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Understanding social behaviours across neurodiverse young people: roles of social cognition and self-regulation

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Background

Differences in social behaviours are common in young people with neurodevelopmental conditions (NDCs). Recent research challenges the long-standing hypothesis that difficulties in social cognition explain social behaviour differences.

Aims

We examined how difficulties regulating one's behaviour, emotions and thoughts to adapt to environmental demands (i.e. dysregulation), alongside social cognition, explain social behaviours across neurodiverse young people.

Method

We analysed cross-sectional behavioural and cognitive data of 646 6- to 18-year-old typically developing young people and those with NDCs from the Province of Ontario Neurodevelopmental Network. Social behaviours and dysregulation were measured by the caregiver-reported Adaptive Behavior Assessment System Social domain and Child Behavior Checklist Dysregulation Profile, respectively. Social cognition was assessed by the Neuropsychological Assessment Affect-Recognition and Theory-of-Mind, Reading the Mind in the Eyes Test, and Sandbox continuous false-belief task scores. We split the sample into training ($n = 324$) and test ($n = 322$) sets. We investigated how social cognition and dysregulation explained social behaviours through principal component regression and hierarchical regression in the training set. We tested social cognition-by-dysregulation interactions, and whether dysregulation mediated the social cognition–social behaviours association. We assessed model fits in the test set.

Results

Two social cognition components adequately explained social behaviours (13.88%). Lower dysregulation further explained better social behaviours ($\beta = -0.163$, 95% CI −0.191 to −0.134). Social cognition-by-dysregulation interaction was non-significant ($\beta = -0.001$, 95% CI −0.023 to 0.021). Dysregulation partially mediated the social cognition–social behaviours association (total effect: 0.544, 95% CI 0.370–0.695). Findings were replicated in the test set.

Conclusions

Self-regulation, beyond social cognition, substantially explains social behaviours across neurodiverse young people.

Keywords

Neurodevelopmental disorders; social functioning; self-regulation; social cognition; social behaviour.

Neurodevelopmental conditions (NDCs; see terminology choice in Acknowledgements section), such as autism, attention-deficit/hyperactivity disorder (ADHD) and paediatric obsessive–compulsive disorder (OCD), share overlapping aetiological, biological and phenotypic characteristics.[1] For instance, social communication differences, inattention, hyperactivity/impulsivity and restricted/repetitive behaviours are present across children and adolescents with autism and/or ADHD.[2] Heterogeneity also exists within each NDC diagnosis. For example, only some young people diagnosed with autism have inattention characteristics as those with ADHD.[2] The complex manifestations of NDCs motivate a transdiagnostic dimensional approach to complement findings from case–control categorical comparisons. This approach models the extent that cognitive–psychological processes underpin phenotypic characteristics cutting across NDC categories along continuums.[3]

The roles of social cognition in shaping social behaviours

Difference in social behaviours (i.e. how one interacts with others and adapts to the social environment differently – for example, some find it difficult to make friends, join conversations or identify when to say thank you or laugh at jokes) is an early-emerging feature commonly shared in many young people with NDCs.[4] The prevailing hypothesis that social cognition underpins social behaviour variability stems from the idea that cognitive processes fill the explanatory gap between brain and behaviour.[5] For example, the Theory of Mind (ToM) hypothesis of autism poses that autistic behaviours result from social cognitive difficulties in inferring one's own and others' mental states.[6,7] However, recent research shows an overlap in social cognitive abilities across typically developing and NDC people.[8,9] Although meta-analyses report, on average, atypical social cognition in people diagnosed with ADHD and OCD,[10,11] a large-scale study on autism shows substantial interindividual variability in social cognition among autistic individuals but no on-average group differences between autistic and typically developing individuals.[9] Unsupervised clustering also identifies a high-performance social cognition group comprising both typically developing and autistic adults.[8] These findings suggest that extreme impairments in social cognition may influence between-group differences observed in case–control designs, yet in their absence, group differences between typically developing individuals and those with NDCs may not be apparent.[8] Thus,

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variability in social cognition can be further understood through dimensional lenses in addition to case–control categorical approaches.[3] Still, the wide variety of measures assessing social cognition poses challenges to harmonising findings.

The roles of self-regulation in shaping social behaviours

Self-regulation encompasses affective, behavioural and cognitive regulatory processes that modify an individual's cognitive–psychological processes to adapt to environmental stimuli, thereby facilitating adaptive goal-directed behaviours.[12] Impaired self-regulation ('dysregulation'; e.g. 'becom[ing] upset too easily', 'demanding attention' or 'having explosive outbursts') is commonly observed in people with NDCs.[13,14] Dysregulation has even been posed as a core feature of ADHD.[15] Meta-analytic evidence shows that adaptive self-regulation abilities are related to better social cognition skills across clinical and non-clinical populations.[16] Dysregulation, on the contrary, is linked to social behaviour difficulties across typically developing young people and those diagnosed with autism, ADHD or OCD.[17–19] Higher parent-reported dysregulation is associated with more observations of peer conflicts and worse classroom adjustment in pre-school typically developing children.[20] Similarly, higher parent-reported dysregulation is related to more parent-reported social behaviour difficulties across children with NDCs.[21] Interventions promoting self-regulation seem to improve social cognition and social behaviours.[22,23] Taken together, dysregulation may play a critical role in the relationship between social cognition and social behaviours.

Aims

Accordingly, to understand how the relationship between social cognition and dysregulation is linked to social behaviours in neurodiverse young people, we used a transdiagnostic approach to address four objectives through a series of planned secondary data analyses, using the Province of Ontario Neurodevelopmental Network (POND) cohort. First, we aimed to identify parsimonious metrics across four available social cognition tasks, to examine how their shared variance accounts for social behaviour differences across neurodiverse young people aged 6–18 years. Second, we tested whether dysregulation explained additional variance in social behaviours on top of social cognition. Third, to explore potential mechanistic relations, we evaluated if the interaction between social cognition and dysregulation explained social behaviours. Finally, in the same vein, we examined if dysregulation mediated the association between social cognition and social behaviours. We hypothesised that across typically developing young people and those diagnosed with NDCs, dysregulation would influence social behaviours over and above social cognition, dampening one's capacity to leverage social cognitive abilities to achieve adaptive social behaviours.

Method

Participants

Analyses were conducted with archived cross-sectional data from 646 typically developing young people and those diagnosed with NDCs, aged 6–18 years, from the POND Network, a collaborative research network across five Ontario hospitals (https://pond-network.ca/our-network/). POND data are openly available in Brain-CODE; any researcher can apply to use the data through the Ontario Brain Institute. Ethical approval was obtained from each site (Queens: TRAQ #6005107 PSIY-121-01; Holland Bloorview: eREB 281; SickKids: REB #1000000346; Lawson: REB #103326; McMaster: REB #15634); written informed consent was obtained from caregivers and directly from capable young people. Data used in this study were collected between 2012 and 2022. All procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2013.

Initially, 338 typically developing young people and 1298 young people diagnosed with NDCs were available for analysis: 516 were autistic, 543 had ADHD, 206 had OCD and 33 had other diagnoses ($n = 2$ fragile X syndrome, $n = 10$ intellectual disability, $n = 7$ Tourette syndrome, $n = 1$ Down syndrome and $n = 13$ other non-specified NDCs). POND included individuals with OCD because of its shared characteristics with other NDCs.[1] The initially available typically developing group comprised participants without clinical NDC diagnoses, including four individuals with siblings with NDCs, 111 with subthreshold ADHD symptoms and four with subthreshold OCD symptoms (Supplementary Table 1 available at https://doi.org/10.1192/bjo.2024.831). However, not all initially available participants completed all social cognition or dysregulation assessments, so those without these data were excluded from the main analyses after normative modelling (see below). Participants who completed all social cognition and dysregulation assessments did not differ from non-completers in full-scale IQ ($t = -0.760$, $P = 0.447$), sex assigned at birth ($\chi^2 = 0.110$, $P = 0.739$) or NDC versus typically developing distributions ($\chi^2 = 1.170$, $P = 0.279$), but completers were significantly younger ($t = 3.644$, $P < 0.01$). Table 1 shows the sociodemographic characteristics of the 646 participants (151 typically developing and 495 NDCs), with complete data for the main analyses. See the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement in Supplementary Table 2.

Diagnostic and cognitive assessments

Clinical diagnoses were initially made by community-based physicians or clinical psychologists before enrolment in POND. Research-confirmed diagnoses for NDC group classification in the present study were further derived from research-reliable assessments administered by trained and reliable assessors in the POND Network. The Autism Diagnostic Observation Schedule-2 and the Autism Diagnostic Interview-Revised confirmed autism diagnoses.[24,25] The Parent Interview for Child Symptoms confirmed ADHD and Tourette syndrome diagnoses.[26] The Kiddie-Schedule for Affective Disorders and Schizophrenia and Children's Yale-Brown Obsessive Compulsive Scale confirmed OCD diagnosis.[27,28]

IQ

The Wechsler Abbreviated Scales of Intelligence (WASI-I or WASI-II), Wechsler Intelligence Scale for Children Fourth Edition (WISC-IV) or Wechsler Preschool and Primary Scale of Intelligence (WPPSI-III or WPPSI-IV) assessed full-scale IQ.[29–31] When the above instrument was not applicable, the Stanford-Binet Intelligence Scale Fifth edition was used.[32] Most young people (559 out of 646; 86.53%) completed full-scale IQ assessments.

Social behaviours

Social behaviours were evaluated through caregiver reports, using the Adaptive Behavior Assessment System Second Edition Social domain (ABAS-II-Social), which evaluates one's ability to navigate social situations and interact with others effectively.[33]

Dysregulation

The caregiver-rated Child Behavior Checklist dysregulation profile (CBCL-DP) captured behavioural, cognitive and affective aspects of

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Table 1 Sociodemographic characteristics of participants included in the main analyses

Sociodemographic characteristics, cognitive and behavioural measures	Typically developing ($n = 151$)	NDCs ($n = 495$)				Statistics
		Autism ($n = 214$)	ADHD ($n = 224$)	OCD ($n = 50$)	Other ($n = 7$)	
Mean age in years (s.d.)	10.52 (3.08)	10.62 (3.10)	10.28 (2.87)	11.74 (2.67)	11.28 (2.13)	**$F = 3.91$**** OCD > ADHD
Number of males (sex assigned at birth) (%)	95 (63.87%)	165 (77.1%)	164 (72.88%)	23 (46%)	5 (71.42%)	**$\chi^2 = 16.89$****
Number of females (sex assigned at birth) (%)	56 (36.13%)	49 (22.9%)	60 (27.12%)	27 (54%)	2 (25%)	**$\chi^2 = 16.89$****
Mean full-scale IQ (s.d.)	106.89 (13.15)	98.12 (18.03)	100.85 (13.97)	110.71 (12.47)	81.67 (15.49)	**$F = 13.02$**** OCD, typically developing > autism, ADHD
Social cognition: normative modelling *z*-scores						
Mean RMET total (s.d.)	−0.097 (1.07)	−0.46 (1.20)	−0.362 (1.18)	−0.28 (1.22)	−0.091 (132)	$F = 2.35$
Mean RMET positive (s.d.)	−0.05 (1.05)	−0.25 (1.06)	−0.26 (1.02)	0.35 (0.99)	−0.25 (1.28)	**$F = 3.18$****, OCD > ADHD
Mean RMET negative (s.d.)	0.05 (0.99)	−0.25 (1.00)	−0.27 (1.12)	−0.72 (0.95)	0.11 (1.10)	**$F = 5.02$****, typically developing > ADHD, OCD
Mean RMET neutral (s.d.)	−0.15 (1.02)	−0.44 (1.12)	−0.27 (1.11)	−0.12 (1.2)	0.21 (1.10)	$F = 1.17$
Mean NEPSY-II-AR total (s.d.)	−0.09 (1.04)	−0.19 (1.04)	−0.28 (1.12)	−0.3 (1.11)	0.45 (0.85)	$F = 0.60$
Mean NEPSY-II-AR happy (s.d.)	−0.09 (1.12)	−0.05 (1.09)	−0.32 (1.34)	−0.27 (1.31)	0.33 (0.21)	$F = 1.33$
Mean NEPSY-II-AR sad (s.d.)	0.05 (0.97)	−0.08 (0.91)	−0.2 (0.95)	−0.14 (0.91)	−0.50 (0.80)	$F = 0.56$
Mean NEPSY-II-AR neutral (s.d.)	−0.11 (1.06)	−0.045 (1.09)	−0.22 (1.13)	0.27 (1.23)	−0.17 (1.75)	$F = 2.37$
Mean NEPSY-II-AR angry (s.d.)	−0.08 (1.03)	−0.15 (0.95)	−0.01 (1.04)	−0.18 (1.07)	−0.40 (1.19)	$F = 1.81$
Mean NEPSY-II-AR fear (s.d.)	−0.14 (1.10)	0.14 (1.01)	−0.09 (1.10)	0.14 (1.03)	−0.12 (0.87)	$F = 2.16$
Mean NEPSY-II-AR disgust (s.d.)	0.11 (1.02)	0.14 (0.95)	0.15 (1.01)	0.13 (0.99)	0.23 (1.36)	$F = 0.49$
Mean NEPSY-II-ToM total (s.d.)	−0.39 (1.12)	−0.56 (1.39)	−0.33 (1.32)	−0.41 (1.40)	0.43 (1.54)	$F = 1.26$
Mean NEPSY-II-ToM-Verbal (s.d.)	−0.41 (1.14)	−0.56 (1.36)	−0.38 (1.35)	−0.43 (1.36)	−0.20 (1.76)	$F = 1.08$
Mean NEPSY-II-ToM-Context (s.d.)	−0.14 (1.03)	−0.2 (1.06)	−0.04 (1.04)	−0.05 (1.05)	0.63 (1.38)	$F = 0.58$
Mean Sandbox egocentric bias (s.d.)	−0.05 (1.05)	−0.07 (1.12)	0.003 (1.04)	0.09 (1.06)	−0.17 (1.35)	$F = 1.14$
Social behaviour: normative modelling *z*-scores						
Mean ABAS-II-Social (s.d.)	−0.57 (1.30)	−1.23 (1.48)	−0.89 (1.47)	−1.74 (1.56)	0.54 (1.68)	**$F = 10.45$****, typically developing > autism, ADHD, OCD
Dysregulation: normative modelling *z*-scores						
Mean CBCL-DP (s.d.)	−0.94 (2.99)	−2.59 (3.17)	−1.68 (3.26)	−3.27 (3.09)	4.02 (6.96)	**$F = 11.31$****, typically developing > autism, ADHD, OCD
Number of individuals with co-occurring conditions (%) (autism, ADHD, OCD, communication disorders, down syndrome, intellectual disabilities, motor disorders, anxiety disorders)						
Zero co-occurring conditions	100	110 (51.40%)	135 (60.26%)	45 (90%)	4 (57.14%)	**$\chi^2 = 21.52$****
One co-occurring conditions	0	43 (20.09%)	33 (14.73%)	4 (4%)	1 (14.28%)	**$\chi^2 = 26.73$****
Two co-occurring conditions	0	42 (19.62%)	32 (14.28%)	6 (6%)	1 (14.28%)	**$\chi^2 = 22.74$****
Three co-occurring conditions	0	12 (5.6%)	16 (7.14%)	0	1 (14.28%)	**$\chi^2 = 13.11$****
Four co-occurring conditions	0	2 (0.93%)	6 (2.67%)	0	0	$\chi^2 = 5.28$
Five co-occurring conditions	0	5 (2.33%)	2 (0.89%)	0	0	$\chi^2 = 4.5$
Race and ethnicity in %						
Latin American/Hispanic	4.34	5.47	2	7.69	Not applicable	$\chi^2 = 3.45$
White	87.26	83.58	95	92.30	Not applicable	$\chi^2 = 0.87$
Black	3.41	4.47	2	0	Not applicable	$\chi^2 = 4.53$
Asian	7.76	11.94	1	0	Not applicable	**$\chi^2 = 18.67$****
Jewish	3.72	3.48	3	0	Not applicable	$\chi^2 = 3.5$
Arab	0.62	0.99	0	0	Not applicable	$\chi^2 = 1.78$
Aboriginal	7.76	6.96	11.11	0	Not applicable	**$\chi^2 = 10.11$***

The typically developing sample includes siblings ($n = 4$), subthreshold OCD ($n = 4$) and subthreshold ADHD ($n = 31$). Other NDCs comprise Tourette syndrome ($n = 2$), intellectual disabilities ($n = 1$) and non-specified NDCs ($n = 4$). Full-scale IQ and race/ethnicity data were only available in a subset of participants ($n = 531$): 139 typically developing, 191 autism, 164 ADHD, 31 OCD and six other NDCs. Race and ethnicity percentages do not add up to 100% since one participant can identify with more than one group. Race and ethnicity were collected in line with the way how the Canadian Census data were collected. Statistics reflect tests of differences between the typically developing, autism, ADHD and OCD groups. Co-occurring conditions were operationalised as the count of participants with a given number of co-occurring conditions within each diagnostic group. NDCs, neurodevelopmental conditions; ADHD, attention-deficit/hyperactivity disorder; OCD, obsessive–compulsive disorder; RMET, Reading the Mind in the Eyes Test (child version); NEPSY-II-AR, Neuropsychological Assessment Affect Recognition subscale; NEPSY-II-ToM Neuropsychological Assessment Theory of Mind subscale; ABAS-II, Adaptive Behavior Assessment System 2nd Edition; CBCL-DP, Child Behavior Checklist Dysregulation Profile. Significant effects appear in bold. *$P < 0.05$, **$P < 0.01$.

dysregulation by the sum of scores of CBCL Aggression, Attention and Anxiety/Depression subscales.[34] The CBCL-DP has been used widely to measure dysregulation across typically developing young people and those diagnosed with NDCs.[35]

Social cognition

Social cognition was assessed by four cognitive tasks implemented in POND. All young people completed the Reading the Mind in the Eyes Test (RMET) Child Version,[36] endorsed by the US National Institute of Mental Health to probe social processes. This task comprises 28 photographs displaying the eye region of diverse faces, prompting participants to choose one of four adjectives best fitting the person's mental state. Subscores reflect performance on positive, neutral and negative mental states.[37] A subset of the initially available dataset completed the NEPSY-II Theory-of-Mind (NEPSY-II-ToM) and Affect-Recognition (NEPSY-II-AR) tasks,[38] along with the Sandbox continuous false-belief task.[39] The NEPSY-II-ToM, consisting of Verbal and Contextual subscales, involves 16 scenarios where participants identify characters' mental states. The NEPSY-II-AR presents 26 emotional facial images and asks participants to match them with one of four other facial images showing the same emotion. Six subscores track the number of errors for each emotion: happiness, anger, sadness, fear, disgust and neutral. Finally, the Sandbox task assesses participants' ToM, i.e. understanding that others can hold false beliefs, in a continuous manner.[39] An egocentric-bias score is computed by measuring the distance between crosses drawn on non-false-belief from false-belief conditions. A higher egocentric-bias score (i.e. larger distance) indicates lower understanding of others' false beliefs.[39] In brief, we included 12 non-overlapping social cognition metrics in the main analyses (Table 2).

Statistical analysis

Analyses were performed with R for macOS version 4.2.1 (see https://cran.r-project.org/bin/macosx/) (Fig. 1; Supplementary Table 3 describes references and R packages guiding statistical analyses).

Data preparation: normative modelling

Because the data came from a wide age range (6–18 years) and some had no available population-based standardised scores (e.g. RMET, Sandbox), we implemented a data transmutation strategy across all measures to account for sex- and age-related effects (Fig. 1(a)). Normative modelling offered the advantage to account for age and sex (assigned at birth) effects on all measures, by generating a deviation score that represents where the individual is compared with the population that the model was estimated on. The sex-stratified reference cohorts to derive RMET, ABAS-II-Social and CBCL deviation scores comprised 127 typically developing males and 92 typically developing females. Smaller reference cohorts (46 typically developing males, 70 typically developing females) were used for NEPSY-II-AR, NEPSY-II-ToM and Sandbox egocentric-bias scores, because of the later inclusion of these measures in POND. Here, typically developing young people with subthreshold ADHD or subthreshold OCD, and unaffected siblings were excluded from the reference cohorts to increase the accuracy of the predicted scores.

To ensure scalability across measures, we first converted each measure's raw scores into percentage scores by scaling each raw score from 0 to 100%, relative to the maximum possible score for that measure. Then, least squares regression models were fitted to the sex-stratified reference cohorts to model linear and quadratic effects of age from the percentage scores of each measure. We derived deviation z-scores for every participant on each measure by dividing the error (i.e. difference between actual [y_{ij}] and predicted [$\hat{y}_{ij}$] value) by the square root of the sum of the reference cohort variance (σ^2_{ij}) and the predictive variance (σ^2_{nj}). The final deviation z-scores represent how much each individual deviates from the reference typically developing trajectory, given their sex and age.

$$z_{ij} = \frac{y_{ij} - y_{ij}}{\sqrt{\sigma^2_{ij} + \sigma^2_{nj}}}$$

CBCL-DP deviation scores were calculated by adding deviation z-scores of the Anxiety/Depression, Attention and Aggression

Table 2 Variance explained by each social cognition principal component and social cognition component loadings

Number of components	Intercept	1	2	3	4	5	6	7	8	9	10	11	12
Predicted root mean s.e.													
Cross-validated	1.985	1.981	1.853	1.86	1.863	1.868	1.873	1.875	1.883	1.89	1.898	1.9	1.9
Adjusted cross-validated	1.985	1.981	1.853	1.86	1.863	1.868	1.873	1.875	1.883	1.89	1.897	1.9	1.9
% of social cognition variance explained													
Proportion	Not applicable	16.022	13.438	10.93	9.03	8.7	8.26	6.61	6.47	6.1	5.45	5.06	4
Cumulative	Not applicable	16.022	29.46	40.39	49.42	58.12	66.38	72.92	79.39	85.49	90.94	96.00	100
% of ABAS-II-Social variance explained													
Proportion	Not applicable	1.33	12.55	0.01	0.03	0.05	0.29	0	0.04	0.01	0	0.03	0.57
Cumulative	Not applicable	1.33	13.88	13.89	13.92	13.97	14.26	14.26	14.30	14.31	14.31	14.61	15.18
Social cognition component loadings													
RMET positive	Not applicable		0.431						−0.647				
RMET negative	Not applicable		0.446						0.532				−0.499
RMET neutral	Not applicable		0.571										−0.724
NEPSY-II-AR happy	Not applicable				0.361	0.664	−0.362		0.364				
NEPSY-II-AR sad	Not applicable			−0.420		0.388					−0.481		
NEPSY-II-AR neutral	Not applicable			−0.528				−0.704					
NEPSY-II-AR fear	Not applicable				0.476		0.567	0.437		−0.354			
NEPSY-II-AR angry	Not applicable	0.454								0.465	0.613		
NEPSY-II-AR disgust	Not applicable				−0.604					−0.634			
NEPSY-II-ToM-Verbal	Not applicable		−0.367								−0.355	−0.492	
NEPSY-II-ToM-Context	Not applicable	0.381		0.419								0.676	
Sandbox egocentric bias	Not applicable					0.488	−0.619	0.367					

We only report loadings ≥0.35. Note that positive correlations between principal components and the original subscores indicate that as scores in each measure increase, principal component loadings increase and *vice versa*. Negative correlations indicate inverse relationships between the social cognition component and the respective measures, such that when scores in a specific measure increase, principal component loadings decrease. ABAS-II, Adaptive Behavior Assessment System 2nd Edition; RMET, Reading the Mind in the Eyes Test (child version); NEPSY-II-AR, Neuropsychological Assessment Affect Recognition subscale; NEPSY-II-ToM Neuropsychological Assessment Theory of Mind subscale.

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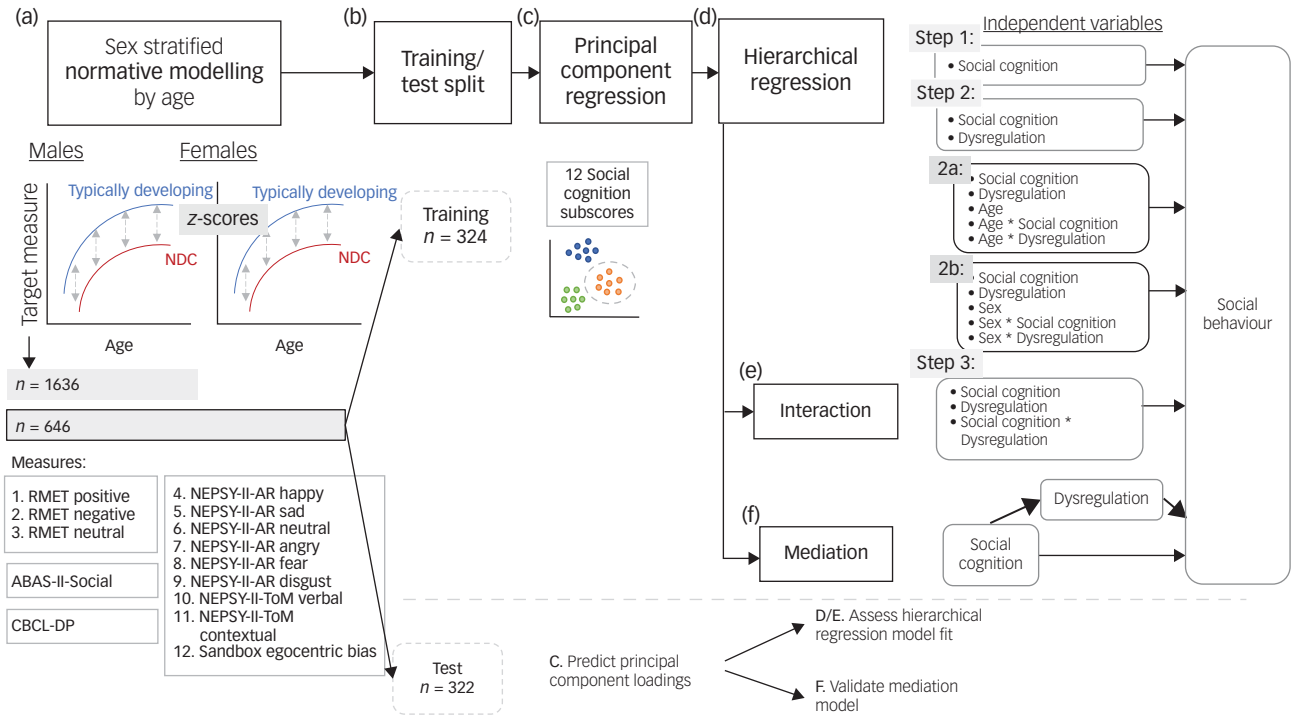

Fig. 1 Analysis flow chart. (a) Sex-stratified normative models were used to standardise all measures by age. (b) The total sample was split into a training and a test set, ensuring a balanced representation of diagnostic conditions, age and sex across sets. (c) Principal component regression was run to reduce dimensionality across the 12 social cognition *z*-scores and test their capacity to explain the variance in ABAS-II-Social *z*-scores. (d) In addition to the social cognition principal component that explained most social behaviour variance (step 1), the ability of CBCL-DP sum of *z*-scores to explain further variance in ABAS-II-social *z*-scores (step 2) was tested through hierarchical regression analysis, alongside further testing of the interaction effects of social cognition and dysregulation with age or sex, and age or sex main effects, in two separate hierarchical regression steps (steps 2a and 2b). (e) Then, we tested whether the interaction between CBCL-DP sum of *z*-scores and the social cognition principal component was significant (step 3). (f) Finally, based on our hypothesis, we tested whether dysregulation is a mediator between the social cognition principal component and ABAS-II-Social *z*-scores. Principal component loadings were predicted in the test set based on the trained principal component regression model. The predicted principal component loadings were selected to test the stability of the hierarchical regression, interaction and mediation models in the test set. ABAS-II, Adaptive Behavior Assessment System Second Edition; CBCL-DP, Child Behavior Checklist Dysregulation Profile; NDC, neurodevelopmental conditions; NEPSY-II-AR, Neuropsychological Assessment Affect Recognition Subscale; NEPSY-II-ToM, Neuropsychological Assessment Theory of Mind Subscale; RMET, Reading the Mind in the Eyes Test (child version).

subscales (Supplementary Table 3). Higher dysregulation sum of *z*-scores represent greater dysregulation. NEPSY-II-AR subscale *z*-scores and Sandbox *z*-scores, reflecting number of errors per emotion and egocentric bias, respectively, were reversed, so higher *z*-scores represent better performance across all social cognition measures. As a sanity check, our CBCL and ABAS-II deviation *z*-scores were very highly correlated with their population-based T-scores (Spearman correlations ranged from 0.966 to 0.994; Supplementary Table 4), reflecting negligible idiosyncrasies within our reference cohorts.

Main analyses

We split the sample (151 typically developing and 495 NDCs; Table 1) into training ($n = 324$) and test ($n = 322$) sets (Fig. 1(b)), ensuring balance by sex, age and diagnostic conditions (Supplementary Table 5). Supplementary Table 6 shows the correlations between age, full-scale IQ, social cognition, dysregulation and social behaviour deviation *z*-scores scores after normative modelling.

A principal component regression (PCR) with scaling and leave-one-out cross-validation was used in the training set (Fig. 1(c)) to balance the trade-off between reducing high dimensionality across the 12 social cognition metrics and capturing their differential abilities to explain social behaviour differences (Supplementary Table 3). This PCR involved principal component analysis (PCA) followed by partial least squares regression, with PCA-derived social cognition principal components as independent variables and ABAS-II-Social *z*-score as the dependent variable. Notably, although PCA is an unsupervised approach for dimensionality reduction and identification of latent constructs, PCR is supervised and deals with high-dimensional predictor data whose outcome components are interpreted with respect to the dependent variable, rather than the latent construct they might reflect. We scaled the social cognition predictors to ensure each contributed equally to the PCR model, and used leave-one-out cross-validation to guarantee our PCR model would generalise to other data-sets by systematically excluding one data point when training the model. We determined the optimal number of components accounting for variance in ABAS-II-Social *z*-scores by using the one-sigma heuristic and permutation approach (Supplementary Table 3). Since the training set sample size was over 300, we considered loadings over 0.35 as significantly contributing to each component (Supplementary Table 3). We assessed PCR model fit in the test set by examining R^2, root mean standard error and mean standard error. We used the trained PCR model to predict the social cognition principal component loadings in the test set.

In step 1, a hierarchical partial least squares regression (Fig. 1(d)) was run on the training set, with ABAS-II-Social *z*-

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score as the dependent variable and the optimal social cognition components identified in PCR as the independent variables, controlling for NDC diagnosis. In step 2, CBCL-DP sum of *z*-score was added as an additional independent variable. We also introduced two parallel steps (2a, 2b) to additionally explore main effects of age and sex, and their interactions with each social cognition component and dysregulation. We examined adjusted R^2 to understand which step/model best explained social behaviours. The one with the highest adjusted R^2 in the training set was assessed in the test set. Based on the best model, in step 3 we tested if social cognition principal components and CBCL-DP sum of *z*-scores further interacted in explaining social behaviours (Fig. 1(e)). If the interaction term was significant, we assessed model fit in the test set.

Finally, we ran a hypothesis-driven mediation analysis (Fig. 1(f)) with 1000 bootstrapped samples to test whether social cognition explains social behaviours via dysregulation on the training set, controlling for NDC diagnosis. The probability of detecting a true mediation effect was checked through Monte Carlo power analysis for indirect effects with 20 000 simulations. We re-ran the mediation model on the test set, using the predicted regression coefficients of the social cognition component that explained most variance in social behaviours in the training set.

Sensitivity analyses

We conducted four additional sensitivity analyses. First, we assessed whether any temporal bias occurred in our cross-sectional mediation analysis in the training set. Second, the indicated upper age for clinical NEPSY-II scoring is 16 years, but our sample included 17- and 18-year-old participants. Additionally, only a subset of participants completed full-scale IQ assessments. Besides, those with intellectual disabilities might drive the main finding. Thus, we reran the hierarchical regression and mediation analyses (Figs 1(d) and 1(f)), covarying for full-scale IQ on the training and test subsets of participants aged ≤16 years with complete full-scale IQ assessments, excluding those with a full-scale IQ <70. Third, we re-ran step 2 of the hierarchical regression and used a linear mixed-effects model to account for the random effect of the research site. Fourth, to assess the robustness of our findings, we re-ran the PCR and hierarchical regression analyses, alternating the training and test sets.

Results

In the training set ($n = 324$), PCR revealed that the optimal number of social cognition principal components was two. The first component explained 16.02% and the second 13.44% of the variance in social cognition. The NEPSY-II-AR Angry and NEPSY-II-ToM Contextual subscales contributed to the first social cognition component, with principal component loadings of 0.454 and 0.381, respectively. The RMET positive, negative and neutral, and NEPSY-II-ToM Verbal subscales contributed to the second social cognition component, with principal component loadings of 0.431, 0.446, 0.571 and −0.367, respectively (Table 2). Regarding social behaviours (ABAS-II-Social *z*-scores), the first component explained 1.33% and the second explained 12.55% of the variance. The MSE of the PCR model in the training set was 3.433. The PCR model fitted to the test set ($n = 322$) demonstrated an adequate fit (mean s.e. = 3.649) (Table 2; Supplementary Fig. 1).

In the training set, the hierarchical regression in step 1 explained 18.07% of social behaviour variance. The first and second social cognition principal components were positively corelated with ABAS-II-Social *z*-scores (first: $\beta = 0.180$, $P = 0.013$; second: $\beta = 0.543$, $P = 2.83e{-}11$) (Table 3). The CBCL-DP sum of *z*-scores in step 2 further explained ABAS-II-Social *z*-scores ($\beta = -0.163$, $P < 2e{-}16$); increased dysregulation correlated with poorer social behaviours. The explained social behaviour variance by the model increased to 41.36% in step 2, with a mean s.e. of 2.248. Residuals were normally distributed ($W = 0.992$, $P = 0.087$), with no concerning multicollinearity (Supplementary Fig. 2). Sex and age did not show significant main effects, nor did they moderate the associations between social cognition or dysregulation and social behaviours (Steps 2b and 2a). Thus, the step 2 model provided the best fit for the training set. This model applied to the test set explained 27.99% of the variance in ABAS-II-Social *z*-scores, with minimal increase in modelling error (mean s.e. = 2.806).

In step 3, the added interaction terms comprising the first or second social cognition principal components with CBCL-DP sum of *z*-scores were not significant in the training set (first: $\beta = -0.010$, $P = 0.317$; second: $\beta = 0.001$, $P = 0.920$).

We ran the mediation analysis using the second social cognition principal component because it explained the most variance in social behaviours. Monte Carlo power analysis for indirect effects showed that the probability of detecting a true mediation effect in the training set was very high (0.94). In the training set, the association between social cognition and social behaviours was partially mediated by dysregulation. We found a significant average direct effect ($\beta = 0.397$, $P \leq 2e{-}16$; i.e. higher social cognition correlated with better social behaviours), alongside a significant average causal mediated effect through CBCL-DP sum of *z*-scores ($\beta = 0.147$, $P = 0.002$; i.e. lower social cognition correlated with higher dysregulation, which correlated with poorer social behaviours). This partial mediation was replicated in the test set (Fig. 2; Supplementary Table 7).

Sensitivity analyses

First, presumably in a longitudinal context, the indirect effect mediated by dysregulation would have been significant, as long as the cross-lagged correlation between CBCL-DP and ABAS-II-Social remained medium and negative (<−0.300), and CBCL-DP scores remained stable over time (>0.450 autoregressive stability). Second, in the training subset aged 16 years and younger and with full-scale IQ ≥70 ($n = 262$), full-scale IQ did not explain significant variance in social behaviours ($\beta = 0.002$, $P = 0.670$). The first social cognition component ($\beta = -0.066$, $P = 0.322$) no longer significantly explained social behaviours, whereas the second social cognition component ($\beta = 0.408$, $P = 1.45e{-}07$) and CBCL-DP sum of *z*-scores ($\beta = -0.171$, $P < 2e{-}16$) continued to significantly explain social behaviours. The model's ability to explain social behaviours improved with respect to the main analyses (Supplementary Table 8). The model adequately fitted the test subset ($n = 264$) (mean s.e. = 2.738). The association between social cognition and social behaviours remained partially mediated by dysregulation in the training and test subsets (Fig. 2; Supplementary Table 9). Third, when re-running step 2 of the hierarchical regression using a linear mixed-effects model accounting for the research site as the random intercept, we found that the second social cognition component and dysregulation remained significantly related to social behaviours ($\beta = 0.166$, $P = 0.002$ and $\beta = -0.165$, $P = 6.15e{-}11$, respectively). The random intercept for the research site had a variance of 0.25, indicating that there was variability in baseline performance across sites. However, the residual variance was 1.329, suggesting that individual differences within sites accounted for the majority of the variance. A likelihood ratio test comparing the full model with a reduced model excluding the random effect for site indicated that the random intercept did not significantly improve model fit ($\chi^2 = 22.625$, $P = 0.066$). Fourth, when alternating the training and test sets, the optimal number of social cognition components was still two, identical to the

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Table 3 Hierarchical regression testing the effects of social cognition, CBCL-DP and their interaction on ABAS-II-Social scores, controlling for diagnosis

Coefficients	Estimate	s.e.		*t*-value	*P*-value
Step 1					
(Intercept)	−1.140	0.206		−5.536	**6.47e−08**
SC Comp 1	0.180	0.072		2.497	**0.013**
SC Comp 2	0.543	0.079		6.900	**2.83e−11**
Autism	−1.155	0.270		−4.285	**2.03e−05**
ADHD	−0.589	0.267		−2.208	**0.028**
OCD	−1.166	0.414		−2.817	**0.005**
Other NDC	0.487	0.830		0.587	0.616
R^2			0.196		
Adjusted R^2			0.181		
F			**12.91***		
Step 2					
(Intercept)	−0.596	0.181		−3.295	**0.001**
SC Comp 1	0.147	0.061		2.399	**0.017**
SC Comp 2	0.397	0.068		5.860	**1.16e−08**
CBCL-DP	−0.163	0.014		−11.282	**<2e−16**
Autism	−0.571	0.234		−2.442	**0.015**
ADHD	−0.017	0.232		−0.073	0.942
OCD	−0.358	0.358		−1.004	0.316
Other NDC	1.032	0.704		1.467	0.143
R^2			0.426		
Adjusted R^2			0.414		
F			**33.64***		
Step 2b					
(Intercept)	−0.343	0.250		−1.371	0.171
Sex	−0.353	0.269		−1.313	0.190
SC Comp 1	0.250	0.116		2.156	**0.032**
SC Comp 2	0.241	0.025		1.973	**0.049**
CBCL-DP	−0.203	0.122		−8.019	**2.15e−14**
SC Comp 1 × Sex	−0.144	0.137		−1.051	0.294
SC Comp 2 × Sex	0.228	0.147		1.550	0.122
CBCL-DP × Sex	0.057	0.030		1.904	0.058
Autism	−0.574	0.236		−2.431	**0.016**
ADHD	0.024	0.233		0.103	0.918
OCD	−0.346	0.360		−0.960	0.338
Other NDC	1.061	0.706		1.503	0.134
R^2			0.438		
Adjusted R^2			0.418		
F			**22.14***		
Step 2a					
(Intercept)	−0.073	0.436		−0.168	0.866
Age	−0.051	0.038		−1.321	0.188
SC Comp 1	0.029	0.221		0.131	0.896
SC Comp 2	0.532	0.267		1.994	**0.047**
CBCL-DP	−0.189	0.048		−3.943	**9.95e−05**
SC Comp 1 × Age	0.012	0.020		0.581	0.562
SC Comp 2 × Age	−0.013	0.025		−0.506	0.614
CBCL-DP × Age	0.003	0.005		0.555	0.579
Autism	−0.550	0.235		−2.342	**0.020**
ADHD	−0.018	0.233		−0.079	0.937
OCD	−0.320	0.364		−0.879	0.380
Other NDC	0.991	0.707		1.401	0.162
R^2			0.431		
Adjusted R^2			0.411		
F			**21.59***		

Significant effects appear in bold. CBCL-DP, Child Behavior Checklist Dysregulation Profile; ABAS-II, Adaptive Behavior Assessment System 2nd Edition, SC Comp, social cognition component; ADHD, attention-deficit/hyperactivity disorder; OCD, obsessive–compulsive disorder; NDC, neurodevelopmental conditions.
*$P < 0.001$.

primary result. The findings of steps 1 and 2 of the hierarchical regression also remained stable (Supplementary Table 10). The model fit of step 2 was also adequate for the other half set, with a minor increase in the mean s.e. from 3.627 to 3.92.

Discussion

Across a large, neurodiverse sample of young people, with split-half validation, we found robust evidence through PCR, hierarchical regression and mediation analyses that social cognition and dysregulation contributed additively, but not interactively, to individual differences in social behaviours, transdiagnostically across typically developing individuals and people diagnosed with NDCs. Furthermore, the level of dysregulation partially mediated the positive association between social cognition and social behaviours, with lower social cognition relating to higher dysregulation, and higher dysregulation relating to poorer social behaviours. The findings are replicable and robust when considering full-scale IQ effects, research site differences and the sequence of training and testing

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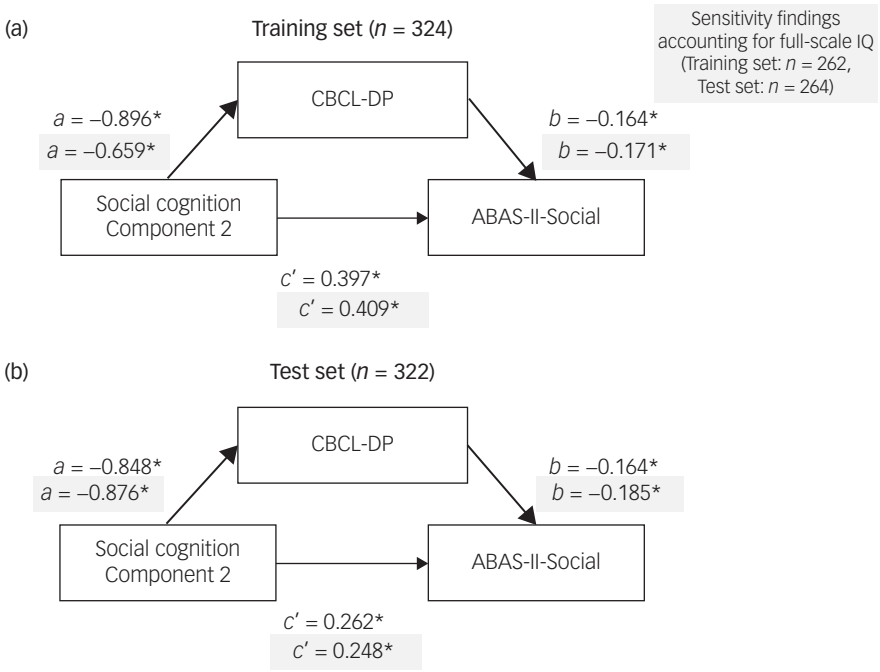

Fig. 2 Mediation analyses. Regression coefficients (βs) from the mediation models represent the indirect effect of social cognition on social behaviours (ABAS-II-Social) via dysregulation (CBCL-DP) in the training and test sets, controlling for age, sex and diagnostic condition as covariates. Partial mediation effects are in bold. Significant effects were found on paths a (from social cognition component 2 to CBCL-DP) and b (from CBCL-DP to ABAS-II-Social). Path a: having an autism diagnosis was significantly associated with poorer social behaviours in the training set ($\beta = -0.544$, $P = 0.021$), but not the test set. Having other NDC diagnoses was not significantly associated with social behaviours in the training or test set. Full-scale IQ was not significantly associated with social behaviours in the training or test sets. Path b: having autism or OCD diagnoses was significantly associated with higher dysregulation in the training and test sets (autism effect on training set: $\beta = 3.563$, $P < 0.01$; autism effect on test set: $\beta = 2.263$, $P < 0.01$; OCD effect on training set: $\beta = 4.931$, $P < 0.01$; OCD effect on test set: $\beta = 4.406$, $P < 0.01$). Having an ADHD diagnosis was significantly associated with higher dysregulation in the training set ($\beta = 3.599$, $P < 0.01$), but not in the test set. Having other NDC diagnoses was not significantly associated with dysregulation in the training or test set. Having a higher full-scale IQ was significantly associated with higher dysregulation in the training set ($\beta = 0.046$, $P = 0.047$), but not in the test set. ABAS-II, Adaptive Behavior Assessment System Second Edition; ADHD, attention-deficit/hyperactivity disorder; CBCL-DP, Child Behavior Checklist Dysregulation Profile; NDC, neurodevelopmental conditions; OCD, obsessive–compulsive disorder. *$p < 0.05$.

in the split-half samples. Across neurodiverse young people, in addition to the variability in social cognition, one's level of dysregulation is also associated with, and potentially mechanistically linked to, their social behaviours.

During the modelling optimisation, we attempted to address issues of heterogeneous measurement of social cognition. Using normative modelling combined with PCR allows us to incorporate a wide array of available metrics and account for potential sex and age effects on social cognitive performances. In contrast to an earlier study using a subset of the POND sample,[37] we noted no significant diagnostic differences in RMET total scores after normative modelling (with some differences in positive and negative valence scores; Table 1). In fact, we found no differences between diagnostic groups on other social cognition performances after normative modelling either (Table 1), echoing recent findings that social cognition differences between NDC and typically developing groups in large-scale data-sets are not as remarkable as previously thought.[8,9] This finding reinforces the notion that the variability across typically developing young people and those diagnosed with NDCs could be further understood with a dimensional approach that is complementary to a categorical framework.[3]

We used PCR to identify parsimonious social cognition components that best explained social behaviours in the training set. Two components explained cumulatively 13.88% of the variance of social behaviours with the least uncertainty, adding to the emerging evidence that social cognition only explains small-to-medium variance in social behaviours across neurodiverse young people.[40] We focused on the interpretation of the second component's measure loadings because it explained the most variance in social behaviours. Consistent with previous findings,[37] RMET positive, negative and neutral subscores positively correlated with social behaviours. Unexpectedly, increased NEPSY-II-ToM Verbal abilities correlated with worse social behaviours. This could be reminiscent of the observation that a subset of young people with NDCs is characterised by good ToM despite poor social behaviours.[41] Differences in loadings might also be explained by the RMET involving processing of specific facial stimuli versus general situation processing in the NEPSY-II-ToM. Future research should still examine how various domains of social cognition differentially contribute to social behaviours.

We used the validated CBCL-DP score to capture overall behavioural, affective and cognitive dysregulation. Beyond social cognition, caregiver-rated dysregulation also significantly explained the variance in social behaviours; higher dysregulation correlated with poorer social behaviours. Accounting for dysregulation substantially increased the model fit. This finding is robust in the test set and across all sensitivity analyses. Adding to previous findings that increased dysregulation is related to poorer social behaviours in young people diagnosed with autism or ADHD,[13,14,17,18] our findings further suggest that to understand social behaviours

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across neurodiverse young people, both social cognition and dysregulation should be considered.

The crucial roles of dysregulation and social cognition in social behaviours are likely consistent across ages and sexes. The normative modelling of social cognition, dysregulation and social behaviour metrics allowed us to consistently characterise individual capabilities with respect to normative age- and sex-related distributions. Additionally, age and sex did not significantly explain variance in social behaviours, nor moderate the relations between social cognition, dysregulation and social behaviours. Nonetheless, future studies should examine how gender-related variables (e.g. gender identity, gender role) and sex-related biological factors (e.g. sex hormone levels), beyond the sex label assigned at birth, influence the extent and direction of the relations among social cognition, dysregulation and social behaviours. Longitudinal designs are also needed to properly disambiguate age, developmental and cohort effects.

We found that dysregulation consistently partially mediated the association between social cognition and social behaviours. This novel finding advances the current understanding that social cognition decreases as dysregulation increases across clinical and non-clinical populations.[16] It further supports our hypothesis that dysregulation could mechanistically mediate the association between social cognition and social behaviours, by dampening the capacity to leverage social cognitive abilities to be socially adaptive. Although much intervention research in NDCs has focused on social cognitive skills, whether such interventions successfully improve real-life social adaptation remains inconclusive.[42,43] Our findings suggest that interventions targeting at reducing dysregulation (whether via individual- or context-focused approaches) may improve social behaviours across neurodiverse young people.[22,23]

Although we found social cognition and dysregulation consistently explained social behaviours across the training and test sets, NDC diagnostic effects were inconsistent. Specifically, having an autism diagnosis was related to worse social behaviours after accounting for dysregulation in the training set (Table 3; Fig. 2). An autism diagnosis was, however, not related to worse social behaviours after accounting for dysregulation when we alternated the training and test sets in our sensitivity analyses (Fig. 2, Supplementary Table 10). Despite the unstable categorical effects of autism in the current data-set, a previous study suggests that the association between dysregulation and worse social behaviours is stronger in autistic compared to non-autistic children.[21] Future research should probe how other psychological processes beyond social cognition and dysregulation contribute to social behaviours in the autistic populations, and how it may or may not differ in other categorical NDC groups. The unstable effect of autism diagnosis in this study also highlights that social cognition and dysregulation profiles are likely more robust predictors of social behaviours than specific NDC diagnoses. This finding supports the transdiagnostic perspective,[3] and suggests that addressing dysregulation in addition to promoting social cognition could be an efficient strategy to enhance adaptive social behaviours across neurodiverse young people. This transdiagnostic perspective also aligns with the neurodiversity paradigm, recognising that humans vary along continuums of neurological make-up and cognitive processing without holding 'typically developing' as the ideal of functioning.[44]

This study represents an attempt to comprehensively examine how dysregulation intersects with social cognition to explain social behaviours across neurodiverse young people. Therefore, our analytic approach aimed to establish a thorough conceptual understanding while reducing multiple testing. Future research should assess how specific social cognitive and self-regulation mechanisms promote social behaviours using more fine-grained analyses (e.g. structural equation modelling). There are additional study limitations. First, the mediation findings are based on cross-sectional data; definitive causal inferences should be based on longitudinal or interventional findings. Second, female and Black, Asian and minority ethnic individuals were relatively underrepresented in some diagnostic groups. Third, there could be intrinsic differential item functioning and measurement non-invariance problems when using the same measure across different populations, which are inherent challenges of transdiagnostic research. Fourth, social cognition was measured based on the young person's one-time task performance, whereas dysregulation and social behaviours were based on caregiver's observations over time, which might result in heightened reporting of children's problematic behaviours.[45] The variance explained in social behaviours by social cognition and dysregulation might be partly attributable to common-method variance across dysregulation and social behaviour metrics (i.e. both via caregiver reports). The findings should be further assessed using real-life interactive measurements. Finally, individuals with severe or profound intellectual disabilities were not well-represented in the sample. Nonetheless, full-scale IQ did not significantly explain social behaviour variance, and findings mostly maintained after accounting for full-scale IQ effects.

In conclusion, we provided new empirical evidence to support the updating of earlier cognitive theories seeking to fill the explanatory gaps between brain and behaviour,[5,44] such as the ToM account.[6] We found that self-regulation, on top of social cognition, is substantially associated with social behaviours across neurodiverse young people. Social cognition and self-regulation should be jointly considered to accurately understand and support adaptive social behaviours across typically developing young people and young people diagnosed with NDCs.

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Supplementary material

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Data availability

The POND data that support the findings of this study are available on request through BrainCODE (https://www.braincode.ca/).

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We use the term 'neurodevelopmental conditions (NDCs)' to refer to autism, ADHD, OCD, intellectual disabilities and other conditions conventionally labelled as 'neurodevelopmental disorders'. We use the term 'neurodiverse' to encompass typically developing people and individuals diagnosed with NDCs. For further clarity on these definitions, please refer to: https://neuroqueer.com/neurodiversity-terms-and-definitions/. We understand and value the diversity of perspectives and evolving language choices. We would like to thank the families who participated in the POND Network studies, Alana Iaboni for assistance with data curation, and members of the MCLab for fruitful discussions that inspired this work.

Author contributions

I.I.-S. contributed to study conceptualisation, methodology, formal analysis, data validation, project administration and data visualisation, wrote the original draft of the manuscript and reviewed and edited the manuscript. E.A. contributed to study conceptualisation, investigation, resources, data curation, methodology, supervision and funding acquisition. M.A.F. contributed to study conceptualisation, methodology and supervision. J.C., R.S., R.N., S.G., E.K., J.J. and J.B. contributed to the study investigation and resources. H.-Y.L. contributed to study conceptualisation, methodology, software and study supervision, wrote the original draft of the manuscript and reviewed and edited the manuscript. M.-C.L. contributed to study conceptualisation, investigation, resources, methodology, project administration, supervision and funding acquisition, wrote the original draft of the manuscript and reviewed and edited the manuscript. All authors read and approved the final manuscript.

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Declaration of interest

None.

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