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Supplementary information

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Limited generalizability of dynamic fMRI correlates of adolescent rumination

In the format provided by the authors and unedited

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Supplementary Note 1. Factors Influencing Rumination and Related Brain Circuitry

Individual-level factors that affect rumination, depression, and related brain circuitry have been identified in adolescents. Sex differences emerge during this period, with girls showing a greater increase in brooding compared to boys,[1] which may account for the greater increase in depression in girls relative to boys during the adolescent years.[2] Previous research found a sex-specific association between salience network (SN) coherence and ruminative brooding in girls, where increased SN coherence, driven by dorsal anterior cingulate cortex (dACC) connectivity within the SN, was linked to higher levels of rumination.[3] Throughout puberty, significant changes occur in brain organization and network connectivity, which are closely tied to age and pubertal status.[4,5] This developmental period is characterized by heightened neural plasticity compared to adults, with pubertal youth exhibiting more dynamic changes in functional connectivity patterns as their brains mature. As a result, experiences during this time lead to more pronounced changes in neural circuitry.[6]

Parental genetic factors may contribute to the development of rumination and psychopathology in adolescents. Rumination has been shown to be moderately heritable and demonstrates shared genetic variability with depressive symptoms.[7] Environmental influences, such as negative life events, also play a role in shaping ruminative tendencies during adolescence,[8] which highlights the complex interplay between genetic and environmental factors in adolescent brain development. Moreover, rumination is comorbid with other depressive symptoms such as anhedonia, and both have been found to be associated with disruptions in cortico-limbic circuitry.[9,10]

Supplementary Note 2. Network Properties of Temporal Poles

We examined static functional connectivity for all participants in **Studies 1-4** between the 20 DMN seeds and 280 Fan connectome regions and calculated network properties of the seeds to investigate whether there were special features of the temporal poles. First, the left and right temporal poles showed on average smaller degrees than other DMN seeds (M_L = 13.64, M_{Ave} = 29.59, $t(344) = -23.29$, $p < 0.001$) and (M_R =20.52, M_{Ave} = 29.59, $t(344) = -11.59$, $p < 0.001$). They showed the same pattern in terms of decreased betweenness. The right temporal pole showed higher clustering coefficients than average DMN seeds (M_R = 0.64, M_{Ave} = 0.57, $t(344) = 5.24$, $p < 0.001$). The left temporal pole showed lower local efficiency than average DMN seeds (M_L = 0.63, M_{Ave} = 0.72, $t(344) = -4.33$, $p < 0.001$).These results support the interpretation that the temporal poles are less connected and hub-like than the other DMN seeds and that the right temporal pole is especially locally connected and segregated.

Supplementary Note 3. External Validity Analyses

External validation

The external test dataset was harmonized using COMBAT, and then used to evaluate the selected random forest models (Left Temporal Pole, Right Temporal Pole, whole-brain). None were significant when examining the full distribution of rumination scores (ps > 0.35,

Supplementary Table 15), and there were no interactions with puberty (*p*s > 0.2). However, the external test dataset, **Study 5,** had considerably lower scores on the CRSQ (Welch's *t*(225.36) = -7.62, $p < 0.001$, $d = -0.70$). To account for this, we restricted analyses to CRSQ scores > 20, which corresponds to approximately 1 SD below the mean in the training set (it is reasonable to assume that the model fit from the training dataset is best for scores within 1 SD of the mean). We used 20 based on observations of the breakpoint in the distribution instead of the exact SD value. Given this admittedly arbitrary cut-off, the model showed a positive relationship that was similar in magnitude to the validation dataset ($r = 0.26$, $p = 0.064$).

More suggestive evidence of the value of the right temporal model comes from the non-harmonized dataset, where we found a significant ***negative*** relationship for the Right Temporal Pole model (**Supplementary Table 15)**, again suggesting that there is valuable information in that particular model but that factors like scanner, scan protocol, and population affect predictive performance. Differences between the external dataset and internal dataset connectivity distributions were assessed. Particularly notable were dynamic differences for highest positive importance regions (**Supplementary Fig. 6)**, which showed an average Cohen's $d = -0.45$ in the external dataset. For the significant networks (**Supplementary Fig. 7**), the DAN and Cerebellum showed even larger dynamic differences across datasets (*ds*= 0.68, 1.34, respectively), but the DMN was similar. Finally, static connectivity distributions were significantly different within these networks (**Supplementary Fig. 8)**. These results support the harmonization procedures here and suggest that improvements may be made in future work to account for static connectivity differences as well.

Supplementary Note 4. Deviations from Preregistration

- We added two additional datasets. One study, **Study 4,** is ongoing and similar in procedures to **Studies 1-3,** and we included the 71 adolescents in the training and validation datasets. During review, we added a completed external dataset consisting of 119 adolescents (99 after quality control) with CRSQ data, see **Study 5.**
- EMA responses were on different scales for **Study 1 & 2 vs Study 3 & 4,** so we z-scored within each dataset (maintaining test and train independence).
- We conducted puberty control analyses and conducted stratified training-test splits.
- We conducted an additional exploratory nonlinear analysis using seed-based analysis (DMN seeds), and upon reviewer request, a whole-brain nonlinear analysis.
- We did not conduct elastic net regression as it was deemed similar enough to the main lasso approach that it would not provide novel predictive power.
- We conducted connectome predictive modelling using the Brainnetome whole-brain atlas, not the Shen atlas, for comparability across methods.

Supplementary Note 5. Rumination Distributions

CRSQ scores were non-normally distributed across **all studies** ($W = 0.96$, $p < 0.001$) (**Extended Data Fig. 1** and **Supplementary Fig. 4**). EMA rumination scores were non-normally distributed across **Studies 1-4** ($W = 0.91$, $p < 0.001$), and not collected in **Study 5** (**Supplementary Fig. 1**).

Supplementary Note 6. fMRI Quality Assurance Check

Quality assurance (QA) checks were performed using the CONN Toolbox on all studies, to ensure data reliability. The preprocessing parameters were consistent with those employed by Kim et al., maintaining methodological consistency. Key metrics such as motion parameters, spatial normalization, and QC-FC were examined, and QA plots were constructed and reviewed to exclude any low-quality scans that could impact subsequent analyses.

Supplementary Note 7. Ecological Momentary Assessment (EMA) Sampling

For the **Study 1 & 2** datasets, we collected 2-3 EMA reports per day over 5 days. For **Study 3**, we collected EMA 4 times per day over 30 days. Finally, for **Study 4**, we collected EMA 3 times per day over 3 days. The differing timeframes reflect the different constraints of the studies – we sought to get EMA data that was relatively pure and free from intervention effects. In both studies, participants were first asked: "Think about the most stressful or negative thing that happened since you completed the last survey (or if this is your first survey, then the last 24 hours). At the worst point, how stressed did you feel?" and then asked the two rumination questions:

(1) "After this stressful or negative thing happened, I was dwelling on my mistakes, failures or losses",

(2)"After this stressful or negative thing happened, I kept thinking about something negative that happened."

Study 5 did not include EMA.

Supplementary Note 8. Scanner Protocols

Study 1-3 Collection

Images were collected on two separate scanners, one Siemens Tim Trio 3.0 Tesla MRI with a 32-channel coil (for a small subset of **Study 1 & 2**), and one Siemens PRISMA 3.0 Tesla MRI with a 64-channel coil (for remaining **Study 1 & 2** and all of **Study 3**). The protocol was the same across scanners and consisted of a T1-weighted scan, one 6.5-minute resting-state scan, and a single gradient-echo fieldmap acquisition. Functional images were acquired with a multiband sequence (TR = 720 ms, TE = 30 ms, FOV = 212 mm, multiband accelerator factor = 6, voxel size = 2.5 × 2.5 × 2.5).

Study 4 Collection

Images were collected using a Siemens Prisma 3.0 Tesla MRI equipped with a 64-channel coil. The protocol consisted of a T1-weighted scan, three 6-minute, eyes-open, resting-state scans, and a single gradient-echo fieldmap acquisition. Functional images were acquired with a multiband sequence (TR = 720 ms, TE = 30 ms, FOV = 212 mm, multiband accelerator factor = 6, voxel size = 2.5 × 2.5 × 2.5).

Study 5 Collection

Images were collected using a Siemens Tim Trio 3.0 Tesla MRI equipped with a 32-channel coil. The protocol consisted of a T1-weighted scan, one five-minute eyes open resting-state scan, and a single gradient-echo fieldmap acquisition. Functional images were acquired with a multiband sequence (TR = 1300 ms, TE = 32.2 ms, FOV = 212 mm, multiband accelerator factor = 8, voxel size = 2.0 × 2.0 × 2.0).

Supplementary Note 9. Connectome Predictive Modelling

This procedure is similar to that followed by previous CPM studies.[11,12] We calculated correlations between CRSQ and EMA and the whole-brain features. We then selected edges with correlations that were below $p = 0.01$, assembling them into positive and negative networks, respectively. The positive network is summed to calculate "positive network strength" and the negative network is summed to calculate "negative network strength," and they may be subtracted to calculate "overall strength." They then may be correlated with rumination scores in held-out data. This was conducted 10-times (10-fold CV), and the correlation coefficient was compared to shuffled data. We shuffled the data 1000 times, and if the true correlation was greater than 95% of the shuffles ($p < 0.05$), we considered it to meet the selection threshold. For the selected models, we generated the positive and negative networks in a single fold and then tested these models in the held-out data. This procedure was also conducted using partial correlations. In analysis 1, we controlled for head motion. In analysis 2, we controlled for head motion and puberty. A final analysis consisted of conducting the same methods but using average DCC features, which are similar to static connectivity.[13]

Supplementary Tables

Supplementary Table 1. Demographic and Clinical Characteristics of the Study 1 Sample

Sample Characteristics						
	S1 Anh (*n* = 38)		**S1 HC (*n* = 41)**		**All S1 (*n* = 79)**	
	N	**%**	**N**	**%**	**N**	**%**
Biological Sex						
Female	24	63.2	27	65.9	51	64.6
Male	14	36.8	14	34.1	28	35.4
Race						
American Indian	0	0	0	0	0	0
Asian	3	7.9	4	9.8	7	8.9
Black	1	2.6	6	14.6	7	8.9
Pacific	0	0	1	2.4	1	1.3
White	30	78.9	28	68.3	58	73.4
Multiracial	4	10.5	2	4.9	6	7.6
Ethnicity						
Hispanic	1	2.6	2	4.9	3	3.8
Not Hispanic	37	97.4	38	92.7	75	94.9
Unknown	0	0	1	2.4	1	1.3
Current Diagnoses (DSM-V)						
MDD	23	60.5	0	0	23	29.1
GAD	9	23.7	0	0	9	11.4
SAD	5	13.2	0	0	5	6.3
Panic Disorder	1	2.6	0	0	1	1.3
Specific Phobia	1	2.6	0	0	1	1.3
ADHD	3	7.9	0	0	3	3.8
ODD	2	5.3	0	0	2	2.5
PTSD	1	2.6	0	0	1	1.3
Medication						
Psychotropic Medication	7	18.4	0	0	7	8.9
	M (Range)	**SD**	**M (Range)**	**SD**	**M (Range)**	**SD**
Age (years)	15.82 (13 – 18)	1.90	16.24 (13 – 18)	1.70	16.04 (13 – 18)	1.80
Family Income (dollars)	129,718.8 (20,000 – 350,000)	67,317.88	151,685.8 (28,000 – 400,000)	88,856.71	141,194.1 (20,000 – 400,000)	79,486.67

Note. Anh: anhedonic individuals, selected by elevated scores on the KSADS interview. HC: healthy controls. MDD: Major Depressive Disorder. GAD: Generalized Anxiety Disorder. SAD:

Social Anxiety Disorder. ADHD: Attention-Deficit/Hyperactivity Disorder. ODD: Oppositional Defiant Disorder. PTSD: Post-Traumatic Stress Disorder.

Supplementary Table 2. Demographic and Clinical Characteristics of the Study 2 Sample

Sample Characteristics						
	S2 Anh (*n* = 36)		**S2 HC (*n* = 43)**		**All S2 (*n* = 79)**	
	N	**%**	**N**	**%**	**N**	**%**
Biological Sex						
Female	27	75.0	29	67.4	56	70.9
Male	9	25.0	14	32.6	23	29.1
Race						
American Indian	0	0	0	0	0	0
Asian	4	11.1	6	14.0	10	12.7
Black	5	13.9	1	2.3	6	7.6
Pacific	1	2.8	0	0	1	1.3
White	19	52.8	30	69.8	49	62.0
Multiracial	4	11.1	5	11.6	9	11.4
Other race	2	5.6	1	2.3	3	3.8
Unknown	1	2.8	0	0	1	1.3
Ethnicity						
Hispanic	6	16.7	4	9.3	10	12.7
Not Hispanic	30	83.3	39	90.7	69	87.3
Unknown	0	0	0	0	0	0
Current Diagnoses (DSM-V)						
MDD	0	0	0	0	0	0
GAD	5	13.9	0	0	5	6.3
SAD	3	8.3	0	0	3	3.8
Panic Disorder	1	2.8	0	0	1	1.3
Specific Phobia	0	0	0	0	0	0
ADHD	1	2.8	1	2.3	2	2.5
OCD	2	5.6	0	0	2	2.5
PTSD	0	0	0	0	0	0
Medication						
Psychotropic Medication	2	18.4	0	0	2	2.5
	M (Range)	**SD**	**M (Range)**	**SD**	**M (Range)**	**SD**
Age (years)	16.03 (12 – 18)	2.09	15.84 (12 – 18)	2.13	15.92 (12 – 18)	2.10
Family Income (dollars)	146,424.20 (0 – 500,000)	100,959.50	179,459.50 (40,000 – 400,000)	81,817.75	163,885.70 (0 – 500,000)	92,171.48

Note. Anh: anhedonic individuals, selected by elevated scores on the KSADS interview. HC: healthy controls. MDD: Major Depressive Disorder. GAD: Generalized Anxiety Disorder. SAD: Social Anxiety Disorder. ADHD: Attention-Deficit/Hyperactivity Disorder. OCD: Obsessive Compulsive Disorder. PTSD: Post-Traumatic Stress Disorder.

Supplementary Table 3. Demographic and Clinical Characteristics of the Study 3 Sample

Sample Characteristics						
	S3 HR (*n* = 49)		**S3 LR (*n* = 67)**		**All S3 (*n* = 116)**	
	N	**%**	**N**	**%**	**N**	**%**
Biological Sex						
Female	31	63.3	28	41.8	59	50.9
Male	18	36.7	39	58.2	57	49.1
Race						
American Indian	1	2.04	0	0	1	0.86
Asian	0	0	3	4.48	3	2.59
Black	1	2.04	2	2.99	3	2.59
Pacific	0	0	0	0	0	0
White	41	83.7	55	82.1	96	82.8
Multiracial	5	10.2	7	10.4	12	10.3
Unknown	1	2.04	0	0	1	0.86
Ethnicity						
Hispanic	7	14.3	2	2.99	9	7.76
Not Hispanic	42	85.7	65	97.0	107	92.2
Current Diagnoses (DSM-V)						
GAD	1	2.04	0	0	1	0.86
Specific Phobia	1	2.04	0	0	1	0.86
ODD	0	0	0	0	0	0
OCD	1	2.04	0	0	1	0.86
PTSD	1	2.04	0	0	1	0.86
Medication						
Psychotropic Medication	0	0	0	0	0	0
	M (Range)	**SD**	**M (Range)**	**SD**	**M (Range)**	**SD**
Age (years)	13.5 (12-15)	1.02	13.6 (12-15)	1.09	13.6 (12-15)	1.1
Family Income (dollars)	163,532 (20,500-900,000)	184,269.7	143,414 (45,000-650,000)	110,582.0	151,078 (20,500-900,000)	142,451.9

Note. HR: High-Risk. LR: Low-Risk. GAD: Generalized Anxiety Disorder. ODD: Oppositional Defiant Disorder. OCD: Obsessive Compulsive Disorder. PTSD: Post-Traumatic Stress Disorder. The following diagnoses were not included due to no participants having met criteria, Major Depressive Episode, Social Anxiety Disorder, Panic Disorder, Selective Mutism,

Attention-Deficit/Hyper-Activity Disorder, Binge Eating Disorder, Alcohol/Substance-Use Disorders, Oppositional Defiant Disorder.

Supplementary Table 4. Demographic and Clinical Characteristics of the Study 4 Sample

Sample Characteristics		
	N (*n* = 71)	**%**
Biological Sex		
Female	53	74.6
Male	18	25.4
Race		
American Indian	1	1.4
Asian	9	12.7
Black	5	7.0
Pacific	1	1.4
White	43	60.6
Multiracial	9	12.7
Unknown	3	4.2
Ethnicity		
Hispanic or Latino	9	12.7
Not Hispanic or Latino	62	87.3
Current Diagnoses (DSM-V)		
MDD	6	8.5
GAD	31	43.7
SAD	20	28.2
Panic Disorder	7	9.9
Specific Phobia	5	7.0
Selective Mutism	1	1.4
ADHD	1	1.4
ODD	3	4.2
OCD	2	2.8
Binge Eating Disorder	3	4.2
PTSD	1	1.4
Alcohol / Substance Use Disorders	0	0.0
Medication		
Psychotropic Medication	4	5.6
Handedness		
Right	44	62.0
Left	7	9.9
Unknown	20	28.2
	M (Range)	**SD**
Age (years)	15.8 (13 -18)	1.6

Family Income (dollars)	186,017.9 (3,000 - 500,000)	113,751.3

Note. One participant was excluded for head motion, but was included here for ease of reporting. MDD: Major Depressive Disorder. GAD: Generalized Anxiety Disorder. SAD: Social Anxiety Disorder. ADHD: Attention-Deficit/Hyperactivity Disorder. ODD: Oppositional Defiant Disorder. OCD: Obsessive Compulsive Disorder. PTSD: Post-Traumatic Stress Disorder.

Supplementary Table 5. Demographic and Clinical Characteristics of the Study 5 Sample

Sample Characteristics		
	N (*n* = 99)	**%**
Biological Sex		
Female	64	64.6
Male	35	35.4
Race		
American Indian	0	0.0
Asian	5	5.1
Black	3	3.0
Pacific	0	0.0
White	81	81.8
Multiracial	10	10.1
Unknown	0	0.0
Ethnicity		
Hispanic or Latino	5	5.1
Not Hispanic or Latino	94	94.9
Current Diagnoses (DSM-V)		
All	0	0.0
Family Income (dollars)		
50,000 – 75,000	8	8.1
75,000 – 100,000	16	16.2
Greater than 100,000	65	65.7
Unknown	10	10.0
	M (Range)	**SD**
Age (years)	13.0 (12 -14)	0.8

Note. All participants were right-handed. Due to exclusionary criteria, no participants were taking psychotropic medications, and no participants met threshold criteria for a current psychiatric diagnosis (DSM-IV). MDD: Major Depressive Disorder. GAD: Generalized Anxiety Disorder. SAD: Social Anxiety Disorder. ADHD: Attention-Deficit/Hyperactivity Disorder. ODD: Oppositional Defiant Disorder. OCD: Obsessive Compulsive Disorder. PTSD: Post-Traumatic Stress Disorder.

Supplementary Table 6. Correlations between model predictions and CRSQ rumination scores, by subgroup

Group	Sample size, n (denoised)	R-value	P-value	FDR-p
S1_Anh	37	-.03	.86	.97
S1_HC	40	.13	.41	.72
S2_Anh	28	.01	.97	.97
S2_HC	40	-.07	.67	.94
S3_HR	48	.14	.34	.72
S3_HC	67	-.13	.31	.72
S4	69	.20	.10	.69

S1-S4: samples 1 to 4. Anh: anhedonic based on KSADS score. HR: high risk, parental history of MDD. * $p < .05$.

Supplementary Table 7. Correlations between model predictions and EMA rumination scores, by subgroup

Group	Sample size, n (denoised)	R-value	P-value	FDR-p
S1_Anh	37	-.23	.16	.30
S1_HC	41	.02	.88	.91
S2_Anh	31	-.25	.17	.30
S2_HC	38	.16	.35	.49
S3_HR	48	-.02	.91	.91
S3_HC	64	-.28	**.02***	.16
S4	68	.25	**.04***	.16

S1-S4: samples 1 to 4. Anh: anhedonic based on KSADS score. HR: high risk, parental history of MDD. * $p < .05$.

Supplementary Table 8. Full Lasso model reporting for CRSQ in training dataset

Seed Area	*p*-values	FDR-*p*
DMN_l_HF	0.2232	0.576
DMN_l_LTC	0.1901	0.576
DMN_l_PCC	0.6066	0.6066
DMN_l_PHC	0.1025	0.576
DMN_l_Rsp	0.2543	0.576
DMN_l_TPJ	0.5425	0.6066
DMN_l_TempP	0.5183	0.6066
DMN_l_aMPFC	0.4171	0.6066
DMN_l_pIPL	0.5698	0.6066
DMN_r_HF	0.2098	0.576
DMN_r_LTC	0.3168	0.576
DMN_r_PCC	0.4123	0.6066
DMN_r_PHC	0.0314*	0.576
DMN_r_Rsp	0.2047	0.576
DMN_r_TPJ	0.5955	0.6066
DMN_r_TempP	0.2981	0.576
DMN_r_aMPFC	0.3102	0.576
DMN_r_pIPL	0.5985	0.6066
DMN_vMPFC	0.0684	0.576
DMN_DMPFC	0.4529	0.6066

Non-parametric *ps* for lasso models trained on dynamic connectivity from the seeds to the Brainnetome regions to predict CRSQ. * $p < 0.05$, passes selection threshold

Supplementary Table 9. Full Lasso model reporting for EMA in training dataset

Area	*p*-values	FDR-p
DMN_l_HF	0.3588	0.650
DMN_l_LTC	0.2042	0.650
DMN_l_PCC	0.1619	0.650
DMN_l_PHC	0.366	0.650
DMN_l_Rsp	0.9245	0.994
DMN_l_TPJ	0.7521	0.956
DMN_l_TempP	0.9482	0.994
DMN_l_aMPFC	0.1855	0.650
DMN_l_pIPL	0.3902	0.650
DMN_r_HF	0.7801	0.956
DMN_r_LTC	0.8008	0.956
DMN_r_PCC	0.0109*	0.218
DMN_r_PHC	0.8124	0.956
DMN_r_Rsp	0.3753	0.650
DMN_r_TPJ	0.1282	0.650
DMN_r_TempP	0.9944	0.994
DMN_r_aMPFC	0.3572	0.650
DMN_r_pIPL	0.7323	0.956
DMN_vMPFC	0.2566	0.650
DMN_DMPFC	0.3688	0.650

Non-parametric *ps* for lasso models trained on dynamic connectivity from the seeds to the Brainnetome regions to predict EMA. * $p < 0.05$, passes selection threshold

Supplementary Table 10. Non-parametric significance for CPM models in the training set

Measure	Pos Network	Neg network	Both	MEAN Connectivity
No partials				
CRSQ RUM	-0.13	0.11 (p = 0.033) *	0.12 (p = 0.063)	-0.076
EMA RUM	-0.0014	0.025	0.033	0.10
Partial HM				
CRSQ RUM	-0.13	0.12 (p = 0.025)*	0.12 (p = 0.043)*	-0.054
EMA RUM	-0.030	0.062	0.07	0.10
Partial HM and puberty				
CRSQ RUM	-0.17	0.18 (p = 0.004)**	0.19 (p = 0.006)**	-0.021
EMA RUM	-0.069	0.089 (p = 0.075)	0.095	0.10

Models were trained on dynamic and mean connectivity from all Brainnetome regions to predict CRSQ and EMA. Mean connectivity column shows overall network performance (pos-neg). One positive network model for mean connectivity passed selection threshold (p = 0.0375), but did not generalize. Only models that passed selection threshold display p-values, * p < 0.05, ** p < 0.01. HM : head motion, puberty: total puberty score on tanner scale.

Supplementary Table 11. Non-parametric significance for random forest models predicting CRSQ in the training set

Area	*p*-value	FDR-*p*
DMN_l_HF	0.004*	0.013
DMN_l_LTC	0.008*	0.018
DMN_l_PCC	0.004*	0.013
DMN_l_PHC	0.188	0.221
DMN_l_Rsp	0.564	0.564
DMN_l_TPJ	0.008*	0.018
DMN_l_TempP	0.004*	0.013
DMN_l_aMPFC	0.012	0.024
DMN_l_pIPL	0.084	0.129
DMN_r_HF	0.340	0.358
DMN_r_LTC	0.104	0.149
DMN_r_PCC	0.004*	0.013
DMN_r_PHC	0.004*	0.013
DMN_r_Rsp	0.284	0.316
DMN_r_TPJ	0.016*	0.029
DMN_r_TempP	0.048*	0.080
DMN_r_aMPFC	0.172	0.220
DMN_r_pIPL	0.008*	0.018
DMN_vMPFC	0.004*	0.013
DMN_DMPFC	0.176	0.220

* $p < 0.05$, passes selection threshold.

Supplementary Table 12: Network importances of Right Temporal Pole model

Networks	Mean Importances	P-value	FDR-P
Brainstem	0.019	0.678	0.678
Cerebellum	0.028	0.034*	0.112
dAttention	0.031	0.014*	0.068
Default	0.028	0.013*	0.068
Frontoparietal	0.021	0.079	0.197
Limbic	-0.011	0.348	0.435
Somatomotor	0.020	0.149	0.266
Subcortical	-0.015	0.218	0.311
vAttention	0.008	0.448	0.498
Visual	0.015	0.159	0.266

Importances for all regions in a network were statistically compared to zero with single sample *t*-tests , * $p < 0.05$

Supplementary Table 13. Non-parametric significance for random forest models predicting EMA in the training set

Area	p-values	FDR-p
DMN_l_HF	0.752	0.904
DMN_l_LTC	0.168	0.480
DMN_l_PCC	0.060	0.240
DMN_l_PHC	0.724	0.904
DMN_l_Rsp	0.500	0.855
DMN_l_TPJ	0.556	0.855
DMN_l_TempP	0.932	0.981
DMN_l_aMPFC	0.044*	0.220
DMN_l_pIPL	0.132	0.440
DMN_r_HF	0.740	0.904
DMN_r_LTC	0.768	0.904
DMN_r_PCC	0.040*	0.220
DMN_r_PHC	0.524	0.855
DMN_r_Rsp	0.400	0.855
DMN_r_TPJ	0.004*	0.040
DMN_r_TempP	0.992	0.992
DMN_r_aMPFC	0.004*	0.040
DMN_r_pIPL	0.920	0.981
DMN_vMPFC	0.216	0.540
DMN_DMPFC	0.552	0.855

* $p < 0.05$, passes selection threshold. Held-out data performance of selected models was non-significant.

Supplementary Table 14. Non-parametric significance for whole-brain random forest models predicting CRSQ and EMA in training set.

Measure	Both	MEAN Connectivity
CRSQ RUM	0.12 (p = 0.012*)	0.13 (p = 0.012*)
EMA RUM	-0.02 (p = 0.38)	0.02 (p = 0.16)

* $p < 0.05$, passes selection threshold

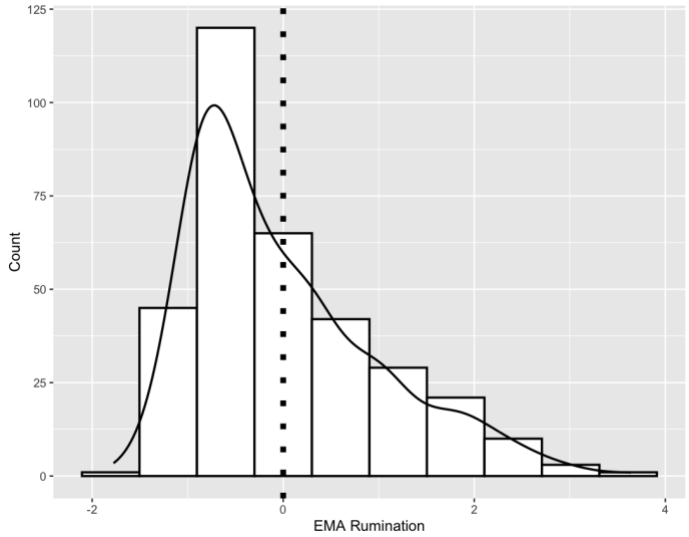
Supplementary Table 15. External test dataset performance for selected random forest models (with and without harmonization)

Model	Harmonized	Non-harmonized
LTP	0.05 (p = 0.61)	-0.02 (p = 0.84)
RTP	0.09 (p = 0.39)	-0.23* (p = 0.02)
Whole-brain	-0.01 (p = 0.85)	-0.04 (p = 0.67)

Sample size = 99. Pearson's correlations and p values (parentheses) are shown. * $p < 0.05$

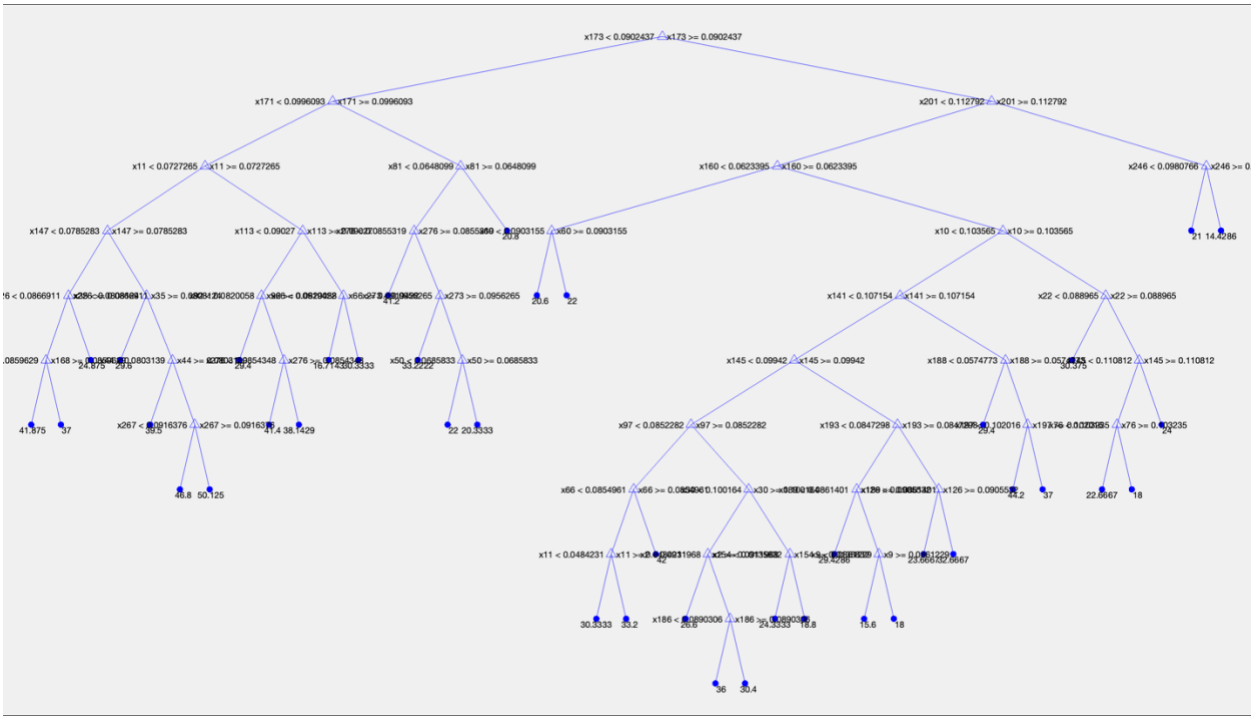
Supplementary Figures

Supplementary Figure 1. EMA Rumination score distribution across Studies 1-4



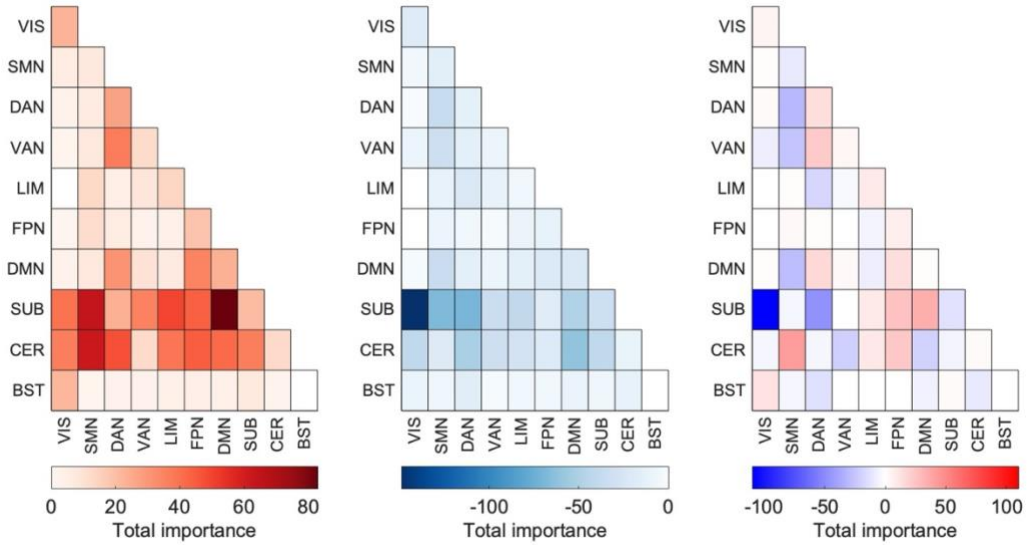
EMA: ecological probes of rumination, Count: number of participants. Dotted line represents mean (*n* = 344). Values are z-scored within datasets (maintaining train-test independence). No EMA was present in **Study 5.**

Supplementary Figure 2. Example Random Forest Decision Tree



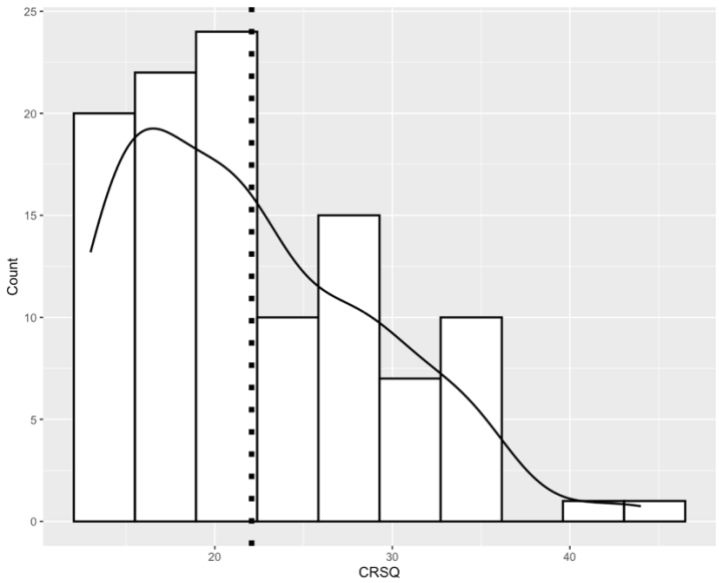
A regression tree from the left temporal pole model. Each branch reflects a rule based on varDCC for that feature (e.g. the value of varDCC for temporal pole and node 270). The final values are the outputted predictions.

Supplementary Figure 3. Whole-brain random forest model importances



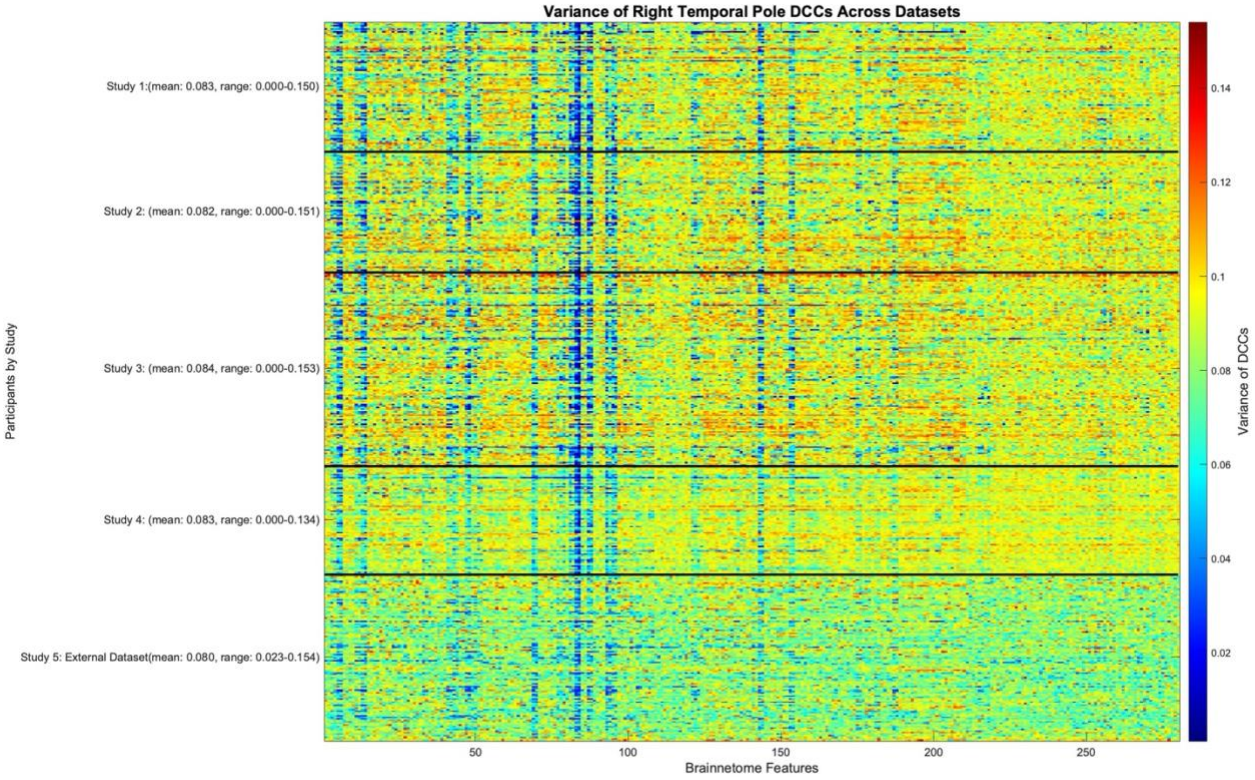
Total importance is the total weight of all out-of-bag importances from the random forest model for a given set of connections between networks from the Brainnetome atlas. On left, positive sum weights, middle negative sum weights, and right overall sum weights. More red represents higher positive weights, more blue represents lower negative weights.

Supplementary Figure 4. CRSQ Rumination score distribution in Study 5



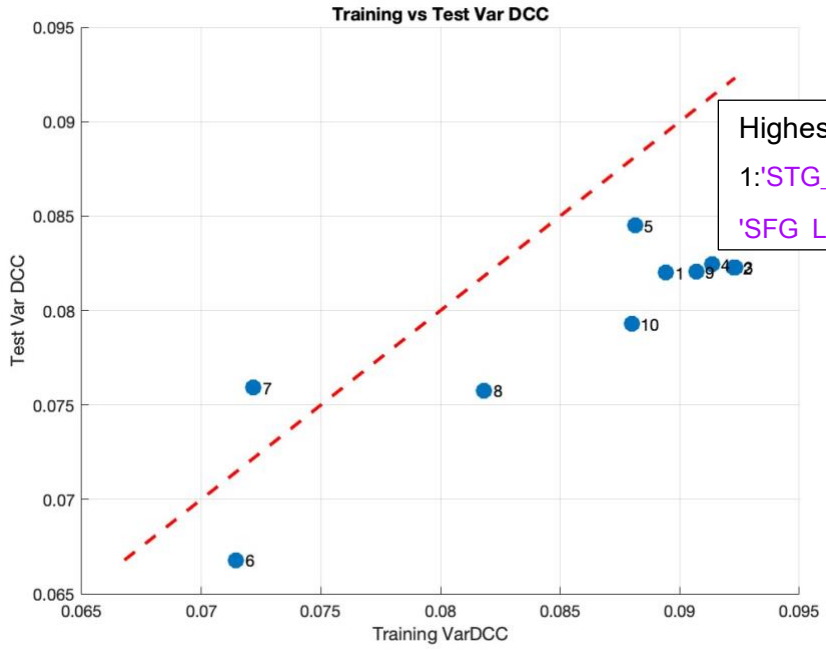
CRSQ: child response style questionnaire, Count: number of participants. Dotted line represents mean (*n* = 99 participants).

Supplementary Figure 5. DMPFC seed dynamic conditional correlations



Example Right Temporal Pole dynamic connectivity features across the 443 subjects (y-axis) and 280 Brainnetome regions (x-axis). **Studies 4 and 5** involved different scan protocols. Red represents higher variance of dynamic connectivity, blue represents lower variance.

Supplementary Figure 6. Right temporal pole DCC differences between training and external test set for highest positive and negative importance regions



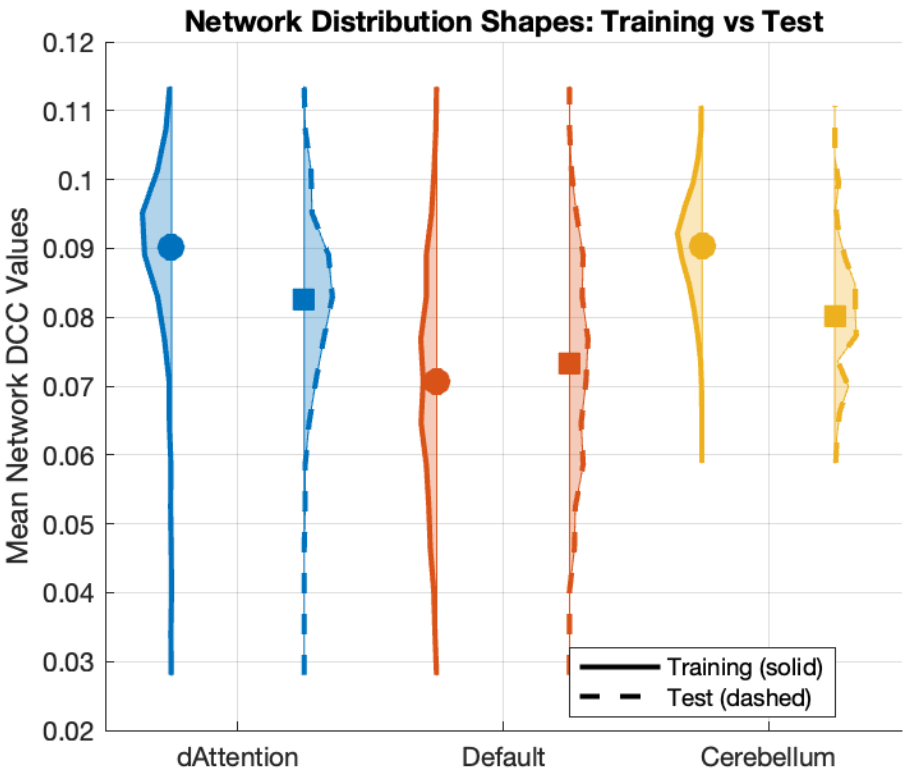
Highest positive importance regions

1:'STG_R_6_4',2: 'PCun_R_4_2', 3:

'SFG_L_7_5',4:'Cb_Right_V',5:'IFG_L_6_1'};

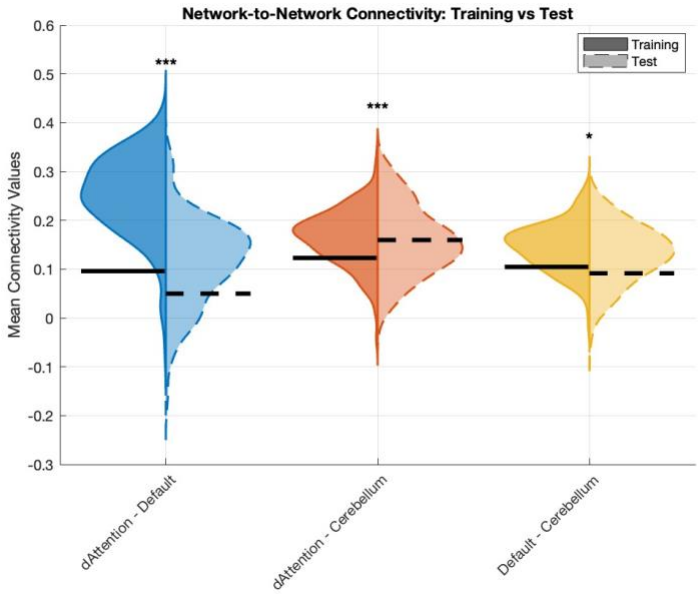
Points represent Training (x-axis) and External Test (y-axis) feature values for high importance regions. The red line represents equal feature values. Smaller DCC values may be observed for the regions in the external test set.

Supplementary Figure 7. Right temporal pole DCC differences between training and external test set for significant positive importance networks



High importance networks shown for training and external data (test) splits. The variance of dynamic connectivity is averaged across all RTP – network edges.

Supplementary Figure 8. Static connectivity differences between training and external test set



Network static connectivity differences for training and external test set. Connections between high importance networks are shown as they exemplify differences. $p < 0.05$ *** $p < 0.001$

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