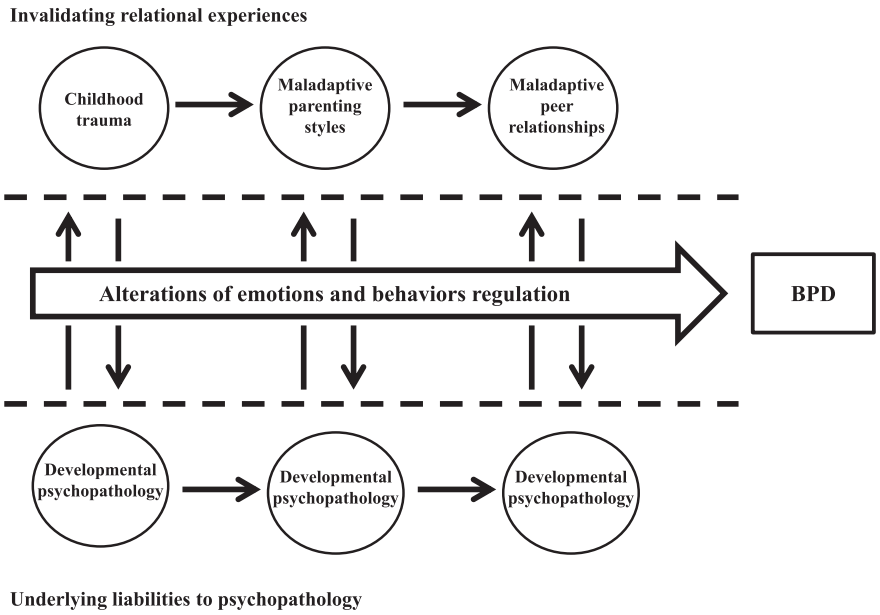

Fig. 3. The transactional developmental model of BPD.

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CRediT authorship contribution statement

Marco Cavicchioli: Writing – original draft, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Andrea Scalabrini:** Writing – original draft, Methodology, Investigation, Formal analysis, Conceptualization. **Ilaria Palumbo:** Writing – original draft, Methodology, Investigation, Formal analysis, Conceptualization. **Francesco Benedetti:** Writing – original draft, Methodology, Investigation, Formal analysis, Conceptualization. **Federica Galli:** Writing – review & editing. **Cesare Maffei:** Writing – review & editing.

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