Childhood Adversity and Emerging Psychotic Experiences: A Network Perspective

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Background and Hypothesis: Childhood adversity is associated with a myriad of psychiatric symptoms, including psychotic experiences (PEs), and with multiple psychological processes that may all mediate these associations. *Study Design*: Using a network approach, the present study examined the complex interactions between childhood adversity, PEs, other psychiatric symptoms, and multiple psychological mediators (ie, activity-related and social stress, negative affect, loneliness, threat anticipation, maladaptive cognitive emotion regulation, attachment insecurity) in a general population, adolescent sample (*n* = 865, age 12–20, 67% female). *Study Results*: Centrality analyses revealed a pivotal role of depression, anxiety, negative affect, and loneliness within the network and a bridging role of threat anticipation between childhood adversity and maladaptive cognitive emotion regulation. By constructing shortest path networks, we found multiple existing paths between different categories of childhood adversity and PEs, with symptoms of general psychopathology (ie, anxiety, hostility, and somatization) as the main connective component. Sensitivity analyses confirmed the robustness and stability of the networks. Longitudinal analysis in a subsample with Wave 2 data (*n* = 161) further found that variables with higher centrality (ie, depression, negative affect, and loneliness) better predicted follow-up PEs. *Conclusions*: Pathways linking childhood adversity to PEs are complex, with multifaceted psychological and symptom-symptom interactions. They underscore the transdiagnostic, heterotypic nature of mental ill-health in young people experiencing PEs, in agreement with current clinical recommendations.

Key words: childhood adversity/psychotic experiences/network analysis/adolescents

Introduction

Psychotic experiences (PEs) are common in the general population,[1] with highest pooled incidence rate (ie, 5 per 100) and persistence rate (ie, 35.8%) in adolescence.[2] Although PEs are transient for most people, about 20% develop persistent PEs and 7% go on to develop a psychotic disorder,[3] suggesting a phenomenological and temporal continuity between PEs and psychotic disorders. Particularly, the typical onset of primary psychotic disorders is in the teens to mid-twenties,[4] stressing the importance of understanding emerging PEs in adolescence.

Childhood adversity is robustly associated with the development of PEs across this continuum, extending from low-level experiences to a full-blown psychotic disorder.[5–10] Extensive literature shows that exposure to adversity in childhood may contribute to an enduring liability to PEs by impacting on affective and cognitive processes, as well as dysfunctional attachment.[11–22] Indeed, PEs are associated with enhanced threat anticipation,[23,24] maladaptive cognitive emotion regulation,[25] dysfunctional attachment,[26,27] elevated emotional reactions to daily life stressors[23,24,28,29] and comorbid psychopathology,[30] characteristics that have also been repeatedly reported in individuals with a history of childhood adversity.[17,21,22,31–34] Evidence in fact suggests that negative affect in daily life,[31] as well as feelings of loneliness,[21] emotional symptoms (eg, anxiety and depression),[21,22,35] emotional

dysregulation,[21,36,37] enhanced threat anticipation,[31] and attachment insecurity,[21] may all mediate the association between childhood adversity and PEs.

Importantly however, these different psychological mediators do not act independently, but are mutually interrelated.[15,18,38] For instance, the effect of stress on PEs is mediated by negative affect in daily life and threat anticipation[23]; attachment insecurity is associated with altered behavioral and neural patterns of emotion regulation[39]; and threat anticipation, maladaptive cognitive emotion regulation, and attachment insecurity are all related to general psychopathology.[32,40–43] Thus, apparently, pathways linking childhood adversity to PEs are complex, with multifaceted psychological and symptom-symptom interactions. The identification of key mediators, as well as understanding the interplay among these different factors, is crucial to develop the most effective intervention and prevention strategies. To this end, it is necessary to systematically map and test the full complexity of etiological pathways. A network approach may be best suited for this purpose.[44–46]

The network theory conceptualizes mental disorders as complex systems, in which the causal interactions between symptoms themselves constitute the disorder.[44] For instance, Fried et al[45] compared 2 hypotheses about the effects of loss (ie, bereavement) on depressive symptoms and found that, instead of being mediated through a latent variable, these effects could be better explained via a network perspective: loss mainly affected the symptom loneliness, which in turn was correlated to other symptoms. Symptoms with such a key role in a network are referred to as central symptoms, as these symptoms may potentially induce other symptoms, whereas decreasing them may help to resolve the whole symptom network. In a longitudinal study with 501 healthy adults, Boschloo et al[46] prospectively examined the onset of DSM-IV major depressive disorder after a 2-, 4-, and 6-year follow-up. The authors found that symptoms with the highest centrality (ie, strength centrality) at baseline, such as loss of interest, fatigue, depressed mood, and concentration problems, strongly predicted the onset of major depressive disorder, whereas symptoms with lowest centrality (such as a decrease in weight/appetite, hypersomnia, and suicidal thoughts) were not or only weakly predictive. Thus, early intervention strategies targeting central symptoms in particular hold considerable promise in terms of prevention and treatment success.

In a hallmark study, Isvoranu et al used network analysis to examine the pathways linking childhood adversity and negative and positive psychotic symptoms in a sample of adult patients with a psychotic disorder.[35] The authors found that distinct adversity types and psychotic symptoms were connected via general psychopathology, with anxiety as a major connective component. Important gaps in knowledge still remain, however. First, it is unknown how these complex interactions evolve in the early stages of emerging PEs, ie, in adolescence, where they have the highest pooled incidence and persistence rate[2] and can be studied free from the biases of treatment, illness progression, or perceived stigma. Second, no study so far has examined the role, centrality and interplay of psychological processes such as stress in daily life, negative affect, feeling of loneliness, threat anticipation, maladaptive cognitive emotion regulation, attachment insecurity, as well as general psychopathology, which are closely associated with PEs and exposure to adversity. The current study therefore aimed to map the complex network linking childhood adversity and PEs by including key psychological mediators in a community sample of adolescents between 12 and 20 years old.

Methods

This study has been post-registered on the Open Science Framework (OSF, https://osf.io/ygchd). Formal analyses deviated from the registered analyses: (1) loneliness was included as a separate variable instead of ascribing it to negative affect as in previous studies, which have demonstrated the mediating role of loneliness between childhood adversity and PEs[21,47]; (2) we did not include the global severity index score, but the subdimensions of the 53-item Brief Symptom Inventory[48] to investigate the mediating role of different categories of psychopathology; (3) we included 4, instead of 9, cognitive emotion regulation strategies, since these 4 have been associated the most with psychopathological symptoms[43,49,50]; (4) we scored childhood adversity by summing up the items within each module instead of coding each module as binary data (ie, yes or no) to better capture the degree of exposure, consequently, a Gaussian graphical model (GGM) using extended Bayesian information criterion (EBIC), rather than a Mixed Graphical Model using cross validation, was fitted to the data. The R codes used in this manuscript are provided on OSF (https://osf.io/8urg5/).

Participants

The sample used in the current article pertain to the first wave (29/1/2018–3/6/2019) of the SIGMA study.[51] 1913 adolescents were recruited through 22 mainstream secondary schools (first year = 1048, third year = 424, fifth year = 441) across all 5 provinces in Flanders, Belgium. Our main measure of PEs in this sample is the Prodromal Questionnaire-16 (PQ-16),[52,53] since it has good internal consistency in both adolescent help-seeking and general populations.[54,55] The PQ-16 was omitted for first-year students following data collection from the first school, as it became clear that the first-year students were unable to complete the full questionnaire battery within the time allowed for the testing session. This resulted in a final sample of 865 adolescents. Written informed consent was obtained from participants and their parents/

caregivers prior to the participation. UZ/KU Leuven Medical Ethics Committee has approved this study (number S61395).

Measures

In the present study, we integrated classical questionnaire data with measures derived through Experience Sampling Method, ie, a digital diary method requesting participants to enter responses on their momentary context, feelings and thoughts 10 times a day during 6 consecutive days. Measures of all domains were translated to Dutch and are described in table 1.

Statistical Analysis

Statistical analyses were performed using the *R*-statistical software, version 4.1.3.[71] Missing values of the raw data (ie, at the item level) were handled using a random forest imputation algorithm, implemented using the *R-package missForest*, version 1.4.[72] Descriptive statistics of each item in the raw and imputed data are reported in the file "Supplementary_PQ_Description.xlsx."

Network estimation was performed on the imputed data. Firstly, scores of the 26 variables were computed using the relevant items and then transformed with nonparanormal transformation[73] to relax the normality assumption. Thereafter, an undirected network with all 26 variables was constructed with GGM using the *R-package bootnet*, version 1.5.[74] Within the network, each variable is represented by a node and an edge indicates a partial correlation between 2 variables, while a missing edge indicates an absent correlation, after conditioning on all other variables. To estimate a sparse (more interpretable) network and control for potential spurious connections, we applied an L1-penalty regularization selected by EBIC[75–77] with the default regularization parameter lambda (0.5, which was shown to yield accurate network estimations). The network was visualized using the *R-package qgraph*, version 1.9.2[78] with the Fruchterman and Reingold layout. To assess the variability of edge-weight accuracy, we performed supplementary analyses of nonparametric bootstrapped 95% confidence intervals and difference tests (see Appendix S2) as suggested by Epskamp et al.[79]

Node centrality was estimated with strength centrality, quantifying how well a given node is directly connected to other nodes by summing the absolute values of the weights on the edges connected to that node.[79] Supplementary analyses of correlation-stability coefficient (*CS* coefficient), difference tests, and expected influence were conducted to check the estimation (see Appendix S3). As, closeness and betweenness, 2 other centrality indices, have been suggested to be less stable and applicable,[44,80] we chose not to interpret these in the current article (see Appendix S3).

Bridge strength indicates the importance of a given node in bridging communities/clusters[81] and was estimated during the exploratory analyses, using R-package *networktools*, version 1.4.0.[82] Items in each domain (eg, general psychopathology, attachment insecurity) were defined as a separate cluster, based on which we calculated bridge strength by summing the absolute value of every edge that connected a given node in a certain cluster to nodes in other clusters. Supplementary analyses of *CS* coefficient, difference tests, and bridge expected influence were conducted to assess the estimation of bridge strength (see Appendix S4). We also performed cluster detection using the "walktrap" algorithm in the *R-package EGAnet*, version 1.1.0,[83] ie, exploring clusters of densely connected nodes within the network structure, while not considering any theoretically determined clusters (see Appendix S5).

Shortest paths[84] were computed using Dijkstra's algorithm[85] to indicate the minimum number of steps needed to go from each category of childhood adversity to the specific PEs. These could give a clear view of potential pathways and mediating variables between different categories of childhood adversity and PEs.[35]

Supplementary sensitivity analyses were also performed. First, to check the stability of the nonzero edges due to the approach to impute missing data, we used the stability selection technique[86] to randomly select half of the cases of the sample size to perform the imputation with random forest and to estimate the network with GGM, over 100 iterations. Then the frequency of each edge present in the 100 iterations was calculated (Appendix S6). Second, we constructed another network quantifying PEs by the 5 items of the Diagnostic Interview Schedule for Children (DISC-C[87]; Appendix S7), which serves as a further validation of the estimation of the PQ-16 network by increasing the sample size (DISC-C were conducted in all 1913 adolescents). Third, since cross-sectional data do not allow for causal inference, we examined prospective associations with PEs at Wave 2 and the baseline variables used in the network analysis as the predictors using the least absolute shrinkage and selection operator (lasso) regression analysis[88] (Appendices S7 and S8).

Results

Demographics

Table 2 displays the demographic information. Of 865 participants, *n* = 582 participants identified as female, *n* = 279 as male, and *n* = 4 as "Other."

Network Estimation

The estimated network is displayed in figure 1a. Of 325 possible edges, 144 (44.31%) nonzero edges emerged, with a mean weight of 0.077. 140 (97.22%) edges were positive and 4 (2.78%) edges were negative. The scores of

Table 1. Information for All Measures Included in the Analyses

Domain	Measures[a]
Childhood adversity	The questionnaire (child self-administered version of the Juvenile Victimization Questionnaire—2nd Revision[56,57]) covers 5 adversity modules: conventional crime, child maltreatment, peer and sibling victimization, sexual victimization, and witnessing and indirect victimization, with each item with answer options "Yes" or "No." The conventional crime module was excluded as it was only administered in a limited number of participants due to time constraints. The remaining 4 modules were scored by summing up the relevant items.
Stress (ESM)	Based on the vulnerability-stress model, evidence has suggested that elevated emotional reactions to daily life minor stressors mediate the relationship between childhood adversity and PEs.[12,19,24,58–60] For social stress,[24,28,29] participants were firstly asked to indicate "Who is with me?." If alone, they then rated the following 2 items "I find being alone pleasant" (reversed) and "I prefer to have company" (from 1 "Not at all" to 7 "Very much"). If they are in company, they rated "I feel comfortable in this company" (reversed) and "I prefer to be alone." Average score across a 6-day period of either 2 items was used to indicate the social stress level. For activity stress,[24,28,29] participants were firstly asked "what are you doing?." Then they rated current activity with 3 items "I would rather do something else," "This is difficult for me" and "I can do this well" (reversed). Average score across the 6-day period and 3 items was used to index the activity stress level.
Negative affect (ESM)	For emotional reactivity in daily life, negative affect was included quantified by the average score across a 6-day period on 6 ESM items: "I feel irritated," "I feel anxious," "I feel insecure," "I feel sad," "I feel stressed," "I feel restless" (from 1 "Not at all" to 7 "Very much").
Loneliness (ESM)	Feeling of loneliness is another pathway suggested to mediate the relationship between childhood adversity and PEs.[21] Here, we quantified loneliness with the average score across a 6-day period with the ESM item "I feel lonely" (from 1 "Not at all" to 7 "Very much").
General psychopathology	Nonpsychotic symptoms, especially anxiety and depression, have been demonstrated to mediate the link between childhood adversity and PEs, especially in general population.[21,61] Here, we quantified general psychopathology using the 53-item Brief Symptom Inventory,[48,62] which asked participants to rate on a 5-point Likert scale (from 0 "Not at all" to 4 "Extremely") to indicate to which extent they experienced those difficulties in the past 7 days. It covers 9 symptom dimensions, of which paranoid ideation and psychoticism are closely related to psychotic symptoms, thus excluded from the current analyses. The remaining 7 dimensions, including somatization, obsession-compulsion, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, were used to indicate a person's level of symptomatology by summing up the relevant items.
Maladaptive cognitive emotion regulation	Affective dysregulation is another relevant mediator in relation to childhood adversity and PEs. Here we used the 18-item short version of the Cognitive Emotion Regulation Questionnaire[50,63] to quantify participants' cognitive way of handling the emotional arousal situation. Participants were asked to rate on a 5-point Likert scale (from 1 "Almost never" to 5 "Almost always") to indicate their thought following a negative experience. It covers 9 dimensions: positive refocusing, planning, positive reappraisal, putting into perspective, acceptance, self-blame, other-blame, rumination, and catastrophizing. The latter 4 dimensions are generally suggested as maladaptive and to be associated the most with psychopathological symptoms,[43,49,50] thus were included in the current analyses.
Attachment insecurity	Based on attachment theory, the link between attachment insecurity and PEs has been well established[14,17] and attachment style could mediate the association between childhood adversity and PEs.[13,33] Here, we quantified attachment insecurity with the 36-item short version of the Inventory of Parent and Peer Attachment Revised,[64,65] which asked participants to rate on a 4-point Likert scale (from 1 "Almost never" to 4 "Almost always"). The questionnaire covers attachment security to father, mother, and peers across 3 subdimensions (Communication, Trust, and Alienation). As the subdimension Alienation was only administered in a limited number of participants, we summed the scores of Communication and Trust reversely to indicate the degree of attachment insecurity to father, mother, and peer, separately.
Threat anticipation	Increasing evidence supports the role of threat anticipation in PEs and the association between childhood adversity and PEs.[23,24,31,66,67] Here, we quantified threat anticipation with the 10-item short version of the Availability Test,[38,68] which asked participants to indicate the possibility that each of 5 negative and 5 positive events described in the items would occur in the coming week on a 7-point Likert scale (from 1 "Very unlikely" to 7 "Very likely"). Negative items were included to indicate threat anticipation by summing up the scores.
Psychotic experiences	The Prodromal Questionnaire-16[52,53] asked participants to indicate the presence of PEs through their lifetime as "True" or "False," with True answers then rated the burden for that experience on a 4-point Likert scale (from 0 "Not burdensome" to 3 "Very burdensome"). We combined the endorsement and burdensome level to represent the continuum of PEs (0 = False, 1 = True and Not burdensome, 2 = True and Mild burdensome, 3 = True and Moderate burdensome, 4 = True and Very burdensome).[53,69,70] Three subdimensions (perceptual abnormalities, unusual thought content, and negative symptoms) have been suggested as a good fit in a general population[70] and were included in the current analyses, with each subdimension calculated by summing up the relevant items.

Note: ESM, Experience Sampling Method; PEs, psychotic experiences.
[a]All questionnaire items included an extra response option (ie, I do not wish to answer) due to the requirements from the participating schools. The proportion of each item with the extra response is reported in file "Supplementary_PQ_ProportionExtraResponse.csv" and data with the extra response were coded as missing data.

Table 2. Descriptive Information for All Variables

Variable	Mean	SD	Observed Range	Theoretical Range	Available Data
Age	15.50	1.33	12–20		860
Stress					
Social stress	2.80	0.89	1–5.62	1–7	780
Activity stress	3.09	0.78	1–7	1–7	778
Negative affect	2.28	0.94	1–7	1–7	783
Loneliness	1.89	0.91	1–7	1–7	781
Childhood adversity					
Child maltreatment	0.82	1.00	0–4	0–4	735
Peer and sibling victimization	1.63	1.00	0–6	0–6	730
Sexual victimization	0.89	1.32	0–7	0–7	703
Witnessing and indirect victimization	1.40	1.38	0–8	0–8	697
Threat anticipation	11.89	4.76	5–33	5–35	777
General psychopathology					
Anxiety	6.11	4.57	0–23	0–24	722
Somatization	6.00	5.03	0–26	0–28	724
Obsession-compulsion	7.41	4.92	0–24	0–24	717
Hostility	5.25	4.07	0–20	0–20	727
Phobic anxiety	3.49	3.64	0–20	0–20	725
Depression	6.21	5.64	0–24	0–24	709
Interpersonal sensitivity	5.39	3.96	0–16	0–16	726
Attachment insecurity					
Father attachment insecurity	19.77	3.78	9–28	8–32	617
Mother attachment insecurity	21.92	3.55	11–29	8–32	617
Peer attachment insecurity	22.12	4.12	8–31	8–32	623
Maladaptive cognitive emotion regulation					
Rumination	6.35	2.06	2–10	2–10	697
Self-blame	5.28	2.08	2–10	2–10	676
Catastrophizing	4.95	2.22	2–10	2–10	669
Other-blame	3.95	1.66	2–10	2–10	661
Psychotic experiences					
Negative symptoms	1.57	1.94	0–8	0–8	**639**
Unusual thought content	4.10	3.57	0–18	0–20	**614**
Perceptual abnormalities	4.50	4.79	0–28	0–36	**597**

The available data are reported due to missingness and those for psychotic experiences are highlighed in bold.

all the edges and of the plotted edges are reported in file "Supplementary_PQ_pcor.csv" and file "Supplementary_PQ_pcor_plot.csv," respectively.

Node and Bridge Centrality

Strength centrality analysis (figure 1b; see raw scores in file "Supplementary_PQ_StrengthScore.csv") revealed that depression (15), anxiety (10), negative affect (3), and loneliness (4) (in decreasing order) had the highest centrality. The *CS* coefficient (supplementary figure S3) was 0.75, indicating high stability. Indeed, the difference tests (supplementary figure S4) revealed that depression and anxiety were significantly different from all other nodes, except from each other, and negative affect and loneliness were significantly different from most of the other nodes in the network. Expected influence (supplementary figure S5) was strongly correlated with strength centrality ($r = 0.997$; $P < .001$), further confirming the results.

Bridge centrality analysis (figure 1b; see raw scores in file "Supplementary_PQ_BridgeStrengthScore.csv") revealed nodes with highest bridge centrality were negative affect (3), loneliness (4), and threat anticipation (9). Visual inspection revealed that, for negative affect and loneliness, these were mainly driven by the connections negative affect (3)-loneliness (4), activity stress (2)-negative affect (3), and social stress (1)-loneliness (4), indicating strong interplay among stress, negative affect, and loneliness. Indeed, supplementary cluster detection without considering theory ascribed these 4 nodes into 1 cluster (supplementary figure S9). For threat anticipation, these were mainly driven by the connections between peer and sibling victimization (6)-threat anticipation (9), threat anticipation (9)-self-blame (21), and threat anticipation (9)-other blame (23). The *CS* coefficient (supplementary figure S6) was 0.75, indicating high stability. The difference tests (supplementary figure S7) revealed that negative affect and loneliness were significantly different from all the other nodes, except from each other, and threat anticipation was significantly different from almost all the other nodes. Bridge expected influence (supplementary figure S8) was strongly correlated with bridge strength centrality ($r = 0.998$, $P < .001$).

Shortest Paths in the Network

Four networks presenting the shortest paths between each category of childhood adversity and PEs are shown in figure 2. For childhood maltreatment (5) (figure 2a), the connections to PEs ran via other categories of childhood adversity, ie, connecting to negative symptoms (24) via peer and sibling victimization (6), anxiety (10), and phobic anxiety (14), to unusual thought content (25) via sexual victimization (7), and to perceptual abnormalities (26) via witnessing and indirect victimization (8) and hostility (13). Peer and sibling victimization (6) (figure 2b) either connected to unusual thought content (25) directly, or to negative symptoms (24) via anxiety (10) and phobic anxiety (14) and to perceptual abnormalities (26) via anxiety (10) and somatization (11). Sexual victimization (7) (figure 2c) directly connected to unusual thought content (25), through which it then connected to negative symptoms (24) and perceptual abnormalities (26). Witnessing and indirect victimization (8) (figure 2d) connected to perceptual abnormalities (26) via hostility (13), then connected to negative symptoms (24) and unusual thought content (25).

Supplementary sensitivity analysis (supplementary figure S10) showed that nonzero edges with strong weights in the constructed network were present in most of the 100 iterated estimations, indicating high stability.
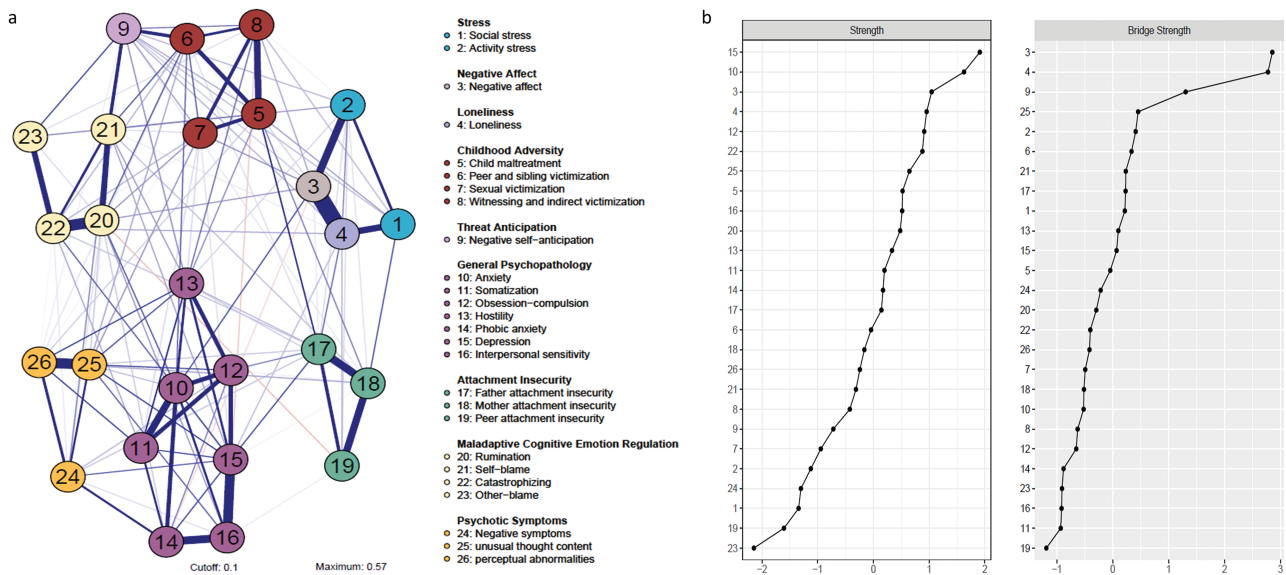

Fig. 1. (a) Network model of childhood adversity, psychotic experiences, and potential psychological processes. Each node displays a variable, with variables in the same domain plotted by the same color. Each edge corresponds to a partial correlation between 2 nodes, the thicker the edge, the stronger the connection. Blue edges display positive connections and red edges display negative ones. (b) Centrality indices of the network: strength centrality (left) and bridge strength centrality (right). Variables are shown as standardized z-scores and sorted in decreasing order.
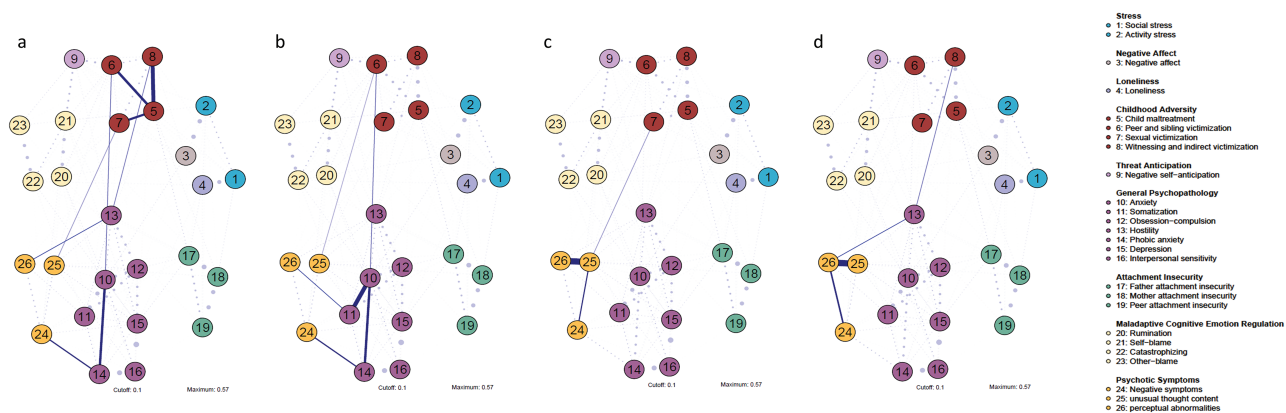

Fig. 2. Networks displaying shortest paths between (a) child maltreatment (5), (b) peer and sibling victimization (6), (c) sexual victimization (7), and (d) witnessing and indirect victimization (8) and psychotic experiences. Dashed lines indicate connections existing within the network framework, but are less relevant when investigating shortest paths.

Sensitivity Analyses Using Psychotic Symptoms Measured by DISC-C

The DISC-C network structure (supplementary figure S11) was well aligned with the PQ-16 network. The estimation of strength centrality (supplementary figure S14) revealed that depression (15) and anxiety (10) were still the first 2 nodes with the highest strength. While negative affect (3) and loneliness (4) were the third and fourth in the strength centrality order in the PQ-16 network, respectively, here they were the fifth and eighth. Thus, though the orders of node strength in the 2 networks were not exactly the same, these nodes were of higher order in both networks, especially anxiety and depression.

The estimation of bridge centrality (supplementary figure S17) revealed that negative affect (3), loneliness (4), and threat anticipation (9) were still the first 3 nodes with highest bridge centrality, confirming the results in the PQ-16 network with larger sample.

The shortest path networks (supplementary figure S20) revealed that the connection to PEs was via hostility (13) from childhood maltreatment (5), via threat anticipation (9) and hostility (13) from peer and sibling victimization (6), via depression (15) and hostility (13) from sexual victimization (7), and via childhood maltreatment (5) and hostility (13) from witnessing and indirect victimization (8). Given PEs were measured in different dimensions in the 2 networks, it is not possible to compare them to each other. However, notably, hostility (13) appeared on the shortest paths estimated within both networks.

Supplementary Longitudinal Analysis

For the lasso regression model including PEs measured by PQ-16, 161 participants (15–21 years; 119 females and 40 males) had complete data at Wave 2. Variables remained after regularization and the coefficients are reported in table 3. In general, the results showed that variables with higher strength centrality in the baseline network, including depression, negative affect, and loneliness, predicted follow-up PEs with larger effect sizes than other variables. The same was true for variables with higher bridge centrality, ie, negative affect, loneliness, and threat anticipation. For the model using PEs measured by DISC-C, 312 participants (13–21 years; 228 females and 80 males) with complete data at Wave 2 were available for analysis. The results (supplementary table S4) showed that loneliness (a variable with high strength and bridge centrality), threat anticipation (a variable with high bridge centrality), and hostility (lying on the shortest paths in the baseline network) remained after regularization.

Discussion

Findings

Using a network approach, the current study investigated the associations between childhood adversity and PEs by

Table 3. Results of the Lasso Regression Analysis With PEs Measured by PQ-16.

Measures at Wave 2	Predicting Variables at Wave 1	Coefficient β
Negative affect	Social stress	0.113
	Negative affect	**0.240**
	Loneliness	**−0.255**
	Peer and sibling victimization	0.029
	Threat anticipation	−0.067
	Hostility	0.005
	Phobic anxiety	0.092
	Depression	**0.385**
	Mother attachment insecurity	−0.110
	Catastrophizing	0.002
	Other-blame	−0.017
	Perceptual abnormalities	−0.015
Unusual thought content	Activity stress	0.038
	Negative affect	**0.177**
	Phobic anxiety	0.001
	Catastrophizing	0.035
	Unusual thought content	**0.125**
	Perceptual abnormalities	0.010
Perceptual abnormalities	**Loneliness**	**0.109**
	Child maltreatment	**0.220**
	Threat anticipation	0.106
	Somatization	0.028
	Hostility	0.036
	Interpersonal sensitivity	**−0.102**
	Catastrophizing	0.007
	Other-blame	0.026
	Perceptual abnormalities	**0.276**

Note: PEs, psychotic experiences; PQ-16, Prodromal Questionnaire-16. Predicting variables with higher coefficients are highlighted in bold.

including multiple psychological mediators in a general population, adolescent sample. Our results revealed that depression, anxiety, negative affect, loneliness, and threat anticipation displayed highest centrality in the formation of the network. We also found evidence for a connective role of anxiety, hostility, and somatization in the shortest paths linking childhood adversity and PEs. These findings move beyond previous studies in 2 crucial aspects: (1) by recruiting multiple previously proposed mediators linking childhood adversity and PEs, we were able to detect those with a central role (ie, high centrality) in these complex associations; (2) by focusing on adolescence, we were able to better grasp the processes linking childhood adversity and emerging PEs in the earliest stages.

Depression, Anxiety, Negative Affect, and Loneliness as Central Elements of the Network

Depression and anxiety were detected as 2 nodes with the highest strength centrality, ie, they had the most and strongest connections with other nodes within the network. Previous studies have demonstrated that depression and anxiety often precede the onset and persistence of PEs.[89,90] Negative affect and loneliness were the next

2 nodes with the highest strength centrality and the first 2 nodes with the highest bridge strength centrality. Our supplementary lasso regression analysis further found that variables with higher centrality in the network (ie, depression, negative affect and loneliness) and higher bridge centrality (ie, negative affect, loneliness, and threat anticipation) predicted follow-up PEs with higher effect sizes, further validating these findings. Together, they underscore the transdiagnostic, heterotypic nature of mental ill-health in young people, in agreement with current clinical recommendations.[91–93]

Bridging Role of Threat Anticipation Between Childhood Adversity and Maladaptive Cognitive Emotion Regulation

Threat anticipation and maladaptive cognitive emotion regulation are 2 psychological processes suggested to mediate the relationship between childhood adversity and PEs.[21–23,36] A recent study[67] with samples from the same SIGMA study demonstrated that threat anticipation was positively associated with childhood adversity and may mediate the association between childhood adversity, general psychopathology, and PEs in adolescents. Our study extends these findings by demonstrating that threat anticipation may be particularly associated with peer and sibling victimization, which was also the most common category of childhood adversity in our sample. Additionally, our results suggest a strong connection between threat anticipation and maladaptive cognitive emotion regulation strategies, especially self-blame, warranting further investigation.

General Psychopathology as the Main Connective Component in the Shortest Paths

By constructing the shortest path networks, our results supported and extended previous findings,[35] indicating that multiple pathways may exist between childhood adversity and PEs, particularly via symptoms of general psychopathology. One of the symptoms playing a main connective role is anxiety (anxiety and phobic anxiety), which has been demonstrated to predict the onset of later PEs.[89,94,95] Another main connective component identified is somatization, which lay on the pathway between peer and sibling victimization and perceptual abnormalities. These findings are consistent with those from Isvoranu et al in an adult clinical sample,[35] despite notable differences in mean age and sex (75.7% of their sample was male, while 67.3% of our samples were girls), demonstrating a connective role of anxiety and somatic concern between childhood adversity and PEs. These findings point a possible stability of symptom-symptom correlations across the psychosis continuum, suggesting they may be inextricably linked in the context of childhood adversity, in agreement with reports linking childhood adversity to

co-occurring psychotic, affective, and anxiety symptoms[96] or even a general psychopathology (p-) factor.[97] In addition, hostility was identified as another connective component in our study, which is further validated by our supplementary analysis. Pooled evidence has found that around 30% of patients with first-episode psychosis commit at least one act of violence, while one of the factors associated with violence of any severity is hostile affect.[98] Furthermore, sensitivity to hostility (eg, angry faces) has been suggested in maltreated children.[99] The role of hostility in linking childhood aversity and psychosis, however, has been rarely studied, warranting further research.

Limitations

Our study has some limitations. First, our analyses do not necessarily capture all aspects of the link between childhood adversity and PEs and the choice for certain mediators, but not others, may be arbitrary to a degree. For example, potential mediation by symptoms of post-traumatic stress disorder has been proposed[21] but was not included in the current analysis. Nevertheless, our analyses include a wide array of potential mediators suggested in previous research and theoretical frameworks. Second, our samples were not representative in terms of sex, with the majority being girls. There is evidence that the characteristics and influence of childhood adversity on psychopathology is different for boys vs girls.[100–102] Future research with sufficient data could estimate the difference of the networks between boys and girls. Third, our analyses were largely based on cross-sectional data, limiting possible causal inference, although the limited longitudinal analyses were generally supportive of our cross-sectional findings. Nevertheless, our results provide a compelling insight into the complex connections between childhood adversity, PEs, and multiple psychological mediators. Future studies with longitudinal design could further explore the causal relationship. Fourth, we used a self-report, retrospective measure of childhood adversity, which may be liable to memory bias and social desirability. Moreover, as the participants were in school while filling out the questionnaires, their data may have been influenced by others observing them, a possible source of bias known as the Hawthorne effect. Lastly, though the network approach is a promising tool helping the understanding of psychiatric disorders, methodological concerns and challenges,[103,104] such as the generalizability problem due to the essentially data-driven analysis and the still limited insight into the relationship between the estimated network structure and the "true structure" of causal systems, still remain.

Conclusions

In summary, the present study uncovered the complex associations between childhood adversity and PEs by examining multiple psychological mediators during adolescence.

Our results highlight the central role of depression, anxiety, negative affect, and loneliness within this complex system and the bridging role of threat anticipation between childhood adversity and maladaptive cognitive emotion regulation, especially between peer and sibling victimization and self-blame. The shortest path networks demonstrated multiple existing paths between childhood adversity and PEs, with symptoms of general psychopathology (ie, anxiety, hostility, and somatization) as main connective components. Together, these findings emphasize a broad, holistic approach to understanding the complex interactions and core variables/pathways underlying the link between childhood adversity and emerging PEs.

Supplementary Material

Supplementary material is available at https://academic.oup.com/schizophreniabulletin/.

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