European Psychiatry 61 (2019) 111–118

Contents lists available at ScienceDirect

European Psychiatry

journal homepage: http://www.europsy-journal.com

Original article

The association between childhood maltreatment and eating disorder psychopathology: A mixed-model investigation

Alessio Maria Monteleone[a,1,*], Giammarco Cascino[b,1], Francesca Pellegrino[a], Valeria Ruzzi[a], Giuseppina Patriciello[a], Luigi Marone[a], Gianmarco De Felice[a], Palmiero Monteleone[b], Mario Maj[a]

[a] Department of Psychiatry, University of Campania "Luigi Vanvitelli", Naples, Italy
[b] Department of Medicine, Surgery and Dentistry 'Scuola Medica Salernitana', University of Salerno, Salerno, Italy

ARTICLE INFO

Article history:
Received 27 May 2019
Received in revised form 8 August 2019
Accepted 9 August 2019
Available online 19 August 2019

Keywords:
Eating disorders
Network analysis
Mediation analysis
Psychopathology
Childhood maltreatment

ABSTRACT

Background. Childhood maltreatment (CM) is recognized as a non-specific risk factor for Eating Disorders (EDs), but the mechanisms explaining this association have been insufficiently assessed. We aim to explore the psychological pathways through which CM experiences promote ED core symptoms.

Methods. Two-hundred-twenty-eight people with EDs, 94 with anorexia nervosa restricting (ANR) type and 134 with binge-purging (BP) symptoms (including 23 with AN purging type and 111 with bulimia nervosa), completed the Eating Disorder Inventory-2, the State-Trait Anxiety Inventory and the Childhood Trauma Questionnaire. The variables provided by these questionnaires were included in a network analysis to identify the shortest pathways between CM nodes and ED core symptoms. Then mediation analysis was performed in order to confirm the mediation role of the nodes included in the shortest pathways from CM to ED core symptoms.

Results. All types of CM experiences were connected to the ED psychopathology through emotional abuse. In the ANR group, interoceptive awareness was included in the shortest path between emotional abuse and drive to thinness and mediated this relationship. In the BP group, the shortest routes between CM and ED core symptoms included both ineffectiveness and interoceptive awareness.

Conclusions. Combining the network analysis approach with the mediation analyses provides for the first time a putative hybrid model, which reveals that all CM types converge towards ED symptoms through emotional abuse and that interoceptive awareness and ineffectiveness mediate these connections in people with ANR and BP symptoms, respectively. These findings may have possible implications for both research and treatment of EDs.

1. Introduction

Childhood maltreatment (CM) has largely been recognized as an important non-specific risk factor for Eating Disorders (EDs) [1]. Compared to both healthy subjects and people suffering from other psychiatric disorders, people with EDs report a higher prevalence of CM, regardless of its nature and of the type of the ED, although the association between CM and EDs seems to be less pronounced for subjects with anorexia nervosa (AN) restricting type [2]. In addition, maltreated individuals with EDs were seen to be characterized by a more severe clinical presentation in terms of earlier age at onset, symptom severity, psychiatric comorbidities and suicidal/self-harm behaviors [3].

Among the different types of CM emotional abuse has shown a direct connection with ED symptoms while sexual and physical abuse have been associated to ED psychopathology through the mediation of one or more psychiatric comorbidities. The association between emotional abuse and EDs is corroborated also by the findings of Molendijk et al [3] who have reported that AN and BN are more strongly connected with emotional abuse than with other types of CM. The potential mechanisms sustaining the association between CM and the risk of developing an ED have been partially investigated. From a psychopathological perspective, Trottier and MacDonald [4] suggested that emotion dysregulation or maladaptive beliefs promoted by early adverse experiences are the mechanisms more closely associated with the ED symptoms. From a neurobiological point of

* Corresponding author at: Department of Psychiatry, University of Campania "Luigi Vanvitelli", Largo Madonna delle Grazie, 80138, Napoli, Italy.
E-mail address: alessio.monteleone@fastwebnet.it (A.M. Monteleone).
[1] These authors contributed equally to the paper.

http://dx.doi.org/10.1016/j.eurpsy.2019.08.002
0924-9338/

view, the endogenous stress response system seems a putative mediator between CM and the risk of developing an ED, as recently highlighted by two studies showing a reduced cortisol awakening response and an impaired response of the hypothalamus-pituitary-adrenal axis to an acute psychosocial stressor in maltreated people with EDs [5,6]. In addition, reduced grey matter volume in the right paracentral lobule and in the left inferior temporal gyrus as well as reduced white matter integrity seem to be potential biological scars of CM in EDs [7]. Thus, psychopathological, biological and clinical data appear to suggest the presence of a maltreated *ecophenotype* in EDs like in other psychiatric conditions [8]. Despite this evidence, it is not clear whether each type of CM is a risk factor for specific or for all ED symptoms nor which clinical pathways account for these associations.

In the last few years, the network analysis (NA) approach has been changing the conceptualization of psychopathology and also providing prognostic and therapeutic implications [9,10]. Indeed, the network theory considers mental disorders as the product of symptom interactions. Their interplay constitutes feedback loops, which maintain a state of prolonged activation of symptoms that is phenomenologically recognized as the disorder [11]. However, the network theory does not rule out that common causes may induce some symptoms that, in turn, promote the onset of others and together contribute to the maintenance of the disorder [12]. In these so-called "hybrid models", a common cause accounts for certain symptoms, which then promote other symptoms through a network process [13]. Thus, symptoms may be influenced by external factors, which are conditions outside the psychopathological network but not outside the person [9]. A NA investigates the relationships between symptoms, identifies the strongest connections as patterns of co-activation between symptoms, and explores the associations of symptoms with variables in the external field [9]. The subjective experience of CM is a potential variable belonging to the external field. Therefore, the use of NA may allow us to identify the possible pathways between each CM type and ED symptoms. A possible limitation of this approach is the inability to draw causal inference from the interactions among variables assessed as cross-sectional data [12]. Thus, in this study, we have integrated the NA with the mediation analysis approach [14] in order to build up a comprehensive investigation of the pathways through which CM experiences operate to promote the activation of ED symptoms. Indeed, the mediation analysis is a highly validated method, which makes possible to elucidate how the potential causal effect of CM experiences on ED symptoms operates through the psychopathological nodes identified in these pathways by the network analysis. Given the lack of agreement in literature data regarding the association between CM types and ED-specific symptoms and/or diagnoses [2,3], we have considered our study exploratory and no a priori hypotheses were formulated.

2. Methods

2.1. Participants

Consecutive patients attending the Eating Disorder Center of the Department of Psychiatry at the University of Campania "Luigi Vanvitelli" were included in the study if they met the following criteria: a) female gender; b) age $\geq$ 18; c) current diagnosis of AN, atypical AN or bulimia nervosa (BN), according to DSM-5 and confirmed by the Structured Clinical Interview for DSM-5 Disorders–Research Version (SCID) [15]; d) absence of current/lifetime comorbid diagnosis of schizophrenia, bipolar disorder, substance abuse disorder; e) willingness to cooperate in the experimental procedures and to sign a written informed consent. All patients were tested before entering specific treatment programs. Diagnostic assessment was made at the moment of referral to the outpatient unit by a trained psychiatrist (A.M.M.) who first made the diagnosis through a routine clinical interview and then used the SCID to confirm the ED diagnosis and, eventually, psychiatric comorbidity.

The ED sample was composed of 228 women: 80 suffering from AN restrictive type (ANR), 14 with atypical AN, 23 with AN purging type and 111 suffering from BN. Given the reported less pronounced prevalence of CM in patients with AN restricting type in comparison to other EDs [2,3], we have distinguished participants with ANR from those with AN purging type, and because of the low number of the latter in our sample, we have merged these patients with those suffering from BN in a single "bingeing-purging" (BP) group. This is reasonable on the light of the evidence that people with AN purging type have a pattern of association to CM more similar to people with BN than to those with AN restricting type [3].

2.2. Procedure

Sociodemographic, psychopathological and clinical data were collected through a face-to-face interview by expert psychiatrists.

Each participant in the study was asked to fill in the following questionnaires: 1) the *Eating Disorders Inventory*-2 (EDI-2) [16]; 2) the *State-Trait Anxiety Inventory* (STAI) [17]; 3) the *Childhood Trauma Questionnaire* (CTQ) [18].

The EDI-2 [16] evaluates ED symptomatology and psychopathology. The questionnaire includes 11 subscales: ineffectiveness, social insecurity, drive to thinness, interoceptive awareness, maturity fear, body dissatisfaction, perfectionism, interpersonal distrust, impulsivity, bulimia and ascetism. In our sample, Cronbach's values ranged from 0.75 (maturity fears) to 0.94 (ineffectiveness).

The STAI [17] is a self-report 40-item questionnaire which evaluates two types of anxiety: state anxiety, referring to temporary feelings perceived at the time of a threat, and trait anxiety, indicating how people feel in typical everyday situations. Cronbach's values resulted to be 0.86 for STAI-state subscale and 0.91 for the trait subscale.

The CTQ [18] explores a self-report recall of childhood trauma experiences. It is a 28-item questionnaire which identifies childhood experience across five types of CM: emotional neglect (EN) (cut-off score $\geq$15; Cronbach's α = 0.88), emotional abuse (EA) (cut-off score $\geq$10; Cronbach's α = 0.85), sexual abuse (SA) (cut-off score $\geq$ 8; Cronbach's α = 0.74), physical neglect (PN) (cut-off score $\geq$ 8; Cronbach's α = 0.78) and physical abuse (PA) (cut-off score $\geq$ 8; Cronbach's α = 0.71). The reported cut-off scores indicate the occurrence of that type of maltreatment [19]. In our network we included a sum-score for each sub-scale [20] given that continuous scales are considered the best measures for network analyses [12]. The use of continuous measures to evaluate trauma is largely documented in literature studies [4] and also employed in network analysis studies [21].

The study was approved by the Institutional Board of the University of Campania L. Vanvitelli.

2.3. Statistical analysis

An independent samples *t*-test was used to compare age, BMI, illness duration, age at onset, EDI-2, CTQ scores between the AN restricting group and the binge-purging group. The presence/absence of each type of CM was compared between the study groups by the Chi-square test.

2.4. Network analyses

NA was performed through R, version 3.4.4, using *qgraph* package [22]. A network is composed of nodes, which represent

https://doi.org/10.1016/j.eurpsy.2019.08.002 Published online by Cambridge University Press

the measured variables, and edges, which are the connections among them. We have included the following nodes in our network: the EDI-2 subscores, the CTQ items and the state and trait scores of the STAI. Network associations are undirected, i.e. there is no direction in the association. The thickness of an edge graphically represents the magnitude of the association. In this study, we have estimated partial-correlation networks, where the association between two nodes is controlled for the influence of all other variables [23]. We have built two separate networks: one for the sample with ANR and one for the BP group. In order to retain only meaningful associations, we applied a "least absolute shrinkage and selection operator" (LASSO) regularization [24], which shrinks small partial correlations and sets them to zero [10]. The Extended Bayesan Information Criterion (EBIC) [25], a parameter that sets the degree of regularization/penalty applied to sparse correlations, was set to 0.5 in these analyses.

We then estimated the accuracy of edge-weights, by drawing bootstrapped confidence intervals calculated through "nonparametric" bootstrapping (nboots = 2500) [26], by means of the *bootnet* package [27].

Next, we computed two networks (one for each study group) showing the shortest paths between each childhood trauma node, as assessed by the CTQ, and the ED core symptoms (drive to thinness, body dissatisfaction and bulimia) evaluated by means of the EDI-2. The shortest path between two nodes represents the minimum number of steps needed to go from one node to the other [28], computed using Dijkstra's algorithm [29]. This kind of network allows us to identify the mediating variables between early traumatic experiences and specific symptoms of EDs. Indeed, it detects the shortest path between two nodes, i.e. the quickest out of all the routes connecting these two nodes. The undirected edges used in this network indicate conditional dependence between two variables: the edge-weight parameters reflect the strength of unique associations between variables, which in turn may highlight potential causal relationships [26,21].

2.5. Mediation analyses

Mediation analyses were conducted using the PROCESS macro for SPSS version 3.1 [14]. We followed the Baron and Kenny method [30] to confirm whether the CM experiences (the independent variables) predict ED core symptoms (body dissatisfaction, drive to thinness and bulimia; the dependent variables) through the mediation of those nodes identified in our network analyses as those included in the shortest pathways between CM and ED core symptoms. Indeed, mediation analysis focuses on specific pathways of influence on one or more dependent variables that need to be identified a priori. Since we had not a robust priori hypothesis from literature data, we used the network analysis to identify those symptoms that, in the global structure of ED psychopathology, convey the association between CM experiences and ED core symptoms. The statistical significances of the mediating and indirect effects were assessed using bootstrapped bias-corrected percentile based confidence interval of 5000 bootstrap draws [31]. All analyses were performed using SPSS version 25 (SPSS Inc, Chicago, IL).

3. Results

3.1. General results

Clinical characteristics of the study samples are reported in Table 1. A comorbid anxiety disorder was detected in 61 subjects while 68 individuals were diagnosed with a comorbid depressive disorder. The independent samples *t*-test (Table 1) showed that, compared to women with ANR, people with BP behaviors reported higher age and BMI and scored higher in the body dissatisfaction, drive to thinness, bulimia, interoceptive awareness and ascetism sub-scores of the EDI-2 questionnaire. Higher values were also detected in the BP sample for CTQ emotional neglect and physical abuse scores and for STAI trait value.

Table 1
Demographic and clinical characteristics of the study groups and independent sample *t*-test.

	ANR (*M*±*SD*)	BP (*M*±*SD*)	*t*	*P*
Age	25.94 ± 7.64	28.59 ± 9.44	−2.13	0.03
Age of Onset	17.61 ± 4.19	18.65 ± 5.3	−1.47	0.14
Illness Duration	7.87 ± 7.55	10.15 ± 8.47	−1.94	0.05
BMI	17.44 ± 2.69	22.48 ± 7.01	−7.12	<0.01
EDI2 IN	11.81 ± 7.46	13.77 ± 7.86	−1.89	0.06
EDI 2 MF	10.02 ± 5.84	8.72 ± 5.86	1.65	0.10
EDI 2 SI	8.96 ± 4.69	8.54 ± 4.35	0.68	0.49
EDI 2 BD	13.43 ± 6.85	16.29 ± 6.94	−3.09	<0.01
EDI 2 P	7.16 ± 4.33	7.19 ± 4.5	−0.05	0.95
EDI 2 ID	8.15 ± 4.68	7.40 ± 4.82	1.17	0.24
EDI 2 IR	7.93 ± 6.44	10.52 ± 7.55	−2.79	<0.01
EDI 2 DT	13.50 ± 6.96	15.31 ± 5.49	−2.09	0.03
EDI 2 BU	2.38 ± 3.18	10.69 ± 5.99	−13.54	<0.01
EDI 2 IA	12.26 ± 7.15	15.14 ± 7.31	−2.97	<0.01
EDI 2 ASC	7.71 ± 4.49	9.31 ± 4.27	−2.70	<0.01
CTQ EN	11.30 ± 4.91	13.33 ± 4.55	−3.17	<0.01
CTQ EA	8.97 ± 4.4	9.80 ± 4.41	−1.40	0.16
CTQ SA	5.76 ± 2.63	6.28 ± 3.01	−1.37	0.17
CTQ PN	6.54 ± 2.72	6.90 ± 2.04	−1.11	0.26
CTQ PA	5.91 ± 2.29	6.65 ± 2.69	−2.21	0.02
STAI-S	57.26 ± 11.96	56.64 ± 13.45	0.35	0.72
STAI-T	56.84 ± 9.66	59.45 ± 9.05	−2.05	0.04

BMI, Body Mass Index; EDI, Eating Disorders Inventory; IN, Ineffectiveness; MF, Maturity Fear; SI, Social Insecurity; BD, Body Dissatisfaction; P, Perfectionism; ID, Interpersonal Distrust; IR, Impulse Regulation; DT, Drive for Thinness; BU, Bulimia; IA, Interoceptive Awareness; ASC, Asceticism; CTQ, Childhood Trauma Questionnaire; EN, Emotional Neglect; EA, Emotional Abuse; SA, Sexual Abuse; PN, Physical Neglect; PA, Physical Abuse; STAI-S, State and Trait Anxiety Inventory – State; STAI-T, State and Trait Anxiety Inventory – Trait.

The Chi-square results showed that the distribution of each CM type was not statistically different between the ANR and the BP groups, with the exception of emotional neglect and sexual abuse, which exhibited, respectively, a higher ($X^2 = 4.38$, $p = 0.03$) and a trend towards higher prevalence in the BP group ($X^2 = 3.52$, $p = 0.06$) (Table 2).

3.2. Network analyses

The network in Fig. 1 shows the connections between childhood trauma experiences and the EDI-2 and STAI scores in the ANR group. From the analysis of the network, it emerged that there were no direct connections between CM nodes and ED symptoms, except for the connection between emotional abuse and interoceptive awareness. This node was also directly connected with impulse regulations and with the ED core symptom drive to thinness. Moreover, all CM nodes were highly interconnected between each other, while the STAI nodes had weak connections only with EDI-2 ineffectiveness and social insecurity nodes.

Table 2
Frequency (%) of different types of trauma assessed by Childhood Trauma Questionnaire in the Anorexia Nervosa restricting subtype group (ANR) and in the binge-purging group (BP).

	AN R (n = 94)	BP (n = 134)	P*
Emotional Abuse	39.4	41.8	0.713
Emotional Neglect	25.5	38.8	0.036*
Physical Abuse	14.9	23.9	0.096
Physical Neglect	23.4	33.6	0.097
Sexual Abuse	8.5	17.2	0.061

* Pearson's chi-squared test.

https://doi.org/10.1016/j.eurpsy.2019.08.002 Published online by Cambridge University Press

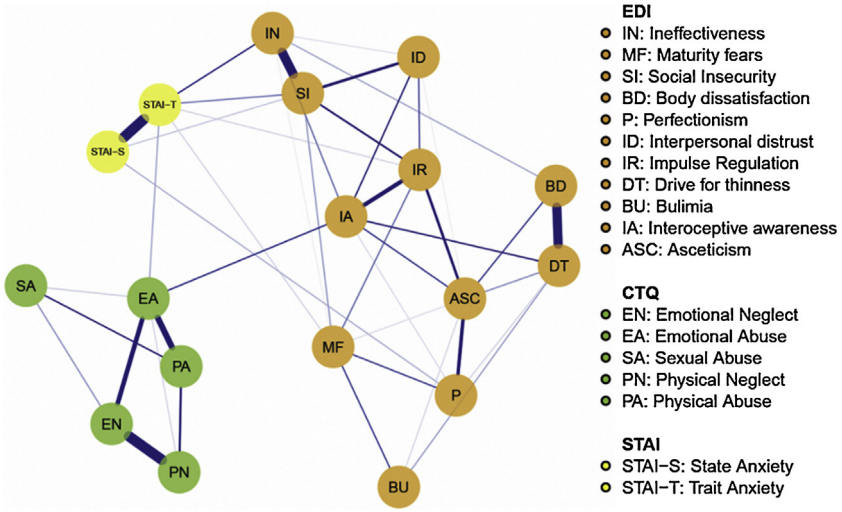

Fig. 1. Restrictive Group Network.
Estimated network of the restrictive AN group depicting the 5 dimensions of Childhood Trauma Questionnaire, the Eating Disorders Inventory-2 subscores and State and Trait Anxiety Inventory scores. Each edge within the network corresponds to a partial correlation between two nodes. For color version, see this figure online.

The network in Fig. 2 illustrates the connections between childhood trauma experiences and EDI-2 and STAI scores in the BP group. As in the ANR network, CM nodes were highly connected between each other and not connected with ED symptoms, except for emotional abuse that was connected with ineffectiveness. This node, in turn, was positively associated with interoceptive awareness, body dissatisfaction and social insecurity.

The *shortest pathways* analysis identified the shortest route between the CTQ nodes and the EDI-2 core symptoms. Fig. 3 illustrates these pathways in the ANR group. In this network, the shortest path to reach drive to thinness from each type of CM was via emotional abuse and interoceptive awareness. Interoceptive awareness was also in the shortest path between emotional abuse and ineffectiveness or interpersonal distrust.

Fig. 4 depicts pathways between trauma and ED psychopathology in the BP group. The shortest path to reach body dissatisfaction and drive to thinness from CM nodes included emotional abuse and ineffectiveness, while the route to reach bulimic symptoms was via emotional abuse, ineffectiveness and interoceptive awareness. Similar to findings in the ANR group, all the pathways from CM nodes to ED psychopathology passed through emotional abuse. Contrary to the ANR group, ineffectiveness, instead of interoceptive awareness, was included in all the pathways from CM to ED symptoms.

In both study groups, anxiety nodes were not in the pathways between CM and ED core symptoms.

The bootstrapped confidence intervals of estimated edge-weights are reported in Supplementary Fig. 1 and 2.

3.3. Mediation analyses

The network analyses conducted in both ED samples pointed to emotional abuse as the node from which all other CM types can evoke ED symptoms.

In the ANR group interoceptive awareness was in the shortest route between childhood emotional abuse and AN core-symptoms (Fig. 3). Thus, we assessed whether the association between emotional abuse and drive to thinness was explained by an indirect effect of interoceptive awareness. A total mediation effect was

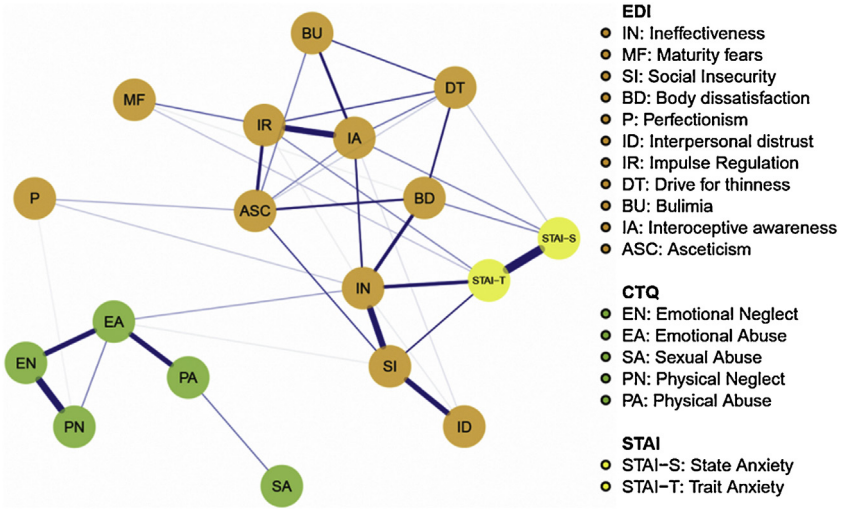

Fig. 2. Binge-purging Group Network.
Estimated network of the binge-purging group depicting the 5 dimensions of Childhood Trauma Questionnaire, the Eating Disorders Inventory-2 subscores and State and Trait Anxiety Inventory scores. Each edge within the network corresponds to a partial correlation between two nodes. For color version, see this figure online.

https://doi.org/10.1016/j.eurpsy.2019.08.002 Published online by Cambridge University Press

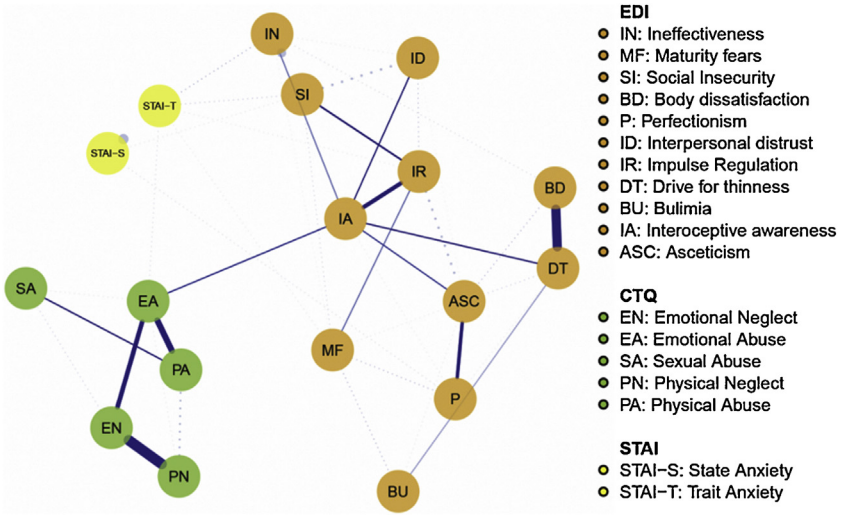

Fig. 3. Restricting Group Shortest Pathways.
Network illustrating the shortest path between childhood trauma dimensions and eating disorder symptoms in the restrictive group. For color version, see this figure online.

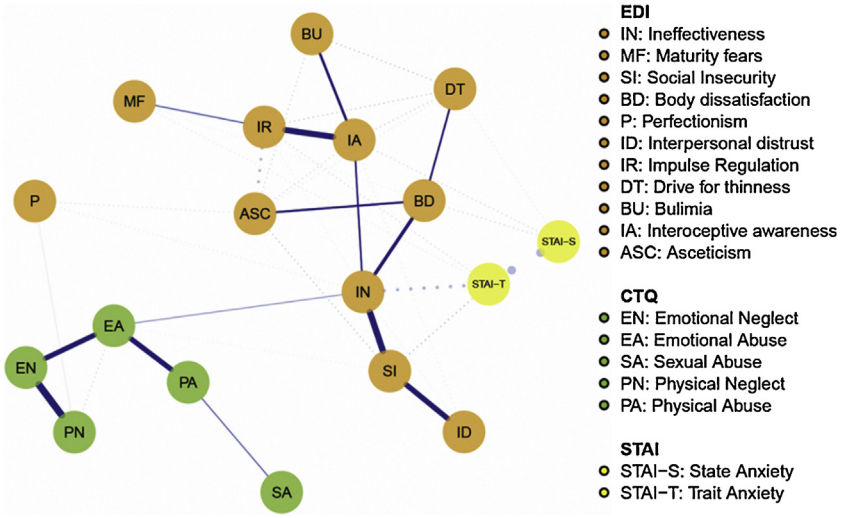

Fig. 4. Binge-purging Group Shortest Pathways.
Network illustrating the shortest path between childhood trauma dimensions and eating disorder symptoms in the binge-purging group. For color version, see this figure online.

revealed pointing to the association between emotional abuse and drive to thinness as completely mediated by the interoceptive awareness (Indirect Effect: $b = 0.28$, 95% C.I.: 0.11 – 0.54).

In the BP group (Fig. 4) the shortest pathway between emotional abuse and body dissatisfaction included ineffectiveness while both ineffectiveness and interoceptive awareness were in the shortest path between emotional abuse and bulimic symptoms. Thus, two mediation models were run. In the first, ineffectiveness totally mediated the relationship between emotional abuse and body dissatisfaction (Indirect Effect: $b = 0.29$, 95% C.I.: 0.13 – 0.49). The second one was a serial multiple mediator model [17]: emotional abuse was found to predict bulimic symptoms trough the contemporary mediation of ineffectiveness first and, then, interoceptive awareness (Indirect Effect: $b = 0.09$, 95% C.I.: 0.03 – 0.17). Ineffectiveness and interoceptive awareness were not significant independent mediators since the 95% CI for their indirect effect (between emotional abuse and bulimic symptoms) included zero (Indirect Effect for Ineffectiveness: $b = -0.01$, 95% C.I.: -0.1 – 0.05; Indirect Effect for Interoceptive Awareness: $b = 0.001$, 95% C.I.: -0.07 – 0.08).

4. Discussion

The present study provides, for the first time, network-based evidence of the associations between CM and ED psychopathology. By mean of the shortest pathways analysis, we have identified the childhood emotional abuse experience as the common node mediating the association between each type of CM and ED core symptoms. Moreover, in the ANR group interoceptive awareness was included in the shortest pathway between emotional abuse and drive to thinness, while in the BP group ineffectiveness was the psychopathological variable in the shortest route from emotional abuse to body dissatisfaction, and both ineffectiveness and interoceptive awareness were in the shortest pathway between emotional abuse and bulimic symptoms. The possible mediating role of these clinical variables (ineffectiveness and interoceptive awareness) between childhood adverse experiences and ED core symptoms has been confirmed through mediation analysis. By combining network and mediation analysis approaches we have potentially constructed a *hybrid* model, which accounts for the role of emotional abuse as a variable mediating between different early

https://doi.org/10.1016/j.eurpsy.2019.08.002 Published online by Cambridge University Press

adverse experiences and some ED symptoms whose interplay may, in turn, maintain a state of prolonged activation of further ED symptoms.

In our pathway analyses we have observed that the effect of each CM type can propagate to ED core symptoms exclusively through emotional abuse. This is in line with previous studies suggesting that different types of CM do not have the same impact on ED symptoms [32–34]. In particular, a recent study indicated a direct association between emotional abuse and ED core symptoms while the associations of other CM types with ED symptoms were dependent on co-occurring psychiatric comorbidities [35]. In addition, other studies [36,37] have hypothesized that emotional abuse promotes emotional deregulation, which significantly contributes to ED symptoms onset [38].

Present data do not show any difference between ANR and BP groups in the pathways from each CM type and ED psychopathology. In particular, sexual abuse was associated with emotional abuse through physical abuse in both groups, which is surprising given the literature data [2] pointing to a higher prevalence of sexual abuse in subjects with BP symptoms, as we have also observed in our sample. Therefore, although ED groups may differ in terms of the prevalence of some CM types [2], our study points out that the experience of early adversities, assessed from a dimensional perspective, may generate similar pathways accounting for the associations to ED psychopathology.

Our findings also show that some differences occur between people with ANR type and those with BP symptoms. In the former group, the shortest pathway between CM (emotional abuse) and ED core symptoms (drive to thinness) included interoceptive awareness while in the latter group ineffectiveness alone mediated the effects of emotional abuse on body dissatisfaction and both ineffectiveness and interoceptive awareness were in the pathway leading to bulimic symptoms. Moreover, in the BP group, the mediation analyses showed that ineffectiveness mediated the relationship between emotional abuse and bulimic symptoms only through the mediation of interoceptive awareness. Therefore, we can suggest that interoceptive awareness plays a central role in the complex interactions between CM and ED core symptoms. This is in line with Bruch's model [39] of AN, which has been corroborated both in a recent review [40] and in a previous study from our group [41]. Indeed, according to Bruch's theory, it seems plausible that early humiliating and self-demeaning behaviors as well as not responding to the needs of the children may compromise their ability to discriminate body sensations and feelings such as hunger and satiety. Moreover, interoceptive awareness reflects the ability to discriminate not only between hunger and satiety perception but also different body sensations and feelings [42]. This is particularly relevant on the light of the *somatic markers* theory, which claims that the recognition of the internal state is the foundation of our emotional feelings and is associated with the capacity to regulate behaviors [43,44]. In line with this, a wide range of studies has suggested that early trauma impact on emotion regulation skills and, in turn, may contribute to the development and maintenance of eating symptoms [4]. Conversely, unlike in the ANR group, in the BP group the central role of ineffectiveness provides a slightly different route to bulimic symptoms suggesting an interplay between emotional abuse, feelings of inadequacy and interoceptive recognition ability in this clinical condition.

The central role played by interoceptive awareness in the pathways from CM to ED core symptoms in both the ANR group and the BP group allows us to rule out, at least in part, a previous hypothesis suggesting that the link between certain types of CM and EDs is mediated by the occurrence of psychiatric comorbidities [35], since interoceptive awareness is a specific ED psychopathological dimension [45] and, in our NA, anxiety symptoms were not in the pathways from CM to ED core symptoms. However, our network did not include other general psychiatric symptoms (i.e. depressive or post-traumatic stress symptoms) which may also be involved in these pathways. Although this limitation, it is interesting to remark that a general psychopathological symptom, ineffectiveness, was found to be involved in the relationships between CM and ED core symptoms in BP individuals. In line with this evidence, previous studies showed that self-criticism or inadequacy, but not depression or anxiety, mediated between early emotional abuse and ED symptoms [46,36]. Moreover, our finding may partially explain the nature of CM as a non-specific risk factor for different psychopathological conditions [47,48,8]. The associations between ineffectiveness and ED core symptoms is in accordance with findings from recent NA studies which showed that feelings of worthlessness span ED symptoms and comorbid psychopathological variables [49,50] and predict treatment outcomes [49,51]. Our findings supplement this evidence with the possibility to consider early emotional abuse as a possible variable associated with self-negative evaluation in both clinical assessment and therapeutic interventions.

Some limitations of the present study need to be acknowledged. First, the use of cross-sectional data outlines the need to exercise caution when interpreting conclusions regarding the direction of causality effects although, as explained below, this limitation has been partially overcome by our statistical model. Second, in our study we were not able to include general psychiatric symptoms, such as depressive symptoms, which are frequent in EDs [52] and may be possible mediators of the connections between CM experiences and ED core symptoms. Third, a larger sample would have been appropriate to improve the strength of our findings in each ED group and in particular subsamples such as, for example, people with defined past event of sexual abuse or those who had a transdiagnostic cross-over or a diagnostic stability. Fourth, other variables, such as self-harm behaviors or quality of life, which have been found to be important outcomes when assessing the effects of CM in EDs [35,3], or PTSD symptoms, emotion deregulation or dissociation, which are often associated with early trauma, were not explored in our sample. Finally, the CTQ, that we used to assess early traumatic experiences, relies on the subjects' recollections without specification of the context and of the time of trauma occurrence: as a result, we cannot rule out that some participants had distorted memories of their early environment or chose not to disclose their maltreatment. This may have been responsible for some bias, although this limitation is not thought to be as critical as some might expect [53].

Our work presents some strengths also. Above all, it is the first research assessing the potential pathways between CM and ED core symptoms through a NA combined with pathways analysis. In addition, on the basis of the NA results, we have also run a mediation analysis yielding a possible *hybrid* model for the first time. The latter identifies symptoms connected to a possible common cause (CM) and those which directly perpetuate other symptoms. Second, we have performed our analyses in both the ANR group and the BP group, in accordance with literature recommendations [2,3], and we have highlighted similarities as well as differences between the two ED groups. Third, we share all the codes used so as to enable reproducibility of the study design (see the appendix).

To conclude, our study provides the first evidence for possible pathways mediating the relationships between childhood trauma exposure and ED core symptoms. Future studies are recommended to further explore these connections including data deriving from multiple levels of analyses (i.e. biological, behavioral and psychophysiological data) and putative resilience factors for CM, such as the quality of parental bonding [34]. From a clinical point of view, the present work provides significant therapeutic implications as it sheds light on putative interventions in the *external field*

https://doi.org/10.1016/j.eurpsy.2019.08.002 Published online by Cambridge University Press

[9] which refers to the associations between CM experiences and specific features of ED psychopathology. Indeed, in a recent study maltreated and non-maltreated ED patients showed different outcomes, particularly in terms of comorbid psychiatric symptoms persistence, two years after the completion of a cognitive-behavioral therapy [54]. Furthermore, a meta-analysis [55] highlighted the lack of superiority for cognitive-behavioral treatment in comparison to other bona fide therapies when assessing outcomes such as self-esteem and interpersonal problems in people with EDs. Thus, our findings suggest that, in the presence of childhood maltreatment, it may be important to extend intervention strategies beyond ED core symptoms, focusing on the person's self-esteem development, abilities to recognize inner body states and emotions and taking into account their putative mediating role between childhood adverse experiences and ED symptoms.

Funding sources

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Declaration of Competing Interest

None.

Acknowledgements

None.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.eurpsy.2019.08.002.

References

[1] Jacobi C, Hayward C, de Zwaan M, Kraemer HC, Agras WS. Coming to terms with risk factors for eating disorders: application of risk terminology and suggestions for a general taxonomy. Psychol Bull 2004;130(1):19.
[2] Caslini M, Bartoli F, Crocamo C, Dakanalis A, Clerici M, Carrà G. Disentangling the association between child abuse and eating disorders. Psychosom Med 2016;78(1):79–90, doi:http://dx.doi.org/10.1097/PSY.0000000000000233.
[3] Molendijk ML, Hoek HW, Brewerton TD, Elzinga BM. Childhood maltreatment and eating disorder pathology: a systematic review and dose-response meta-analysis. Psychol Med 2017;47(8):1402–16, doi:http://dx.doi.org/10.1017/S0033291716003561.
[4] Trottier K, MacDonald DE. Update on psychological trauma, other severe adverse experiences and eating disorders: state of the research and future research directions. Curr Psychiatry Rep 2017;19:45, doi:http://dx.doi.org/10.1007/s11920-017-0806-6.
[5] Monteleone AM, Monteleone P, Volpe U, De Riso F, Fico G, Giugliano R, et al. Impaired cortisol awakening response in eating disorder women with childhood trauma exposure: evidence for a dose-dependent effect of the traumatic load. Psychol Med 2018;48(6):952–60, doi:http://dx.doi.org/10.1017/S0033291717002409.
[6] Monteleone AM, Patriciello G, Ruzzi V, Cimino M, Del Giorno C, Steardo L, et al. Deranged emotional and cortisol responses to a psychosocial stressor in anorexia nervosa women with childhood trauma exposure: evidence for a "maltreated ecophenotype"? J Psychiatr Res 2018;104:39–45, doi:http://dx.doi.org/10.1016/J.JPSYCHIRES.2018.06.013.
[7] Monteleone AM, Monteleone P, Esposito F, Prinster A, Ruzzi V, Canna A, et al. The effects of childhood maltreatment on brain structure in adults with eating disorders. World J Biol Psychiatry 2017;1–10, doi:http://dx.doi.org/10.1080/15622975.2017.1395071.
[8] Teicher MH, Samson JA. Childhood maltreatment and psychopathology: a case for ecophenotypic variants as clinically and neurobiologically distinct subtypes. Am J Psychiatry 2013;170(10):1114–33, doi:http://dx.doi.org/10.1176/appi.ajp.2013.12070957.
[9] Borsboom D. A network theory of mental disorders. World Psychiatry 2017;16(1):5–13, doi:http://dx.doi.org/10.1002/wps.20375.
[10] McNally RJ. Can network analysis transform psychopathology? Behav Res Ther 2016;86:95–104, doi:http://dx.doi.org/10.1016/j.brat.2016.06.006.
[11] Cramer AOJ, Waldorp LJ, van der Maas HLJ, Borsboom D. Comorbidity: a network perspective. Behav Brain Sci 2010;33(2-3):137–50, doi:http://dx.doi.org/10.1017/S0140525X09991567.
[12] Levinson CA, Vanzhula IA, Brosof LC, Forbush K. Network analysis as an alternative approach to conceptualizing eating disorders: implications for research and treatment. Curr Psychiatry Rep 2018;20(9):67, doi:http://dx.doi.org/10.1007/s11920-018-0930-y.
[13] Bringmann LF, Eronen MI. Don't blame the model: reconsidering the network approach to psychopathology. Psychol Rev 2018;125(4):606–15, doi:http://dx.doi.org/10.1037/rev0000108.
[14] Hayes AF, Rockwood NJ. Regression-based statistical mediation and moderation analysis in clinical research: observations, recommendations, and implementation. Behav Res Ther 2017;98:39–57, doi:http://dx.doi.org/10.1016/j.brat.2016.11.001.
[15] First MB, Williams JB. SCID-5-CV: structured clinical interview for DSM-5 disorders: clinician version. Arlington, NE: American Psychiatric Association; 2016. https://www.appi.org/Structured_Clinical_Interview_for_DSM-5_Disorders-Clinician_Version_SCID-5-CV.
[16] Garner DM. Eating disorder inventory-2. Odessa, FL: Psychological Assessment Resources; 1991, doi:http://dx.doi.org/10.1177/1073191111428761.
[17] Spielberger CD, Gorsuch R, Lushene R, Vagg PR, Jacobs GA. Manual for the state-trait anxiety inventory (Form Y). Palo Alto, CA: Consulting Psychologists Press; 1983.
[18] Bernstein DP, Stein JA, Newcomb MD, Walker E, Pogge D, Ahluvalia T, et al. Development and validation of a brief screening version of the Childhood Trauma Questionnaire. Child Abuse Negl 2003;27(2):169–90, doi:http://dx.doi.org/10.1016/S0145-2134(02)00541-0.
[19] Walker EA, Gelfand A, Katon WJ, Koss MP, Von Korff M, Bernstein D, et al. Adult health status of women with histories of childhood abuse and neglect. Am J Med 1999;107(4):332–9, doi:http://dx.doi.org/10.1016/S0002-9343(99)00235-1.
[20] Bernstein DP, Fink L. Childhood trauma questionnaire : a retrospective self-report : manual. Orlando: Harcourt Brace & Co.; 1998.
[21] Epskamp S, Cramer AOJ, Waldorp LJ, Schmittmann VD. Borsboom D. qgraph: network visualizations of relationships in psychometric data. J Stat Softw 2012;48(4):1–18, doi:http://dx.doi.org/10.18637/jss.v048.i04.
[22] Epskamp S, Fried EI. A tutorial on regularized partial correlation networks. Psychol Methods 2018;23(4):617–34, doi:http://dx.doi.org/10.1037/met0000167.
[23] Friedman J, Hastie T, Tibshirani R. Glasso: graphical lasso-estimation of Gaussian graphical models. R package version 2014;1:.
[24] Chen J, Chen Z. Extended Bayesian information criteria for model selection with large model spaces. Biometrika 2008;95(3):759–71, doi:http://dx.doi.org/10.1093/biomet/asn034.
[25] Epskamp S, Borsboom D, Fried EI. Estimating psychological networks and their accuracy: a tutorial paper. Behav Res Methods 2018;50(1):195–212, doi:http://dx.doi.org/10.3758/s13428-017-0862-1.
[26] Epskamp S, Maris G, Waldorp L, Borsboom D, Irwing P, Hughes D, et al. Handbook of psychometrics. New York, NY: Wiley; 2016.
[27] Brandes U. On variants of shortest-path betweenness centrality and their generic computation. Soc Networks 2008;30(2):136–45, doi:http://dx.doi.org/10.1016/J.SOCNET.2007.11.001.
[28] Dijkstra EW. A note on two problems in connexion with graphs. Numer Math 1959;1(1):269–71.
[29] Isvoranu AM, van Borkulo CD, Boyette LL, Wigman JTW, Vinkers CH, Borsboom D. A network approach to psychosis: pathways between childhood trauma and psychotic symptoms. Schizophr Bull 2017;43(1):187–96, doi:http://dx.doi.org/10.1093/schbul/sbw055.
[30] Baron RM, Kenny DA. The moderator–mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. J Pers Soc Psychol 1986;51(6):1173–82.
[31] Preacher KJ, Hayes AF. Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. Behav Res Methods 2008;40:879–91, doi:http://dx.doi.org/10.3758/BRM.40.3.879.
[32] Fischer S, Stojek M, Hartzell E. Effects of multiple forms of childhood abuse and adult sexual assault on current eating disorder symptoms. Eat Behav 2010;11(3):190–2, doi:http://dx.doi.org/10.1016/J.EATBEH.2010.01.001.
[33] Kent A, Waller G, Dagnan D. A Greater role of emotional than physical or sexual abuse in predicting disordered eating attitudes: the role of mediating variables. Int J Eat Disord 1999;25:159–67 doi:10.1002/(SICI)1098-108X(199903)25:2<159::AID-EAT5>3.0.CO;2-F..
[34] Monteleone AM, Ruzzi V, Patriciello G, Pellegrino F, Cascino G, Castellini G, et al. Parental bonding, childhood maltreatment and eating disorder psychopathology: an investigation of their interactions. Eat Weight Disord 2019;1–13, doi:http://dx.doi.org/10.1007/s40519-019-00649-0.
[35] Guillaume S, Jaussent I, Maimoun L, Ryst A, Seneque M, Villain L, et al. Associations between adverse childhood experiences and clinical characteristics of eating disorders. Sci Rep 2016;6:35761, doi:http://dx.doi.org/10.1038/srep35761.
[36] Groleau P, Steiger H, Bruce K, Israel M, Sycz L, Ouellette AS, et al. Childhood emotional abuse and eating symptoms in bulimic disorders: an examination of possible mediating variables. Int J Eat Disord 2012;45(3):326–32.
[37] Racine SE, Wildes JE. Emotion dysregulation and anorexia nervosa: an exploration of the role of childhood abuse. Int J Eat Disord 2015;48(1):55–8, doi:http://dx.doi.org/10.1002/eat.22364.
[38] Aldao A, Nolen-Hoeksema S, Schweizer S. Emotion-regulation strategies across psychopathology: a meta-analytic review. Clin Psychol Rev 2010;30(2):217–37, doi:http://dx.doi.org/10.1016/J.CPR.2009.11.004.

[39] Bruch H. Anorexia Nervosa: therapy and theory. Am J Psychiatry 1982;139:1531–8, doi:http://dx.doi.org/10.1176/ajp.139.12.1531.
[40] Treasure J, Cardi V. Anorexia Nervosa, Theory and Treatment: Where Are We 35 Years on from Hilde Bruch's Foundation Lecture? Eur Eat Disord Rev 2017;25(3):139–47, doi:http://dx.doi.org/10.1002/erv.2511.
[41] Monteleone AM, Castellini G, Ricca V, Volpe U, De Riso F, Nigro M, et al. Embodiment mediates the relationship between avoidant attachment and eating disorder psychopathology. Eur Eat Disord Rev 2017;25(6):461–8, doi:http://dx.doi.org/10.1002/erv.2536.
[42] Pollatos O, Kirsch W, Schandry R. On the relationship between interoceptive awareness, emotional experience, and brain processes. Cogn Brain Res 2005;25:948–62, doi:http://dx.doi.org/10.1016/j.cogbrainres.2005.09.019.
[43] Craig AD. Interoception and emotion: a neuroanatomical perspective. In: Lewis M, Haviland-Jones JM, Barrett LF, editors. Handbook of emotions. New York: The Guilford Press; 2008. p. 272–92.
[44] Damasio AR. The feeling of what happens: body and emotion in the making of consciousness. New York: Harcourt Brace; 1999.
[45] Brown TA, Berner LA, Jones MD, Reilly EE, Cusack A, Anderson LK, et al. Psychometric evaluation and norms for the multidimensional assessment of interoceptive awareness (MAIA) in a clinical eating disorders sample. Eur Eat Disord Rev 2017;25(5):411–6, doi:http://dx.doi.org/10.1002/erv.2532.
[46] Feinson MC, Hornik-Lurie T. 'Not good enough:' exploring self-criticism's role as a mediator between childhood emotional abuse & adult binge eating. Eat Behav 2016;23:1–6, doi:http://dx.doi.org/10.1016/j.eatbeh.2016.06.005.
[47] Leverich GS, McElroy SL, Suppes T, Keck PE, Denicoff KD, Nolen WA, et al. Early physical and sexual abuse associated with an adverse course of bipolar illness. Biol Psychiatry 2002;51(4):288–97.
[48] Young EA, Abelson JL, Curtis GC, Nesse RM. Childhood adversity and vulnerability to mood and anxiety disorders. Depress Anxiety 1997;5(2):66–72.
[49] Elliott H, Jones PJ, Schmidt U. Central symptoms predict post-treatment outcomes and clinical impairment in anorexia nervosa: a network analysis in a randomized-controlled PsyArXiv. , doi:http://dx.doi.org/10.31234/OSF.IO/HW2DZ.
[50] Smith KE, Mason TB, Crosby RD, Cao L, Leonard RC, Wetterneck CT, et al. A comparative network analysis of eating disorder psychopathology and co-occurring depression and anxiety symptoms before and after treatment. Psychol Med (Paris) 2019;49(2):314–24, doi:http://dx.doi.org/10.1017/S0033291718000867.
[51] Olatunji BO, Levinson C, Calebs B. A network analysis of eating disorder symptoms and characteristics in an inpatient sample. Psychiatry Res 2018;262:270–81, doi:http://dx.doi.org/10.1016/J.PSYCHRES.2018.02.027.
[52] Spindler A, Milos G. Links between eating disorder symptom severity and psychiatric comorbidity. Eat Behav 2007;8(3):364–73, doi:http://dx.doi.org/10.1016/j.eatbeh.2006.11.012.
[53] Brewin CR, Andrews B, Gotlib IH. Psychopathology and early experience: a reappraisal of retrospective reports. Psychol Bull 1993;113(1):82–98.
[54] Castellini G, Lelli L, Cassioli E, Ciampi E, Zamponi F, Campone B, et al. Different outcomes, psychopathological features, and comorbidities in patients with eating disorders reporting childhood abuse: a 3-year follow-up study. Eur Eat Disord Rev 2018;26(3):217–29, doi:http://dx.doi.org/10.1002/erv.2586.
[55] Grenon R, Carlucci S, Brugnera A, Schwartze D, Hammond N, Ivanova I, et al. Psychotherapy for eating disorders: a meta-analysis of direct comparisons. Psychother Res 2018;1–13, doi:http://dx.doi.org/10.1080/10503307.2018.1489162.