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ADHD and autism symptoms in youth: A network analysis.

Abbreviated title: ADHD and autism symptoms: a network analysis

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ABSTRACT

Background: Previous research investigating the overlap between attention-deficit/hyperactivity disorder (ADHD) and autism spectrum disorder (henceforth, autism) symptoms in population samples have relied on latent variable modelling in which averaged scores representing dimensions were derived from observed symptoms. There are no studies evaluating how ADHD and autism symptoms interact at the level of individual symptom items.

Methods: We aimed to address this gap by performing a network analysis on data from a school survey of children aged 6-17 years old (N = 7,405). ADHD and autism symptoms were measured via parent-report on the Swanson, Nolan, Pelham-IV questionnaire and the Childhood Autism Spectrum test, respectively.

Results: A relatively low interconnectivity between ADHD and autism symptoms was found with only 10.06% of possible connections (edges) between one ADHD and one autism symptoms different than zero. Associations between ADHD and autism symptoms were significantly weaker than those between two symptoms pertaining to the same construct. Select ADHD symptoms, particularly those presenting in social contexts (e.g. ‘talks excessively’, ‘frequently blurts out answers’, ‘does not wait turn’), showed moderate-to-strong associations with autism symptoms, but some were considered redundant to autism symptoms.

Conclusion: The present findings indicate that individual ADHD and autism symptoms are largely segregated in accordance with diagnostic boundaries corresponding to these conditions in children and adolescents from the community. These findings could improve our clinical conceptualization of ADHD and autism and guide advancements in diagnosis and treatment.

Keywords: attention-deficit hyperactivity disorder (ADHD), autism spectrum disorder (ASD), network analysis, comorbidity, neurodevelopmental

Attention-deficit/hyperactivity disorder (ADHD) and autism spectrum disorder (henceforth, autism) are two neurodevelopmental disorders that emerge early in childhood. ADHD is defined by a persistent pattern of inattention and/or hyperactivity-impulsivity (Posner, Polanczyk, & Sonuga-Barke, 2020) whereas autism is characterized by social communication and interaction deficits and restricted, repetitive patterns of behaviors, interests or activities (Lord et al., 2020). Both conditions are relatively prevalent in children and adolescents, with estimates of 5.3% for ADHD (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007) and 1.85% for autism (Maenner et al., 2020). Both conditions also tend to co-occur; the prevalence rates for ADHD in the context of autism (~22%) (Lai et al., 2019) and autism in the context of ADHD (~21%) (Hollingdale, Woodhouse, Young, Fridman, & Mandy, 2019) are significantly higher than those reported for children in the general population.

Given the co-occurrence of ADHD and autism, clinicians may experience difficulties distinguishing the symptoms of these two conditions when evaluating children in clinical practice (Grzadzinski, Dick, Lord, & Bishop, 2016; Yerys et al., 2017). For instance, children with autism may ‘often seem not to listen when spoken to directly’ because they are focused on their perserverative interests; they may ‘often talk excessively’ or ‘interrupt or intrude on others’ due to unilateral social-communication skills. Likewise, children with ADHD may also present with autism symptoms such as social difficulties and language impairments. Thus, children with autism may intially be misdiagnosed with ADHD (Perry, 1998) and vice versa (Grzadzinski et al., 2016). In this scenario, research aimed at clarifying how ADHD and autism symptoms relate to each other in children and adolescents could be of value.

Previous studies (Pinto, Rijsdijk, Ronald, Asherson, & Kuntsi, 2016; Ronald, Larsson, Anckarsater, & Lichtenstein, 2014; Ronald, Simonoff, Kuntsi, Asherson, & Plomin, 2008; Taylor, Charman, & Ronald, 2015) have relied on latent variables to investigate the overlap between ADHD and autism symptoms in population samples. In contrast to latent variable analysis, network analysis methods enable the visualization of relationships between individual symptom items, without the need to derive an average score of symptom dimensions (Borsboom & Cramer, 2013; Cramer, Waldorp, van der Maas, & Borsboom, 2010). In network analysis, symptoms constitute *nodes* and the coefficients obtained from multiple regression models in which one symptom is regressed on all others simultaneously constitute the *edges* connected to that symptom. Thus, network analysis can be employed to evaluate the interdependencies between individual ADHD and autism symptoms, providing a novel perspective to the current knowledge on the co-occurrence of these symptoms in children and adolescents.

Network analysis methods have been employed to investigate relationships between core autism symptoms (Anderson, Montazeri, & de Bildt, 2015) as well as between core autism symptoms and depression (Montazeri, de Bildt, Dekker, & Anderson, 2019b), anxiety (Montazeri, de Bildt, Dekker, & Anderson, 2019a) and obsessive-compulsive disorder (Ruzzano, Borsboom, & Geurts, 2015) symptomatology. To the best of our knowledge, no previous study has employed network analysis to characterize the relationships between individual symptoms of ADHD and autism. To address this issue, we aimed to investigate the network structure of inattentive and hyperactive-impulsive ADHD symptoms as well as social-communication and inflexible/stereotyped language

and behavior symptoms of autism as distributed in a population sample of children and adolescents from Brazil.

METHODS

Participants and survey procedures

The participants for this study were children aged between 6 and 17 years who were enrolled in public schools in the city of *São Caetano do Sul* in 2014. *São Caetano do Sul* is located in the metropolitan area of *São Paulo*, the largest city in Brazil. *São Caetano do Sul* has a Human Development Index of 0.862, ranking first among the cities in Brazil, and an estimated population of 149,263 inhabitants according to the national census from 2010. Protocols consisting of consent forms, information leaflets and questionnaires were sent to parents of every child aged between 6 and 17 years enrolled in public schools of *São Caetano do Sul*. Overall, 10,525 questionnaires were sent to parents. Of those, 7,723 who had signed the consent form were considered eligible for inclusion in the study. Protocols were fully anonymized to protect the identity of participants and the project was approved by the secretary of education of *São Caetano do Sul* and by the ethics committee of the *Faculdade de Medicina da Universidade de São Paulo* (project # 1.900.291).

Measures

The school survey was completed by parents regarding their child's behavior; questions included in the survey asked about sociodemographic characteristics (sex, date of birth, ethnicity, religious affiliation, maternal and paternal education level and employment), medical history (height, weight, if

ever received treatment for a behavioral problem), ADHD symptoms (Swanson, Nolan and Pelham – IV questionnaire, SNAP), autism symptoms (Childhood Autism Spectrum Test, CAST), anxiety symptoms (modified version of the Multidimensional Anxiety Scale for Children, MASC) and general psychopathology (Children Behavior Checklist, CBCL). The CBCL assesses 120 emotional, behavioral and social problems reported by parents of children aged 6 to 18 years old and comprises an 8-syndrome structure which reflects different dimensions of internalizing and externalizing symptoms (Ivanova et al., 2007).

Swanson, Nolan and Pelham - IV questionnaire (SNAP-IV) (Bussing et al., 2008) - the SNAP-IV is an ADHD rating scale that includes 18 items pertaining to two symptom domains: inattention (9 items) and hyperactivity/impulsivity (9 items). The parent-report version of the SNAP-IV was employed in this study; parents rated their children's behavior on a 4-point Likert scale: 0 ("Never or rarely"), 1 ("Sometimes"), 2 ("Often") and 3 ("Very often"). Higher scores reflect greater ADHD symptoms.

Childhood Autism Spectrum Test (CAST) (Scott, Baron-Cohen, Bolton, & Brayne, 2002) **–** the CAST is a screening instrument measuring autistic traits and is composed of 39 items, 31 of which cover autistic symptoms pertaining to two symptom dimensions: social-communication and inflexible/stereotyped language and behaviors. Items were completed by parents in a dichotomized manner (yes/no scores); for some items, e.g. 'does s/he join in playing games

with other children easily?' (CAST 1), endorsing 'no' represents the individual scored 1 to indicate the presence of an autistic symptom.

Data analysis

Statistical analyses were performed using the computing environment R (R Core Team, 2019). The list of R-packages employed as well as the full R script with the code for the analyses of this study are available in the supplementary information (Appendix S1). The network was based on 49 symptoms, 31 symptoms of autism and 18 of ADHD. The description of each ADHD and autism item is presented in Table S1 (Appendix S2) as well as the number of individuals who endorsed each one of the items.

Missing data – To be included in our study, participants had to provide answers to at least 80% of the ADHD and autism items; therefore, participants with less than 15 answers on the SNAP or less than 25 answers on the CAST were excluded from the study. To avoid listwise deletion of individuals who were eligible for inclusion but who had missing data on up to 3 ADHD symptoms or 6 autism symptoms, we adopted an expectation-maximization with bootstrapping algorithm to perform multiple imputation (Honaker, King, & Blackwell, 2011).

Network estimation – we investigated the network structure of ADHD and autism symptoms by employing *eLasso* procedures to estimate undirected networks from binary data (van Borkulo et al., 2014). Answers from the SNAP, which had been given on a 4-point Likert scale, were recoded to indicate the presence (raw scores of 2 or 3) or absence (raw scores of 0, 1) of each ADHD

symptom. *eLasso* uses regression to compute the degree of association between each item score within and between ADHD and autism symptoms while partialling out the association between each symptom and all other symptoms, resulting in a (31x18) matrix of regression coefficients. Matrix cells with very small regression coefficients, indicating almost no association between the symptoms, are set to zero through L1-penalty to increase the specificity of the edges (connections) identified in the network. This method ensures that the edge weight between a pair of nodes (symptom items) is not an artifact of other interactions from those nodes.

Relations between constructs – To investigate whether symptoms from the same construct (ADHD or autism) were more closely associated with each other than with symptoms from the other construct, we analyzed the interconnectivity of the network by comparing the difference in edge weights through non-parametric Wilcoxon test or Kruskal-Wallis test for two and three groups, respectively. When the Kruskal-Wallis test was significant, a multiple pairwise-comparison Wilcoxon test was subsequently used to determine which pairs of groups were significantly different from one another while accounting for the multiple comparisons.

Node centrality – to identify which nodes were central in the ADHD/autism symptom network, we estimated the expected influence (EI) of each node, i.e. the sum of all positive edges subtracted by the sum of all negative edges. We also computed the bridge EI to identify *bridge symptoms*, i.e. symptoms from one construct that were more strongly associated with symptoms of the other

construct (Jones, Ma, & McNally, 2019). We also evaluated whether there existed pairs of ADHD and autism symptoms which could be considered 'redundant', i.e. a pair of ADHD and autism symptoms establishing similar connections with other nodes in the network. We employed the goldbricker function to calculate the proportion of correlations that were significantly different for pairs of symptoms (Jones, 2017); if less than 95% of the correlations were considered significantly different, the pair of symptoms was considered redundant.

Network stability and accuracy – Accuracy and stability of the ADHD/autism symptom network was evaluated according to the procedures described by Epskamp, Borsboom, and Fried (2018). Non-parametric bootstrapping was used to calculate 95% confidence-intervals (CIs) for edge weights. Narrow CIs and lack of overlap between them indicate significant differences between edges, whereas wide CIs which overlap significantly make the interpretation of edge weights difficult. We also estimated whether differences between edge weights were significant using the bootstrap difference test. Node-dropping subset bootstrap was employed to quantify the stability of the EI through the correlation stability coefficient (CS-coefficient); CS-coefficients > 0.25, but preferably CS-coefficients > 0.5, are required to consider centrality parameters as stable.

RESULTS

Sample characteristics

Of the 7,723 eligible participant protocols, 318 had missing responses to more than 3 ADHD and/or 6 autism items, and, therefore, were excluded from the analyses. Individuals who were excluded from the study (N = 318) did not differ from those who were included (N = 7,405) in sex (percentage of boys = 52.65% vs. 50.72%, χ = 2, p = 0.15), age (mean [SD] = 11.03 [2.92] vs. 11.02 [2.66], t = -0.02, p = 0.98), CBCL internalizing (mean [SD] = 7.82 [7.48] vs. 7.97 [7.02], t = 0.25, p = 0.80) and CBCL externalizing (mean [SD] = 9.51 [9.41] vs. 8.12 [7.45], t = -1.76, p = 0.08) psychopathology.

Of the 7,405 individuals who were included, 4,189 (56.5%) did not have any missing data and an additional 1,680 individuals (22.7%) left only one item unanswered. The frequencies of missing data for each ADHD and autism symptom are presented in Table S1 (Appendix S2). Briefly, for ADHD symptoms, the percentage of missingness varied from 0.66% (SNAP 17) to 2.30% (SNAP 1); for autism symptoms, the percentage of missingness varied from 0.33% (CAST 2) to 4.75% (CAST 37).

Network estimation

Of 1176 potential edges, 349 (29.67%) edges different than zero were identified with a mean weight of 0.09. Almost all edges (N = 314, 89.97%) were positive; few edges were negative (N = 35, 10.03%). No node was unconnected. Figure 1 illustrates the estimated network; nodes were colored according to whether they belong to the ADHD (green) or autism (orange) construct. As depicted, the network was characterized by stronger connections between two symptoms from the same construct and by weaker connections between two symptoms from different constructs. Figure S1 (Appendix S3)

represents the same network with nodes colored according to the symptom dimension of the ADHD (inattention, hyperactivity/impulsivity) and autism (social communication and inflexible, restricted repetitive behaviors) constructs.

FIGURE 1

Relations between constructs

There were 99 non-zero edges (64.07% of all 153 possible edges) between two ADHD symptoms, 194 non-zero edges (41.72% of all 465 possible edges) between two autism symptoms, and 56 non-zero edges (10.03% of all 558 possible edges) between one ADHD and one autism symptom. Non-parametric Kruskal-Wallis test indicated significant differences in edge weight between ADHD-ADHD, autism-autism and ADHD-autism connections (χ = 45.88, $p < 0.0001$); pairwise Wilcox test accounting for multiple comparisons indicated ADHD-autism connections were significantly weaker than ADHD-ADHD ($p < 0.0001$) and autism-autism ($p = 0.03$) connections. ADHD-ADHD connections were significantly stronger than autism-autism connections ($p < 0.0001$). Figure S2 (Appendix S3) illustrates a line plot of the mean edge weight for ADHD-ADHD, autism-autism and ADHD-autism connections.

Of the 56 non-zero ADHD-autism connections, inattention symptoms and hyperactivity symptoms did not differ in number of edges (29 vs. 27) and group edge weight (Figure S3) (Appendix S3) ($W = 395$, $p = 0.96$). Similarly, social-communication and inflexible/stereotyped language and behaviors did not differ in number of edges (26 vs. 25) and group edge weight (Figure S4) (Appendix

S3) (W = 327, p = 0.97). Table S2 (Appendix S2) presents the connections with edge weights between ADHD and autism symptoms.

Considering ADHD symptoms individually, the items ‘does not sustain attention’ (SNAP 2), ‘Fidgets hands or feet; squirms in seat’ (SNAP 10), ‘talks excessively’ (SNAP 15), ‘does not wait turn’ (SNAP 17) and ‘intrudes on others’ (SNAP 18) were the only ones that established moderate or strong connections with autism symptoms. Considering autism symptoms individually, the items ‘Does not join in playing’ (CAST 1), ‘Is bad at taking turns in conversations’ (CAST 23), ‘Has unusual repetitive movements’ (CAST 28) and ‘Turns conversations to favorite subjects’ (CAST 36) were the only ones that established moderate or strong connections with ADHD symptoms. Of note, autism symptoms ‘Does not understand polite behavior’ (CAST 18) and ‘Loses the listener because of not explaining’ (CAST 32) established a considerable number – 6 and 7, respectively - of weak connections with ADHD symptoms.

Node centrality

Figure 2 presents a graphical representation of EI (Figure 2A) and bridge EI (Figure 2B) values for each of the ADHD and autism symptoms. The specific values for EI and bridge EI for each symptom are presented in Table S3 (Appendix S2). Overall, ADHD symptoms displayed higher values of EI than autism symptoms.

The five ADHD symptoms with stronger bridge EI values, i.e., that were more strongly associated with autism symptoms, were ‘fidgets hands or squirms in seat’ (SNAP 10) (bridge EI = 1.21), ‘avoids tasks that require sustained attention’ (SNAP 6) (bridge EI = 1.03), ‘does not wait turn’ (SNAP 17) (bridge EI

= 1.02), ‘intrudes on others’ (SNAP 18) (bridge EI = 1.01) and ‘Does not follow through on instructions’ (SNAP 4) (bridge EI = 0.80). The five autism symptoms with stronger bridge EI values were ‘is bad at taking turns in conversations’ (CAST 23) (bridge EI = 2.08), ‘turns conversations to favorite subjects’ (CAST 36) (bridge EI = 1.39), ‘does not understand polite behavior’ (CAST 18) (bridge EI = 1.07), ‘loses the listener because of not explaining’ (CAST 32) (bridge EI = 0.84) and ‘does not join in playing easily’ (CAST 1) (bridge EI = 0.80).

FIGURE 2

The goldbricker function identified 130 pairs of nodes which could be considered redundant. Only 4 redundant pairs were between one autism and one ADHD symptom. Specifically, ‘Is bad at taking turns in conversations’ (CAST 23) with ‘intrudes on others’ (SNAP 18) (proportion of different correlations = 85%), ‘Is bad at taking turns in conversations’ (CAST 23) with ‘Often runs about or climbs excessively in situations in which it is inappropriate. (SNAP 12)’ (proportion of different correlations = 91%), ‘Is bad at taking turns in conversations’ (CAST 23) with ‘does not wait turn’ (SNAP 17) (proportion of different correlations = 93%) and ‘turns conversations to favorite subjects’ (CAST 36)’ with ‘does not wait turn’ (SNAP 17) (proportion of different correlations = 93%).

Network stability and accuracy

Figure S5 and Figure S6 (Appendix S3) illustrate the results from the non-parametric bootstrapping and indicate the order of edges can be

interpreted with certainty. Figure S7 (Appendix S3) illustrates the results from the node-dropping subset bootstrapping. The CS-coefficients for EI and bridge EI were 0.75 and 0.59, respectively, indicating the order of centrality parameters can be estimated with confidence.

DISCUSSION

In this study, we performed network analysis on parent-reported ADHD and autism symptoms from data of a Brazilian school survey. Estimation of the network structure indicated there was a small proportion of non-zero connections between ADHD and autism symptoms (10.03% of possible edges), which were significantly weaker than connections between two ADHD or two autism symptoms. Select ADHD symptoms established moderate-to-strong connections with autism symptoms and could be considered bridge symptoms, i.e. psychopathological ‘markers’ of the co-occurrence of ADHD and autism symptomatology. Nonetheless, some of these ADHD bridge symptoms were considered redundant to autism symptoms.

Our findings indicate that individual ADHD and autism symptoms are profusely and strongly associated with other symptoms pertaining to the same construct, but sparsely and weakly associated with symptoms pertaining to the other construct in children and adolescents from the community. The presence of moderate and strong connections between two ADHD or two autism symptoms was expected given both disorders are characterized by a constellation of, rather than isolated, core symptoms (Rommelse, Visser, & Hartman, 2018); strong connections between two symptoms reflect they are likely to co-occur in the same individual as one symptom ‘leads to’ the other.

The presence of weak connections between ADHD and autism symptoms was not expected given the well-documented overlap between the two neurodevelopmental disorders, but caution is required when interpreting this finding. As described in the methods section, the *eLasso* procedures employed in our study to estimate the network structure of ADHD/autism symptoms shrinks small coefficients to zero. Therefore, *eLasso* guarantees specificity at the expense of sensitivity of the identified connections; in other words, while the connections identified in our study represent the true association between two symptoms, relevant edges might have been missed through penalization. Considering the high rates of comorbidity between ADHD and autism in clinical samples, we hypothesize that a larger number of moderate and strong connections would be identified if a clinical sample was included in the analyses. Nonetheless, given the high specificity of the *eLasso* procedure and the adequate accuracy of the estimated ADHD/autism network, the identified connections in the present study can still be interpreted with confidence, and may have some clinical implications. This perspective is in line with a dimensional approach to psychopathology (Coghill & Sonuga-Barke, 2012), with clinical diagnoses of ADHD and autism representing the upper extremes of behavioral continuums in which symptoms are distributed in a uni-dimensional trait-disorder overlap in the population of individuals (Thapar, Cooper, & Rutter, 2017). Understanding how ADHD and autism symptoms relate to each other at different ends of this continuum, beyond the categorical disorders, is also of value for clinical researchers.

The present study also provides some insights on the overlap between ADHD and autism symptoms as endorsed by parents. Our findings indicated

ADHD symptoms presenting in social contexts, such as ‘talks excessively’, ‘frequently blurts out answers’, ‘does not wait turn’ and ‘intrudes on others’, were strongly related to autism symptoms. However, some of these ADHD symptoms were also identified as redundant to autism symptoms because they shared a high proportion of similar connections with other symptoms in the network. Whether the observed node redundancy between these ADHD and autism symptoms represents the existence of (a) shared underlying causes for social impairments across neurodevelopmental disorders, (b) shared semantic references across the items of ADHD and autism rating scales, or (c) merely a general response tendency, is unclear. This issue of node redundancy or item content overlap has been raised previously regarding the relationship between social communication items of the CAST and items assessing ADHD symptoms, and more broadly in the autism context in general (Taylor et al., 2015). Currently, our findings suggest that clinicians should be alert to the possible presence of autism symptoms when an individual presents with parent-reported ADHD symptoms that only manifest in a social context.

This study is the first, to the best of our knowledge, to employ network analysis to characterize the overlap of ADHD and autism symptoms in a population sample, as previous studies (Pinto et al., 2016; Ronald et al., 2014; Ronald et al., 2008; Taylor et al., 2015) have relied on latent variables to investigate the co-occurrence of ADHD and autism symptoms. The network theory of psychopathology advocates that the association between two symptoms is established by a causal relationship between them; therefore, it refutes the existence of a latent variable which would account for the shared variability of the observed symptoms. This study is not capable of providing a

definitive conclusion about whether the observed associations between symptoms result from shared variability due to a latent variable or from a causal interplay between ADHD and autism symptoms. Both interpretations are possible and sensible.

On the one hand, recent research has identified several underlying genetic (Ghirardi et al., 2018; Miller et al., 2019), neuroimaging (Ameis et al., 2016; Aoki et al., 2017) and neurocognitive features (Visser, Rommelse, Greven, & Buitelaar, 2016) that could help explain the co-occurrence of ADHD and autism. On the other hand, ADHD and autism symptoms might influence and reinforce each other directly, thereby creating causal chains including feedback loops which culminate in the disorders (Borsboom & Cramer, 2013). For instance, a child who is bad at taking turns (CAST 23) could frequently intrude on others (SNAP 18) because of the inability to understand when it is their time to speak rather than due to pure hyperactivity-impulsivity. Because our study analyzed cross-sectional data, the identified edges between ADHD and autism symptoms were *undirected*, i.e. edges represent partial correlation coefficients and simple associations between symptoms. Longitudinal data enables the estimation of *directed* edges, which would permit inferences to be made about how symptoms influence each other in a causal manner. Previous research in child and adolescent mental health has reported interesting findings on how psychopathological symptoms in school-aged girls might influence the later emergence of anxiety and depression in adolescence (Rouquette et al., 2018). By employing network analysis methods in longitudinal data, future research could help advance the current understanding of how ADHD and autism symptoms are causally related to one another.

This study has several strengths, such as the large sample size and the analytical plan conducted in accordance with previous network analysis research (de Haan et al., 2020; McElroy & Patalay, 2019). Importantly, this study also presents a unique perspective to the presence and characteristics of neurodevelopmental traits in Brazil. The behavioral manifestation of ADHD and autism are inherently related to the expectations and demands of the surrounding environment (e.g. home and/or school) on the child, which is dependent on cultural aspects. In this way, the standards for “typical” and “atypical” behaviors and conceptualizations of ADHD and autism symptoms could vary across cultures. There is strong evidence for the cross-cultural validity of ADHD in Brazil (Rohde et al., 2005), but significantly less research has been devoted to autism (Paula, Fombonne, Gadia, Tuchman, & Rosanoff, 2011; Teixeira et al., 2010). Therefore, this study also provides a stepping stone to the characterization of autism symptoms in a school sample of children and adolescents from Brazil. Additional research on the overlap between autism and ADHD in Brazil is warranted.

Several limitations to this study should be highlighted. The study did not include a clinical sample and therefore the clinical implications of our findings are largely hypothetical, and further research investigating the network structure of ADHD and autism symptoms in clinical samples is needed. Our findings may have been influenced by selection bias given that 2,802 parents did not return the questionnaires. Answers to the SNAP were transformed from an ordinal scale to a binomial variable, which resulted in some loss of information. Potential cross-cultural differences in the presentation of ADHD and autism

symptoms in Brazil could impact the generalizability and replicability of the findings from this study.

CONCLUSION

In conclusion, the present study reports a network analysis of data from a large population sample to demonstrate that ADHD and autism symptoms manifest along multiple interconnected domains. By quantifying the ADHD/autism symptom network structure, our study demonstrates ADHD and autism symptoms may be partially segregated in accordance with diagnostic boundaries corresponding to these conditions in children and adolescents from the community.

KEY POINTS

- A network analysis was conducted in a large population sample to investigate connections between individual ADHD and autism symptoms in children and adolescents from the community.
- Some individual symptoms of ADHD and autism correlated positively with each other, but correlations between two ADHD symptoms or two autism symptoms were more frequent and stronger than those between one ADHD and one autism symptom, indicating these neurodevelopmental symptoms segregate in accordance with diagnostic boundaries in children and adolescents from the community.
- ADHD symptoms presenting in a social-context (e.g. ‘does not wait turn’ and ‘intrudes on others’) which showed moderate-to-strong connections with autism symptoms were mostly found to be redundant to social-communication autism symptoms.
- The extent to which these findings can be extrapolated to individuals with diagnoses of ADHD and/or autism and the causes for redundancy between connected ADHD and autism symptoms require direct examination in clinical samples in future research.

RELEVANCE

The present study is the first to provide an item-level network perspective to the psychopathology of ADHD and autism among individuals from the population. The insights from this study reinforce the importance of considering item level data in clinical research in child and adolescent mental health.

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FIGURES

Figure 1. The network structure of ADHD, autism symptoms colored according to disorder. Each node (circle) corresponds to an individual ADHD or autism symptom. Green nodes indicate autism symptoms and orange nodes indicate ADHD symptoms. The connections (lines) between symptoms represent edges. Width of each connection indicates the strength of the association between symptoms. Blue indicates a positive correlation whereas red indicates a negative correlation.

Figure 2. Expected influence (Figure 2A) and bridge expected influence (Figure 2B) for ADHD and autism symptoms.