Research Square

Preprints are preliminary reports that have not undergone peer review. They should not be considered conclusive, used to inform clinical practice, or referenced by the media as validated information.

ADHD and OCD Symptom Co-Occurrence in adolescent: A Bridge Network Analysis

Lianjingyi Liang
Shandong Second Medical University

Wanling Zhang
Beijing Children's Hospital, Capital Medical University, National Center for Children Health

Hui Xu
Beijing Children's Hospital, Capital Medical University, National Center for Children Health

Yanyu Wang
Shandong Second Medical University

Ying Li
liying@bch.com.cn

Beijing Children's Hospital, Capital Medical University, National Center for Children Health

Research Article

Keywords: ADHD, OCD, co-occurrence, gender, network analysis

Posted Date: October 15th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-9620394/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License. Read Full License

Additional Declarations: No competing interests reported.

ADHD and OCD Symptom Co-Occurrence in adolescent: A Bridge Network Analysis

Lianjingyi Liang[1,2], Wanling Zhang[2,*], Hui Xu[3,*], Yanyu Wang[1], Ying Li[2,*]

[1] School of Psychology, Shandong Second Medical University, Weifang, Shandong, China

[2] Department of Psychosomatic Medicine, Beijing Children's Hospital, Capital Medical University, National Center for Children Health, Beijing, China

[3] Big Data Center, Beijing Children's Hospital, Capital Medical University, National Center for Children's Health, Beijing, China

Correspondence

Ying Li, Beijing Children's Hospital, Capital Medical University, National Center for Children Health, Beijing 100046, China

Email address: liying@bch.com.cn

Wanling Zhang, Beijing Children's Hospital, Capital Medical University, National Center for Children Health, Beijing 100046, China

Email address: linya-chang@foxmail.com

Hui Xu, Beijing Children's Hospital, Capital Medical University, National Center for Children Health, Beijing 100045, China

Email address: xuhuiyunyun@126.com

Abstract:

Background: Attention deficit hyperactivity disorder (ADHD) and obsessive compulsive disorder (OCD) frequently co-occur in adolescents, and this co-occurrence exhibits gender differences. Network analysis provides an effective approach to examine these symptom level interactions; however, the specific bridging symptoms linking ADHD and OCD and their potential gender variations remain unclear. This study aims to identify these bridging symptoms and examine their gender differences.

Methods: This study utilized data from 11,807 adolescents at baseline in the Adolescent Brain Cognitive Development (ABCD) study, with a subset of 616 meeting criteria for co-occurring subclinical ADHD and OCD symptoms. Symptoms were measured using the Child Behavior Checklist (CBCL) ADHD and OCD subscales. Bridge network analysis was applied to construct symptom level networks at ages 9–10, 10–11, and 11–12 years. The analysis aimed to identify core and bridge symptoms connecting ADHD and OCD clusters and to examine gender differences in these network structures.

Results: Network analysis revealed stable core symptoms within each disorder cluster ("can't concentrate" in ADHD; "strange behavior" in OCD). Key bridge symptoms connecting the clusters included "loud," "can't get their mind off certain thoughts," "strange behavior," and "impulsive," though these evolved over time, particularly during early adolescence. The subclinical comorbid group exhibited a more densely connected and stable network structure than the full sample. Gender differences were observed, with males showing stronger behavioral bridge symptoms and females exhibiting stronger cognitive and affective connections.

Conclusion: This study found that across four years, core symptoms of ADHD and OCD remained stable, whereas bridge symptoms evolved dynamically, with early

adolescence emerging as a key period for network reorganization. Gender differences were pronounced: males predominantly showed connections through OCD related symptoms, whereas females exhibited stronger ADHD related bridging. Thus, these findings provide precise targets for developmentally timed and personalized interventions.

Trial registration:

Not applicable.

Keywords: ADHD; OCD; co-occurrence; gender; network analysis

Background:

Attention deficit hyperactivity disorder (ADHD) and obsessive compulsive disorder (OCD) are both prevalent and impairing conditions that frequently emerge during childhood and adolescence [1, 2]. ADHD is characterized by persistent patterns of inattention, hyperactivity, and impulsivity that interfere with functioning, whereas OCD is defined by the presence of time consuming obsessions and compulsions [3]. Notably, these disorders co-occur at clinically significant rates in adolescents. Approximately 5.5% of adolescent with ADHD are diagnosed with comorbid OCD, a prevalence substantially higher than that in the general population. Within this population, comorbid OCD appears more common in girls than in boys [4, 5]. This co-occurrence is of considerable clinical importance. It complicates diagnostic assessment by obscuring the core symptoms of each disorder and is associated with greater functional impairment and poorer treatment response compared to either condition alone [6, 7]. These inherent challenges are further compounded by specific treatment difficulties, as standard interventions for one disorder are often less effective in the presence of the other [6]. Consequently, elucidating the symptomatic interplay between ADHD and OCD in adolescents is fundamental to developing more effective clinical strategies.

Building on the significant clinical challenges posed by ADHD-OCD co-occurrence, emerging evidence suggests shared neurobiological vulnerabilities may

underpin this overlap. Specifically, dysfunction in fronto-striatal circuits and common genetic factors within a neurodevelopmental framework are frequently implicated [8, 9]. At the symptomatic level, a complex bidirectional interplay is evident and exacerbates impairment. For instance, core ADHD features like attentional dysregulation and impulsivity may lower distress tolerance, potentially intensifying compulsive urges as maladaptive coping mechanisms [10, 11]. Conversely, the time consuming nature of OCD rituals can severely disrupt focus and task persistence, thereby amplifying ADHD like inattentiveness [8]. This reciprocal reinforcement creates a vicious cycle, leading to greater overall symptom severity and functional decline than either disorder alone [10, 12]. Despite recognition of this symptomatic interaction, the precise symptom level associations through which they influence each other remain inadequately mapped. Relatively few studies have systematically investigated the core bridging symptoms that may dynamically connect ADHD and OCD symptom clusters within an individual [13, 14]. Importantly, there is emerging evidence that the symptom expression and comorbidity profiles of these disorders differ by gender [15]. For example, among adolescent with ADHD, boys tend to exhibit more externalizing problems, whereas girls show a more pronounced pattern of comorbid internalizing disorders such as anxiety and specific phobias [16]. Traditional diagnostic approaches, which focus on disorder level overlap, are not designed to identify which specific symptoms (e.g., particular types of intrusive thoughts, executive dysfunctions) might act as primary conduits for symptom exacerbation across diagnostic boundaries [12, 17]. Consequently, a fuller elucidation of these interactions would greatly facilitate the development of targeted intervention strategies [8, 18].

To directly address this gap and move beyond disorder level descriptions, bridge network analysis emerges as a methodological solution [18, 19]. This approach statistically identifies and quantifies the bridge centrality of individual symptoms, pinpointing those that exhibit the strongest connections between the ADHD and OCD clusters [2, 9]. Recent applications of network psychopathology have demonstrated its utility in unpacking complex comorbidities by revealing direct symptom to symptom

associations. By modeling these dynamic relationships, bridge analysis can empirically reveal which symptoms act as the most influential bridges [20], thereby elucidating the core symptoms driving the comorbid presentation and informing potential intervention targets. Critically, applying this framework to adolescent populations and importantly, to those with subclinical symptoms is essential for a complete understanding. Subclinical symptoms often precede full disorder onset and share underlying genetic and neurobiological mechanisms with clinical diagnoses [14, 21]. Examining these dimensional symptom interactions offers greater sensitivity for identifying early risk markers and potential bridging symptoms, capturing the continuum of impairment [22]. Understanding how subclinical ADHD traits interact with emerging OCD tendencies can reveal the earliest symptoms to comorbidity [23], thereby informing preventative strategies long before significant functional decline occurs [24].

In summary, while the clinical co-occurrence and symptom level interactions between ADHD and OCD in adolescents are evident, the precise core bridging symptoms that facilitate this symptom overlap remain unclear. Bridge network analysis offers a powerful methodological framework to directly address this gap. The present study employs this approach by leveraging data from the Adolescent Brain Cognitive Development (ABCD) Study, a large scale, longitudinal cohort following over 11,800 U.S. children from age 9-10 with deep phenotyping [25, 26]. Most prior network studies of ADHD-OCD have focused on clinical samples; this study shifts the focus to the understudied subclinical and dimensional manifestations in a population based cohort. Applying bridge network analysis to the ABCD data, this study aims to: (1) construct symptom level network models of ADHD-OCD co-occurrence; (2) identify stable and dynamic bridging symptoms that connect the two symptom clusters over time; and (3) examine gender differences in these bridging symptoms and network structures during adolescence. Achieving these objectives will elucidate the bridging symptoms that connect ADHD and OCD symptom clusters, informing the development of more targeted and developmentally sensitive prevention and early intervention strategies.

Method:

Sample:

The data for this study were drawn from the Adolescent Brain Cognitive Development (ABCD) (Data Release 5.0; NDAR-DOI: 10.15154/1503209) study, a large scale, longitudinal cohort in the United States. The ABCD study recruited over 11,800 children aged 9–10 years at baseline [27], aiming to establish a nationally representative sample through a multi stage probability sampling design [28]. The current analyses utilize data from the 3 year follow up assessment (Data Release 5.0). All study procedures were approved by the central Institutional Review Board, with written informed consent obtained from a parent or guardian and assent from each child. Detailed recruitment and study design have been described in prior publications [17].

This study utilized parent reported Child Behavior Checklist (CBCL) data from the ABCD study. Given the longitudinal design and varying participation across waves, we employed a cross sectional network approach, estimating symptom networks independently for the baseline (T0, ages 9–10), 1 year (T1), and 2 year (T2, ages 11–12) follow ups using data available at each respective assessment. Participants were included in a given timepoint's analysis if they had valid CBCL data at that wave, resulting in differing sample sizes across T0, T1, and T2. At each timepoint, two analytic samples were defined: (1) The full sample included all participants with valid data, used to model population wide networks; and (2) The subclinical sample, a subset of the full Sample, comprised participants scoring above pre-defined thresholds on both the DSM-oriented ADHD and OCD scales at that wave, enabling focused analysis of bridging symptoms within co-occurring traits. At baseline (T0), the full sample comprised 11,807 participants, with a subclinical sample of 616. The number of valid cases at each timepoint is detailed in Table 1.

Demographic characteristics for the overall analytic sample are summarized in Table S1.

Measurement:

The Children's Behavior Checklist was used to evaluate children in the ABCD cohort of dimensional psychopathology and children's adaptive function[29]. The English version consists of 113 items, aiming to assess emotional and behavioral problems. Each item is scored on a 3 point scale: 0 (incorrect), 1 (rarely correct), and 2 (usually correct). We used the AHDH subscale and OCD subscale in CBCL to evaluate ADHD and OCD, among which the CBCL-ADHD contained 7 items (ADHD04 "Fails to finish", ADHD08 "Can't concentrate", ADHD10 "Can't sit still", ADHD41 "Impulsive", ADHD78 "Inattentive", ADHD93 "Talks much", ADHD104 "Loud")[28]. The CBCL-OCD contains 8 items (OCD09 "Can't get their mind off certain thoughts", OCD31 "Feels they might think or do something bad", OCD32 "Feels they have to be perfect", OCD52 "Feels too guilty", OCD66 "Repeats certain acts over and over", OCD84 "Strange behavior", OCD85 "Strange ideas", OCD112 "Worries")[14]. The specific items of CBCL-ADHD and CBCL-OCD can see in Table 1.

INSERT Table 1: The items of ADHD and OCD in the CBCL

The CBCL-OCD subscale consists of 8 items. The total score is obtained by adding up the scores of these 8 items. The total score range is limited to between 5 and 16 points. The cut off point for subclinical screening is 3.5 points or above, which has reasonable specificity (67.2%)[30]. The CBCL-ADHD scale consists of 7 items. The total score is obtained by adding up the scores of these 7 items. The total score range is limited to between 4 and 10 points, and the cut off point for subclinical screening is 3.5 points or above[31].

Statistical analysis:

Network analyses were performed using R software (version 4.3.0). The network estimation and visualization were implemented through the " bootnet " and " qgraph " packages [32]. The network construction employed EBIC graphical LASSO regularization with the tuning parameter set to 0.5, which effectively reduced false positive connections by producing sparse network representations. Node centrality was quantified using strength centrality to identify the most influential symptoms within the network configuration [33].

To examine cross symptoms connections, bridge symptoms were identified using the "networktools" package through bridge centrality indices and bridge strength metrics. The reliability of network parameters was verified using bootstrap procedures to assess estimation accuracy and stability. Additionally, the case dropping method was applied to evaluate the stability of centrality indices, requiring a minimum correlation coefficient of 0.7 between the original and subset based centrality rankings [7].

The correlation stability coefficient (CS-coefficient) was computed to determine the robustness of centrality indices, representing the maximum proportion of cases that can be excluded while maintaining a correlation of at least 0.7 with the original centrality values [8]. According to established methodological standards, CS-coefficients exceeding 0.50 indicate excellent stability, values above 0.25 are considered acceptable, while values below 0.25 suggest that centrality interpretations should be treated with caution [18].

Results:

Descriptive Statistics:

The gender distribution of adolescents in the entire sample and the subclinical sample for each year is shown in Table 2. The full sample comprised 11,807 participants at baseline (T0), maintaining approximately 10,000 participants through the 3 year follow up (T3). The subclinical group with co-occurring ADHD-OCD

symptoms included 616 participants at baseline, with 463 remaining at the final assessment. Both samples demonstrated consistent gender distributions across all time points. In the full sample, males consistently accounted for 52.2%–52.6% of participants. A more pronounced gender disparity was observed in the subclinical group, where males represented 63.3% of participants at baseline and maintained a majority (56.2%–63.3%) throughout the study period. The specific demographic characteristics are presented in Supplementary Table S1. Before network analysis, we checked mean item level, item informativeness, and item relevance (see Supplementary Figure S1).

INSERT Table 2: The demographic and behavioral characteristics of the full sample and the OCD subclinical sample with comorbid ADHD.

Network Characteristics:

Network analyses were conducted for the full sample at four time points. Regularized partial correlation networks are shown in Figure 1, and centrality in Figure 2. At baseline, of the 105 edges, 92 were non-zero with an average weight of 0.06. Global strength was 3.95, and the correlation stability coefficient (CS) for expected influence was 0.75. The strongest connections were within disorder clusters: between "can't concentrate" and "inattentive" (edge stability = 0.58) in the ADHD domain, and between "strange behavior" and "strange ideas" (edge stability = 0.51) in the OCD domain. Centrality analysis identified "can't concentrate" (EI = 1.59), "inattentive" (EI = 1.23), and "strange behavior" (EI = 1.47) as the most influential symptoms. At T1, connectivity increased (global strength = 4.03) while maintaining similar structure (CS = 0.75), with the same central symptoms: "can't concentrate" (EI = 1.58), "inattentive" (EI = 1.18), and "strange behavior" (EI = 1.40). By T2, the network consolidated further (global strength = 4.25). At T3, the structure stabilized (global strength = 4.16), with consistent centrality patterns and excellent stability (CS = 0.75 for expected influence).

INSERT Figure 1. Network constructed via the graphical LASSO depicting regularized partial correlations between pairs of OCD and ADHD in the full sample

INSERT Figure 2. Centrality for the network analysis in the full sample

The subclinical ADHD-OCD group showed the same network characteristics as the full sample in the first three assessments (networks in Figure 3, centrality in Figure 4; stability CS = 0.75). At T3, the network changed: global strength was 2.77, CS for expected influence remained 0.75. The strongest within cluster connections were between "strange behavior" and "strange ideas" (edge stability = 0.55) in the OCD domain, and between "can't concentrate" and "inattentive" (edge stability = 0.52) in the ADHD domain. Centrality analysis identified "can't concentrate" (EI = 2.15), "can't sit still" (EI = 1.05), and "strange ideas" (EI = 0.18) as the most influential symptoms. Edge strength stability and confidence intervals for centrality estimates are shown in Figures S2 and S3, respectively.

INSERT Figure 3. Network constructed via the graphical LASSO depicting regularized partial correlations between pairs of OCD and ADHD in the subclinical sample

INSERT Figure 4. Centrality for the network analysis in the subclinical sample

Bridge Symptom Identification:

Bridge expected influence analysis in the full sample at baseline identified three primary connectors between ADHD and OCD symptom clusters: "loud" (bridge EI = 0.25), "can't get their mind off certain thoughts" (bridge EI = 0.29), and "strange behavior" (bridge EI = 0.27). The correlation stability coefficient for bridge centrality was adequate (CS = 0.75). At T1, the bridge symptom profile remained consistent, with "impulsive" (bridge EI = 0.24), "can't get their mind off certain thoughts" (bridge

EI = 0.27), and "strange behavior" (bridge EI = 0.26) as connectors (CS = 0.75). T2 showed complete consistency with T1. By T3, the bridge profile evolved: "impulsive" (bridge EI = 0.23), "can't get their mind off certain thoughts" (bridge EI = 0.24), and "repeats certain acts over and over" (bridge EI = 0.23) emerged as the most influential connectors, with stability remaining excellent (CS = 0.75). The bridging network structure is shown in Figure 5, and centrality patterns in Figure 6.

INSERT Figure 5. Bridge Network constructed of OCD and ADHD in the full sample

INSERT Figure 6. Bridge centrality for the network analysis in the full sample

In the subclinical group, the bridge symptom profile showed greater dynamic changes. At baseline, "impulsive" (bridge EI = 0.21), "can't get their mind off certain thoughts" (bridge EI = 0.31), and "strange behavior" (bridge EI = 0.24) were the main connectors (CS = 0.59). T1 maintained this pattern. By T2, the profile evolved: "can't concentrate" (bridge EI = 0.22), "can't get their mind off certain thoughts" (bridge EI = 0.33), and "strange behavior" (bridge EI = 0.30) became primary connectors (CS = 0.52). At T3, the pattern further evolved to "can't concentrate" (bridge EI = 0.23), "can't get their mind off certain thoughts" (bridge EI = 0.35), and "repeats certain acts over and over" (bridge EI = 0.23), with stability remaining at CS = 0.52. The bridging network structure for the subclinical group is shown in Figure 7, and centrality patterns in Figure 8. Differences in edge weights and centrality are shown in Figures S4 and S5, respectively.

INSERT Figure 7. Bridge Network constructed of OCD and ADHD in the subclinical

INSERT Figure 8. Bridge centrality for the network analysis in the subclinical sample

Gender differences in network characteristics:

Network comparison tests revealed distinct gender patterns. In the full sample, males and females showed generally consistent network structures across assessments, with no significant differences in global strength (all $p > 0.05$) and only marginal structural variation at T3 ($p = 0.04$). Both genders maintained excellent stability (CS = 0.68–0.75). The subclinical group exhibited more pronounced gender differences. At baseline, significant structural divergence emerged ($p = 0.02$) despite comparable global strength (males: 2.56, females: 2.64, $p = 0.48$). Both genders shared "can't concentrate" and "strange behavior" as core symptoms, while males additionally featured "inattentive". Bridge symptoms showed distinct patterns: males exhibited OCD focused bridges (intrusive thoughts, unusual behaviors), whereas females showed ADHD related bridges (impulsivity, verbal symptoms). These qualitative differences persisted throughout the study with adequate stability (CS = 0.52–0.59). Longitudinal analysis indicated that initial structural differences in the subclinical group diminished at T1–T3 (all $p > 0.05$), but gender specific symptom patterns remained stable. Both genders maintained higher global strength than the full sample, with consistent stability (CS = 0.52 – 0.65) across time points. Gender network comparisons are shown in Figure S6.

Discussion:

This study examined ADHD and OCD symptom networks across four annual assessments in adolescent, revealing three key patterns. First, "can't concentrate" as the central ADHD symptom and "strange behavior" as the central OCD symptom were stable across both the full and subclinical samples, suggesting these symptoms serve as transdiagnostic anchors independent of symptom severity [6, 34]. Second, in the subclinical group, the primary ADHD related bridge shifted from "impulsive" at baseline to "can't concentrate" at later assessments, while "can't get their mind off certain thoughts" strengthened and "repeats certain acts over and over" emerged as a

key connector. This pattern suggests a maturation of cognitive control alongside persistent intrusive phenomena [14, 35]. Third, gender differences were observed: males showed stronger connections through OCD related symptoms, whereas females exhibited stronger ADHD related bridges [28]. These gender specific patterns persisted across the study period, even as overall structural differences diminished [5, 8]. Together, these findings highlight the interplay between stable core symptoms and dynamic bridge symptoms in adolescent ADHD-OCD symptom co-occurrence, underscoring the importance of developmental timing, symptom specific connections, and gender in understanding the etiology and progression of these symptom clusters.

This study reveals a stable core symptom architecture within ADHD-OCD symptom networks that persists across different population groups. The central position of attention symptoms in ADHD networks underscores the fundamental role of executive function deficits [36], consistent with neurocognitive models identifying attention regulation as a core impairment in ADHD [37]. The anchoring function of behavioral symptoms in OCD networks highlights the diagnostic importance of observable compulsive behaviors, which may serve as reliable indicators for early identification of obsessive compulsive tendencies in adolescent [6]. The cross group consistency in network structure supports dimensional models of psychopathology, suggesting that ADHD-OCD symptom overlap exists on a continuum rather than representing distinct categorical entities [35, 38]. This finding aligns with recent cognitive models proposing that certain thought patterns may simultaneously contribute to both attentional difficulties and compulsive behaviors through shared mechanisms [7, 20]. The high stability coefficients further validate the reliability of this core architecture, indicating that these symptom relationships remain robust across different developmental stages and population characteristics.

The shift from "impulsive" to "can't concentrate" as the primary ADHD related bridge during early adolescence aligns with executive function maturation, particularly cognitive control development in the dorsolateral prefrontal cortex [39, 40]. This period (ages 10–12) corresponds with fronto-striatal reorganization, where

improved response inhibition may paradoxically reveal sustained attention inefficiencies [41, 42]. The emergence of "repeats certain acts over and over" as an additional bridge symptom reflects striatal habit system maturation, suggesting compulsive behaviors become more cognitively mediated during this window [43]. These patterns highlight neurodevelopmental vulnerabilities that could be targeted with executive function training before cognitive bridge symptoms consolidate [44].

The persistent role of "can't get their mind off certain thoughts" as a stable bridge symptom across all assessments suggests it represents a fundamental mechanism in ADHD-OCD symptom overlap. This stability aligns with neurobiological evidence indicating shared neural substrates in fronto-striatal circuits, particularly involving the anterior cingulate cortex and orbitofrontal-striatal regions that regulate both intrusive thoughts and attentional control [45]. Functional connectivity studies have shown that intrusive thoughts in adolescent with co-occurring ADHD-OCD symptoms are associated with altered default mode network salience, which may simultaneously disrupt attention regulation and promote repetitive thinking [46]. The observed strengthening of this bridge connection over time, especially in the subclinical group, supports cognitive models proposing that intrusive thoughts may serve as a common mechanism through which executive dysfunction manifests across both symptom domains [8]. This persistent transdiagnostic role highlights the importance of targeting intrusive thoughts early in intervention, potentially through cognitive strategies that address thought action fusion and metacognitive beliefs [47].

The gender differentiated bridge patterns in the subclinical group provide evidence for distinct phenotypic expressions. These gender specific symptom connection patterns offer new directions for personalized interventions. The male predominant OCD focused connection pattern suggests an internalizing expression of symptom overlap, characterized by cognitive rituals and behavioral compulsions that may reflect particular difficulties with cognitive control and habit formation [20, 40]. Conversely, the female dominant ADHD related connection pattern indicates an externalizing expression, primarily manifesting through impulse control deficits and

verbal impulsivity that may involve distinct reward processing mechanisms [3, 12]. The persistence of these gender differences despite overall structural convergence reveals stable phenotypic variations maintained across development [48].

This study examined ADHD and OCD symptom networks across four annual assessments, revealing stable core symptoms (attention in ADHD, behavioral symptoms in OCD) alongside a shift from behavioral impulsivity to cognitive difficulties as bridge symptoms in the subclinical group, as well as gender specific symptom connection patterns. However, several limitations should be considered. First, the four year observation period captures only a portion of developmental trajectories, leaving patterns beyond early adolescence unexplored. Second, the absence of neurobiological measures limits our understanding of the neural mechanisms underlying the identified symptom networks. Future research should extend the observation period into later developmental stages and incorporate neuroimaging techniques to elucidate the biological substrates of these network characteristics.

Conclusion:

This study examined ADHD and OCD symptom networks across four annual assessments in adolescent. Results showed stable core symptoms (attention difficulties in ADHD, behavioral symptoms in OCD), dynamic bridge symptoms (shifting from impulsivity to concentration difficulties), early adolescence as a critical period, and gender specific patterns (males: OCD focused connections; females: ADHD related bridges). These findings demonstrate network analysis as a valuable tool for understanding symptom interactions and informing targeted early interventions.

Declarations:

Ethics approval and consent to participate

The anonymized data used in the preparation of this article (secondary data analysis) were obtained from the Adolescent Brain Cognitive Development℠ (ABCD) Study (DOI: 10.15154/1523041), held in the NIMH Data Archive (NDA). The ABCD Study was conducted in accordance with the Declaration of Helsinki and was reviewed and approved by a central Institutional Review Board at the University of California, San Diego (UCSD IRB; protocol number: 160091), which acts as the single IRB for all ABCD Study sites. In addition, all participating sites obtained any necessary local or regional ethical approvals. Written informed consent was obtained from the parents or legal guardians of all participating children and adolescents, and written assent was obtained from the participating children themselves.

Consent for publication

Not Applicable

Availability of data and materials

The anonymized data analyzed in this study were obtained from the Adolescent Brain Cognitive Development℠ (ABCD) Study (DOI: 10.15154/1523041), held in the NIMH Data Archive (NDA). The ABCD Study data are publicly available and can be accessed upon request and approval by the NDA at https://nda.nih.gov/abcd. Further information on how to access ABCD data through the NDA is available on the ABCD Study data-sharing webpage: https://abcdstudy.org/scientists_data_sharing.html

Competing interests

The authors declare that they have no competing interests.

Funding

This work was supported by the Science and Technology Innovation Development Project of Beijing Children's Hospital, Capital Medical University, National Center for Children's Health (Grant No. YN2020409 to Ying Li) and the National Natural Science Foundation of China (Grant No. 82301731 to Dr. Hui Xu).

Authors' contributions

Lianjingyi Liang wrote the manuscript, revised the format, and performed data curation; Wanling Zhang was responsible for conceptualization, formal analysis, visualization, and writing – review & editing; Hui Xu contributed to data curation and formal analysis; Yanyu Wang and Ying Li supervised the project, handled project administration and validation, and performed writing – review & editing. All authors read and approved the final manuscript.

Acknowledgements

The authors thank the Adolescent Brain Cognitive Development℠ (ABCD) Study participants, their families, and all investigators and staff for making the de-identified data publicly available. The authors also acknowledge the National Institute of Mental Health Data Archive (NDA) for providing data access services.

References:

1. Association AP. American psychiatric association. 2019.
2. Goodman SJ, Burton CL, Butcher DT, Siu MT, Lemire M, Chater-Diehl E *et al*. Obsessive-compulsive disorder and attention-deficit/hyperactivity disorder: distinct associations with DNA methylation and genetic variation. Journal of neurodevelopmental disorders. 2020;12(1-15).
3. Abramovitch A, Dar R, Mittelman A, Wilhelm S. Comorbidity between attention deficit/hyperactivity disorder and obsessive-compulsive disorder across the lifespan: a systematic and critical review. Harvard review of psychiatry. 2015;23(4):245-262.
4. Njardvik U, Wergeland GJ, Riise EN, Hannesdottir DK, Öst L-G. Psychiatric comorbidity in children and adolescents with ADHD: A systematic review and meta-analysis. Clinical Psychology Review. 2025;102571.
5. Çelebi F, Koyuncu A, Ertekin E, Alyanak B, Tükel R. The features of comorbidity of childhood ADHD in patients with obsessive compulsive disorder. Journal of Attention Disorders. 2020;24(7):973-980.
6. Fineberg NA, Hollander E, Pallanti S, Walitza S, Grünblatt E, Dell'Osso BM *et al*. Clinical advances in obsessive-compulsive disorder: a position statement by the International College of Obsessive-Compulsive Spectrum Disorders. International clinical psychopharmacology. 2020;35(4):173-193.
7. Mersin Kilic S, Dondu A, Memis CO, Ozdemiroglu F, Sevincok L. The clinical characteristics of ADHD and obsessive-compulsive disorder comorbidity. Journal of attention disorders. 2020;24(12):1757-1763.
8. Miyauchi M, Matsuura N, Mukai K, Hashimoto T, Ogino S, Yamanishi K *et al*. A prospective investigation of impacts of comorbid attention deficit hyperactivity disorder (ADHD) on clinical features and long-term treatment response in adult patients with obsessive-compulsive disorder (OCD). Comprehensive Psychiatry. 2023;125(152401).
9. Sheppard B, Chavira D, Azzam A, Grados MA, Umaña P, Garrido H *et al*. ADHD prevalence and association with hoarding behaviors in childhood-onset OCD. Depression and anxiety. 2010;27(7):667-674.
10. Karakoç S, Avcil S, Aksu H, Gürbüz Özgür B, Güleş Z, Öztürk S. Comparison of executive functions in attention deficit hyperactivity disorder, obsessive compulsive disorder and pathological internet use in children and adolescents. TURKISH JOURNAL CLINICAL PSYCHIATRY. 2023;26(2):104-112.
11. De Mathis MA, Diniz JB, Hounie AG, Shavitt RG, Fossaluza V, Ferrao Y *et al*. Trajectory in obsessive-compulsive disorder comorbidities. European Neuropsychopharmacology. 2013;23(7):594-601.
12. Yang Z, Xiao S, Su T, Gong J, Qi Z, Chen G *et al*. A multimodal meta-analysis

of regional functional and structural brain abnormalities in obsessive–compulsive disorder. European Archives of Psychiatry and Clinical Neuroscience. 2024;274(1):165-180.

13. Norman LJ, Carlisi CO, Christakou A, Murphy CM, Chantiluke K, Giampietro V *et al*. Frontostriatal dysfunction during decision making in attention-deficit/hyperactivity disorder and obsessive-compulsive disorder. Biological Psychiatry: Cognitive Neuroscience and Neuroimaging. 2018;3(8):694-703.
14. Sharma E, Sharma LP, Balachander S, Lin B, Manohar H, Khanna P *et al*. Comorbidities in obsessive-compulsive disorder across the lifespan: a systematic review and meta-analysis. Frontiers in psychiatry. 2021;12(703701).
15. Kao P-H, Ho C-H, Huang CL-C. Sex differences in psychiatric comorbidities of attention-deficit/hyperactivity disorder among children, adolescents, and adults: A nationwide population-based cohort study. Plos one. 2025;20(1):e0315587.
16. Sun H-L, He F, Rao W-W, Qi Y, Rao S-Y, Ho TI *et al*. Gender differences in behavioral and emotional problems among school children and adolescents in China: National survey findings from a comparative network perspective. Journal of Affective Disorders. 2025;369(227-233.
17. Jacobs GKR: Investigating heterogeneity and overlapping clinical and neurobiological features across early psychosis and neurodevelopmental disorders in children and adolescents. University of Toronto (Canada); 2022.
18. Jones PJ, Ma R, McNally RJ. Bridge centrality: a network approach to understanding comorbidity. Multivariate behavioral research. 2021;56(2):353-367.
19. Peterson BS, Pine DS, Cohen P, Brook JS. Prospective, longitudinal study of tic, obsessive-compulsive, and attention-deficit/hyperactivity disorders in an epidemiological sample. In: editor. Obsessive-Compulsive Disorder and Tourette's Syndrome. Routledge; 2022. p. 107-117.
20. Poletti M, Gebhardt E, Pelizza L, Preti A, Raballo A. Neurodevelopmental antecedents and sensory phenomena in obsessive compulsive disorder: a systematic review supporting a phenomenological-developmental model. Psychopathology. 2023;56(4):295-305.
21. Solberg BS, Halmøy A, Engeland A, Igland J, Haavik J, Klungsøyr K. Gender differences in psychiatric comorbidity: a population-based study of 40 000 adults with attention deficit hyperactivity disorder. Acta Psychiatrica Scandinavica. 2018;137(3):176-186.
22. Robinaugh DJ, Hoekstra RH, Toner ER, Borsboom D. The network approach to psychopathology: A review of the literature 2008–2018 and an agenda for future research. Psychological medicine. 2020;50(3):353-366.
23. Liu J, Cao L, Li H, Gao Y, Bu X, Liang K *et al*. Abnormal resting-state functional connectivity in patients with obsessive-compulsive disorder: A systematic review and meta-analysis. Neuroscience & Biobehavioral Reviews. 2022;135(104574).
24. Anderson D, Lord C, Risi S, DiLavore P, Shulman C, Thurm A *et al*.

American Psychiatric Association.(2013). Diagnostic and statistical manual of mental disorders . Washington, DC: Author. The Linguistic and Cognitive Effects of Bilingualism on Children with Autism Spectrum Disorders. 2017;21(175).

25. Volkow ND, Koob GF, Croyle RT, Bianchi DW, Gordon JA, Koroshetz WJ *et al*. The conception of the ABCD study: From substance use to a broad NIH collaboration. Developmental cognitive neuroscience. 2018;32(4-7).
26. Barch DM, Albaugh MD, Avenevoli S, Chang L, Clark DB, Glantz MD *et al*. Demographic, physical and mental health assessments in the adolescent brain and cognitive development study: Rationale and description. Developmental cognitive neuroscience. 2018;32(55-66).
27. Pagliaccio D, Durham K, Fitzgerald KD, Marsh R. Obsessive-compulsive symptoms among children in the adolescent brain and cognitive development study: clinical, cognitive, and brain connectivity correlates. Biological Psychiatry: Cognitive Neuroscience and Neuroimaging. 2021;6(4):399-409.
28. Efe A, Kaba D, Canlı M, Temeltuerk RD. Impact of attention-deficit/hyperactivity disorder comorbidity on phenomenology and treatment outcomes of pediatric obsessive-compulsive disorder. Journal of Child and Adolescent Psychopharmacology. 2022;32(6):337-348.
29. Benatti B, Girone N, Celebre L, Vismara M, Hollander E, Fineberg NA *et al*. The role of gender in a large international OCD sample: A Report from the International College of Obsessive-Compulsive Spectrum Disorders (ICOCS) Network. Comprehensive psychiatry. 2022;116(152315).
30. Jiang Z, Wang D, Xu H, Zhang A, Zhao Q, Yan J *et al*. Diagnostic efficiency and psychometric properties of CBCL DSM-oriented scales in a large sample of Chinese school-attending students aged 5–16. Asian Journal of Psychiatry. 2023;88(103724).
31. Li F, Cui Y, Li Y, Guo L, Ke X, Liu J *et al*. Prevalence of mental disorders in school children and adolescents in China: diagnostic data from detailed clinical assessments of 17,524 individuals. Journal of Child Psychology and Psychiatry. 2022;63(1):34-46.
32. Faraone SV, Asherson P, Banaschewski T, Biederman J, Buitelaar JK, Ramos-Quiroga JA *et al*. Attention-deficit/hyperactivity disorder. Nature reviews Disease primers. 2015;1(15020-15020).
33. Fayyad J, Sampson NA, Hwang I, Adamowski T, Aguilar-Gaxiola S, Al-Hamzawi A *et al*. The descriptive epidemiology of DSM-IV adult ADHD in the world health organization world mental health surveys. ADHD Attention Deficit and Hyperactivity Disorders. 2017;9(47-65).
34. Smárason O, Weidle B, Höjgaard DR, Torp NC, Ivarsson T, Nissen JB *et al*. Age differences in children with obsessive-compulsive disorder: symptoms, comorbidity, severity and impairment. Nordic Journal of Psychiatry. 2021;75(sup1):S11-S11.
35. Norman LJ, Taylor SF, Liu Y, Radua J, Chye Y, De Wit SJ *et al*. Error processing and inhibitory control in obsessive-compulsive disorder: a meta-

analysis using statistical parametric maps. Biological Psychiatry. 2019;85(9):713-725.
36. Carta A, Fucà E, Guerrera S, Napoli E, Valeri G, Vicari S. Characterization of clinical manifestations in the co-occurring phenotype of attention deficit/hyperactivity disorder and autism spectrum disorder. Frontiers in Psychology. 2020;11(861).
37. Mathes BM, Morabito DM, Schmidt NB. Epidemiological and clinical gender differences in OCD. Current psychiatry reports. 2019;21(1-7).
38. Tremblay S, Willcutt EG. Implications of Co-occurrence Between ADHD and Anxiety in a Community-based Child Sample. Journal of Attention Disorders. 2025;10870547251324806.
39. Zelazo PD. Executive function and psychopathology: A neurodevelopmental perspective. Annual review of clinical psychology. 2020;16(1):431-454.
40. Smárason O, Selles RR, Højgaard DR, Best JR, Melin K, Ivarsson T *et al*. Exploring latent clusters in pediatric OCD based on symptoms, severity, age, gender, and comorbidity. European Child & Adolescent Psychiatry. 2024;1-14.
41. Breda V, Rohde LA, Menezes AMB, Anselmi L, Caye A, Rovaris DL *et al*. The neurodevelopmental nature of attention-deficit hyperactivity disorder in adults. The British Journal of Psychiatry. 2021;218(1):43-50.
42. Garaigordobil M. Educational psychology: the key to prevention and child-adolescent mental health. Psicothema. 2023;35(4):327-339.
43. Gillan CM. Recent developments in the habit hypothesis of OCD and compulsive disorders. The neurobiology and treatment of OCD: Accelerating progress. 2021;147-167.
44. Marklin M, Killion B, O'Connor E, Renschler K, Freeman JB. Comorbid OCD and ADHD in youth: Clinical implications and treatment recommendations. The Brown University Child and Adolescent Behavior Letter. 2023;39(11):1-4.
45. Tamon H, Fujino J, Itahashi T, Frahm L, Parlatini V, Aoki YY *et al*. Shared and specific neural correlates of attention deficit hyperactivity disorder and autism spectrum disorder: a meta-analysis of 243 task-based functional MRI studies. American Journal of Psychiatry. 2024;181(6):541-552.
46. Harikumar A, Evans DW, Dougherty CC, Carpenter KL, Michael AM. A review of the default mode network in autism spectrum disorders and attention deficit hyperactivity disorder. Brain connectivity. 2021;11(4):253-263.
47. Newman MG, Jacobson NC, Erickson TM, Fisher AJ. Interpersonal problems predict differential response to cognitive versus behavioral treatment in a randomized controlled trial. Behavior Therapy. 2017;48(1):56-68.
48. Melis İ, Izmir SBI, Türkçapar MH, Özdel K, Ardiç ÜA, Ercan ES. Psychiatric Comorbidity in the Subtypes of ADHD in Children and Adolescents with ADHD According to DSM-IV. Archives of Neuropsychiatry. 2020;57(4):283.

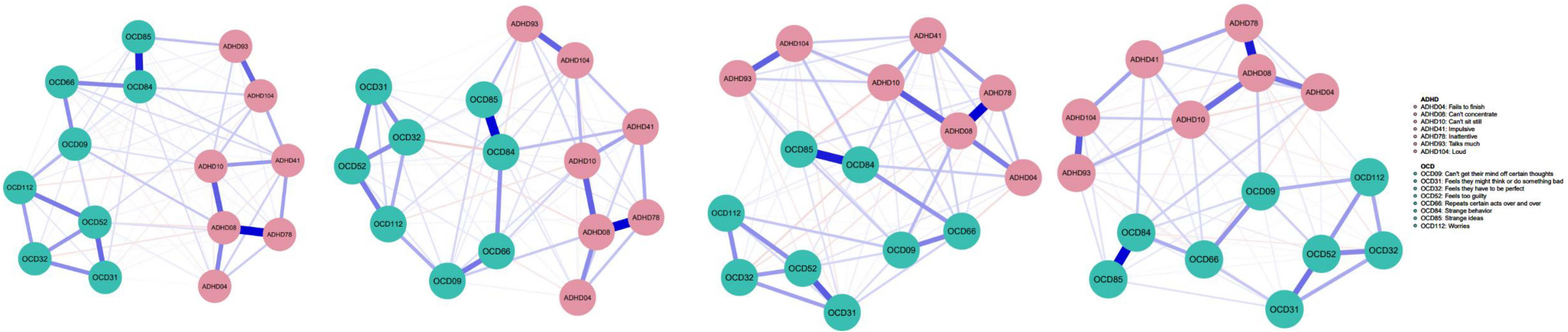
Table 1 The items of ADHD and OCD in the CBCL.

Item	Content
ADHD04	Fails to finish
ADHD08	Can't concentrate
ADHD10	Can't sit still
ADHD41	Impulsive
ADHD78	Inattentive
ADHD93	Talks much
ADHD104	Loud
OCD09	Can't get their mind off certain thoughts/obsessions
OCD31	Feels they might think or do something bad
OCD32	Feels they have to be perfect
OCD52	Feels too guilty
OCD66	Repeats certain acts over and over ; compulsions
OCD84	Strange behavior
OCD85	Strange ideas
OCD112	Worries

Table 2 The demographic and behavioral characteristics of the full sample and the OCD subclinical sample with comorbid ADHD.

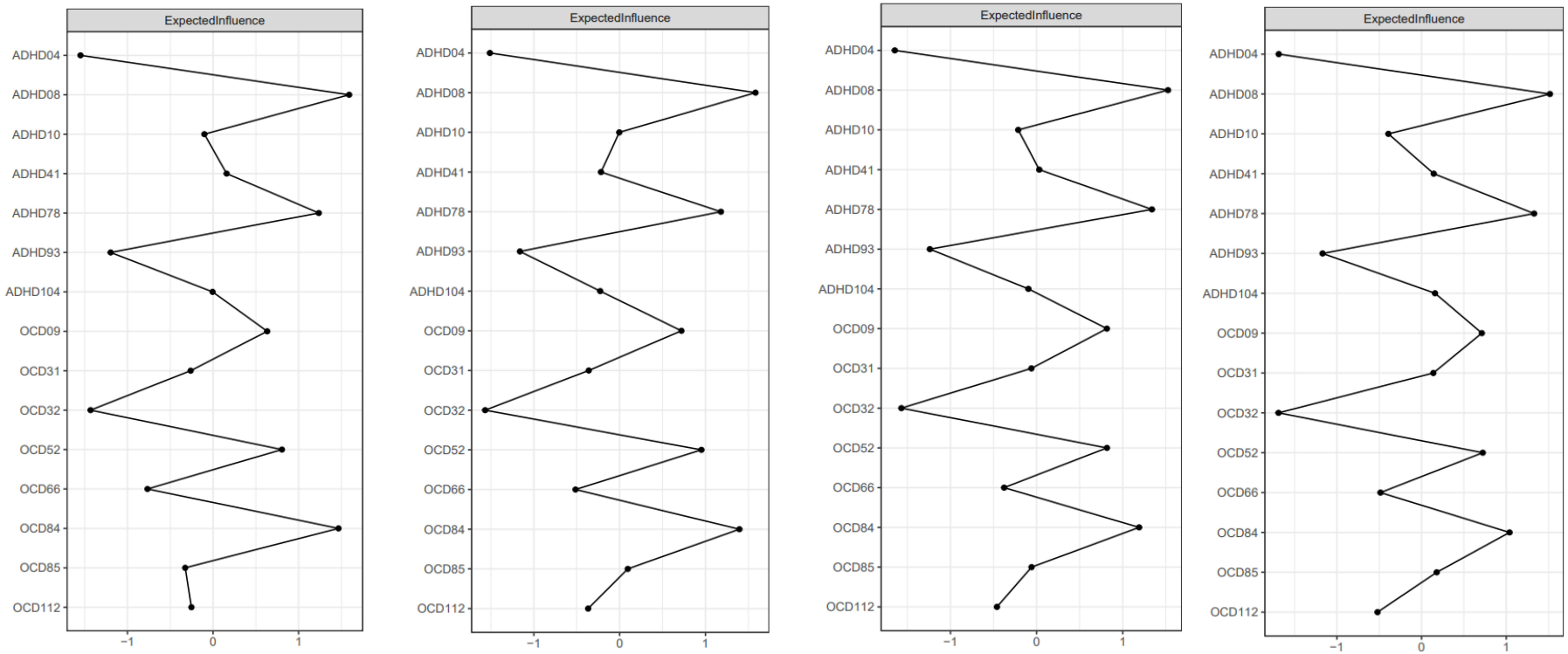
Time point	Full sample			Subclinical sample		
	Total	Male	Female	Total	Male	Female
T0	11807	6163(52.2%)	5644	616	390(63.3%)	226
T1	11182	5851((52.3%)	5331	552	338(61.2%)	214
T2	10879	5711(52.4%)	5168	444	269(60.5%)	175
T3	10079	5307(52.6%)	4772	463	26056.2%)	203

Note. T0: indicates the baseline, T1: indicates the 1-year follow-up, T2: indicates the 2-year follow-up, T3: indicates the 3-year follow-up. OCD: Obsessive-compulsive disorder. ADHD: Attention Deficit Hyperactivity Disorder. All the expressions in the following tables are consistent.



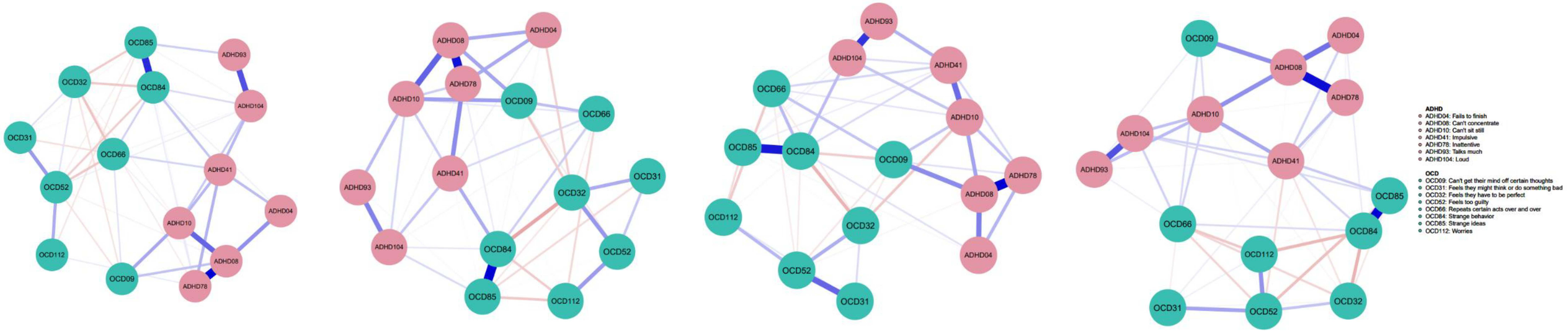
a. T0 **b. T1** **c. T2** **d. T3**

Figure 1. Network constructed via the graphical LASSO depicting regularized partial correlations between pairs of OCD and ADHD in the full sample.



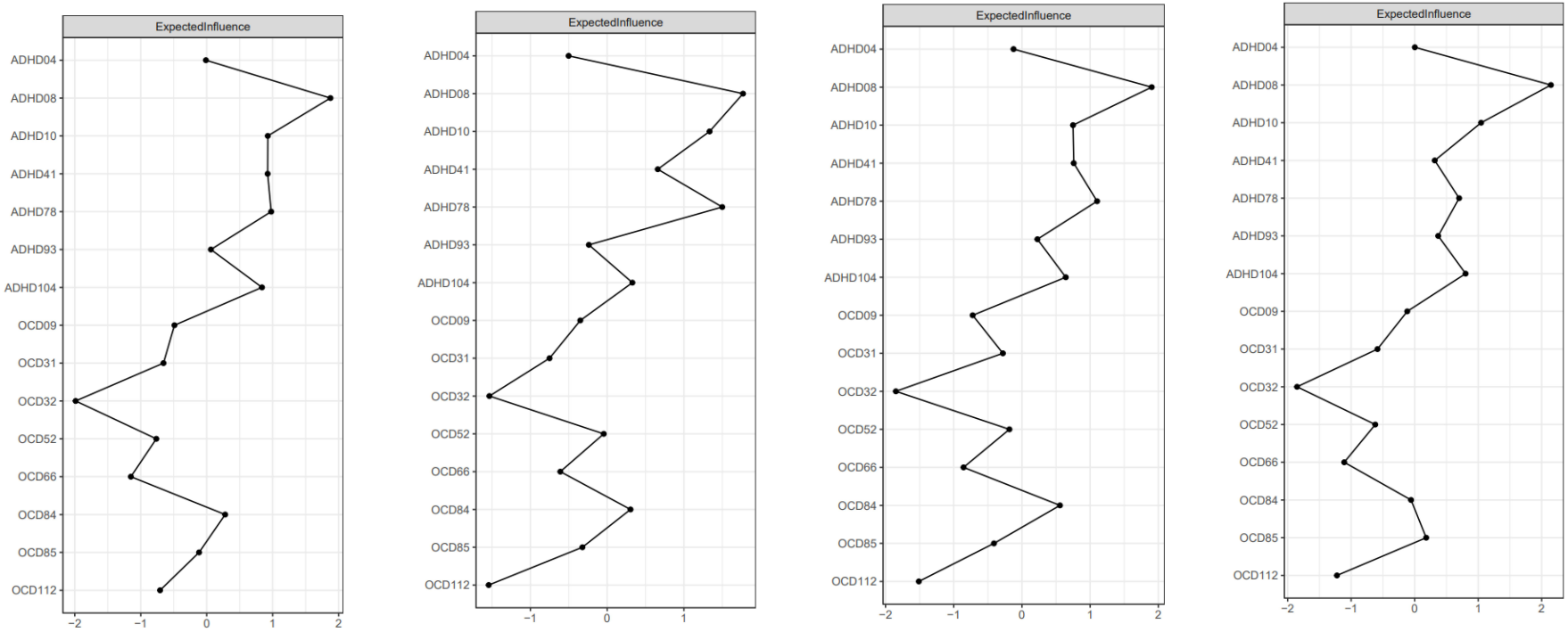
a. T0 **b. T1** **c. T2** **d. T3**

Figure 2. Centrality for the network analysis in the full sample.



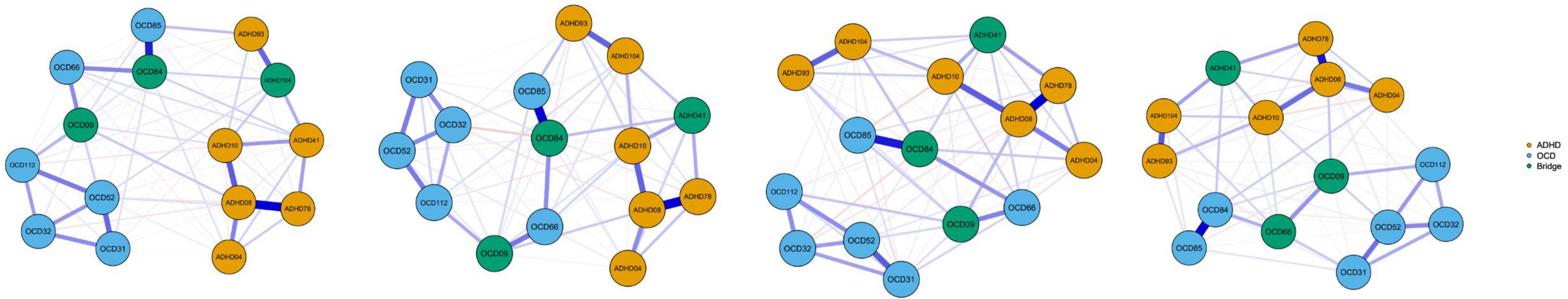
a. T0 **b. T1** **c. T2** **d. T3**

Figure 3. Network constructed via the graphical LASSO depicting regularized partial correlations between pairs of OCD and ADHD in the subclinical sample.



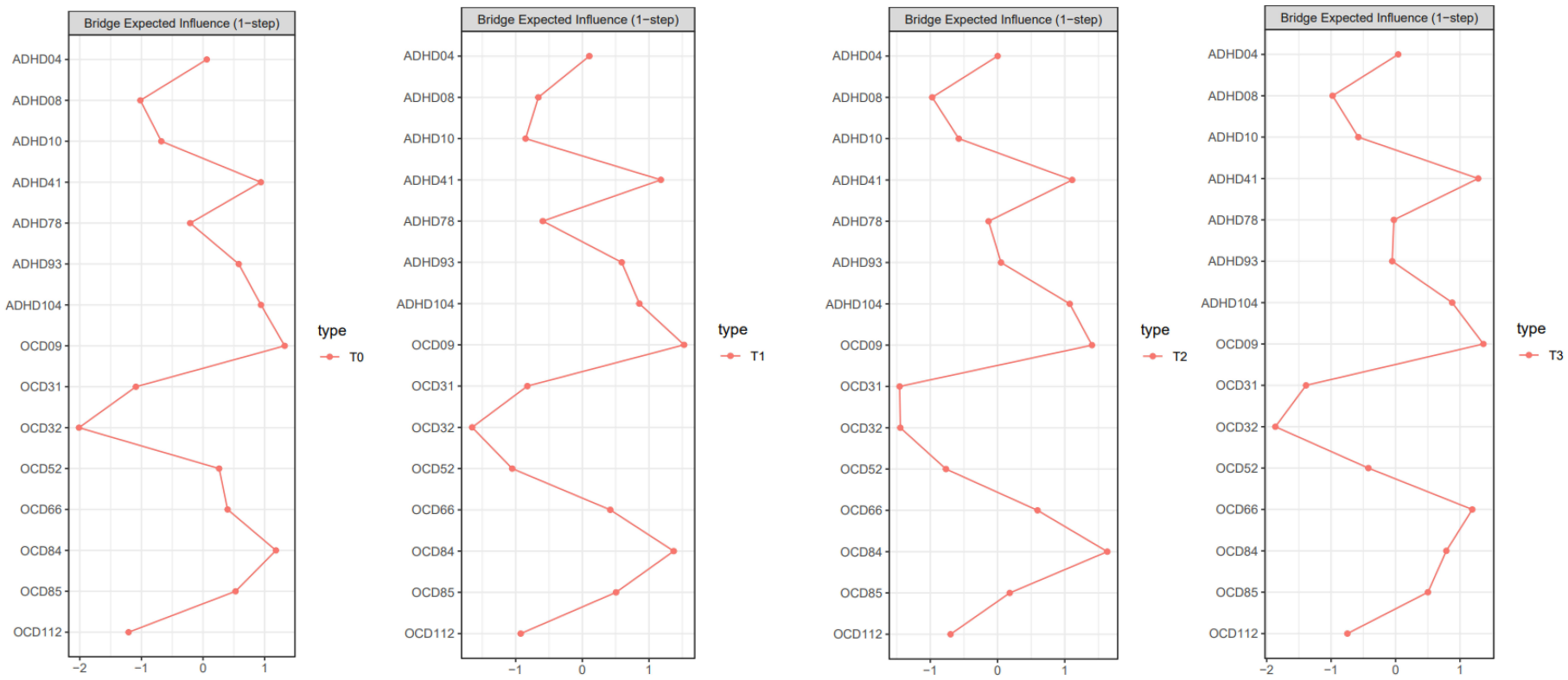
a. T0 **b. T1** **c. T2** **d. T3**

Figure 4. Centrality for the network analysis in the subclinical sample.



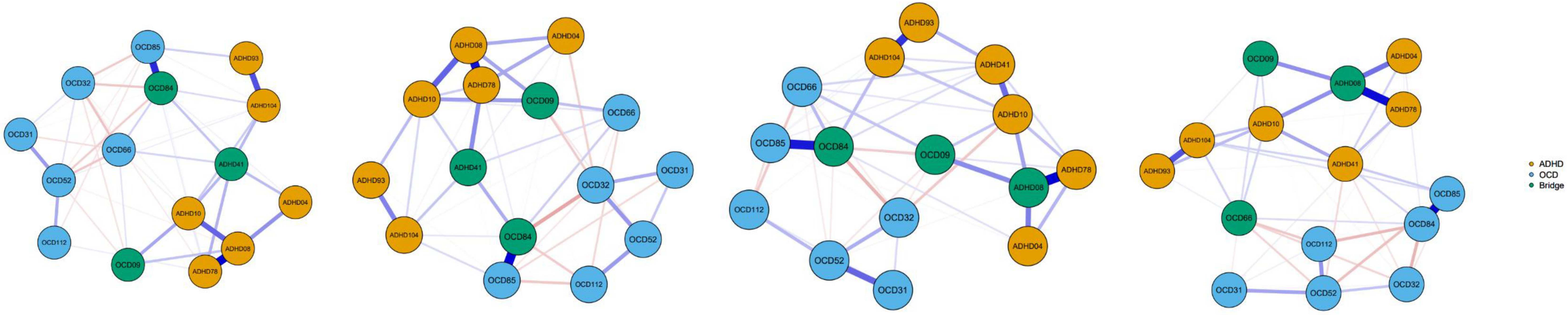
a. T0 **b. T1** **c. T2** **d. T3**

Figure 5. Bridge Network constructed of OCD and ADHD in the full sample.



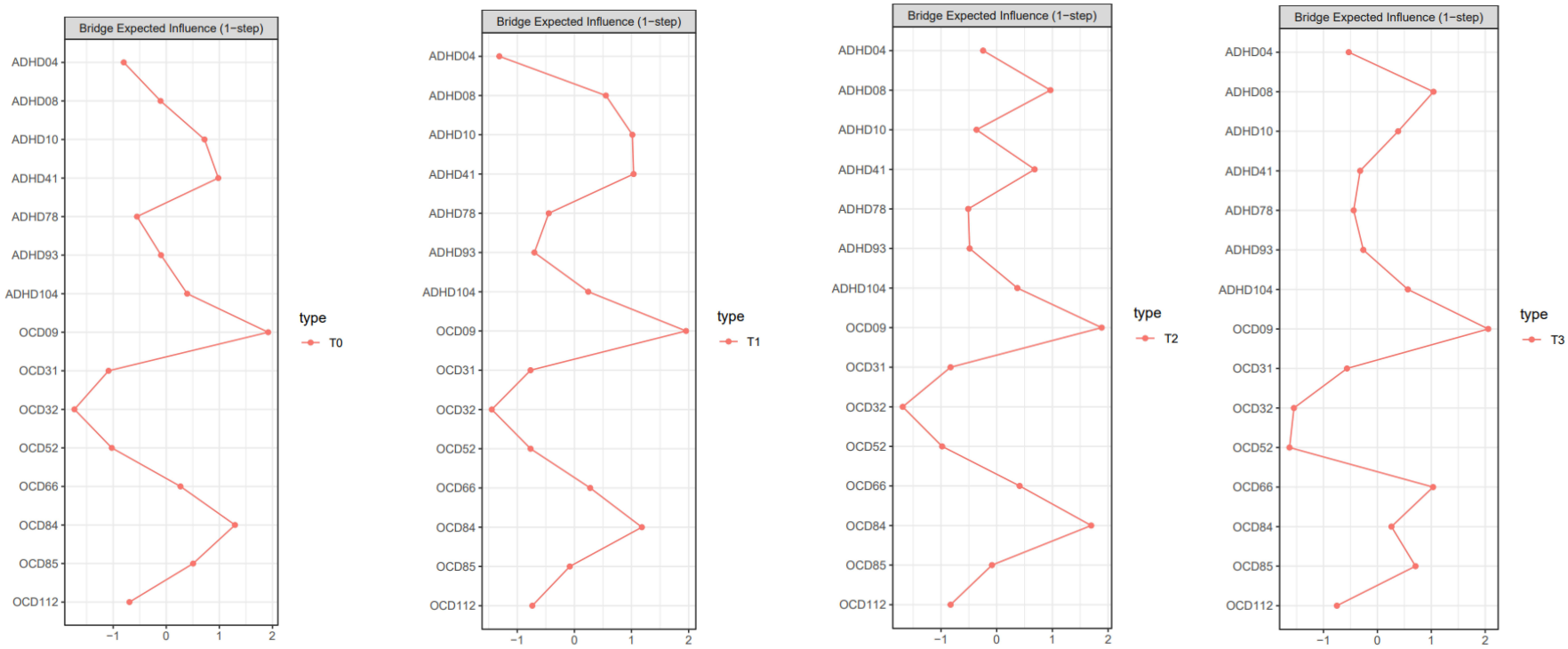
a. T0 b. T1 c. T2 d. T3

Figure 6. Bridge centrality for the network analysis in the full sample.



a. T0 **b. T1** **c. T2** **d. T3**

Figure 7. Bridge Network constructed of OCD and ADHD in the subclinical.



a. T0 **b. T1** **c. T2** **d. T3**

Figure 8. Bridge centrality for the network analysis in the subclinical sample.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- FigureS1.docx
- FigureS2.docx
- FigureS3.docx
- FigureS4.docx
- FigureS5.docx
- FigureS6F0.pdf
- FigureS6F1.pdf
- FigureS6F2.pdf
- FigureS6F3.pdf
- FigureS6Sub0.pdf
- FigureS6Sub1.pdf
- FigureS6Sub2.pdf
- FigureS6Sub3.pdf
- supplementarymaterialslist.docx
- tableS1.docx