Supplemental Analyses and Results

Missing Subject Analysis

A portion of the current sample is missing data for the emotion recognition memory task (n = 1070; i.e., 22.38% of the current sample). This rate of data loss is likely due to the timing of the emotion recognition memory task. The emotion recognition memory task is administered after a 90-minute neuroimaging protocol and therefore is not administered to participants who either refused to complete the entire neuroimaging protocol, or who refused to continue the session after the prolonged neuroimaging sequence due to discomfort or fatigue. We ran a binary logistic regression model with task completion as the outcome variable and CD symptomatology, sex, race, age, and data collection site as simultaneous predictors to examine if task completion varied by these predictors.

Rates of CD symptomatology, our primary predictor of interest, did not meaningfully differ among those who completed and those who did not, $\beta = -.056$, 95% CI [-.1248 .0125]. However, there were effects for the covariates of non-interest (e.g., demographic factors): sex, $\beta = .216$, 95% CI [.0810 .3536], where female participants were more likely to have emotion recognition task data than male participants; age, $\beta = .139$, 95% CI [.0687 .2108], where older participants were more likely to have emotion recognition task data than where emotion recognition task data than younger participants; and, race, $\beta = .224$, 95% CI [.0755 .3791], where non-white participants were more likely to have emotion recognition task data than white participants. Additionally, there was one data collection site with a higher rate of available emotion recognition task data, $\beta = 1.144$, 95% CI [.0832 1.971], however no other sites meaningfully differed in their rates of available emotion recognition task data (β s ranged from -.769 to .911).

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NIH Toolbox Task Scores

While our *a priori* global hypotheses focus on examining domain-general neurocognitive functioning (as measured by the NIH Toolbox cognition battery total cognition corrected Tscore), it is possible that CD symptomatology is related to more specific aspects of neurocognitive functioning. For the sake of completeness, we ran zero-order correlation and partial correlation analyses examining the relationships among CD symptomatology and the individual NIH Toolbox cognition battery task and composite scores (see Table S1 for descriptive statistics and Tables S2 and S3 for the correlation findings).

Cortical Network Nodes

While our *a priori* node-level analyses focused on examining the relationships between CD symptomatology and subcortical structures, we also completed exploratory analyses examining the impact of CD symptomatology on node-level metrics for each cortical network. These exploratory analyses used the same regression models described for the main node-level analysis (see *Methods: Data Analysis: Node-Level Analysis*). Results from these analyses revealed that CD symptomatology was not meaningfully related to any node-level metrics for cortical network nodes. The complete list of CD-related effects from this exploratory analysis can be found in Table S4.

CD Diagnosis

To investigate whether our CD symptomatology-related effects replicated at a diagnostic group-level, we sorted participants into two groups: youth who met *DSM-5* diagnostic criteria for CD based on the Kiddie Schedule for Affective Disorders and Schizophrenia for school-age children (K-SADS-PL; Kaufman et al., 2013) (CD group; n = 219) and youth who did not

endorse symptoms of psychopathology ("healthy controls" [HC] group; specific criteria for participants classified as HC were taken from Waller et al., [2020]; n = 288). Youth who did not meet criteria for either the CD or HC groups were excluded from these analyses. Then we reran all regression analyses (see *Methods: Data Analysis*) using a group-level CD variable (dichotomously coded, CD vs. HC) as our primary predictor of interest.

Global Analysis.

Global Graph Analysis. The group-level analysis replicated our main global analysis finding that CD is associated with higher global clustering coefficients, F(5, 501) = 3.738, p =.002, $\beta = .117, 98.75\%$ CI [.0020 .2346]. Similarly, as in our main analysis, CD was not related to any other global differences after correcting for multiple comparisons (Degree_{max}, F(5, 501) =2.530, $p = .028, \beta = .087, 98.75\%$ CI [-.0348 .2029]; $BC_{max}, F(5, 501) = 4.085, p = .001, \beta =$.098, 98.75% CI [-.0180 .2156]; efficiency_{global}, $F(5, 501) = 1.868, p = .098, \beta = -.051, 98.75\%$ CI [-.1685 .0668]).

Neurocognitive Functioning. Similar to our main analysis, this grouped analysis replicated that CD was associated with generally lower neurocognitive functioning as measured by the NIH Toolbox cognition battery corrected cognition total composite T-score, F(5, 454) = 9.783, p < .001, $\beta = -.300$, 95% CI [-.3928 -.2070].

Node-Level Analysis.

Node-Level Metrics: Subcortical. The group-level analysis replicated that CD was associated with lower Degree_{subcortical}, but only prior to correction, F(5, 501) = 7.218, p < .001, $\beta = -.091$, 95% CI [-.1830 -.0048], 98.33% CI [-.2024 .0156]. This failure to detect a corrected CD effect may be due to the substantial drop in power from the full sample (n = 4781) to the

subsample (n = 507) used in the grouped analysis. Additionally, CD was not associated with $BC_{subcortical}$, F(5, 501) = 2.526, p = .028, $\beta = .072$, 98.33% CI [-.0367 .1873], or local efficiency_{subcortical}, F(5, 501) = 3.671, p = .003, $\beta = .087$, 98.33% CI [-.0109 .2035], in the grouped analysis.

Emotion Recognition Memory. This grouped analysis replicated the main effect of CD on task performance where youth with CD displayed worse performance on the emotion recognition memory task across all three face stimuli, F(1, 362) = 8.525, p = .004, $\beta = -.233$, 95% CI [-.3356 -.1313]. Also, consistent with our main analysis, there was not a CD X Facial expression interaction, F(2, 724) = 0.135, p = .874.

Demographic Interactions with CD Symptomatology

Prior research has demonstrated that the neurocognitive underpinnings of CD may differ by sex (Decety, Yoder, & Lahey, 2015; Smaragdi et al., 2017) and race (Wiesner et al., 2015). All analyses (see *Methods: Data Analysis*) were rerun with a CD symptomatology X Sex interaction variable or a CD symptomatology X Race interaction variable included in the model.

CD Symptomatology X Sex Interactions.

Global Analysis.

Global Graph Analysis. There were no CD symptomatology X Sex interactions for any of the global graph theory metrics after controlling for multiple comparisons (βs ranged from -.061 through -.005).

Neurocognitive Functioning. There was a CD symptomatology X Sex interaction for the impact of CD symptomatology on neurocognitive functioning as measured by the NIH Toolbox

cognition battery, F(6, 4368) = 15.063, p < .001, $\beta = .074$, 95% CI [.0234 .1253]. Unpacking this interaction, higher levels of CD symptomatology were associated with lower performance on the NIH Toolbox cognition battery for both male, $\beta = -.127$, 95% CI [-.1666 -.0893], and female participants, $\beta = -.089$, 95% CI [-.1281 -.0497], however the effect was stronger in male participants.

Node-Level Analysis.

Node-Level Analysis: Subcortical. There were no CD symptomatology X Sex interactions for any of the node-level graph theory metrics after controlling for multiple comparisons (βs ranged from -.054 through .044).

Emotion Recognition Memory. Neither the CD symptomatology X Sex interaction, F(1, 3687) = 0.792, p = .374, nor the CD symptomatology X Sex X Facial expression interaction, F(2, 7374) = 2.013, p = .134, were meaningful.

CD Symptomatology X Race Interactions.

Global Analysis.

Global Graph Analysis. There were no CD symptomatology X Race interactions for any of the global graph theory metrics (βs ranged from -.035 through .008).

Neurocognitive Functioning. Race did not moderate the effect of CD symptomatology on neurocognitive functioning as measured by the NIH Toolbox cognition battery (CD symptomatology X Race interaction: $\beta = -.037$, 95% CI [-.0823 .0073]).

Node-Level Analysis.

Node-Level Analysis: Subcortical. There was a CD symptomatology X Race interaction on local efficiency_{subcortical}, F(6, 4774) = 5.237, p < .001, $\beta = .065$, 98.33% CI [.0077 .1350]. Unpacking this interaction, white participants do not show an association between CD symptomatology and local efficiency_{subcortical}, $\beta = -.009$, 95% CI [-.0291 .0144]. In contrast, higher CD symptomatology was related to higher local efficiency_{subcortical} in non-white participants, $\beta = .068$, 95% CI [.0163 .1336], suggesting that there may be a relationship between CD symptomatology and heightened local efficiency_{subcortical} unique to non-white youth, possibly reflecting differences in structural societal factors that often differentiate white and non-white youth in the United States and that can impact brain functioning and structure (Freedman & Woods, 2013; Kim, Evans, Chen, Miller, & Seeman, 2018). Race did not moderate the effect of CD symptomatology on either Degree_{subcortical}, F(6, 4774) = 24.439, p < .001, $\beta = .037$, 98.33% CI [-.0974 .0178], or *BC*_{subcortical}, F(6, 4774) = 2.551, p = .018, $\beta = .023$, 98.33% CI [-.0455 .0958].

Emotion Recognition Memory. Neither the CD symptomatology X Race interaction, F(1, 3687) = 0.981, p = .322, nor the CD symptomatology X Race X Facial expression interaction, F(2, 7374) = 0.015, p = .985, were meaningful.

Uniqueness to CD

While the current findings demonstrate that CD is related to various neural and neurocognitive abnormalities, it is possible that these neurocognitive differences are not unique to CD but rather shared across multiple externalizing psychopathologies. To assess whether these effects also are present in other externalizing psychopathologies, we reran all of our regression analyses (see *Methods: Data Analysis*) two times, once with oppositional defiance disorder

(ODD) symptomatology (as measured by the K-SADS-PL)¹ as the primary predictor of interest, and once with attention deficit hyperactivity disorder (ADHD) symptomatology (as measured by the K-SADS-PL)² as the primary predictor of interest. In both cases, CD symptomatology was removed as a factor from the models.

ODD Effects

Global Analysis.

Global Graph Analysis. Unlike CD, ODD symptomatology was not related to global clustering, F(5, 4394) = 9.931, p < .001, $\beta = .011$, 98.75% CI [-.0251 .0491]. Additionally, ODD symptomatology was not related to global Degree_{max}, F(5, 4394) = 6.962, p < .001, $\beta = .029$, 98.75% CI [-.0115 .0698], global BC_{max} , F(5, 4394) = 9.105, p < .001, $\beta = .011$, 98.75% CI [-.0262 0.0490], or global efficiency, F(5, 4394) = 4.430, p = .001, $\beta = .005$, 98.75% CI [-.0324 .0436].

Neurocognitive Functioning. Similar to CD, higher ODD symptomatology was related to generally impaired neurocognitive functioning as measured by the NIH Toolbox cognition battery corrected cognition total composite T-score, F(5, 4007) = 8.199, p < .001, $\beta = -.052$, 95% CI [-.0846 -.0205].

Node-Level Analysis.

Node-Level Metrics: Subcortical. In contrast with CD, ODD symptomatology was not related to Degree_{subcortical}, F(5, 4394) = 22.192, p < .001, $\beta = -.025$, 98.33% CI [-.0623 .0120].

¹ 381 participants were missing data for ODD symptomatology and were excluded from any analyses using ODD symptomatology as a variable of interest.

² 454 participants were missing data for ADHD symptomatology and were excluded from any analyses using ADHD symptomatology as a variable of interest.

Similarly, ODD symptomatology was not related to $BC_{subcortical}$, F(5, 4394) = 1.701, p = .131, $\beta = .019$, 98.33% CI [-.0211 .0660], or subcortical efficiency, F(5, 4394) = 3.186, p = .007, $\beta = .001$, 98.33% CI [-.0336 .0417].

Emotion Recognition Memory. Similar to CD, there was a main effect of ODD symptomatology on task performance where youth higher on ODD symptomatology generally performed worse on the emotion recognition memory task across all three face stimuli conditions, F(1, 3405) = 5.467, p = .019, $\beta = -.040$ 95% CI [-.0733 -.0068]. Also, consistent with the CD findings, there was not a meaningful ODD X Facial expression interaction, F(2, 6810) = 0.329, p = .720.

ADHD Effects.

Global Analysis.

Global Graph Analysis. In contrast to CD, ADHD symptomatology was not related to global clustering coefficient, F(5, 4321) = 11.137, p < .001, $\beta = .016$, 98.75% CI [-.0244 .0565]. However, higher ADHD symptomatology was related to higher global Degree_{max}, F(5, 4321) = 8.865, p < .001, $\beta = .044$, 98.75% CI [.0014 .0843] and higher global BC_{max} , F(5, 4321) = 11.104, p < .001, $\beta = .039$, 98.75% CI [.0011 .0776], indicating that, unlike youth higher on CD and/or ODD symptomatology, youth higher on ADHD symptomatology exhibit larger, largest hubs in terms of both hub size (i.e., number of direct connections to the largest hub) and hub centrality in the global flow of information throughout the brain. ADHD symptomatology was not related to global efficiency, F(5, 4321) = 5.379, p < .001, $\beta = .003$, 98.75% CI [-.0365 .0440].

Neurocognitive Functioning. Similar to CD and ODD, higher ADHD symptomatology was related to a general impairment in neurocognitive functioning as measured by the NIH Toolbox cognition battery corrected cognition total composite T-score, F(5, 3938) = 17.213, p < .001, $\beta = -.114$, 95% CI [-.1450 -.0829].

Node-Level Analysis.

Node-Level Metrics: Subcortical. In contrast with CD, ADHD symptomatology did not relate to Degree_{subcortical}, F(5, 4321) = 25.171, p < .001, $\beta = -.035$, 98.33% CI [-.0732 .0025]. Additionally, ADHD symptomatology was not related to $BC_{subcortical}$, F(5, 4321) = 1.629, p = .149, $\beta = .004$, 98.33% CI [-.0311 .0459], or local efficiency_{subcortical}, F(5, 4321) = 3.275, p = .006, $\beta = .014$, 98.33% CI [-.0241 .0618].

Emotion Recognition Memory. Similar to CD and ODD, there was a main effect of ADHD symptomatology on emotion recognition memory task performance where youth higher on ADHD symptomatology displayed worse performance on the emotion recognition memory task across all three face stimuli conditions, F(1, 3355) = 11.174, p = .001, $\beta = -.064$, 95% CI [-.0954 -.0329]. Also, consistent with the CD and ODD findings, there was not a meaningful ADHD X Facial expression interaction, F(2, 6710) = 0.415, p = .660.

Comorbidities.

Given the high comorbidities across externalizing disorders such as CD, ODD, and ADHD, and the correlations among these phenotypes (correlation between CD symptomatology and ODD symptomatology in the current sample is r = .421, 95% CI [.3775 .4614]; correlation between CD symptomatology and ADHD symptomatology in the current sample is r = .363, 95% CI [.3196 .4040]), it is possible that either ODD or ADHD symptomatology may be

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confounding our CD-related findings. To help ensure our main findings are not being confounded by comorbid externalizing psychopathologies, we ran linear regression models examining our main findings (i.e., the main effect of CD symptomatology on: global clustering, NIH Toolbox cognition battery total cognition score, Degree_{subcortical}, and emotional recognition memory task performance) including CD, ODD, and ADHD symptomatology as simultaneous regressors. The same covariates of non-interest from the main analyses (see *Methods: Data Analysis*) also were included in these models.

Global Analysis.

Global Graph Analysis. The effect of CD symptomatology on global clustering coefficient remained present, F(7, 4188) = 7.997, p < .001, $\beta = .045$, 95% CI [.0117 .0808], even when controlling for ODD and ADHD symptomatology. In contrast, neither ODD, $\beta = -.007$, 95% CI [-.0437 .0290], nor ADHD symptomatology related to global clustering, $\beta = .001$, 95% CI [-.0351 .0378].

Neurocognitive Functioning. The association between higher CD symptomatology and impaired neurocognitive functioning did not meaningfully change when ODD and ADHD symptomatology were included in the model, F(7, 3811) = 14.584, p < .001, $\beta = -.089$, 95% CI [-.1214 -.0568]. Additionally, higher ADHD symptomatology remained related to general neurocognitive impairment, even when controlling for CD and ODD, $\beta = -.097$, 95% CI [-.1340 - .0590]; however, ODD symptomatology did not meaningfully relate to neurocognitive impairment when controlling for other externalizing psychopathologies, $\beta = .033$, 95% CI [-.0043 .0722].

Node-Level Analysis.

Node-Level Metrics: Subcortical. Including ODD and ADHD symptomatology in the model did not meaningfully change the relationship between CD symptomatology and Degree_{subcortical}, F(7, 4188) = 17.148, p < .001, $\beta = .042$, 95% CI [-.0794 -.0077]. In contrast, neither ODD, $\beta = .011$, 95% CI [-.0252 .0468], nor ADHD symptomatology related to Degree_{subcortical}, $\beta = -.023$, 95% CI [-.0585 .0125].

Emotion Recognition Memory. The relationship between CD symptomatology and impaired emotion recognition memory task performance did not meaningfully change when ODD and ADHD were included in the model, F(7, 3271) = 12.044, p < .001, $\beta = -.042$, 95% CI [-.0797 -.0030]. Similarly, higher ADHD symptomatology still related to worse emotion recognition memory task performance, $\beta = -.049$, 95% CI [-.0877 -.0118], even when controlling for CD and ODD. However, the unique variance of ODD symptomatology did not appear to relate to emotion recognition memory task performance, $\beta = -.002$, 95% CI [-.0425 .0385].

Socioeconomic Status

Prior research indicates that CD may be related to socioeconomic status (SES; Loeber, Green, Keenan, & Lahey, 1995). Moreover, given the modest correlation between CD symptomatology and SES (as measured by self-reported minimum household income divided by number of household members; McLoyd, 1998)³ in the current sample (r = -.129, 95% CI [-.1532 -.1029]), it is possible that SES may be confounding our CD-related findings. To help ensure our main findings are not being confounded by SES, we ran linear regression models examining our four main findings (i.e., the main effect of CD symptomatology on: global clustering, NIH Toolbox cognition battery total cognition score, subcortical degree, and

³ 482 participants were missing data for SES were excluded from any analyses using SES as a variable of interest.

emotional recognition memory task performance) including CD symptomatology as our regressor of interest, the covariates of non-interest used in the main analyses (see *Methods: Data Analysis*), and SES as an additional covariate of non-interest.

Global Analysis.

Global Graph Analysis. The effect of CD symptomatology on global clustering coefficient remained present when controlling for SES, F(6, 4278) = 10.228, p < .001, $\beta = .032$, 95% CI [.0016 .0641].

Neurocognitive Functioning. CD symptomatology remained linked with general neurocognitive impairments even when controlling for SES, F(6, 3915) = 23.138, p < .001, $\beta = -$.087, 95% CI [-.1164 -.0572].

Node-Level Analysis.

Node-Level Metrics: Subcortical. Controlling for SES did not meaningfully impact the link between CD symptomatology and decreased Degree_{subcortical}, F(6, 4278) = 21.087, p < .001, $\beta = -.046$, 95% CI [-.0818 -.0132].

Emotion Recognition Memory. Including SES in the model did not meaningfully change the relationship between CD and reduced performance on the emotion recognition memory task, $F(6, 3350) = 21.087, p < .001, \beta = -.044, 95\%$ CI [-.0752 -.0118].

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Supplementary Tables

	Whole Sample $(N = 4781)$				HC $(N = 288)$				CD (N = 219)						
Variable	n	Min	Max	Mean	Std. Dev	n	Min	Max	Mean	Std. Dev	n	Min	Max	Mean	Std. Dev
Picture Vocab.	4481	16	131	52.54	10.97	262	26	101	53.97	10.81	208	20	81	49.76	9.81
Flanker	4480	10	101	46.42	9.21	262	17	82	47.39	9.06	209	16	81	45.35	9.58
List Sorting	4473	14	90	49.52	9.76	262	29	78	51.64	9.41	208	18	73	47.42	9.45
Card Sorting	4483	26	100	47.49	9.65	262	29	86	49.03	10.25	209	28	86	45.97	9.63
Pattern Comp.	4476	-5	100	45.16	14.46	262	-1	100	47.27	15.40	209	10	81	42.00	13.41
Picture Sequence	4481	15	96	49.47	10.87	262	28	86	51.07	10.77	209	22	81	46.95	9.04
Oral Reading	4479	27	122	49.48	11.48	262	33	95	51.04	10.90	209	30	109	47.13	11.67
Fluid Cog.	4378	12	105	45.89	11.10	252	19	92	48.78	11.09	208	14	75	42.68	10.14
Crystallized Cog.	4391	20	120	51.12	11.20	252	31	102	52.97	10.76	209	27	99	48.18	11.08
Total Cog.	4375	13	126	47.91	11.13	252	29	88	50.83	10.72	208	20	79	44.08	10.71

Table S1. Descriptive Statistics for the NIH Toolbox Cognition Battery

HC = Participants classified as "healthy controls" by the criteria put forth in Waller et al., (2020); CD = Participants who met diagnostic criteria for conduct disorder as assessed by the Kiddie Schedule for Affective Disorders and Schizophrenia for school-age children (Kaufman et al., 2013); NIH Toolbox = National Institutes of Health (NIH) Toolbox cognition battery; Picture Vocab = NIH Toolbox Picture Vocabulary Test, Fully-Corrected T-score; Flanker = NIH Toolbox Flanker Inhibitory Control and Attention Test, Fully-Corrected T-score; List Sorting = NIH Toolbox List Sorting Working Memory Test, Fully-Corrected T-score; Card Sorting = NIH Toolbox Dimensional Change Card Sort Test, Fully-Corrected T-score; Pattern Comp = NIH Toolbox Pattern Comparison Processing Speed Test, Fully-Corrected T-score; Picture Sequence = NIH Toolbox Picture Sequence Memory Test, Fully-Corrected T-score; Oral Reading = NIH Toolbox Oral Reading Recognition Test, Fully-Corrected T-score; Fluid Cog = NIH Toolbox Fluid Cognition Composite, Fully-Corrected T-score; Total Cog = NIH Toolbox Total Cognition Composite, Fully-Corrected T-score.

					Zero-	Order Co	rrelation	IS			
Variable	1	2	3	4	5	6	7	8	9	10	11
Clinical Variable											
1. CD Symptoms	—	07*	03	08*	06*	06*	06*	08*	10*	08*	11*
NIH Toolbox											
2. Picture Vocab.		—	.18*	.25*	.15*	.09*	.15*	.43*	.25*	.83*	.67*
3. Flanker			—	.18*	.37*	.32*	.17*	.18*	.62*	.21*	.51*
4. List Sorting					.22*	.14*	.28*	.30*	.55*	.32*	.53*
5. Card Sorting						.39*	.19*	.17*	.68*	.19*	.53*
6. Pattern Comp							.15*	.12*	.71*	.12*	.51*
7. Picture Sequence								.17*	.56*	.19*	.46*
8. Oral Reading								_	.29*	.85*	.70*
9. Fluid Cog.										.32*	.81*
10. Crystallized Cog.											.81*
11. Total Cog.											

Table S2. Zero-Order Correlations for NIH Toolbox Cognition Battery Scores

Correlation analyses were limited to participants with complete NIH Toolbox data (n = 4368). Bootstrapped CIs (5000 samples) were used to assess the meaningfulness of the correlations. To correct for multiple comparisons, 99.5% CIs were evaluated to match Bonferroni corrected p-values (1-[.05/10] = .995). CD Symptoms = Number of conduct disorder symptoms present as assessed by the Kiddie Schedule for Affective Disorders and Schizophrenia for school-age children (Kaufman et al., 2013); NIH Toolbox = National Institutes of Health (NIH) Toolbox cognition battery; Picture Vocab = NIH Toolbox Picture Vocabulary Test, Fully-Corrected T-score; Flanker = NIH Toolbox Flanker Inhibitory Control and Attention Test, Fully-Corrected T-score; List Sorting = NIH Toolbox List Sorting Working Memory Test, Fully-Corrected T-score; Card Sorting = NIH Toolbox Dimensional Change Card Sort Test, Fully-Corrected T-score; Pattern Comp = NIH Toolbox Pattern Comparison Processing Speed Test, Fully-Corrected T-score; Picture Sequence = NIH Toolbox Picture Sequence Memory Test, Fully-Corrected T-score; Oral Reading = NIH Toolbox Oral Reading Recognition Test, Fully-Corrected T-score; Fluid Cog = NIH Toolbox Fluid Cognition Composite, Fully-Corrected T-score; Crystallized Cog = NIH Toolbox Crystallized Cognition Composite, Fully-Corrected T-score; Total Cog = NIH Toolbox Total Cognition Composite, Fully-Corrected T-score.

* 99.5% Bootstrapped CI (5000 samples) does not contain 0.

		Partial Correlations									
Variable	1	2	3	4	5	6	7	8	9	10	11
Clinical Variable											
1. CD Symptoms	—	06**	03*	08**	06**	06**	06**	07**	09**	07**	10**
NIH Toolbox											
2. Picture Vocab.		—	.18**	.26**	.14**	.09**	.15**	.42**	.25**	.83**	.67**
3. Flanker			—	.18**	.37**	.32**	.17**	.18**	.62**	.21**	.51**
4. List Sorting				—	.22**	.15**	.28**	.30**	.55**	.33**	.54**
5. Card Sorting					—	.39**	.19**	.17**	.68**	.18**	.53**
6. Pattern Comp						—	.15**	.12**	.71**	.12**	.51**
7. Picture Sequence								.17**	.56**	.19**	.46**
8. Oral Reading									.29**	.85**	.70**
9. Fluid Cog.									_	.32**	.81**
10. Crystallized Cog.										—	.81**
11. Total Cog.											—

Table S3. Partial Correlations for NIH Toolbox Cognition Battery Scores

Correlation analyses were limited to participants with complete NIH Toolbox data (n = 4368). Partial correlations controlled for race (dichotomously coded, white vs. non-white), sex (dichotomously coded, female vs. male), age, and site effects. Bootstrapped CIs (5000 samples) were used to assess the meaningfulness of the correlations. To correct for multiple comparisons, 99.5% CIs were evaluated to match Bonferroni corrected p-values (1-[.05/10] = .995). CD Symptoms = number of conduct disorder symptoms present as assessed by the Kiddie Schedule for Affective Disorders and Schizophrenia for school-age children (Kaufman et al., 2013); NIH Toolbox = National Institutes of Health (NIH) Toolbox cognition battery; Picture Vocab = NIH Toolbox Picture Vocabulary Test, Fully-Corrected T-score; Flanker = NIH Toolbox Flanker Inhibitory Control and Attention Test, Fully-Corrected T-score; List Sorting = NIH Toolbox List Sorting Working Memory Test, Fully-Corrected T-score; Card Sorting = NIH Toolbox Dimensional Change Card Sort Test, Fully-Corrected T-score; Picture Sequence = NIH Toolbox Picture Sequence Memory Test, Fully-Corrected T-score; Oral Reading = NIH Toolbox Oral Reading Recognition Test, Fully-Corrected T-score; Fluid Cog = NIH Toolbox Fluid Cognition Composite, Fully-Corrected T-score; Crystallized Cog = NIH Toolbox Crystallized Cognition Composite, Fully-Corrected T-score; Total Cog = NIH Toolbox Total Cognition Composite, Fully-Corrected T-score.

* 95% Bootstrapped CI (5000 samples) does not contain 0.

** 99.5% Bootstrapped CI (5000 samples) does not contain 0.

		Default Network						
		β	t	CI Lower	CI Upper			
Degree								
DC	CD Symptomatology	-0.003	-0.232	-0.0509	0.0417			
BC	CD Symptomatology	0.010	0.651	-0.0337	0.0609			
Efficiencylocal	CD Symptomatology	0.016	1.079	-0.0270	0.0771			
		Do	rsal Atte	ntion Netv	vork			
		β	t	CI Lower	CI Upper			
Degree								
	CD Symptomatology	-0.031	-2.108	-0.0837	0.0182			
BC	CD Commence to 1	0.000	0.550	0.0250	0.0(14			
Teraianan	CD Symptomatology	0.008	0.559	-0.0358	0.0614			
Efficiency _{local}	CD Symptomatology	0.035	2.414	-0.0168	0.1225			
		Fr	onto-Par	ietal Netw	Network			
		β	t	CI Lower	CI Upper			
Degree								
	CD Symptomatology	0.019	1.313	-0.0307	0.0637			
BC	CD Symptomatology	-0.004	-0.307	-0.0475	0.0392			
Efficiencylocal								
	CD Symptomatology	0.010	0.709	-0.0316	0.0695			
			Salience	e Network				
		β	t	CI Lower	CI Upper			
Degree								
D <i>G</i>	CD Symptomatology	0.001	0.068	-0.0531	0.0464			
BC	CD Symptomatology	0.014	0.942	-0.0237	0.0720			
Efficiency _{local}								
	CD Symptomatology	0.018	1.257	-0.0316	0.0695			
		Ven	tral Atte	ention Net	work			

Table S4: Exploratory Node-Level Analysis

Degree							
	CD Symptomatology	0.020	1.355	-0.0340	0.0715		
BC	CD Symptomatology	-0.006	-0.401	-0.0405	0.0394		
Efficiencylocal							
	CD Symptomatology	-0.005	-0.366	-0.0372	0.0471		
		Cing	gulo-Ope	rcular Net	twork		
		ß	t	CI	CI		
		Р	·	Lower	Upper		
Degree							
	CD Symptomatology	0.016	1.107	-0.0395	0.0685		
BC							
	CD Symptomatology	-0.008	-0.526	-0.0411	0.0425		
Efficiency _{local}							
	CD Symptomatology	0.014	0.949	-0.0327	0.1260		
		Cin	gulo-Pa	rietal Netv	vork		
		ß	+	CI	CI		
		þ	l	Lower	Upper		
Degree							
	CD Symptomatology	-0.031	-2.112	-0.0865	0.0224		
BC							
	CD Symptomatology	-0.003	-0.215	-0.0470	0.0453		
Efficiency _{local}							
•	CD Symptomatology	0.007	0.491	-0.0306	0.0862		
			Visual	Network			
				CI	CI		
		β	t	Lower	Upper		
Degree							
	CD Symptomatology	-0.015	1.062	-0.0680	0.0338		
BC							
	CD Symptomatology	0.000	0.026	-0.0470	0.0523		
Efficiency _{local}							
	CD Symptomatology	0.054	3.708	-0.0090	0.1249		
			Auditor	y Network	K		
				CI	CI		
		β	t	Lower	Upper		
Degree							
-	CD Symptomatology	0.034	2.353	-0.0149	0.0855		
BC							
	CD Symptomatology	0.013	0.869	-0.0395	0.0811		

Efficiency _{local}									
	CD Symptomatology	0.020	1.404	-0.0203	0.1206				
		Retrosplenial-Temporal Network							
		β	t	CI Lower	CI Upper				
Degree									
	CD Symptomatology	0.010	0.691	-0.0401	0.0569				
BC									
	CD Symptomatology	-0.003	-0.193	-0.0459	0.0532				
Efficiencylocal									
	CD Symptomatology	0.006	0.418	-0.0363	0.0685				
		Sen	sorimoto	or _{hand} Netv	vork				
		β	t	CI Lower	CI Unnor				
Degree				Lower	Opper				
Degree	CD Symptomatology	0.002	0.170	-0.0462	0.0525				
BC	5 1 05								
	CD Symptomatology	-0.003	-0.230	-0.0474	0.0512				
Efficiencylocal									
	CD Symptomatology	-0.007	-0.482	-0.0402	0.0418				
		Sen	sorimoto	rmouth Net	etwork				
		ß	t	CI	CI				
		4		Lower	Upper				
Degree	CD Commence and the large	0.017	1 150	0.0000	0.0429				
R <i>C</i>	CD Symptomatology	-0.017	-1.130	-0.0892	0.0438				
DC	CD Symptomatology	0.024	1 657	-0.0370	0 0994				
Ffficiency,	CD Symptomatology	0.024	1.007	0.0570	0.0774				
Efficiency local	CD Symptomatology	0.044	3.029	-0.0133	0.1828				
			"Other"	" Network					
				CI	CI				
		þ	t	Lower	Upper				
Degree									
	CD Symptomatology	0.004	0.251	-0.0557	0.0588				
BC		0.000	0 6 4 4	0.02/0	0.0702				
	CD Symptomatology	0.009	0.644	-0.0369	0.0693				
Efficiency _{local}	CD Com (1	0.010	1 200	0.02/0	0.0702				
	CD Symptomatology	0.018	1.206	-0.0369	0.0693				

CIs were 99.87% Bootstrapped CIs (5000 samples) to achieve the equivalent of Bonferroni correction for *p*-values when controlling for the 39 exploratory comparisons (1-[.05/39] = 0.9987).



Supplemental Figure

Figure S1. Distribution of CD symptomatology in the current sample. Figure S1 displays a histogram of the number of CD symptoms present in the current sample (as assessed as by K-SADS-PL).