

# Sensory Over-responsivity: A Feature of Childhood Psychiatric Illness Associated With Altered Functional Connectivity of Sensory Networks

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## ABSTRACT

**BACKGROUND:** Sensory over-responsivity (SOR) is recognized as a common feature of autism spectrum disorder. However, SOR is also common among typically developing children, in whom it is associated with elevated levels of psychiatric symptoms. The clinical significance and neurocognitive bases of SOR in these children remain poorly understood and actively debated.

**METHODS:** This study used linear mixed-effects models to identify psychiatric symptoms and network-level functional connectivity (FC) differences associated with parent-reported SOR in the Adolescent Brain Cognitive Development (ABCD) Study, a large community sample (9 to 12 years of age) ( $N = 11,210$ ).

**RESULTS:** Children with SOR constituted 18% of the overall sample but comprised more than half of the children with internalizing or externalizing scores in the clinical range. Controlling for autistic traits, both mild and severe SOR were associated with greater concurrent symptoms of depression, anxiety, obsessive-compulsive disorder, and attention-deficit/hyperactivity disorder. Controlling for psychiatric symptoms and autistic traits, SOR predicted increased anxiety, attention-deficit/hyperactivity disorder, and prodromal psychosis symptoms 1 year later and was associated with FC differences in brain networks supporting sensory and salience processing in datasets collected 2 years apart. Differences included reduced FC within and between sensorimotor networks, enhanced sensorimotor-salience FC, and altered FC between sensory networks and bilateral hippocampi.

**CONCLUSIONS:** SOR is a common, clinically relevant feature of childhood psychiatric illness that provides unique predictive information about risk. It is associated with differences in brain networks that subserve tactile processing, implicating a neural basis for sensory differences in affected children.

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Sensory over-responsivity (SOR) is a pattern of atypical negative reactions to seemingly innocuous sensory stimuli such as clothing textures or appliance sounds. SOR is known to cause distress and functional impairment in childhood and is associated with poor sleep and nutrition, anxiety, negative affect, and impaired family functioning (1–6). SOR is common among children with neurodevelopmental disorders and was recently added to the DSM-5 diagnostic criteria for autism spectrum disorder (ASD) (7–12). Yet SOR is also found in an estimated 15% to 20% of typically developing children (1–4,6), where it is positively associated with common childhood psychiatric symptoms and diagnoses (1,3,4,6,13–20). The etiology and significance of SOR in typically developing children is a topic of considerable ongoing debate, resulting in clinical uncertainty about diagnoses and interventions (21,22).

Scientists and clinicians have put forth conflicting explanations for why SOR may be associated with psychiatric symptoms in children. One account proposes that SOR is a

manifestation of emotion dysregulation, which is an established transdiagnostic correlate of virtually all psychiatric disorders (23–27). By this account, SOR does not reflect differences in sensory processing or provide independent information about psychiatric risk over and above existing psychiatric symptoms. Another account draws on evidence that autistic traits are continuously distributed in the population and positively associated with atypical sensory responses including SOR among nonautistic adults (28–30). Given high rates of psychiatric illness in autistic children and elevated autistic traits in children with psychiatric diagnoses (31–35), this account proposes that links between SOR and psychiatric symptoms in nonautistic children are caused by subthreshold autistic traits. According to this account, SOR would not provide predictive information about psychiatric risk over and above other autistic traits. A third account builds upon evidence that SOR in early childhood predicts worsening anxiety symptoms in both autistic and typically developing children

(6,36). It posits that SOR reflects neurobiological differences in sensory processing that can lead to enhanced anxiety and associated psychopathology (22,37–39). By this account, SOR should provide unique predictive information beyond both psychiatric symptoms and autistic traits. These 3 accounts entail fundamentally different conceptualizations of SOR as a behavioral outcome of psychiatric illness (dysregulation account), a feature of subthreshold autistic traits (autism account), or a specific neurobiological risk factor (sensory-specific account). Resolution of this debate and progress in our understanding of SOR in childhood critically hinge upon how SOR is related to common psychiatric disorders of childhood when controlling for autistic traits, whether SOR provides predictive information about psychiatric illness, and whether SOR is associated with neural differences related to sensory processing (21).

These questions have gone unanswered because most studies investigating SOR focus on autistic populations. Community studies of SOR to date do not control for autistic traits and lack neural measures that could elucidate the neural bases of SOR in these children (1,3,40–42). Functional magnetic resonance imaging (MRI) studies with autistic children have found that SOR is associated with differences in stimulus-evoked activity in the sensory cortex (43–45) and in the functional connectivity (FC) of the sensorimotor cortex and amygdala (46,47). These results from studies of autism, paired with success using FC to predict cognitive traits in the general population (48–50), suggest that FC is a promising imaging modality to study in relation to SOR.

Existing community studies of SOR also include sample sizes ranging from 50 to approximately 1000 (1,3,40–42). Due to sampling variability, smaller datasets tend to produce imprecise risk estimates relative to true population parameters and provide insufficient statistical power to discern effects with multivariate models that include multiple covariates (51,52). This study leverages existing data from the Adolescent Brain Cognitive Development (ABCD) Study to overcome these challenges and provides the first investigation of clinical and neural correlates of SOR in a large ( $N = 11,210$ ) longitudinal community sample (53,54). We used multilevel modeling (MLM) with clinical covariates to specify the relation between SOR and psychiatric symptoms, determine whether SOR signals psychiatric risk, and identify neural differences associated with SOR, thereby testing predictions of conflicting accounts of SOR and clarifying its relevance to childhood psychopathology.

## METHODS AND MATERIALS

### Participants

Data were obtained from the ABCD Study, a multisite, longitudinal study that collects behavioral, clinical, and neuroimaging data from children in the United States beginning at 9 to 10 years of age. Participants were recruited primarily through schools using a selection process designed to maximize sample representativeness and minimize selection biases (55). Children who met diagnostic criteria for ASD were excluded if they required special-needs schooling. Data included the baseline (Y0), year 1 (Y1), and year 2 (Y2) time points. All present analyses were limited to the 11,210 children

with available SOR measures. Specific sample sizes vary by analysis depending on data availability (see [Demographic and Clinical Analysis](#)) (Table 1).

### Assessments

Behavioral and clinical measures were drawn from parent- and self-report instruments included among the ABCD Study assessment battery (53).

**Short-Social Responsiveness Scale.** The Short-Social Responsiveness Scale is an 11-item abbreviated parent-report instrument derived from the Social Responsiveness Scale, Second Edition to measure ASD symptoms (56). It was administered at Y1 when participants were 10 to 11 years of age. The following item was used to identify SOR: “Seems overly sensitive to sounds, textures, or smells.” Parents responded with Not true (“1”), Sometimes True (“2”), Often True (“3”), or Almost Always True (“4”). Three SOR groups were derived to reflect the relative frequency of SOR behaviors according to parent report: no SOR (“1”; not overly sensitive),

**Table 1. Demographic Information About the Full Sample**

Demographic Variables	<i>N</i> <sup>a</sup>	Mean (SD), <i>n</i> , or <i>n</i> (%)
Age		
Age at Y1, months	11,210	131.1 (7.7)
Age at Y2, months	10,175	144.0 (8.0)
Sex at Birth <sup>b</sup>		
Female	11,210	5346 (47.7%)
Male	11,210	5864 (52.3%)
Socioeconomic Status		
Economic disadvantage	11,199	2289 (20.4%)
Pubertal Stage		
Pubertal stage at Y1 <sup>c</sup>	11,138	2.19 (0.86)
Families		
Number of families participating	–	9276
Number of families with >1 child participating	–	1871
Parental Schooling, Highest Obtained		
Did not complete high school	11,181	523 (4.7%)
Obtained high-school degree or equivalent	11,181	998 (8.9%)
Completed some college or obtained associate degree	11,181	2838 (25.4%)
Obtained bachelor's degree	11,181	2891 (25.9%)
Obtained master's degree	11,181	2721 (24.3%)
Obtained professional or doctoral degree	11,181	1210 (10.8%)
Race/Ethnicity (Multiple Responses Permitted)		
Asian/Pacific Islander	11,193	772 (6.9%)
Black/African American	11,193	2269 (20.2%)
Hispanic/Latinx	11,056	2215 (20.0%)
Native American/Alaskan Native	11,193	385 (3.4%)
White	11,193	8437 (75.4%)

Y, year.

<sup>a</sup>For each variable, *N* depends on the number of participants with measures for both sensory over-responsivity and the variable of interest.

<sup>b</sup>Sex categories reflect participant sex identified at birth.

<sup>c</sup>Average of parent and youth report.

## Sensory Over-responsivity in Childhood

mild SOR (“2”; occasionally overly sensitive), and severe SOR (“3” or “4”; frequently overly sensitive). The remaining items were separated into 6 social communication impairment (SCI) items and 4 restricted, repetitive behavior (RRB) items, as identified by prior factor analysis of Social Responsiveness Scale, Second Edition items (57). Total SCI and RRB scores were computed as the sum of ratings for SCI and RRB items, respectively.

**Child Behavior Checklist.** The Child Behavior Checklist (CBCL)/6–18 is a parent-report measure comprising items scored on a 3-point Likert scale that assesses the frequency of behaviors indicative of psychopathology and atypical development in children of ages 6 to 18 years (58). Internalizing and externalizing problem raw scores were used as dimensional measures of internalizing and externalizing symptoms. The depressive problems, anxiety problems, attention-deficit/hyperactivity problems, oppositional defiant problems, conduct problems, and obsessive-compulsive problems raw scores were analyzed as dimensional measures of disorder-specific symptoms. Raw scores were used for all statistical analyses, allowing us to model change in these metrics over time. T scores were only used to summarize and illustrate symptoms by SOR group relative to approximate cutoffs for scores in the borderline clinical ( $65 \leq T < 70$ ) or clinical ( $T \geq 70$ ) range.

**Prodromal Questionnaire-Brief Child Version.** Prodromal Questionnaire-Brief Child Version (PQ-BC) is a 21-item self-report questionnaire assessing psychotic-like experiences administered annually to participants in the ABCD Study (59). The prodromal psychosis score (sum of endorsed items) was used as a dimensional measure of prodromal psychosis symptoms at Y1 and Y2.

### Demographic and Clinical Analysis

We used an MLM approach to examine how the SOR group is associated with variables of interest. Linear mixed-effects models were fitted to assess relationships between the multinomial predictor SOR group (modeled as a multinomial with groups no, mild, and severe SOR) and dichotomous or continuous demographic or clinical outcome variables. All models included random effects for family nested within data collection site and included sex, economic disadvantage, and Y1 age in months as either fixed-effects covariates or outcome variables. MLMs were fitted using PROC GLIMMIX (SAS Institute, Inc.) with a logit link function for dichotomous outcomes and PROC MIXED (version 9.4 statistical software; SAS Institute, Inc.) for continuous outcomes. False discovery rate (FDR)-corrected *p* values were computed using the Benjamini-Hochberg method (60) to preserve an overall type I error rate of  $\alpha = 0.05$  across sets of related models (see the Supplement). Associations between SOR group and demographic variables were tested using models with sex, economic disadvantage, and age in months as outcome variables ( $n = 11,199$ ). Associations between SOR group and autistic traits were assessed using models with SCI and RRB scores as outcome variables ( $n = 11,199$ ).

Associations between SOR group and concurrent psychiatric symptoms were evaluated for symptoms of depression, anxiety disorders, obsessive-compulsive disorder (OCD), attention-deficit/hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), and conduct disorder (CD) (from Y1 CBCL;  $n = 11,194$ ) and prodromal psychosis (from Y1 PQ-BC;  $n = 11,199$ ) using independent models. To assess the specificity of associations between mild or severe SOR and concurrent psychiatric symptoms, we additionally fitted 2 models with the SOR group as a dichotomous outcome variable as follows: no versus mild SOR ( $n = 10,538$ ;  $n = 9150$  no SOR/1388 mild SOR) and no versus severe SOR ( $n = 9806$ ;  $n = 9150$  no SOR/656 severe SOR); Y1 measures of depression, anxiety disorders, OCD, ADHD, ODD, CD, and prodromal psychosis symptoms were simultaneously entered as predictors. To test whether SOR would predict subsequent changes in psychiatric symptoms 1 year later, models were fitted with Y2 depression, anxiety disorders, OCD, ADHD, ODD, CD, and prodromal psychosis symptoms ( $n = 7894$ ) as outcome variables and the corresponding Y1 symptom measure along with other Y1 internalizing and externalizing symptoms as covariates (Table S1).

### Inclusion of Autistic Trait Covariates

Selection of appropriate covariates is necessarily guided by researchers' understanding of the constructs in question. Given existing debate regarding whether SOR should be conceived as a behavioral outcome of psychopathology, a feature of subthreshold autism, or a risk factor for psychiatric illness, we adopted an incremental approach to the inclusion of ASD covariates. To assess the specificity of associations between SOR and psychopathology over and above other autistic traits, we incrementally tested survival of significant effects with inclusion of increasingly related covariate measures, applying FDR correction at each interim step. Prior studies of autistic traits have grouped SOR with RRB (57,61,62); therefore, we covaried for SCI before RRB in this incremental approach. Models were initially fitted as described above. When significant effects were found after FDR correction, models were refitted to include SCI; when significant effects persisted after FDR correction, models were refitted to additionally include RRB. This approach provides added transparency and information about individual associations.

### Imaging Acquisition and Preprocessing

Resting-state functional MRI data were collected with 3T scanners across multiple ABCD sites at Y0 and Y2. Detailed descriptions of the protocol have been published elsewhere (54). Briefly, three or four 5-minute runs of resting-state functional MRI were collected during each scan session, with slices acquired in the axial plane. We used ABCD Study recommendations for data quality control (see the Supplement). To maximize SOR group differences in service of identifying neural correlates, FC analyses were limited to children in the no and severe SOR groups. Following exclusion, we retained 7760 children ( $n = 7296$  no SOR/464 severe SOR) for Y0 exploratory analysis and 5117 children ( $n = 4819$  no SOR/298 severe SOR) for Y2 a priori analysis. Cortical parcels were derived and assigned to 1 of 13 functional networks according

to the Gordon parcellation scheme (63), whereas subcortical regions of interest were identified with atlas-based segmentation (64). Mean pairwise blood oxygen level-dependent signal correlations between pairs of cortical parcels or between cortical parcels and subcortical regions were computed, Fisher transformed, and averaged by cortical network.

### Analysis of FC

Linear mixed-effects models were used to determine how SOR group is associated with FC between brain network/structure pairs while accounting for variance introduced by scanners at different ABCD sites. We analyzed Y0 data in an exploratory fashion to identify FC pairs of interest that were then subjected to a priori testing in the separate Y2 dataset. We fitted two MLM models separately for each FC pair: one with and one without Y0 CBCL internalizing and externalizing raw scores included as covariates (see the [Supplement](#)). To ensure both the face validity and specificity of FC-SOR association results, FC pairs were selected for a priori testing only if FC-SOR associations were statistically significant in both models. The resulting FC pairs of interest and analytic plan for a priori testing in the Y2 dataset were preregistered (<https://osf.io/bzpa4>) prior to data analysis. A priori testing similarly entailed two MLM models per FC pair, one with and one without Y2 internalizing and externalizing covariates, and required statistical significance in both models. Exploratory Y0 analyses and preregistered analyses were carried out using the nlme package (65) available for R (66). Y2 FC pairs that were significantly associated with SOR in the preregistered analyses were subjected to more stringent post hoc testing with models including family, sex, age, economic disadvantage, and autistic traits (see the [Supplement](#)). Post hoc MLMs were fitted using PROC GLIMMIX with a logit link function for dichotomous outcomes in SAS version 9.4 statistical software (SAS Institute Inc.).

## RESULTS

### SOR Results

Most participants (82%;  $n = 9163$ ) fell within the no SOR group. An additional 12% ( $n = 1390$ ) made up the mild SOR group, and 6% ( $n = 657$ ) comprised the severe SOR group. Although children with severe or mild SOR together comprised only 18% of the Y1 sample, they constituted 65% and 52% of children with Y1 CBCL T scores in the clinical range ( $T \geq 70$ ) for internalizing and externalizing behaviors, respectively

(Table 2). Symptom measures by group are summarized in Figure 1, Table 3, and Table S2.

Children with either severe or mild SOR were more likely than those without SOR to be male ( $ps < .0001$ ; odds ratio [OR] = 1.81, 95% CI, 1.50–2.20 and OR = 1.38, 95% CI, 1.21–1.58, respectively), and children with severe SOR were more likely to be male than those with mild SOR ( $p = .04$ ; OR = 1.31, 95% CI, 1.05–1.64). Relative to no SOR, severe SOR was also positively associated with economic disadvantage ( $p = .04$ , OR = 2.61, 95% CI, 1.23–5.52). Age at time of SOR measure was not significantly associated with SOR ( $ps \geq .14$ ).

### Concurrent Clinical Results

SOR was associated with higher autism trait scores (SCI and RRB) in all pairwise group comparisons ( $ps < .0001$ ), indicating a stepwise function across the 3 groups (none < mild < severe). Hereafter, results are reported from models that include both SCI and RRB scores as covariates (see the [Supplement](#) for comprehensive results). Analysis of CBCL disorder-specific scores revealed similar stepwise associations between increasing SOR severity and concurrent symptoms of depression ( $ps \leq .0005$ ), anxiety disorders ( $ps \leq .003$ ), and OCD ( $ps \leq .0006$ ) (Table S3). Mild and severe SOR were significantly associated with greater ADHD symptoms relative to the no SOR group ( $ps \leq .001$ ) but did not significantly differ from one another ( $p = .35$ ). Mild SOR was associated with greater ODD symptoms relative to both no SOR and severe SOR ( $ps \leq .02$ ), which did not significantly differ from one another ( $p = .65$ ). Moreover, severe SOR was negatively associated with CD symptoms relative to no SOR ( $p < .0001$ ). No associations between SOR and psychosis symptoms survived inclusion of autism trait covariates.

In models testing the specificity of associations between SOR and psychiatric symptoms, both mild and severe SOR were positively associated with symptoms of anxiety disorders ( $ps < .0001$ ) and depression ( $ps < .05$ ) and negatively associated with symptoms of CD ( $ps < .0001$ ) (Table S4). Mild SOR was also positively associated with ODD symptoms ( $p = .02$ ).

### Longitudinal Clinical Results

Relative to no SOR, both mild ( $p < .0001$ ) and severe ( $p = .04$ ) SOR positively predicted symptoms of anxiety disorders 1 year later, controlling for concurrent psychiatric symptoms and autistic traits (Table S5). Mild SOR predicted subsequent symptoms of ADHD ( $p = .01$ ) relative to no SOR, whereas

**Table 2. The Majority of Sample Participants With Clinically Significant Psychiatric Symptoms Exhibit SOR**

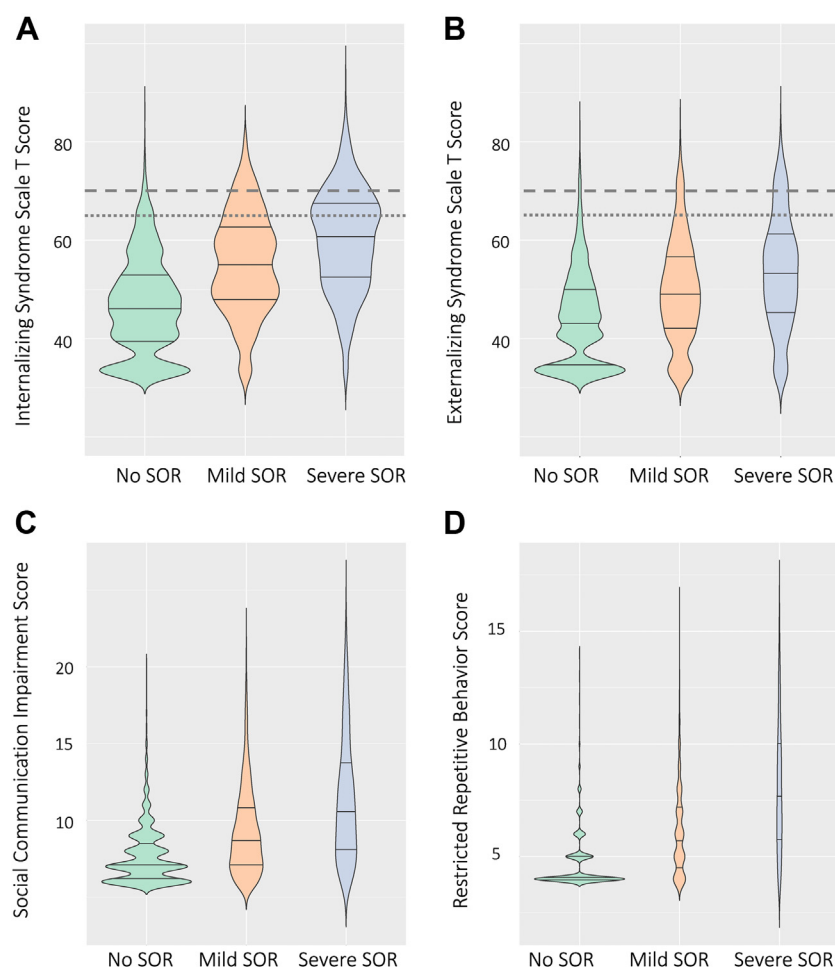
Children by SOR Group	Total Sample, $N = 11,205$	Children in Clinical Range (Internalizing), $n = 384$	Children in Clinical Range (Externalizing), $n = 272$	Children in Clinical Range (Internalizing or Externalizing), $n = 556$
Children in No SOR Group	9159 (81.7%)	133 (34.6%)	131 (48.2%)	239 (43.0%)
Children in Mild SOR Group	1389 (12.4%)	129 (33.6%)	71 (26.1%)	163 (29.3%)
Children in Severe SOR Group	657 (5.9%)	122 (31.8%)	70 (25.7%)	154 (27.7%)
Children in Mild or Severe SOR Group	2046 (18.3%)	251 (65.4%)	141 (51.8%)	317 (57.0%)

Values are presented as  $n$  (%). The total Y1 sample with Y1 CBCL scores and the subsets of sample participants with Y1 internalizing or externalizing scores in the clinical range are shown, broken down by SOR group. Y1 CBCL T scores for internalizing problems and externalizing problems were used to identify symptoms in the clinical range based on the criterion  $T \geq 70$ .

CBCL, Child Behavior Checklist; SOR, sensory over-responsivity; Y, year.



## Sensory Over-responsivity in Childhood



**Figure 1.** Symptom distributions reveal elevated psychopathology in children with sensory over-responsivity (SOR). Distribution of dimensional psychopathology scores by SOR group at year 1 (ages 10–11 years). Violin plots depict distributions of dimensional psychopathology T scores from the Child Behavior Checklist for the no SOR, mild SOR, and severe SOR groups. The 25th, 50th, and 75th percentiles of each group are depicted in horizontal black lines. Children with mild or severe SOR tended to score higher than children with no SOR on both (A) internalizing symptoms and (B) externalizing symptoms. Scores between the gray dotted and dashed lines are considered in the borderline clinical range, whereas scores above the dashed gray line are considered in the clinical range. Children with mild or severe SOR also tended to score higher on measures of autistic traits, specifically (C) social communication impairment and (D) restricted, repetitive behavior.

severe SOR predicted increases in psychosis symptoms relative to both no SOR ( $p = .049$ ) and mild SOR ( $p = .03$ ). No significant associations between SOR and subsequent depression, OCD, ODD, or CD symptoms survived inclusion of autism trait covariates.

### FC Results

We used an exploratory approach to identify FC pairs associated with severe SOR in the Y0 resting-state functional MRI dataset. Of the 161 FC pairs tested, 21 met selection criteria (Table S6) and were preregistered for independent testing in the Y2 dataset; of the 21 pairs, 17 met preregistered validation criteria. Of these, 15 survived more stringent post hoc analyses that included sex, economic disadvantage, age, internalizing and externalizing symptoms, and autistic traits as covariates (Figure 2; Table S7).

Among corticocortical FC pairs, severe SOR was associated with reduced FC within the sensorimotor hand network and between the sensorimotor hand and mouth networks. It was also associated with stronger sensorimotor hand-salience FC and within-ventral attention network FC. Among identified corticosubcortical FC pairs, nearly all exhibited a positive

relation with SOR, reflecting greater FC in children with severe SOR. This included increased FC between the cingulo-opercular network and the right and left amygdalae, between the sensorimotor hand network and several subcortical structures (i.e., right hippocampus, left cerebellum, right caudate), and between the right and/or left hippocampus and the cingulo-opercular, the sensorimotor mouth, and visual networks. A notable exception is that children with severe SOR exhibited reduced FC between the sensorimotor hand network and left hippocampus in both datasets.

### DISCUSSION

This work, which used a sample approximately 10 times larger than existing community studies of childhood SOR, provides new insights into the prevalence, clinical relevance, and neural bases of SOR in late childhood. Results indicate that SOR affects 18% of children overall, yet these individuals make up 57% of children with psychiatric symptoms in the clinical range. Both mild and severe SOR were significantly associated with greater psychiatric symptom burden and predicted subsequent increases in symptoms of anxiety disorders and other specific psychiatric conditions (i.e., ADHD and prodromal

**Table 3. Children With SOR Score Higher on Dimensional Measures of Concurrent Psychiatric Symptoms and Autistic Traits**

Symptom/Trait Scores at Y1	No SOR, Mean (SD)	Mild SOR, Mean (SD)	Glass's $\Delta$ No SOR vs. Mild SOR	Severe SOR, Mean (SD)	Glass's $\Delta$ No SOR vs. Severe SOR
<b>Summary Scores</b>					
Autistic traits	12.3 (2.7)	15.4 (4.4)	1.1	19.4 (6.4)	2.6
Internalizing symptoms	4.1 (4.5)	8.4 (6.6)	1.0	11.8 (8.1)	1.7
Externalizing symptoms	3.5 (4.9)	6.4 (6.8)	0.6	9.1 (8.4)	1.1
Prodromal psychosis symptoms	1.8 (3.1)	2.3 (3.5)	0.2	2.5 (3.8)	0.2
<b>Specific Scores</b>					
Restricted, repetitive behavior	4.7 (1.2)	6.1 (2.0)	1.2	8.1 (2.9)	2.8
Social communication impairment	7.7 (1.9)	9.3 (2.8)	0.8	11.3 (4.0)	1.9
Depressive problems	1.1 (1.7)	2.5 (2.8)	0.8	3.9 (3.5)	1.7
Anxiety problems	1.7 (2.1)	3.5 (2.8)	0.9	4.9 (3.5)	1.6
Obsessive-compulsive problems	1.1 (1.5)	2.3 (2.2)	0.8	3.5 (2.9)	1.6
ADHD problems	2.0 (2.6)	3.8 (3.2)	0.7	5.4 (3.6)	1.3
Oppositional defiant problems	1.5 (1.8)	2.5 (2.2)	0.6	3.3 (2.7)	1.0
Conduct problems	1.0 (2.0)	1.8 (2.8)	0.4	2.5 (3.4)	0.8

For each measure, mean and SD are provided by SOR group, and effect sizes (Glass's  $\Delta$ ) are provided for the mild and severe SOR groups, each relative to the no SOR group. All measures reflect raw scores.

ADHD, attention-deficit/hyperactivity disorder; SOR, sensory over-responsivity; Y, year.

psychosis for mild and severe SOR, respectively) when controlling for concurrent symptoms. Analyses of network-level FC identified reliable FC differences associated with severe SOR across scans collected 2 years apart. Of these identified differences, more than half constituted altered FC of sensorimotor networks that support processing of tactile information.

In this community sample that excluded children with ASD requiring special schooling, 18% of children were identified as exhibiting SOR. This rate is similar to those from several smaller community samples conducted with younger children, which ranged from 14.5% to 21.2% (1,3,4,6). Although most existing studies with smaller community samples have found no significant effect of sex on SOR in children or adults (1,4,6,29,30,67), this study revealed a small but significant effect of sex, such that sex ratios of children with SOR were disproportionately male skewed. Prior studies have also found that SOR is positively associated with other known early-life risk factors such as exposure to poverty and premature birth (1,2,68). Here, we found that severe SOR disproportionately affects children from families facing economic hardship, implicating a role for environmental factors.

These results can inform the debate over SOR as a construct and its relation to psychiatric conditions. Notably, findings are not consistent with either the dysregulation account or the autism account. Whereas emotion dysregulation is associated with virtually all forms of psychopathology (24), the current study demonstrated differential associations between SOR and psychiatric symptoms, including positive associations with symptoms of certain disorders (e.g., anxiety disorders) and negative associations with others (e.g., CD). Although we found large differences in autistic traits between SOR groups, SOR remained significantly associated with psychiatric symptoms after controlling for autistic traits. In addition, SOR predicted increases in anxiety symptoms when controlling for both psychiatric symptoms and autistic traits. Taken together, these results provide strong support for a sensory-specific account.

Although many studies have sought to characterize FC differences in autistic children or children with psychiatric conditions relative to typically developing control subjects, few have investigated FC differences related specifically to SOR. Analyses in this study identified 15 network-level FC differences that were reliably associated with severe SOR across datasets collected 2 years apart and survived inclusion of covariates for autistic traits and psychiatric symptoms. Notably, these included reduced FC within and between sensorimotor networks in children with SOR, which may reflect reduced coordination among brain areas that support tactile processing. Analyses also highlighted increased FC between the sensorimotor hand and salience networks and within the ventral attention network in children with SOR. The salience network is believed to support direction of attention toward behaviorally relevant stimuli (69), whereas the ventral attention network is thought to promote automatic, stimulus-driven orienting of attention (70). Both enhanced salience-sensorimotor FC and enhanced FC within the ventral attention network may promote the atypical allocation of attention toward innocuous or irrelevant sensory stimuli that characterizes SOR.

Analysis of FC between cortical networks and subcortical structures identified 11 FC differences associated with SOR, nearly all of which exhibited enhanced FC in children with severe SOR. Eight of these included either the sensorimotor hand network, which supports tactile processing, or the cingulo-opercular network, which is believed to support conflict or error detection and task set maintenance (71,72). In particular, associations between SOR and increased FC of the cingulo-opercular network and bilateral amygdalae might promote atypical error signaling and not just-right sensory experiences (73). In addition, 5 of the identified FC pairs associated with SOR were between the right or left hippocampi and sensory networks. In light of evidence that the hippocampus supports sensory prediction and modulation of activity in the sensory cortex based on past experience (74,75),

**Figure 2.** Functional connectivity pairs specifically associated with severe sensory over-responsivity (SOR). Matrices depict **(A)** cortical network-cortical network and **(B)** cortical network-subcortical structure pairs between which functional connectivity is significantly associated with SOR status in analyses controlling for autistic traits, psychiatric symptoms, sex, age, economic disadvantage, family, and scanner. Positive associations with SOR are indicated in green. Negative associations with SOR are indicated in red with a dotted texture.

Another limitation arises from our conservative approach to identifying FC differences associated with SOR across 2 time points. Although this approach should improve the replicability of our findings, it prevents us from characterizing changes in FC-SOR associations over time. Finally, the ABCD Study is not specifically designed to recruit, test, and scan children with SOR. Children with auditory SOR might have declined to participate or dropped out prematurely because of discomfort from repetitive scanner noise and therefore may be underrepresented in this sample.

Despite its limitations, this study demonstrates that SOR is common in late childhood, is found in most children with elevated psychiatric symptoms, provides unique information about psychiatric risk, and is associated with differences in brain networks that subserve tactile processing. These results support a sensory-specific account and implicate a neural basis for sensory differences in affected children, highlighting candidate neurocognitive targets for therapeutic intervention. Taken together, the findings suggest that SOR is a clinically relevant marker for childhood psychiatric illness warranting greater attention from both researchers and clinicians.

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## ARTICLE INFORMATION

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## Sensory Over-responsivity in Childhood

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