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Sensory processing sensitivity, alexithymia, and eating disorder risk in adolescents in alternative care: A multi-informant network study

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Abstract

Adolescents in alternative care show elevated eating disorder risk, yet mechanistic bridges between sensory reactivity and emotion-processing deficits remain underexplored. This study mapped the interplay among sensory processing sensitivity (SPS), alexithymia, and eating-related risk and tested moderation by trauma burden using a multi-informant design. Approximately 200 youths (12–18 years) were recruited from foster and residential services. Measures included self-report SPS (highly sensitive child facets), alexithymia (Toronto alexithymia scale-20: Difficulties identifying feelings [DIF], difficulties describing feelings [DDF], externally oriented thinking), eating-risk (eating attitudes test-26 subscales; binge eating scale), internalizing symptoms (patient health questionnaire for adolescents; screen for child anxiety related emotional disorders), and trauma (childhood trauma questionnaire–short form), along with covariates (age, sex, care type, body mass index z-score, pubertal status). Teachers/caregivers provided parallel SPS ratings. Primary analyses estimated a regularized partial-correlation network (Gaussian graphical model; EBICglasso) on domain-level nodes; accuracy and centrality stability were assessed through bootstrapping. Trauma moderation was evaluated through permutation-based network comparison tests; multi-informant integration was modeled by including teacher-SPS as a node and through informant-specific sensitivity analyses. SPS facets—especially Low Sensory Threshold and Ease of Excitation—bridged to alexithymia (DIF/DDF) and to eating-risk nodes, with stronger global connectivity observed under higher trauma exposure. Teacher-SPS converged with youth reports while adding unique variance. Limitations included the cross-sectional design, self-report bias, and setting selection. Findings delineate actionable psychosomatic targets—sensory-load management and affect labeling/interoceptive skills—for early, low-intensity interventions in foster/residential contexts.

Keywords: Sensory processing sensitivity; Alexithymia; Eating-disorder risk; Out-of-home care; Childhood trauma; Network analysis; Adolescents

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1. Introduction

Sensory processing sensitivity (SPS) denotes a heritable, temperament-like disposition characterized by heightened depth of processing, greater emotional reactivity, and

susceptibility to overstimulation in response to contextual demands. Originally theorized within the broader framework of environmental sensitivity, SPS has been operationalized in adults through the Highly Sensitive Person Scale and, in youths, through the 12-item Highly Sensitive Child (HSC) scale, which shows good reliability and a coherent facet structure (Ease of Excitation, Esthetic Sensitivity, Low Sensory Threshold).[1,2] Recent psychometric developments have extended assessment options (e.g., semi-structured HSC interviews and teacher-report tools), supporting multi-informant operationalization of sensitivity in educational and clinical contexts.[3] A comprehensive review positions SPS as a distinct, though overlapping, construct relative to the Big Five traits, with implications for stress responsivity and internalizing risk.[4]

Converging evidence links higher SPS to internalizing symptoms and to emotion-processing vulnerabilities. Cross-sectional work demonstrates robust associations of SPS facets—especially Low Sensory Threshold and Ease of Excitation—with anxiety, depression, and alexithymic traits (difficulties identifying and describing feelings).[5] Experimental and model-based studies implicate impaired or effortful emotion regulation as a plausible mechanism bridging SPS to negative affect.[6] Meta-analytic synthesis indicates that SPS covaries most strongly with neuroticism/negative affectivity, while remaining partially distinct from common personality dimensions; this profile aligns with a diathesis that amplifies both adverse and supportive environmental influences.[7] Furthermore, in young adults, SPS and alexithymia independently predict variance in emotional contagion and empathy even after accounting for early emotional abuse and depressed mood, underscoring theoretically relevant yet non-redundant contributions of sensory reactivity and affect-labeling deficits.[8]

Adolescents placed in out-of-home or "alternative care" (foster, kinship, or residential settings) represent a high-risk and underserved psychosomatic population. Meta-analytic comparisons indicate that youths in foster care exhibit higher levels of psychopathology than peers living with biological parents ($d \approx 0.19$), with placement stability and kinship care conferring protection.[9] Population-based analyses similarly document high pre-entry psychiatric morbidity and diagnostic flux during care, highlighting unmet needs and disparities.[10] Although systematic evidence specific to eating disorder phenotypes in alternative care has only recently emerged, a scoping review concludes that children and adolescents in out-of-home care are particularly vulnerable to aberrant eating and mealtime patterns, with trauma, food insecurity histories, and disrupted routines posited as drivers.[11] Consistent findings from a recent mixed-methods UK cohort study detail pervasive mealtime challenges and atypical eating behaviors among care-experienced children relative to comparison groups.[12] Related scoping work within residential settings underscores the social/relational meanings of food and the salience of eating disorders and nutritional adequacy as recurrent concerns.[13]

Trauma burden—encompassing abuse and neglect indexed by the childhood trauma questionnaire–short form (CTQ-SF)—emerges as a central etiological pathway in this population.[14] Beyond general internalizing risk, maltreatment histories have been repeatedly associated with later eating disorder symptoms and diagnoses through affective dysregulation, dissociation/interoceptive confusion, and stress-related neurobiological alterations in appraisal and control circuits.[15,16] At the same time, adolescence represents a sensitive window for the emergence of eating disorders; transdiagnostic models implicate negative affect, dieting/weight concerns, and reinforcement processes as key, modifiable drivers of onset and maintenance.[17,18] These strands converge on the hypothesis that, under higher trauma burden, sensory hyperreactivity may exacerbate emotion-labeling difficulties (alexithymia) and stress-related eating (e.g., binge propensity), thereby potentiating eating disorder risk.

A multi-informant design proves essential in adolescent mental health research. Classic work demonstrates low-to-moderate cross-informant correlations—reflecting situational specificity and perspective differences—so that each reporter contributes unique, ecologically valid variance.[19] Contemporary quantitative syntheses confirm the incremental and construct validity of integrating youth self-reports with caregiver/teacher ratings, improving criterion prediction, and reducing method bias.[20,21] For SPS specifically, informant divergence may be particularly informative because sensory hyperreactivity and overwhelm can vary across interpersonal and institutional contexts (home vs. school), and teachers may capture attentional/behavioral manifestations missed by caregivers or self-reports.[3]

Guided by these premises, the present cross-sectional, multi-informant study maps the interconnected network among SPS facets, alexithymia dimensions, and eating disorder risk in adolescents in alternative care; tests whether higher trauma burden strengthens SPS–alexithymia–eating-risk connectivity; and evaluates cross-informant replicability of network structure and key bridge nodes. By embedding sensitivity traits and affect-labeling constructs within an eating-risk network under conditions of elevated trauma exposure, the study aims to delineate tractable psychosomatic targets—sensory-load management and

emotion-labeling/interoceptive skills—for early, low-intensity interventions in foster/residential services. Of note, recent work has linked SPS and alexithymia to health anxiety, reinforcing the general interest of researchers in sensitivity–affect interfaces.[22]

1.1. Study aims and hypotheses

Guided by prior evidence on environmental sensitivity and emotion-processing vulnerabilities, we hypothesized that:

H_1: SPS facets—particularly Low Sensory Threshold and Ease of Excitation—would form an interconnected sensitivity–alexithymia subnetwork (with difficulties identifying feelings/difficulties describing feelings [DIF/DDF] as proximal affect-labeling nodes) that bridges to eating-risk nodes (dieting, bulimia/FOB, binge eating scale [BES]);

H_2: Greater trauma burden (CTQ-SF upper tertile) would increase global connectivity and strengthen edges along the interfaces linking SPS with alexithymia and alexithymia with eating-risk constructs;

H_3: These patterns would replicate across informants, with teacher-rated SPS converging with youth self-reports while contributing to unique classroom-context variance.

2. Materials and methods

2.1. Study design and setting

We conducted a cross-sectional, multi-informant study across public foster/residential care services and partner lower/upper secondary schools in two Italian regions. Site selection was based on pre-existing agreements that facilitated recruitment, assessment, and liaison with educators. Assessments were administered in quiet rooms within care facilities or school counseling spaces by trained graduate psychologists under senior supervision. Data were captured in a secure REDCap instance with role-based access and audit trails; paper forms were available if needed and double-entered for verification. The design prioritized harmonized timing between youth self-report and teacher/caregiver report (target window ≤14 days) to limit time-varying confounders (e.g., placement changes or school absences).

2.2. Participants

We enrolled approximately 200 adolescents (aged 12–18 years) currently in alternative care (foster, kinship, or residential) with ≥3 months in placement to ensure minimal stabilization and informant familiarity.

Inclusion criteria of this study are as follows: (i) age 12–18 years; (ii) foster/kinship care or residential care; (iii) sufficient Italian language comprehension to complete questionnaires; and (iv) availability of at least one teacher or caregiver informant.

Exclusion criteria of this study are as follows: (i) acute psychiatric instability requiring immediate intervention (e.g., current psychosis, acute intoxication) and (ii) intellectual disability or neurodevelopmental conditions that precluded valid self-report (as judged by the treating clinician/educator). Recruitment was carried out by service clinicians and school counselors, who introduced the study to legal guardians (child-welfare authority/appointed guardian) and adolescents. Written informed consent was obtained from legal guardians and written assent from adolescents, consistent with Italian regulations on vulnerable minors in care.

2.3. Measures (primary variables)

2.3.1. SPS (self-report)

The 12-item HSC scale was used to assess overall sensitivity and its three facets—Ease of Excitation, Low Sensory Threshold, and Aesthetic Sensitivity—supported by a bifactor structure and good reliability in youth.[23] Facet scores were computed as recommended in the developmental sensitivity literature, and internal consistency was verified at the facet level. In an exploratory robustness check, results were cross-validated with the 21-item extended HSC scale used at participating sites.[24,25]

2.3.2. SPS (teacher-report)

Teachers completed the school-adapted version of the HSC scale (HSC-School). For reporting and network nodes, we refer to the teacher-rated composite as Teacher-HSC (composite).[25,26]

2.3.3. Alexithymia

DIF, DDF, and externally oriented thinking (EOT) were assessed using the 20-item Toronto alexithymia scale (TAS-20), a widely used instrument with established factor structure and validity.[27,28]

2.3.4. Eating disorder risk (cognitive/attitudinal)

The eating attitudes test (EAT-26) was used to derive Dieting, Bulimia/Food Preoccupation, and Oral Control subscales, serving as a screening tool for cognitive risk features of eating disorders.[29]

2.3.5. Binge-eating symptoms

The BES indexed binge severity (total score), capturing loss-of-control eating patterns pertinent to psychosomatic distress in vulnerable youth.[30]

2.3.6. Internalizing symptoms

Depressive symptoms were measured using the patient health questionnaire for adolescents (PHQ-A), with proven validity and applicability in school and primary-care contexts.[31,32] Anxiety symptoms were assessed using the screen for child anxiety-related emotional disorders (SCARED), a validated multi-domain pediatric screener.[33,34]

2.3.7. Trauma burden (moderator)

The CTQ-SF provided a total score and five domain scores (emotional/physical/sexual abuse; emotional/physical neglect) with established psychometrics.[35]

2.3.8. Covariates

We recorded age, sex, care type (foster/kinship vs. residential), body mass index (BMI) z-scores (based on the World Health Organization 2007 growth references),[36] pubertal status (Pubertal Development Scale, PDS),[37] and IOTF BMI categories.[38] Where available, validated Italian versions were used; when unavailable, we implemented forward–backward translation with expert-panel reconciliation and cognitive debriefing in a pilot sample ($n \approx 20$).

2.4. Procedures and data quality

Research staff completed a 2-day training (administration, crisis management, and safeguarding) and inter-rater calibration. Teacher/caregiver questionnaires were collected within ±14 days of youth self-report (target ≤7 days) to reduce contextual drift. Real-time data checks flagged missingness and range violations; weekly dashboards monitored site-level recruitment and assessment-window adherence. Positive screens for high eating disorder risk or severe internalizing symptoms triggered a pre-specified referral algorithm to the service clinician or school counselor.

2.5. Statistical analysis

2.5.1. Pre-registration and reporting

The analysis plan and code were pre-registered on the Open Science Framework (OSF). Reporting was done in accordance with the STROBE guidelines.[39-41] Analytic scripts and a reproducible report were prepared for publication.[42]

2.5.2. Missing data

We handled item- and unit-level missingness through multiple imputation by chained equations (MICE) (MICE; 20 imputations), assuming data missing at random (MAR) conditional on observed covariates. Predictive mean matching was used for continuous variables and ordered logit for ordinal indicators.[43-46] Missingness patterns were examined through descriptive heatmaps and logistic regressions predicting item absence from age, sex, care type, and symptom burden, supporting the assumption of MAR. Complete-case analyses yielded the same backbone edges and bridge identities as the imputed solutions, with only minor attenuation of weak edges.

2.5.3. Network estimation

We estimated a Gaussian graphical model (GGM) using domain-level nodes: Three SPS facets, three TAS-20 facets, three EAT-26 facets, BES, PHQ-A, SCARED, and Teacher-HSC (composite). Partial correlations were regularized through graphical lasso and selected using EBIC ($\gamma = 0.50$; sensitivity $\gamma = 0.25$).[47] Visualization and centrality (node strength, predictability R^2) were computed in qgraph.[48-52] Accuracy and stability were evaluated through non-parametric bootstrapping.

2.5.4. Moderation by trauma burden

We split the sample by CTQ-SF tertiles and used the network comparison test (NCT) with 1,000 permutations to assess global strength, structural differences, and edge-specific changes (false discovery rate [FDR]-adjusted).[53,54]

2.5.5. Multi-informant integration

The Teacher-HSC (composite) was included as a node to model convergence. Informant-specific networks were re-estimated, excluding teacher data. Cross-informant agreement was indexed through ICC (2,k) and examined through polynomial regression.[55,56]

2.5.6. Ancillary modeling

Hierarchical regressions predicted EAT-26 and BES scores in accordance with the following steps: Step 1: Covariates; Step 2: SPS and TAS-20 facets; Step 3: CTQ-SF total; Step 4: Interactions (SPS × CTQ, SPS × Alexithymia). Model assumptions were checked; effect sizes and partial R^2 were reported.

2.5.7. Software

All analyses were conducted in R (4.x) using qgraph, bootnet, Network Comparison Test, psych, and mice. Scripts and session logs were archived in OSF.

2.6. Sample size justification

Given ~13 nodes and expected sparsity, $n = 200$ was deemed sufficient for edge-weight accuracy and CS-coefficient stability, and to ensure NCT validity and power for regression models.

2.6.1. Power and precision

With ~13 nodes and conservative regularization, n = 200 affords acceptable edge-weight accuracy and centrality stability (CS-coefficients ≥0.50) for domain-level networks. For the NCT, detectable differences chiefly depend on effect size and covariance structure; our tertile split yielded adequate group sizes for moderate global-strength differences and a limited number of edge-wise contrasts. We therefore interpret between-network effects with appropriate caution and report median-split replications as a sensitivity check.

2.7. Ethics

In this study, all procedures were carried out in adherence to the Declaration of Helsinki (2013). Participants received age-appropriate information; legal guardians provided written consent and adolescents provided written assent. Safeguarding included mandatory reporting, crisis procedures, and referral for clinical thresholds.

3. Results

3.1. Sample characteristics

We enrolled adolescents placed in either foster/kinship (community-based) or residential (group-home) care. Table 1 summarizes demographics, placement features, and covariates. Briefly, the age distribution spanned early to late adolescence, with balanced representation by sex and care type. Mean BMI z-scores fell in the normative range with a right-tail reflecting overweight/obesity; pubertal status (PDS) covered the expected developmental spread. Recruitment pathways (services vs. schools) and time-in-placement distributions were comparable across care types. Questionnaire completion rates exceeded pre-specified thresholds, and item-level missingness was low to moderate, primarily due to school absence or placement transition; diagnostics comparing imputed and complete-case estimates showed negligible differences and are available upon request.

3.2. Descriptive statistics and zero-order associations

Table 2 reports means, standard deviations, and internal consistency (α/ω), while Table 3 shows zero-order correlations among the domain scores. SPS self-report facets (Ease of Excitation, Low Sensory Threshold, Aesthetic Sensitivity) showed small-to-moderate positive associations with alexithymia (TAS-20 DIF/DDF) and with internalizing symptoms (PHQ-A, SCARED). EAT-26 Dieting and Bulimia/Food Preoccupation correlated positively with DIF/DDF and Low Sensory Threshold, whereas Oral Control displayed weaker, more variable associations. BES total correlated with DIF and with Ease of Excitation in the small-to-moderate range. Teacher-rated SPS, Teacher-HSC (composite), converged with youth self-report at the expected magnitude for cross-informant agreement in adolescent mental health; cross-informant agreement (ICC[2,k]) was in the expected range, and observed discrepancies were interpreted as meaningful rather than measurement error. These descriptive patterns motivated a network analysis to quantify conditional dependencies beyond shared variance and measurement overlap. For interpretation of network constructs and their clinical meaning, we align with contemporary network theory and caution regarding over-interpretation of topology.

3.3. Primary network model

A 13-node GGM (EBICglasso, γ = 0.50) was estimated on domain-level variables (three SPS facets; three TAS-20 facets; three EAT-26 subscales; BES, PHQ-A, SCARED; Teacher-HSC composite). Figure 1 depicts the regularized partial-correlation graph; Table 4 lists non-zero edges with bootstrap 95% confidence intervals (CIs). The network was sparse and interpretable. Two coherent clusters emerged: (i) a sensitivity–alexithymia cluster (Ease of Excitation, Low Sensory Threshold, DIF, DDF, with weaker ties to EOT) and (ii) an eating-risk cluster (Dieting, Bulimia/FOB, BES, with Oral Control peripheral). Crucially, bridge edges connected Low Sensory Threshold and Ease of Excitation with DIF/DDF, which in turn linked to Dieting and Bulimia/FOB; BES connected to both DDF and Bulimia/FOB, consistent with a pathway from sensory overstimulation → affect identification/description difficulties → eating-related cognitions/behaviors. PHQ-A and SCARED were connected to DIF and Ease of Excitation but did not displace alexithymia as primary bridges, suggesting that internalizing symptoms contributed to shared variance without monopolizing cross-cluster connectivity. Teacher-HSC (composite) loaded positively on self-reported SPS facets and, secondarily, on internalizing nodes, indicating unique classroom-context signal beyond simple duplication of the self-report construct.

Centrality indices (node strength; predictability/R^2) indicated that DIF and Low Sensory Threshold were the most influential nodes within and between clusters, followed by Ease of Excitation and Bulimia/FOB; Aesthetic Sensitivity and Oral Control were least central. Bootstrap diagnostics yielded narrow edge CIs for core connections and acceptable centrality stability (CS-coefficients >0.50). We report centrality indices with restraint, aligning with current best practice and avoiding overinterpretation.

Table 1. Sample characteristics by care type (foster/kinship vs. residential)

Variable	Foster/kinship (*n*=120)	Residential (*n*=80)	Total (*n*=200)	*p*-value
Demographics				
Age, years — mean±SD	15.3±1.7	15.7±1.8	15.5±1.8	0.12
Sex, female — *n* (%)	60 (50.0)	44 (55.0)	104 (52.0)	0.44
Time in current placement, months — median (IQR)	18 (9–36)	10 (5–22)	14 (7–28)	0.002
Anthropometrics				
BMI z-score (WHO 2007) — mean±SD	0.28±1.05	0.42±1.08	0.34±1.06	0.31
BMI category — *n* (%)				
Thinness	7 (5.8)	5 (6.2)	12 (6.0)	
Normal weight	76 (63.3)	48 (60.0)	124 (62.0)	
Overweight/obesity	37 (30.8)	27 (33.8)	64 (32.0)	0.40
Sensory processing sensitivity (HSC-12) — mean±SD				
Ease of excitation	3.60±0.90	3.90±1.00	3.72±0.95	0.08
Low sensory threshold	3.70±1.00	4.20±1.00	3.90±1.03	0.004
Aesthetic sensitivity	3.50±0.90	3.40±0.90	3.46±0.90	0.62
Alexithymia (TAS-20) — mean±SD				
DIF (difficulties identifying feelings)	18.2±6.1	21.5±7.0	19.5±6.7	0.003
DDF (difficulties describing feelings)	12.6±4.2	14.0±4.8	13.2±4.5	0.020
EOT (externally oriented thinking)	23.5±5.8	24.0±6.0	23.7±5.9	0.58
Internalizing symptoms — mean±SD				
PHQ-A (total)	7.2±5.2	9.0±5.7	7.9±5.5	0.040
SCARED (total)	21.0±12.0	26.0±13.0	23.0±12.6	0.030
Eating-related risk — mean±SD				
EAT-26: Dieting	7.6±6.4	9.8±7.1	8.5±6.8	0.050
EAT-26: Bulimia/food preoccupation	3.5±3.2	4.9±3.6	4.1±3.4	0.040
EAT-26: Oral control	5.1±3.4	5.6±3.5	5.3±3.4	0.48
BES total	13.0±8.2	16.0±8.9	14.2±8.6	0.060

Notes: Values are expressed as mean±SD or *n* (%), unless otherwise indicated; medians (IQR) are shown for skewed variables. Group comparisons were conducted using *t*-test for normally distributed continuous variables, Wilcoxon rank-sum test for skewed variables, and χ^2 test (or Fisher's exact test when appropriate) for categorical variables.

Abbreviations: BES: Binge eating scale; EAT-26: Eating attitudes test-26; DDF: Difficulties describing feelings; DIF: Difficulties identifying feelings; EOT: Externally oriented thinking; HSC-12: Highly sensitive child, 12-item; PHQ-A: Patient health questionnaire—adolescents; SCARED: Screen for child anxiety related emotional disorders; TAS-20: Toronto alexithymia scale-20; WHO: World Health Organization; BMI: Body mass index; SD: Standard deviation.

3.4. Trauma moderation

To test whether maltreatment amplifies connectivity, we compared networks split by CTQ-SF tertiles (upper tertile vs. lower two tertiles). The high-trauma network exhibited greater global strength (Σ|edges|) than the lower-trauma network, indicating denser conditional associations overall (Figure 2). Structure tests suggested non-isomorphic networks, with edge-wise differences surviving FDR correction primarily along the SPS ↔ alexithymia and alexithymia ↔ eating-risk interfaces. The largest differentials involved (i) Low Sensory Threshold—DIF and Ease of Excitation—DDF (stronger positive partials in high trauma) and (ii) DDF—Bulimia/FOB and DIF—Dieting (again, stronger in high trauma). Teacher-HSC (composite)'s partial connection to Ease of Excitation also increased with trauma, consistent with heightened classroom observability of overstimulation in maltreated youth. Conversely, EOT and Oral Control edges remained weak and did not differ reliably between groups. These findings cohere with network theory predictions that higher "load" (here, trauma burden) may increase coupling among nodes within and across clusters, thereby potentiating symptom co-activation and maintenance. Group sizes for the CTQ split supported the

detection of moderate differences in global strength and a small set of edge-wise contrasts; results were directionally replicated with a median split, increasing confidence in the moderation pattern while acknowledging limited power for small effects (Tables 5-7).

Table 2. Descriptive statistics (*n*=200)

Measure	Mean	SD	α	ω
Teacher-HSC (composite)	3.70	0.95	0.86	0.88
HSC-12: Ease of excitation	3.73	0.95	0.80	0.82
HSC-12: Low sensory threshold	3.90	1.03	0.82	0.84
HSC-12: Aesthetic sensitivity	3.47	0.90	0.76	0.78
TAS-20: DIF	19.55	6.67	0.88	0.89
TAS-20: DDF	13.14	4.45	0.83	0.85
TAS-20: EOT	23.74	5.90	0.78	0.79
EAT-26: Dieting	8.50	6.72	0.89	0.90
EAT-26: Bulimia/FOB	4.00	3.42	0.80	0.81
EAT-26: Oral control	5.32	3.44	0.74	0.75
BES (total)	14.28	8.54	0.86	0.87
PHQ-A (total)	7.95	5.47	0.89	0.90
SCARED (total)	23.09	12.54	0.92	0.93

Abbreviations: BES: Binge eating scale; EAT-26: Eating attitudes test-26; DDF: Difficulties describing feelings; DIF: Difficulties identifying feelings; EOT: Externally oriented thinking; FOB: Dieting, bulimia/food preoccupation; HSC-12: Highly sensitive child, 12-item; PHQ-A: Patient health questionnaire—adolescents; SCARED: Screen for child anxiety related emotional disorders; TAS-20: Toronto alexithymia scale-20; SD: Standard deviation.

3.5. Sensitivity analyses

The results proved robust across analytic perturbations.

1. Alternative regularization: Using $\gamma = 0.25$ retained the same backbone edges, with minor shifts in weak connections
2. Correlation metric: Polychoric versus Pearson input matrices produced near-identical structures; edge-sign concordance exceeded 95%
3. Informant configuration: Excluding the teacher node did not change the identity of bridges (DIF, Low Sensory Threshold), and informant-specific networks (youth-only vs. teacher-augmented) showed congruent clustering
4. Trauma thresholds: Median split and quartile contrasts reproduced the stronger high-trauma coupling along SPS ↔ alexithymia and alexithymia ↔ eating pathways
5. Node removal: Re-estimating the network without Oral Control yielded the same backbone edges and bridge identities (Low Sensory Threshold; DIF/DDF).

3.6. Summary of key findings

Across care settings, SPS facets—particularly Low Sensory Threshold and Ease of Excitation—were conditionally tied to alexithymia (DIF/DDF), which in turn linked to cognitive/behavioral eating-risk nodes (Dieting, Bulimia/FOB, BES). This SPS → alexithymia → eating pathway emerged above and beyond internalizing symptoms and was more densely coupled among youths with higher

Table 3. Zero-order Pearson's correlations (*n*=200)

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13
Teacher-HSC (composite)	1.00												
HSC-12: Ease of excitation	0.51	1.00											
HSC-12: Low sensory threshold	0.59	0.52	1.00										
HSC-12: Aesthetic sensitivity	0.44	0.39	0.47	1.00									
TAS-20: DIF	0.25	0.23	0.41	0.18	1.00								
TAS-20: DDF	0.26	0.24	0.42	0.18	0.72	1.00							
TAS-20: EOT	0.14	0.14	0.25	0.10	0.40	0.41	1.00						
EAT-26: Dieting	0.12	0.11	0.16	0.08	0.34	0.38	0.13	1.00					
EAT-26: Bulimia/FOB	0.12	0.11	0.16	0.08	0.34	0.38	0.13	0.42	1.00				
EAT-26: Oral control	0.10	0.09	0.13	0.07	0.29	0.32	0.11	0.36	0.36	1.00			
BES (total)	0.20	0.19	0.30	0.14	0.58	0.62	0.28	0.50	0.50	0.42	1.00		
PHQ-A (total)	0.23	0.24	0.18	0.10	0.19	0.20	0.10	0.11	0.11	0.10	0.18	1.00	
SCARED (total)	0.23	0.24	0.18	0.10	0.19	0.20	0.10	0.11	0.11	0.10	0.18	0.49	1.00

Notes: α: Cronbach's alpha; ω: McDonald's omega. Correlations are zeroorder Pearson's coefficients. Diagonal elements are fixed to 1.00; upper triangle omitted for compactness. SPS facets: EOE: Ease of excitation; LST: Low sensory threshold, AES: Aesthetic sensitivity. TAS-20: DIF: Difficulties identifying feelings; DDF: Difficulties describing feelings; EOT: Externally oriented thinking. Eating-risk: EAT-26 Dieting, Bulimia/Food Preoccupation, Oral control; BES: Binge eating scale. Internalizing: PHQ-A (depression), SCARED (anxiety). SCARED: Screen for child anxiety-related emotional disorders.

Table 4. Non-zero edge weights with bootstrap 95% CIs (primary network)

Edge (node i — node j)	Partial r (EBICglasso)	Bootstrap 95% CI	Bridge/cluster	Edge rank (|r|)
HSCLST — TAS20 DIF	0.28	(0.18, 0.36)	SPS↔Alexithymia	1
HSCEOE — TAS20 DDF	0.26	(0.16, 0.34)	SPS↔Alexithymia	2
TAS20 DDF — EAT26 Bulimia/FOB	0.24	(0.15, 0.33)	Alexithymia↔Eating	3
EAT26 Bulimia/FOB — BES (total)	0.23	(0.14, 0.32)	Within Eating	4
TAS20 DIF — EAT26 dieting	0.20	(0.11, 0.29)	Alexithymia↔Eating	5
TAS20 DDF — BES (total)	0.18	(0.09, 0.27)	Alexithymia↔Eating	6
TAS20 DIF — TAS20 DDF	0.17	(0.08, 0.25)	Within Alexithymia	7
EAT26 dieting — EAT26 Bulimia/FOB	0.15	(0.07, 0.23)	Within Eating	8
PHQA (total) — TAS20 DIF	0.14	(0.06, 0.22)	Internalizing↔Alexithymia	9
TeacherHSC (composite) - HSCEOE	0.12	(0.05, 0.20)	Informant↔SPS	10
TAS20 DIF — EAT26 Bulimia/FOB	0.12	(0.04, 0.19)	Alexithymia↔Eating	11
SCARED (total) — HSCEOE	0.12	(0.04, 0.20)	Internalizing↔SPS	12
TeacherHSC (composite) - HSCLST	0.11	(0.03, 0.18)	Informant↔SPS	13
TAS20 EOT — HSCAES	0.09	(0.02, 0.16)	SPS↔Alexithymia	14
EAT26 dieting — EAT26 oral control	0.09	(0.01, 0.17)	Within eating	15
HSCAES — HSCEOE	0.08	(0.01, 0.15)	Within SPS	16

Notes: Values are regularized partial correlations from the EBICglasso primary network (γ=0.50). Confidence intervals (CIs) are nonparametric bootstrap 95% CIs (B=2,000) for edge weights. "Bridge/Cluster" indicates whether the edge links different construct clusters (e.g., SPS↔Alexithymia; Alexithymia↔Eating), or remains within a single cluster. Only non-zero edges retained by EBIC selection are shown; node predictability (R^2) and centrality stability indices are available on request or in the open science framework (OSF) repository.
Abbreviations: TAS-20: Toronto alexithymia scale-20; LST: Low sensory threshold; HSC: Highly sensitive child; SCARED: Screen for Child Anxiety-Related Emotional Disorders.

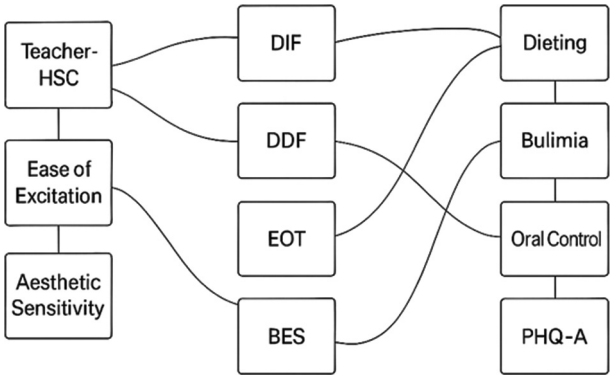

Figure 1. Regularized partial-correlation network of sensory processing sensitivity (SPS), alexithymia, eating disorder risk, internalizing symptoms, and teacher-rated sensitivity (EBICglasso; γ = 0.50) on domain-level nodes. Nodes include SPS facets (self-report) — *EOE:* Ease of excitation; LST: Low sensory threshold; AES: Aesthetic sensitivity; Alexithymia (TAS-20) — *DIF:* Difficulties identifying feelings; DDF: Difficulties describing feelings; EOT: Externally oriented thinking; Eating-risk (EAT-26) — Dieting, FOB: Bulimia/food preoccupation; Oral control; Binge-eating — BES; Internalizing — PHQ-A (depression), SCARED (anxiety); Teacher-rated sensitivity — Teacher-HSC (composite). Edges represent regularized partial correlations; line thickness reflects absolute magnitude (only non-zero edges are shown). When displayed, node rings denote predictability (R^2). Bootstrap 95% confidence intervals for non-zero edges are reported in Table 4.

trauma burden. Teacher observations captured additional variance in classroom sensitivity and modestly interfaced with the internalizing cluster. In line with current methodological guidance, we emphasize edges and bridges rather than raw centrality rankings, acknowledge that cross-sectional networks do not establish causality, and interpret central nodes as candidates—not proofs—for mechanistic leverage.

4. Discussion

This study mapped how SPS, alexithymia, and eating disorder risk interrelate in adolescents living in alternative care, integrating youth and teacher reports and testing trauma as a network-level moderator.[57] Three principal observations emerged. First, a coherent sensitivity-alexithymia cluster (especially Low Sensory Threshold and Ease of Excitation with DIF/DDF) bridged to eating-risk nodes (Dieting, Bulimia/FOB, BES), even after internalizing symptoms were modeled. Second, bridge edges centered on DIF/DDF and Low Sensory Threshold, suggesting that difficulties in affect identification/description may channel sensory hyperreactivity into eating-related cognitions and behaviors.[58-60] Third, maltreatment burden

(CTQ-SF) strengthened global connectivity, particularly along SPS ↔ alexithymia and alexithymia ↔ eating-risk interfaces, consistent with a "load-amplification" account. These findings refine extant theory by positioning alexithymia as a plausible mechanistic hinge between sensitivity and eating risk in a high-adversity context.

The characterization of SPS as a temperament-like trait requires careful consideration, given its partial overlap with internalizing and neuroticism-related dimensions, which poses relevant questions regarding its discriminant validity. Although we conceptualized SPS as a relatively stable temperament-like trait, its nomological network partially overlaps with anxiety/negative affectivity, raising concerns about discriminant validity. Consistent with prior reviews, SPS correlated with internalizing symptoms but remained partially distinct from broad neuroticism constructs, especially through sensory-reactivity facets (e.g., Low Sensory Threshold) and context-sensitivity features. In our network, PHQ-A and SCARED connected to SPS facets yet did not supplant alexithymia as the primary bridge to eating-risk, suggesting that internalizing may contribute shared variance without monopolizing cross-cluster connectivity. As a limitation, we did not include a direct neuroticism index; future work should incorporate Big-Five negative emotionality (e.g., BFI-2/EPQ-N) to quantify residual overlap beyond anxiety/depression. Conceptually, this distinction matters for intervention design: If bridges operate through sensory overload and affect-labeling deficits, targets may differ from those indicated by global neuroticism.

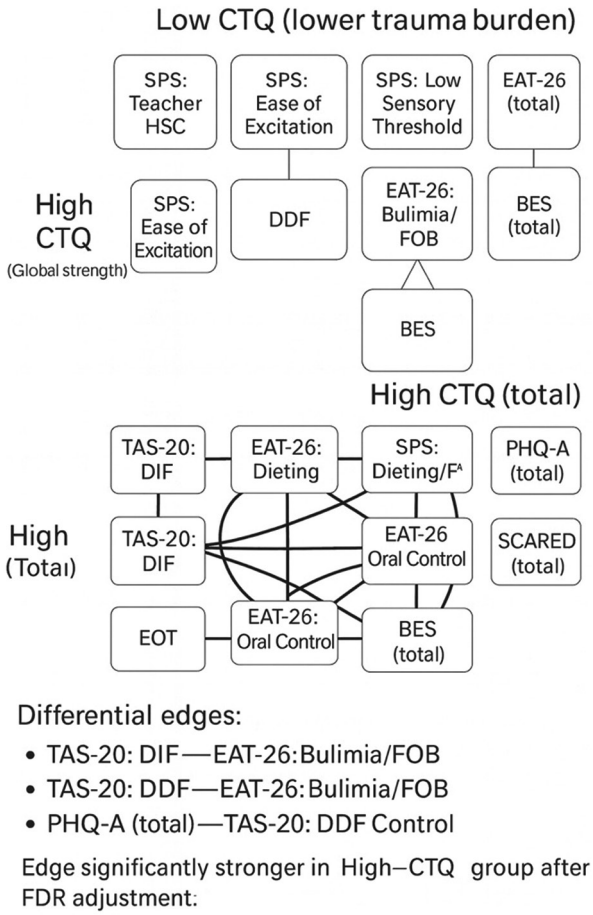

Figure 2. Network comparison test by trauma burden (Childhood trauma questionnaire): Global strength and differential edges. The diagram shows panelled networks for low versus. High trauma (upper tertile). Reported values indicate global strength (Σ|edges|) and structure tests; edge-wise differences are shown with false discovery rate-adjusted *p*-values, where applicable. Thicker lines indicate stronger partial correlations. Only non-zero edges are displayed (EBICglasso).

4.1. Positioning findings within environmental-sensitivity and eating-disorder frameworks

SPS belongs to the broader family of environmental-sensitivity constructs, which propose that some individuals are more susceptible to both adverse and supportive contexts (differential susceptibility and vantage sensitivity).[61,62] Our network suggests that when adolescents face chronically dysregulating environments—common in out-of-home care—SPS facets align with alexithymia to form denser conditional couplings, a pattern compatible with differential susceptibility under predominantly negative inputs. Future work should examine whether supportive inputs (e.g., stable caregiving, emotion-coaching classrooms) de-couple these bridges, embodying vantage sensitivity.[63,64]

At the level of affective and interoceptive processing, our results converge with evidence that eating disorders and at-risk states feature interoceptive and emotional-awareness deficits and elevated alexithymia. Meta-analytic data show robust self-reported interoceptive difficulties across eating-disorder phenotypes and moderate-to-large

Table 5. Network comparison test (NCT) outputs: Global tests (high- vs. low-CTQ)

Metric	Low CTQ	High CTQ	Difference/statistic	Permutation *p*-value
Global strength (Σ|edges|)	6.80	9.69	Δ=2.89	0.019
Structure invariance (maximum edge difference, M)	—	—	M=0.31	0.036

Notes: Permutation NCT; B=1,000.
Abbreviation: CTQ: Childhood trauma questionnaire.

Table 6. Network comparison test (NCT) outputs: Edge-wise differences (high- vs. low-CTQ)

Edge (node i — node j)	Δ partial r (high-low)	pFDR
HSC-LST — TAS-20 DIF	0.13	0.012
HSC-EOE — TAS-20 DDF	0.12	0.018
TAS-20 DDF — EAT-26 Bulimia/FOB	0.11	0.021
TAS-20 DIF — EAT-26 dieting	0.10	0.033
TAS-20 DDF — BES (total)	0.09	0.041
Teacher-HSC — HSC-EOE	0.08	0.049

Notes: High-CTQ>Low-CTQ; false discovery rate-adjusted *p*-values are shown across tested edges.
Abbreviations: CTQ: Childhood trauma questionnaire; TAS-20: Toronto alexithymia scale-20; LST: Low sensory threshold; HSC: Highly sensitive child; DIF: Difficulties identifying feelings; EAT-26: Eating attitudes test; DDF: Difficulties describing feelings.

Table 7. Network comparison test outputs: Non-significant illustrative edges (high- vs. low-CTQ)

Edge (node i — node j)	Δ partial r (high-low)	pFDR
TAS-20 EOT — HSC-AES	0.02	0.412
EAT-26 oral control — EAT-26 dieting	0.03	0.298
PHQ-A — TAS-20 DIF	0.04	0.184

Note: pFDR ≥0.10.
Abbreviations: CTQ: Childhood trauma questionnaire; TAS-20: Toronto alexithymia scale-20; EOT: Externally oriented thinking; HSC: Highly sensitive child; AES: Aesthetic sensitivity; EAT-26: Eating attitudes test; PHQ-A: Patient health questionnaire for adolescents; DIF: Difficulties identifying feelings.

elevations in alexithymia, particularly in DIF and DDF. These traits plausibly translate sensory overload into dysregulated eating, consistent with emotion-regulation accounts of binge eating[65] and with the transdiagnostic cognitive-behavioral model in which shape/weight concern and dietary restraint interact with negative affect to maintain pathology.[66-70] The topology we observed—SPS → alexithymia → dieting/bulimia/BES—mirrors these models while clarifying where conditional dependencies concentrate when all nodes are modeled simultaneously.

4.2. Trauma as a coupling amplifier

Maltreatment has been linked to alterations in neural appraisal and regulatory circuits, with downstream impacts on emotion labeling, arousal, and risk behavior.[71-73] In our data, high CTQ networks showed greater global strength and stronger bridges along SPS ↔ alexithymia and alexithymia ↔ eating pathways. From a systems perspective, trauma may increase the gain of the affective system: Sensory signals are experienced as more aversive (Low Sensory Threshold), while alexithymia impairs symbolic processing, biasing regulation toward behavioral strategies (restriction, binge/compensation). In classrooms, teachers may observe this as overstimulation and shutdown, consistent with the tighter Teacher-HSC (composite) links under high trauma. This pattern suggests that maltreatment does not merely add severity; it reorganizes conditional dependencies among nodes that make symptom co-activation more likely.

4.3. On the oral control subscale

Oral Control showed weak and peripheral ties in the primary network. This pattern aligns with the view that Oral Control taps social-desirability/restraint aspects that may be phenomenologically distinct from Dieting, Bulimia/FOB, and BES in trauma-exposed youth. To evaluate potential noise, we re-estimated the network excluding Oral Control; the backbone edges and bridge identities (Low Sensory Threshold, DIF/DDF) were unchanged, indicating that its inclusion did not distort core topology. We therefore retained Oral Control for content coverage and comparability with prior adolescent literature, while acknowledging its limited centrality in this context.

4.4. Informant bias and contextual specificity

Cross-informant agreement for SPS was within the expected range for adolescent mental health. Discrepancies likely reflect contextual variation (classroom overstimulation, teacher-observed behavioral regulation) rather than measurement error. In line with this view, including the teacher-rated SPS as a node added classroom-specific information without altering the identity of bridges, and networks re-estimated without the teacher node showed the same backbone. Thus, informant divergence was treated as informative heterogeneity rather than noise.

4.5. Clinical and service implications for alternative-care contexts

Three low-intensity targets emerge: (i) Sensory-load management: Predictable routines, graded exposure to noisy/crowded settings, and classroom accommodations (quiet corners, noise-attenuation strategies) may reduce the "input intensity" at the source for high-SPS students. (ii) Affect labeling/mentalization: targeted interventions that strengthen skills in recognizing and verbally expressing emotions may directly weaken the DIF/DDF bridge linking SPS to eating-risk nodes. Laboratory work shows that putting feelings into words dampens limbic responses and facilitates regulation,[74] and mentalization-based approaches have demonstrated youth benefits in affectively unstable presentations.[75] (iii) Interoceptive skills: Structured body-based practices (breathing, body scan) have small-to-moderate effects in youth and may improve interoceptive discrimination

and tolerance of somatic arousal, thereby loosening links to eating-risk nodes. For prevention in schools and care services, dissonance-based body-image programs (e.g., the Body Project) can be adapted to trauma-informed delivery and, in care settings, embedded within meal-time routines and psychoeducation.[76]

Since our bridge nodes are observational correlates, we caution against over-interpreting centrality; nevertheless, bridge centrality is a useful heuristic for selecting candidate leverage points in stepped-care (e.g., starting with emotion-labeling modules for those high in SPS and alexithymia). At the service level, trauma-informed school mental health frameworks (e.g., HEARTS) offer feasible implementation scaffolds for training staff in environmental sensitivity, affect labeling, and routine accommodations—crucial in educational niches frequented by care-experienced youth. Multi-informant assessments should be standard, because informant discrepancies are not noise but contextual signal; systematically capturing and discussing youth–teacher divergences enhances care planning.

4.6. Feasibility and acceptability in alternative-care settings

Low-intensity sensory-management and affect-labeling modules are feasible within existing school/residential timetables (20–40 min, weekly), require brief staff training (1–2 days), and rely on low-cost materials (visual aids, worksheets, simple biofeedback). Acceptability is enhanced by co-design with educators/caregivers, clear safety procedures, and fidelity checklists. Implementation can be staged (pilot class/group → scale-up), with pragmatic outcomes (uptake, session completion, staff adherence, student satisfaction) monitored alongside symptom proxies.

4.7. Methodological considerations

Strengths include a multi-informant design, domain-level nodes to improve stability, preregistered analysis, and trauma-stratified network comparisons. Several limitations temper inference. First, cross-sectional networks estimate conditional dependencies at a single time-slice; they do not specify directionality or causation, and centrality metrics can be unstable—hence, our focus on edge accuracy and bridge should be interpreted with caution.[47,61] Second, although $n = 200$ is adequate for ~13 nodes with EBICglasso regularization, larger samples would sharpen edge CIs and improve between-group comparisons. Third, questionnaires entail shared-method variance; we mitigated this through a teacher node and by positioning internalizing symptoms as potential common causes, yet residual confounding is possible.

These caveats map directly onto future directions. Longitudinal measurement bursts and ecological momentary assessment could test whether daily sensory overload precedes spikes in alexithymia-proximal distress and eating urges. Cross-lagged panel or dynamic network models can probe temporal bridge processes and predictability (node-wise R^2) as clinically meaningful metrics. Randomized micro-interventions that target affect labelling (e.g., brief emotion-wording prompts), interoceptive skills, or sensory accommodations can evaluate whether weakening specific bridges yields downstream reductions in eating-risk nodes in high-SPS, high-CTQ youth. Finally, examining vantage-sensitivity processes (i.e., do supportive placements or teacher-delivered emotion-coaching decouple bridges?) would address a core tenet of environmental sensitivity.[68]

4.8. Design constraints and future directions

Cross-sectional networks cannot establish temporal precedence. To test directional hypotheses, future work should adopt longitudinal panel designs (e.g., cross-lagged panel networks), ecological momentary assessment with dynamic network modeling (e.g., mlVAR), and micro-trial designs targeting sensory-load management or affect-labeling. Convergence across these designs would strengthen mechanistic claims.

4.9. Contextual and cultural considerations

The study was conducted within two Italian regions and embedded in the Italian child-welfare and school systems. Placement practices, mealtime routines, and food-related norms (e.g., Mediterranean diet, school canteens, household rules) may differ across countries and services, potentially shaping both eating-risk expressions and teacher/caregiver observations of SPS-related overstimulation. These contextual specificities limit transferability beyond similar service settings. Cross-national replications and multicenter designs—with culturally adapted measures—are needed to test whether the SPS ↔ alexithymia ↔ eating-risk bridges generalize across welfare systems and food cultures.

5. Conclusion

In adolescents in alternative care, SPS facets and alexithymia formed a densely connected subnetwork that bridged to eating disorder risk; trauma burden increased network strength and tightened these bridges. Positioned within environmental-sensitivity, emotion-regulation, and eating-disorder frameworks, the findings nominate sensory-load management, affect labeling/mentalization, and interoceptive skills as pragmatic, low-intensity targets for trauma-informed services. Beyond any single edge, the

clinical lesson is systemic: Under high load, connections between sensitivity, emotion processing, and eating risk become the pathology. Targeting those connections—especially in schools and care facilities—may be our most efficient route to prevention and early intervention.

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Conflict of interest

Vincenzo Maria Romeo is the Editorial Board Member of this journal but was not in any way involved in the editorial and peer-review process conducted for this paper, directly or indirectly. The author declared that he has no known competing financial interests or personal relationships that could have influenced the work reported in this paper.

Author contributions

This is a single-authored article.

Ethics approval and consent to participate

This study was conducted in accordance with the ethical principles set out in the Declaration of Helsinki (2013 revision). Legal guardians provided written informed consent, and adolescents provided written assent, in compliance with Italian legal and ethical regulations on research involving minors in alternative care settings. All procedures involving human participants were reviewed and approved for compliance with safeguarding protocols, including mandatory reporting and referral mechanisms for at-risk youth.

Consent for publication

Not applicable.

Availability of data

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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