



## Regular Article

# Socioemotional profiles of autism spectrum disorders, attention deficit hyperactivity disorder, and disinhibited and reactive attachment disorders: a symptom comparison and network approach

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### Abstract

Children with autism spectrum disorders (ASDs), attention deficit hyperactivity disorder (ADHD) and disinhibited and reactive attachment disorders (RAD/DAD) often experience socioemotional problems. Elucidating a clear picture of these profiles is essential. Strengths and Difficulties Questionnaires (SDQs) were analysed from cohort of children with ASD (n = 1430), ADHD (n = 1193), and RAD/DAD (n = 39). Kruskal–Wallis Tests and network analytic techniques were used to investigate symptom profiles. Children with ASD experienced more emotional problems, peer problems and fewer prosocial behaviours. Children with ADHD and RAD/DAD had higher levels of hyperactivity and conduct problems. Overall, ASD and ADHD networks were highly correlated ( $r_s = 0.82$ ), and we did not observe a statistically significant difference in terms of global Strength.

**Keywords:** ADHD; ASD; attachment; emotional; social

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Delineating the clinical profiles associated with autism spectrum disorders (ASDs), attention deficit hyperactivity disorder (ADHD) and disinhibited and reactive attachment disorders (RAD/DAD<sup>1</sup>) has been a significant concern for healthcare professionals and researchers alike (Davidson et al., 2015; Follan et al., 2011; Gargaro et al., 2011; Sadiq et al., 2012). It is not uncommon to hear health care professionals discuss the challenges untangling these conditions in practice (Klein et al., 2015; McKenzie & Dallos, 2017). Social and communication atypicalities are a core part of the diagnostic criteria for ASD and RAD/DAD (American Psychiatric Association, 2013; First et al., 2018; World Health Organization, 2018). Although it is not part of the diagnostic criteria, impairments in social functioning are also common in ADHD (Ros & Graziano, 2018). Furthermore, child institutionalisation (a key risk factor for RAD/DAD; American Psychiatric Association, 2013) is also linked with attentional, cognitive, and socioemotional difficulties

(van IJzendoorn et al., 2020) and in some cases quasi-autistic behaviours (Rutter et al., 2007). Indeed, each of these conditions, albeit to varying degrees, are associated with socioemotional and behavioural impairments (Totsika et al., 2011; Wehmeier et al., 2010; Charles H. Zeanah et al., 2016).

Understanding how these socioemotional and behavioural problems manifest is crucial not only in terms of case conceptualisation but also in terms of structuring supports and guiding interventions. For instance, if a child's neurodevelopmental profile is such that they require movement breaks in class, then that would seem like an entirely reasonable adjustment. However, if the child is leaving the classroom frequently because of peer victimisation, then naturally, the intervention changes. By contrast, if a child with ASD has a neurodevelopmental preference/inclination to avoid eye-contact, it would be a mistake to interpret this through a socioemotional lens as a marker for mood problem such as depression.

For clinicians, this challenge is compounded by the fact that mental healthcare provision tends to be diagnostically-focused. Indeed, recent survey work with healthcare professionals (n=1335), from 92 countries, indicates that practitioners most often use psychiatric classifications for administrative reasons (First et al., 2018). Practitioners were found to regard diagnosis as helpful for assigning a diagnosis and facilitating a shared understanding between practitioners, but less informative in terms of treatment and prognosis (First et al., 2018).

One popular dimensional approach to psychopathology is network analysis (Borsboom & Cramer, 2013; Borsboom

<sup>1</sup>Until recently, disinhibited and reactive attachment disorders were considered part of the same condition. However recent work does suggest that they are distinct phenomenon. Nevertheless much of the research comparing symptom profiles across these conditions (i.e. RAD/DAD and ASD or ADHD) has used the RAD/DAD criteria. Thus, in this study the term RAD/DAD is used to refer to children with either condition. This is discussed further in the limitations.

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et al., 2011; Cramer et al., 2010). To date, network techniques have been applied to a myriad of topics including depression (Mullarkey et al., 2019), anxiety (Fried et al., 2016), post-traumatic stress disorder (Armour et al., 2017), personality research (Costantini et al., 2015), neurodevelopmental conditions including ASD and ADHD (Anderson et al., 2015; Ruzzano et al., 2015; Silk et al., 2019), and social psychological assessments of attachment (McWilliams & Fried, 2019). Broadly speaking, the network approach contends that psychological constructs such as disorders as expressions of an interconnected system made up of various elements (e.g. symptoms, genes; Borsboom & Cramer, 2013). It follows, therefore, that understanding how the constituent parts of the system interact with one another is important for understanding the phenomenon of interest. In constituting networks, symptoms, attributes or behaviours are conceptualised as 'nodes', and the existence of relationships between variables is indicated by edges and the magnitude of the relationships is represented by the thickness of the edge.

The current study aims at exploring socioemotional symptom profiles in a clinical cohort of children with a diagnosis of either ASD, ADHD, or RAD/DAD. First, we will compare Strengths and Difficulties Questionnaire (SDQ) scale scores (i.e. Emotional Problems, Conduct Problems, Hyperactivity, Peer Problems, and Prosocial behaviour) across the groups. Then we will present network models based on the individual items of the SDQ. Our overarching objective is to generate hypotheses that might help signpost future research on sharper differentiation between ASD, ADHD, or RAD/DAD.

## Method

### Ethical approval

Ethical approval for analysis of anonymised healthcare records using the Clinical Record Interactive System (CRIS) was granted by The Oxfordshire Research Ethics Committee C (08/H606/71). The current project [Project number 18-039] was approved by the CRIS Oversight committee, which is comprised of patients and professionals. As per the CRIS security model, we did not analyse data in cell sizes lower than  $n=10$  to reduce the threat of inadvertent deanonymisation of participants.

### Setting

Data for this study were drawn from anonymised mental health records at the South London and Maudsley NHS Foundation Trust (slam) Case Register using the CRIS. SLAM provides mental health supports and services to a catchment area of over 1.2 million people in the following four London boroughs: Croydon, Lambeth, Lewisham and Southwark (Perera et al., 2016). Recent estimates indicate that CRIS accesses over 320,000 patient records. For a description of the cohort profile, see Perera et al. (2016).

### Measures

To explore socioemotional symptomology, we analysed SDQ (Goodman et al., 2000) data from the patient records. The SDQ is a popular, freely available screening tool for psychopathology in children. In terms of psychometric properties, the SDQ demonstrates adequate reliability and validity (Goodman, 2001). This questionnaire contains 25 items, each with three response categories ("Not True", "Somewhat True", and "Certainly True"). Scores are collected into the following scales: Emotional Problems, Conduct Problems, Hyperactivity, Peer Problems, and Prosocial

behaviour (strengths scale). Emotional Problems, Conduct Problems, Hyperactivity and Peer Problems can be combined to calculate Total Difficulties. In the current study, we used data from 2-4-year-old and 4-17-year-old parent/caregiver-report versions. In the current sample, the Cronbach's alpha value on the subscales was as follows: Emotional Problems = 0.75; Conduct Problems = 0.72; Hyperactivity = 0.74; Peer problems = 0.64; Prosocial behaviour = 0.77.

### Data extraction

Data were extracted for all children with a primary diagnosis relating to ASD (Codes: 84.0; 84.1; 84.3; 84.5; 84.8; 84.9), ADHD (All codes: F90) and RAD/DAD (i.e. F90.1, F90.2) according to World Health Organisation ICD-10 (World Health Organization, 2010). All patients were aged 17 years or under at the index diagnosis date (i.e. The date diagnosis was entered into the structured fields). Demographic data (i.e. gender and age), diagnostic status, and SDQ scores were extracted from structured fields in CRIS. Extraction took place in June 2020. To support meaningful linkage between SDQ scores and diagnosis, we treated the date of diagnosis as the index date and only selected SDQ scores which were entered six months before diagnosis or up to one month after. It is therefore important to note that we cannot confirm whether the diagnosis was historic or carried over from another service. Additionally, we only included SDQs that were coded as initial assessments. SDQ that were coded as follow up were excluded because these were often conducted following an intervention.

### Statistical analysis

First, we collected descriptive statistics, including co-occurrence, for all children with a diagnosis of ASD, ADHD, or RAD and DAD which are presented in the supplementary materials (see S7). We defined co-occurrence as ever having either a primary or secondary diagnosis of ASD, ADHD, RAD/DAD. We decided to collapse RAD and DAD into one category (i.e. RAD/DAD). Several factors contributed to this decision. RAD and DAD are often considered together under the umbrella of attachment difficulties in other studies (e.g. Davidson et al., 2015; Mayes et al., 2017; Minnis et al., 2020). We also judged that combining the two conditions would safeguard against the possibility of small cell sizes and risk of patient deanonymisation.

Where children had more than one SDQ in this timeframe, we selected the SDQ with the fewest missing items and nearest (in months) the index date of diagnosis. In total,  $k = 289$  additional SDQs were removed so that each child was only represented by one SDQ. In addition,  $n = 382$  cases were removed due to missing data or incomplete assessments. As our aim was to explore these symptom profiles, we excluded cases from the analysis where a child had two or more of the conditions of interest ( $n = 508$ ). See supplementary materials (S8) for participant flow chart.

Item-level SDQ descriptive statistics for each of the groups are presented in supplementary materials. We then compared groups (i.e. ASD, ADHD, RAD/DAD) on SDQ scales (i.e. Emotional Problems, Conduct Problems, Hyperactivity, Peer Problems, Prosocial scale, and Total difficulties) using Kruskal-Willis Tests. Post-hoc analysis was conducted using a Dunn's test (Dunn, 1964) with Bonferroni adjustment. After performing this analysis, we prepared the data for network analysis.

Many previous network analytical studies of psychopathology have used questionnaire data. An important issue has been how best to consider items which measure overlapping phenomena (Fried & Cramer, 2017). For instance, taking an example from research on depression, it could be reasonably speculated that “feeling low” might overlap conceptually with “feeling down”. It has been suggested that the presence of highly correlated items might bias the network structure if they are, in fact, measuring the same construct (Fried & Cramer, 2017). In the current paper, we adopt an approach similar to that of Burger et al. (2020); we explored inter-correlations between items in the full data set ( $n=2662$ ) and any symptom pairs with correlations of  $r_s \geq 0.6$  were combined into a single variable. We identified one pair of items which had a correlation  $r > .6$ . These were “restless/overactive” (i.e. Hyp1) and “Consistently fidgeting or squirming” (i.e. Hyp 2); we collapsed these variables into one variable (i.e. Hyp1.2) by dividing the sum of the scores by two. Combined scores were then rounded to the nearest integer to ensure variables were discrete. After this, we estimated the networks.

First, we estimated a series of partial correlation networks based on Spearman rank correlations. Specifically, we estimated separate networks for each of the groups. Like Burger et al. (2020), we used Spearman rank correlations as opposed to polychoric correlations in an effort to obtain more stable network estimations. Next, in line with reference standard psychometric work (S. Epskamp & Fried, 2018), we applied Least Absolute Shrinkage and Selection Operator (LASSO) Regularisation with Extended Bayesian Information Criterion (EBIC) model selection to the networks in an effort to limit the number of spurious edges in the network. Initially, we set the hyperparameter to 0.5. However, preliminary analysis using this hyperparameter estimated a dense regularised network when applied to the ASD group, which might signal reduced specificity (Epskamp et al., 2012), and a core assumption of the LASSO Regularisation method of model selection is the assumption of sparsity (Epskamp et al., 2017). We increased the parameter to 0.55, which did not yield a dense network. For consistency and in preparation for the network comparison test, we also adjusted the hyperparameter for the ADHD group to 0.55.

By contrast, and potentially a consequence of sample size, LASSO Regularisation with a hyperparameter 0.55 yielded an empty network for the RAD/DAD sample. In response, we decreased the hyperparameter to a more liberal .05. As described by Epskamp and Fried (2018), although reducing the LASSO regularisation hyperparameter to lower values is likely to include more spurious edges, it still yields a sparser network than a partial correlation network and is aligned with our hypothesis-generating aims (Epskamp & Fried, 2018). Given that there are almost as many nodes as participants in the RAD/DAD group it should be considered to have a more exploratory status than analyses of the ASD and ADHD groups and is therefore presented in the supplementary materials (S 1-6).

Centrality estimates were then collected. Specifically, we collected estimates for *Strength*, *Closeness*, and *Betweenness*. Briefly, the *Strength centrality* refers to the sum of absolute weights of edges Opsahl et al., 2010). Thus, the most central node in terms of *Strength* is the node with the highest sum of absolute weights of all edges connecting to a node all edges all edges. By contrast, *Closeness* refers to the inverse of the sum of the shortest paths to all other nodes. *Betweenness* refers to the number of times a node lies on the shortest path between other pairs of nodes in the network (Bringmann et al., 2019; Opsahl et al., 2010). Recent work suggests that centrality estimates, *Betweenness* and *Closeness*, in

particular, might be unstable in cross-sectional data (Bringmann et al., 2019). In the current analysis, therefore, we focus predominantly on *Strength centrality*.

Next, using the approach described by Epskamp et al. (2018) we applied nonparametric bootstrapping techniques to check the stability and accuracy of each of the regularised networks. We bootstrapped 2000 rounds for each network. Bootstrapped edge weights and confidence intervals were visually inspected for each of the networks. We then plotted significant differences between edges. Finally, we used case-dropping Bootstrap to explore the stability of the centrality indices, plotted the stability results, and collected correlation stability co-efficient (Epskamp et al., 2018).

Finally, we compared the networks. First, we plotted and compared the network visually. Then we used network comparisons tests (van Borkulo et al., 2017) to compare the ASD and ADHD networks. Given the uneven sample sizes and the less conservative approach to estimating the RAD/DAD network, we did not run network comparison tests between RAD/DAD and either ASD or ADHD.

Data were prepared, and analysis was conducted in R (R. Core Team, 2020) using the following packages: “psych” (Revelle, 2019); “qgraph” (Epskamp et al., 2012); “plyr” (Wickham, 2011); “dplyr” (Wickham et al., 2020), “ggpubr” (Kassambara, 2020a), “reshape2” (Wickham, 2007); “ggplot2”; (Wickham, 2016); “bootnet” (Epskamp et al., 2017); “rstatix” (Kassambara, 2020b); “OpenMx”(Boker et al., 2020; Hunter, 2018; Neale et al., 2016; Pritikin et al., 2015); “tidyverse” (Wickham, 2019); “car” (Fox & Weisberg, 2019); “RColorBrewer” (Neuwirth, 2014), “jmv” (Selker et al., 2020), “MASS” (Venables & Ripley, 2002); “effects”(Fox & Weisberg, 2019); “jmvcore”(Love, 2020); “igraph” (Csardi & Nepusz, 2006); “EGAnet” (Golino & Christensen, 2020); “Network ComparisonTest” (van Borkulo et al., 2017).

## Results

### *Descriptive statistics and analysis of SDQ scales*

Mean values on SDQ subscales can be found in Figure 1. For item-level means, standard deviations, skewness and Kurtosis see supplement (S9). Differences between SDQ scales were explored in a series of Kruskal–Wallis tests and Dunn’s Tests with a Bonferroni correction (see Table 2). For an illustration of scale scores with total difficulties, see supplement (S10)

In terms of demographics, we did not observe a significant association between gender and diagnosis,  $X^2(NA^2, n = 2662) = 8.56$ ,  $p = 0.1$ , though each group was heavily skewed male (see Table 1). A Kruskal–Wallis test showed significant group differences in terms of age  $H(2) = 46.8$ ,  $p < .001$ . Post-hoc Dunn’s test with a Bonferroni correction identified significant differences between ASD and ADHD ( $p < .001$ ), ASD and RAD/DAD ( $p < .001$ ), and ADHD and RAD/DAD ( $p < .001$ ). Consequently, we conducted a MANCOVA to examine the effect of age on SDQ subscale scores in the ASD and ADHD groups. We did not include RAD/DAD in the multivariate model due to the differences in sample size. A Shapiro Wilks test indicated that the data were not normally distributed ( $W = .998$ ,  $p = 0.007$ ) and a Box’s M-Test for homogeneity of covariance test identified a statistically significant result ( $X^2 = 334$ ,  $p < .001$ ), and regression lines were not parallel. Thus, the outcomes of parametric tests should be interpreted with caution. Nevertheless, we observed a

<sup>2</sup>Simulated p-value (2000 replicates).

**Table 1.** SDQ Sample Descriptive Statistics for the SDQ Sample (n = 2662)

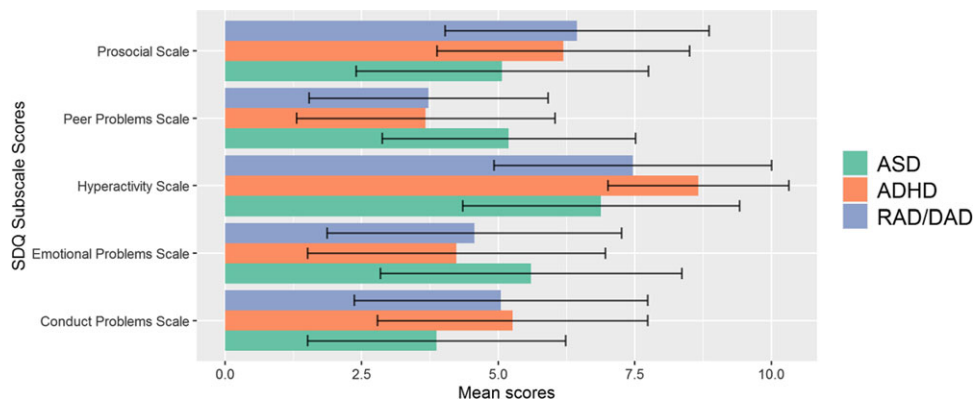
Index Diagnosis	N	Age			Gender
		Mean Age (Years)	Median (Years)	SD	(% Male)
ASD	1430	10.6	10	3.4	74.3%
ADHD	1193	9.9	9	3.2	78.9%
RAD/DAD	39	8.1	8	3.6	74.4%

SD = Standard Deviations.

**Table 2.** SDQ Scale scores

Item	ASD (n = 1430)		ADHD (n = 1193)		RAD/DAD (n = 39)		Kruskal–Wallis Tests						
	Mean	SD	Mean	SD	Mean	SD	Df	H	P	ES (eta <sup>2</sup> )	Asdx ADHD Z value (p.adj)	Asdx RAD/DAD Z value (p.adj)	Adhdx RAD.DAD Z value (p.adj)
Emotional Problems	5.60	2.76	4.23	2.73	4.56	2.69	2	154.76	P < 0.001	0.0574	-12.4 (p < 0.001)	-2.31 (p = 0.628)	0.686 (p = 1)
Conduct Problems	3.87	2.36	5.26	2.47	5.05	2.68	2	202	P < 0.001	0.0752	14.1 (p < 0.001)	3.02 (p = 0.008)	-0.394 (p = 1)
Hyperactivity	6.88	2.53	8.66	1.66	7.46	2.54	2	369.48 <sup>a</sup>	P < 0.001	0.138	19.2 (p < 0.001)	1.78 (p = 0.225)	-2.86 (p = 0.029)
Peer Problems	5.19	2.23	3.67	2.36	3.72	2.19	2	251.81	P < 0.001	0.094	-15.7 (p < 0.001)	-3.72 (p < 0.001)	0.084 (p = 1)
Prosocial Scale	5.07	2.68	6.19	2.31	6.44	2.41	2	119.41	P < 0.001	0.0442	10.8 (p < 0.001)	3.08 (p = 0.006)	0.483 (p = 1)
Total Difficulties	21.54	6.42	21.82	6.25	20.79	7.75	2	1.21	0.55	-0.003	-	-	-

Post-hoc Test were a Dunn’s Test with a Bonferroni adjustment. SD = Standard Deviations; N = number of patients; df = Degrees of freedom; ES = effect size; H = H test statistic; p.adj = adjusted p value. <sup>a</sup> = positive Bartlett’s test.

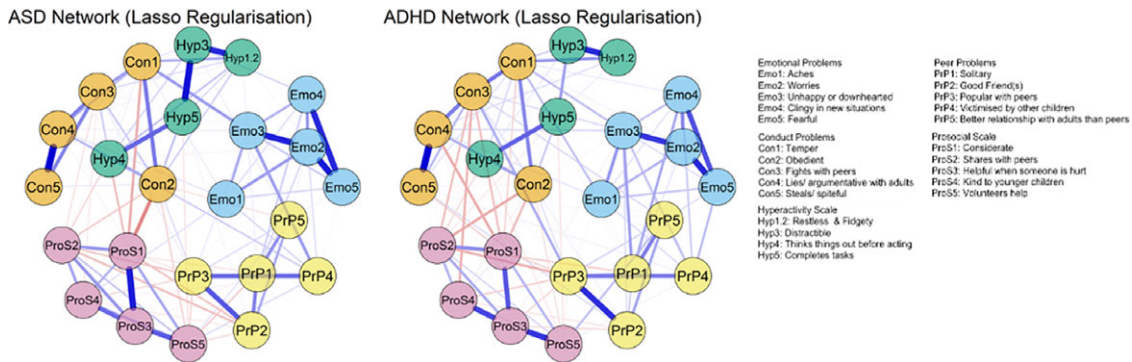


**Figure 1.** Mean scores SDQ scales. Error bars represent standard deviations. ASD (n = 1430), ADHD (n = 1193), RAD/DAD (n = 39).

significant interaction between age and diagnosis ( $F = 9.67, df = 5, 2615, p < .001, \text{partial } n^2 = 0.018$ ). Age (the covariate) was statistically significant ( $F = 47.856, df = 5, 2615, p < 0.001, \text{partial } n^2 = 0.084$ ). We identified a statistically significant difference between ASD and ADHD ( $F = 291.963, df = 5, 2615, p < .001, \text{partial } n^2 = 0.358$ ). Children with ASD had higher mean scores for emotional problems, peer problems and lower prosocial social behaviour, whereas those with ADHD had greater conduct problems and hyperactivity. For adjusted means, see supplementary materials S11).

**Network estimates**

Regularised network graphs for each condition are shown in Figure 2. There are some conspicuous similarities and differences between the networks. For instance, there is a positive edge between “temper” (Con1) and “unhappy or downhearted” (Emo3) in each of the network structures. By contrast, we can see a strong negative association between “lies or argumentative” (Con4) and “considerate” (Pro1) in the ADHD network but not in the ASD network. Edge Strength



**Figure 2.** LASSO Regularised Networks for ASD ( $n = 1430$ ) and ADHD ( $n = 1193$ ).

In these network graphs, blue lines represent positive edges, whereas red lines indicate negative edges. The magnitude of the association is represented by the width and vividness of the line. Nodes are coloured according to their respective SDQ scales. Thus blue nodes represent items from the Emotional Problems Scale. Orange nodes represent items from the Conduct Problems Scale. Green nodes represent items from the Hyperactivity Scale; Yellow nodes represent items from the Peer Problems Scale. Pink nodes represent Prosocial behaviour. Please note EBIC hyperparameter for the ASD and ADHD regularised networks is set to  $= 0.55$ .

centrality estimates for each of the networks are presented in supplementary materials (S12). Centrality estimates including Betweenness and Closeness for each group can be found supplementary materials (S2).

### Network accuracy and stability

Our first step to explore the stability and accuracy of the network was to explore edge weight stability using nonparametric bootstrapping (number of bootstraps = 2000). In the ASD and ADHD groups, the bootstrapped confidence intervals for edge weights were fairly narrow, and the sample means and the bootstrapped means were close (see supplementary materials: S3–S4). This suggests that both ASD and ADHD networks are fairly stable.

After investigating edge weight stability, we analysed the stability of centrality estimates using case-dropping Bootstrap (number of bootstraps = 2000). First, regarding the ASD network, correlation stability analysis (i.e. CS-coefficients) indicated sound levels of stability for the centrality estimates: *Betweenness* (CS-coefficient = .517) and *Closeness* (CS-coefficient = .594). Although these are both acceptable levels of stability, the highest level of stability was reported for *Strength* (CS-coefficient = .75). Taken together this means that 50% of the data could be excluded and we would still be 95% confident that there would be a correlation of at least 0.7 with all of the original centrality coefficients in the ASD network. Further in terms of *Strength centrality*, analysis indicates that 75% of the ASD sample could be dropped and we would still be confident (95%) that there would be a correlation with the original sample. In summary, centrality stability analysis indicated that the ASD network structures were stable. Turning to ADHD, centrality stability analysis found comparable CS-coefficients for *Betweenness* (CS-coefficient = .516), *Closeness* (CS-coefficient = .672) and *Strength* (CS-coefficient = .672). Therefore, like the ASD network, the centrality estimates in the ADHD network appear to be stable. Centrality analysis for each group is presented in supplementary materials (S13).

### ASD network strength centrality

Moving now to consider nodes with the strongest *Strength centrality* in the ASD network, “considerate” (pros1), “worries” (Emo2), “obedient” (Con2), “unhappy or Downhearted” (Emo3)

and “helpful when someone is hurt” (pros3) were the nodes with the greatest degree of *Strength centrality*. Nonparametric bootstrapped differences in *Strength centrality* for each of the nodes in the ASD network can be found in the supplementary materials (S14). Briefly, “considerate”, “worries”, and “obedient” seemed to be particularly central nodes in the ASD network. By contrast, items such as “better relationship with adults than peers” (prp5), “aches” (Emo1), and “steals/spiteful” (Con5) appeared to have little influence in terms of *Strength centrality*. Further, *Strength centrality* seemed to occupy a distinct role from the mean level of symptomology (see supplementary materials for item-level SDQ scores). That is, the highest scores in terms of mean symptomology tended to be associated with Hyperactivity. Moreover, the three most central nodes (i.e. Pros1, Emo2, and Con2) were 16<sup>th</sup>, 8<sup>th</sup>, 12<sup>th</sup>, respectively.

### ADHD network strength centrality

Regarding ADHD, the nodes with the highest *Strength centrality* were “(un)popular with peers” (prp3), “unhappy or downhearted” (Emo3), “lies/argumentative with adults” (Con4), “fights with peers” (Con3) and “helpful when someone is hurt” (pros3). Non-parametric differences ( $\alpha = 0.05$ ) for *Strength centrality* are presented in the supplementary materials (S15). Prp3 (“popular with peers”) appeared to have a stronger influence in the network than other items. However, “unhappy or downhearted”, “lies/argumentative with adults”, “fights with peers”, “helpful when someone is hurt”, “considerate”, “temper” each seemed to have comparable influences in terms of *Strength centrality*. In terms of mean symptomology, the five Hyperactivity items had the highest mean values (and lowest standard deviations) in the ADHD network. By contrast, (un)popularity, the most central node, was 22<sup>nd</sup> in terms of mean symptomology.

### Network comparisons

In order to explore differences between each of the network structures, first, we correlated the adjacency matrices for the networks structures of ASD and ADHD. Regarding ASD and ADHD, we observed a correlation of  $r_s = 0.82$ , which suggests remarkably strong similarities between these networks. Next, we conducted a permutation test using Network Comparison

Test (van Borkulo et al., 2017) for the ASD and ADHD networks. This permutation test did not identify a significant difference between the two networks in terms of global Strength ( $p = 0.78$ ). However, a test of network structure invariance was significant ( $M = 0.20$ , permutations = 2000,  $p < .001$ ).

### Communities of nodes

Upon visual inspection, broadly speaking, it seems that nodes tended to cluster according to their respective SDQ subscales. To investigate this further, we applied a spin glass algorithm (Reichardt & Bornholdt, 2006) using igraph (Csardi & Nepusz, 2006) to construct networks. We the number of possible communities was set to ten. This algorithm identified five communities in the ASD network and only four in the ADHD network. In both graphs, we see a cluster of emotional problems, prosocial behavioural and peer problems. Yet we also see some loose clustering of hyperactivity and conduct problems in the ASD network and a more pervasive clustering of these items in the ADHD network (see supplementary materials S16).

### Subgroup analysis: Gender and age

We also conducted a subgroup analysis to explore whether gender or age had a significant impact on the network structure. First, ASD networks for male ( $n = 1062$ ) and non-male ( $n = 368$ ) were constructed. Comparison of the respective adjacency matrices for the ASD<sub>male</sub> and ASD<sub>non-male</sub> found a strong correlation ( $r_s = 0.76$ ). In addition, the permutation did not identify a significant difference between the networks in terms of global Strength ( $p = 0.59$ ). Along similar lines, a test of network structure invariance was not significant ( $M = 1.97$ , permutations = 2000,  $p = 0.59$ ). Therefore, it seems the networks were reasonably similar. Yet these should be interpreted with some caution due to differences in sample size and relatively small  $n$  in the non-male group. ADHD<sub>male</sub> ( $n = 941$ ) and ADHD<sub>non-male</sub> ( $n = 252$ ) were also constructed. Between these networks, the correlation adjacency matrices were moderate to high ( $r_s = 0.67$ ). Permutation tests on the estimated network objects selected an empty network, and again there were notable differences in sample size between the two groups and the non-male network did not appear to be stable. As such, these networks should be interpreted with caution.

For both ASD and ADHD, age was dichotomised into two groups: i) nine years and under ii) ten years and over. In terms of ASD, network comparisons permutation test (van Borkulo et al., 2017) not identify a significant difference in global Strength ( $p = 0.83$ ) between the nine and under group ( $n = 576$ ) and the ten and over ( $n = 854$ ) groups, and there was a strong correlation between the age groups ( $r_s = 0.81$ ). Similarly, we did not observe a significant difference in global Strength ( $p = 0.62$ ) between the ADHD nine years and under ( $n = 636$ ) and the ten years and older groups ( $n = 557$ ). Again, there was a strong correlation between the networks ( $r_s = 0.78$ ). See supplementary materials for subgroup networks, centrality, and stability details (S17-36).

### Discussion

The aim of this study was to explore socioemotional and behavioural symptom profiles in a clinical cohort of children with a diagnosis of ASD, ADHD, or RAD/DAD. We identified differences in SDQ scales between the groups, with children with ASD experiencing more emotional problems, difficulties with

peers and few prosocial strengths than those with ADHD or RAD/DAD. By contrast, children with ADHD and RAD experienced by hyperactivity and conduct problems than their peers with ASD. Regarding ASD and ADHD, the diagnosis seemed to have a considerable impact on the SDQ profile. We then applied network analytic methods to build network models of the symptom profiles in the ASD and ADHD groups. We identified some differences in terms of central nodes in each of these networks; however, overall, there was a strong correlation between the ASD and ADHD networks. This was, to our knowledge, the first study to investigate socioemotional profiles in children with ASD and ADHD using a network approach.

Concerning SDQ scales, we did not observe significant differences between either ASD, ADHD, or RAD/DAD in terms of total difficulties. We did, however, identify some areas of divergence. For instance, children with ASD were more likely to experience emotional problems, peer problems, and have fewer prosocial strengths than children with ADHD or RAD/DAD. This is consistent with previous work comparing SDQ profiles in ASD and ADHD (Iizuka et al., 2010; Russell et al., 2013). The finding that children with ASD had significantly more peer problems and fewer prosocial behaviours than children with RAD/DAD is potentially illuminating. Elsewhere, Davidson et al. (2015) has described the difficulties in social functioning between RAD/DAD and ASD as “superficially similar”. Yet, our findings suggest that even a brief screening assessment was able to identify significant differences in symptom profiles. One explanation might be that the SDQ measures symptoms that are more direct indices of ASD than RAD/DAD (e.g. “shares readily with other children”). Nevertheless, this does indicate that some differences between attachment-related and ASD profiles can be identified even using brief screening tools.

Considering that RAD/DAD are conditions diagnosed based on impaired social functioning, it was striking that we did not observe statistically significant differences between RAD/DAD and ADHD in terms of peer problems and prosocial behaviour. Although both groups did seem to experience elevated peer problems according to the SDQ thresholds (<https://www.sdqinfo.org/py/sdqinfo/c0.py>) it might have been expected that social issues would be more pronounced in RAD/DAD. However, peer problems are well documented in the ADHD literature (Ros & Graziano, 2018). It might be the case that children with a diagnosis of RAD/DAD might experience comparable difficulties in this regard. It is likely that other symptoms, not captured by the SDQ, help differentiate these presentations in practice (e.g. cuddles with strangers; Follan et al., 2011). It follows, therefore, that it is attachment-specific behaviours that may differentiate RAD/DAD from ASD and ADHD. However, core to the theoretical conceptualisation of RAD/DAD is the idea that the social and emotional difficulties have relationship-specific elements (e.g. minimal comfort-seeking towards primary caregiver) but also extend beyond the context of the relationship (e.g. limited positive affect; American Psychiatric Association, 2013). Therefore, the current findings raise a question about the extent to which social and emotional difficulties associated with RAD/DAD extend beyond the relationship-specific context.

Turning to conduct problems, these were significantly higher in the ADHD and RAD/DAD groups than in the ASD group. Mean values for ADHD ( $M = 5.26$ ) and RAD/DAD ( $M = 5.05$ ) are considered ‘high’ according to SDQ scoring criteria (<https://www.sdqinfo.org/py/sdqinfo/c0.py>). Taken together, the finding that children with ADHD experience greater levels of hyperactivity

and conduct problems in comparison to peers with ASD appear to be in concert with previous work on this topic (Russell et al., 2013). One explanation for the increased rates of conduct problems in the ADHD group might be that hyperkinetic conduct disorder was a distinct subgroup of ADHD in ICD-10 (World Health Organization, 2018). The reason for the elevated rates of conduct problems in children with RAD/DAD is perhaps less clear. Previous work (Allen & Schuengel, 2020; Woolgar & Baldock, 2015) has drawn attention to the fact that conduct problems are sometimes mistakenly identified as markers for attachment disorders and RAD, in particular. As such, it would be beneficial for future studies to explore how practitioners perceive the role of conduct problems in the respective phenotypes of RAD and DAD.

The results of the network analysis tests help shed light on the relationships between socioemotional symptoms in children. Taking each of the networks in turn: in the ASD network, the degree to which the child was described as “considerate” (pro1) was the most central aspect of the network in terms of *Strength centrality*. As we can see from Figure 2, being characterised as considerate was, to varying degrees, positively associated with all of the other items on the prosocial scale and was negatively associated with distinct issues such as problems with “obedience” (Con2), “having at least one good friend” (prp2), “difficulties managing temper” (Con1), “thinking things out before acting” (Hyp4). This is particularly intriguing as few topics in ASD research have generated more debate and discussion than issues surrounding empathy (Baron-Cohen, 2009; Baron-Cohen & Wheelwright, 2004; Fletcher-Watson & Bird, 2020). Elsewhere, Fletcher-Watson and Happé (2019) draw an important distinction between *feeling* and *expressing* empathy in ASD. Though we are unable to draw conclusions about causality, our findings might tentatively suggest that support and interventions designed to promote expressions of empathy in children with ASD might be particularly helpful targets for intervention.

In terms of the ADHD network, peer (un)popularity (prp3) had the highest levels of *Strength centrality*. Here we see robust associations between peer popularity and other items from the peer problems scale, a negative association between (un)popularity and sharing, and a positive association with fighting with peers. Yet a number of other nodes appeared to be almost as influential in terms of *Strength centrality*. For instance, “unhappiness or downhearted” (emo3) seemed to occupy a critical role in the network. Moreover, in addition to links with other items from the emotion problems scale, we also see positive associations between unhappiness and several items from the conduct problems scale including “temper” (con1) and “fighting with peers” (con3), as well peer problems such as “solitary play” (prp1) and “being victimised by other children” (prp4). The link between conduct and mood problems is well established in the child psychiatric literature (Angold & Costello, 1993; Polier et al., 2012). Analysis of the SDQ subscales suggested that children with ADHD had close to average or slightly above average emotional problems. And yet in terms of centrality, it seems that unhappiness had an important role in terms of *Strength*. However, on a more general note, it signals an advantage of network analytic models, which is to identify meaningful connections between symptoms that might have been otherwise overlooked. Lower levels of wellbeing and happiness are commonly associated with ADHD (Peasgood et al., 2016; Stickley et al., 2018). Therefore, it seems that identifying the drivers of unhappiness in ADHD could be a particularly helpful target for intervention.

Previous network analytic work has found variations in symptom profiles in children with ADHD (Silk et al., 2019). One unexpected finding was that although the mean scores for Hyperactivity were high in the ADHD group, the ADHD items were not particularly influential in terms of *Strength centrality*. Indeed, by and large, nor were they particularly strong nodes in the ASD networks. Moreover, ASD items such as “gets on better with adults” were not highly central. It could be argued that these features have less influence on other areas because they are distinctly neurodevelopmental, whereas the other areas are socio-emotional. That would be an intriguing finding and would lend support to nosological assertions regarding the nature of hyperactivity (i.e. distinctly neurodevelopmental; American Psychiatric Association, 2013; World Health Organization, 2018). Then again, some items in the ASD network regarding prosocial behaviour (e.g. “considerate”) were central in the network and are, according to nosological descriptions, neurodevelopmental. Delineating the boundaries between what is socioemotional, behavioural and neurodevelopmental is one of the core challenges in differential conceptualisation.

Within this context, it is interesting to note that the community analysis identified five communities of nodes in the ASD sample and four in the ADHD group. In both networks, items from the emotional problems, peer problems and prosocial behaviour scales seemed to cluster into their respective scales. Indeed, on these scales, there were significant differences between the ASD and ADHD group. Yet, regarding conduct problems and hyperactivity, there was a more complicated picture. In the ASD sample, we see a clustering of hyperactivity items and two items from the conduct problems scale: “temper” and “(dis)obedience”. Meanwhile, in the ADHD group, all items from the conduct problems scale and the hyperactivity scales seemed to cluster together. This could reflect a hyperkinetic conduct constellation for both groups, with some differences regarding latent conduct disorder symptoms such as lying and stealing. This raises the question of whether conduct items could be sorted along the lines of oppositional and conduct behaviours.

It was somewhat unanticipated that such high correlations would be observed in the adjacency matrices between the ASD and the ADHD groups. A network permutations test (van Borkulo et al., 2017) did not find significant differences between the ASD and ADHD groups in terms of global *Strength*. This was surprising considering that significant differences were found in terms of the SDQ subscales. One explanation is that although the subscales are different, the ways in which the symptoms interact in terms of global *Strength* is similar. And yet a test of network structure invariance identified a significant difference in terms of structure. When thinking differentially about these profiles; therefore, it might be helpful to think about the qualitative intensity of behaviours rather than strictly the presence of absence of certain features. Another possibility is that the high correlations between SDQ subscale items within each network disguised more subtle significant differences in the network structures. However, checks were conducted for highly correlated items before running the analysis.

### Limitations

Firstly, given the sample size for RAD/DAD and the CRIS guidance regarding small cell sizes, we were limited in the number of covariates we could include. It is possible that groups might have differed on other factors such as ethnicity and socioeconomic

status or involvement in the care system. We think this would be a crucial area of investigation for future work on the topic of differential diagnosis. Elsewhere (Woolgar & Baldock, 2015) and others (Allen, 2016; Allen & Schuengel, 2020) have spotlighted problems with the overuse of attachment concepts in children in care. Thus, it would be illuminating for future work to explore whether symptom profiles covary by involvement in the care system. One way to do this would be to compare networks of children with a diagnosis of ASD, ADHD, and RAD/DAD who have also been involved with the care system.

Another limitation was the number of RAD/DAD cases, which led us to follow common practice and combine RAD and DAD into one group. Yet it is generally accepted that RAD is more aligned with internalising difficulties, whereas DAD is more aligned with externalising problems (Zeanah & Gleason, 2015). Further, recent work suggests that although both are associated with lower general social functioning and social competence, there might be relevant differences, such as peer victimisation (Guyon-Harris et al., 2019). Moreover, work conducted by Lehmann et al. (2016) indicated a two factor model of socioemotional and behavioural profiles on the SDQ for children with RAD and DAD.

Another limitation is that we only included children with a diagnosis entered into structured fields. Thus we do not have context for the diagnosis being entered into the structured field and the quality of the diagnostic assessment. In addition, the diagnosis might have, for instance, been historical or the diagnosis might have been made in another service. Furthermore, the children might have had other co-occurring mental health conditions and thus might have contributed to the symptom profiles. Finally, due to practical constraints, it was not possible to extract data from unstructured fields such as clinical notes.

An additional limitation of this study is that children who had a dual diagnosis of ASD, ADHD, or RAD/DAD were excluded. This decision was made in order to help us better delineate between profiles and increase confidence in the findings. Yet, at the same time, this does impose certain limitations on the generalisability. It would be beneficial for future work to explore whether children with ADHD and ASD, for instance, experienced a particular profile of socioemotional and behavioural difficulties that is distinct from children without a dual diagnosis of these conditions.

Finally, the data in this study comes from a clinical cohort of children attending mental health services. Therefore, our results might not be applicable to those not involved with mental health services. Other mental health factors (Luo et al., 2019; Simonoff et al., 2008; Thapar & Cooper, 2016) might have been relevant in terms of shaping the socioemotional and behavioural profiles in a community sample.

## Conclusions

This study explored socio-emotional symptom profiles in a clinical cohort of children with a diagnosis of ASD, ADHD, and RAD/DAD. Despite presenting with comparable levels of "Total difficulties", we observed significant differences between the subscales. Emotional problems, peer problems, and fewer prosocial strengths were more aligned with autism, whereas hyperactivity and conduct problems played a more significant role in ADHD and RAD/DAD. We observed considerable effect sizes in terms of ADHD and ASD and SDQ subscales. Yet ASD and ADHD had comparable network structures. Taken together, this illustrates the complex nature of socioemotional and behavioural difficulties

in children with ASD, ADHD, RAD/DAD and whilst also identify some specific lines of divergence.

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**Author contributions.** All authors contributed to the development of the study concept and contributed to the study design. Data analysis was conducted by BC. BC drafted the initial manuscript. MW, MvIJ and RD authors contributed critical revisions. The final draft was approved by all the authors.

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