Data Supplement for Jalbrzikowski et al., Age-Associated Deviations of Amygdala Functional Connectivity in Youths With Psychosis Spectrum Disorders: Relevance to Psychotic Symptoms. Am J Psychiatry (doi: 10.1176/appi.ajp.2018.18040443)

Supplemental Methods

Participants

Luna 1 and Luna 2

For Luna 1 and Luna 2, participants and their first-degree relatives did not have a psychiatric disorder determined by phone screen and a clinical questionnaire (1). Exclusion criteria for all participants included: medical illness affecting the central nervous system function, IQ (determined using the Reynolds Intellectual Assessment Scale [2]) lower than 80, a first-degree relative with a major psychiatric disorder, or any MRI contraindications.

PNC

Data for the Philadelphia Neurodevelopmental Cohort (PNC) was obtained through the Database of Genotypes and Phenotypes platform (Beatriz Luna, #43787-2). The PNC is a population sample consisting of 9498 youth (ages 9-22 years) who participated in neurocognitive and genetic assessment after providing writing informed consent or assent with parental consent (youth under 18 years old [3]). A subset of these youth (N=997) also underwent neuroimaging measures (4). Psychopathology was assessed using a computerized, structured interview (GOASSESS [3, 5]), which is based on the Kiddie Schedule for Affective Disorders and Schizophrenia for School-Age Children Present and Lifetime (KSADS-PL [6]). Categorical and dimensional measures of psychosis were created from clinical symptom responses to GOASSESS, the Structured Interview for Prodromal Syndromes (SIPS [7]), and a 12-item PRIME Screen-Revised questionnaire (PS-R [8]). Categorical psychosis spectrum group was defined as 1) a score that is two standard deviations or greater than age-matched

peers on the SIPS or PS-R, 2) definite or possible hallucinations or delusions reported on the responses to psychosis items in GOASSESS, or 3) a minimum of 1 PS-R item rated 6 (definitely agree) or at least 3 items rated 5 (somewhat agree); this definition is consistent with previous PNC publications (5, 9, 10).

To test specificity of psychosis abnormalities, another group of participants who met DSM-IV criteria for non-psychotic psychopathology was created. We used responses to questions on the GOASSES to determine DSM-IV diagnosis ranking. Similar to previous PNC publications (3), psychopathology was considered to be significant if symptoms endorsed were consisted with frequency and duration of a DSM-IV psychiatric disorder, while correspondingly accompanied by significant distress or impairment (a rating of $\geq$5 on a scale of 0-10).

Pitt

The Pitt sample was recruited from an ongoing Conte Center study examining neurobiological mechanisms of working memory deficits in first episode psychosis (FEP). Exclusion criteria for all participants included: medical illness affecting the central nervous system function, IQ (determined using the Wechsler Abbreviated Scale of Intelligence [11], lower than 75, or any MRI contraindications. Inclusion criteria for FEP were as follows: experiencing one's first psychotic episode and seeking help for his/her psychotic symptoms for the first time and antipsychotic naive or prescribed antipsychotic treatment for less than two months. Diagnoses were determined using all available clinical information and data gathered from a Structured Clinical Interview for DSM-IV (SCID [12]) conducted with a trained clinician. Experienced diagnostician/clinical researchers confirmed diagnoses at consensus meetings. None of the patients met criteria for a DSM-IV substance abuse disorder currently or within the previous 6 months. The inclusion criteria for controls in the Pitt sample were no lifetime history of a major psychiatric disorder or antipsychotic treatment, no first-degree family member with a history of a psychotic disorder, and no significant neurological disorder or head injury or mental retardation as defined by the DSM-IV.

rsfMRI Processing

The first 4 TRs from all scans were removed to allow for BOLD signal normalization. Functional images were warped into MNI standard space using a series of affine and nonlinear transforms. Normalization based on global mode was then calculated on the functional images. Next all functional images were spatially smoothed using a 5-mm full width at half maximum Gaussian kernel. Removal of non-stationary events in the fMRI time series was conducted using Wavelet Despiking (13). To better control nuisance-related variability (14) we then conducted simultaneous multiple regression of nuisance variables and bandpass filtering at 0.009 Hz $< f <$ 0.08. Nuisance regressors included were non-brain tissue (NBT), average white matter signal, average ventricular signal, six head realignment parameters obtained by rigid body head motion correction, and the derivatives of these measures. NBT, average white matter, and average ventricular signal nuisance regressors were created using Freesurfer's automated segmentation program (15) and extracted from each participant's MPRAGE scan. ICA-Aroma was implemented to remove motion artifacts (16, 17). We then removed any remaining high motion volumes via scrubbing procedure. For all subjects, we calculated two quality control measures with respect to head motion: volume-to-volume frame displacement, (FD) and the RMS derivative of fMRI time series (DVARS). We censored and removed volumes that had an FD > 0.3 mm and/or DVARS > 20 (computed after wavelet despiking). By implementing wavelet despiking prior to scrubbing, we were able to use most of the time series data to provide a more reliable estimate of the true correlation. However, because motion is such a critical issue in developmental studies and there were some remaining DVARS values over the identified threshold, after wavelet despiking, these volumes were censored as extra validation to ensure that motion was not contaminating our signal. Subjects were dropped from rsfMRI analyses if more than 20% of their volumes were removed.

Regions of Interest

Centromedial (CM) and basolateral (BL) regions of interest (ROIs) are available in FSL's Juelich histological atlas (18) and have been used in previous studies examining amygdala rsfMRI connectivity (19–21). Because the FSL atlas has a slight bias for its MNI template (22), we first used FNIRT to warp the Jeulich atlas to the standard MNI template space. Voxels with at least a 50% probability of belonging in one of these subregions were included in each ROI and each voxel was only assigned to one subregion.

I***mplementation of AFNI's 3dClustSim***

Analysis was masked to only include voxels with a 50% or greater probability of being grey matter in the MNI-152 template. Results were corrected for multiple comparisons using a combination of cluster size and voxel probability, with parameters determined through a Monte Carlo simulation using AFNI's 3dClustSim program on randomly generated data within the grey matter mask with the same smoothness as the group mean smoothness estimated from first-level residuals for each subregion. This analysis specified that a cluster of 30 contiguous voxels with a single voxel threshold of $p<.001$ are required to achieve a clusterwise corrected $p<.05$.

Supplemental Figures

FIGURE S1. Flowchart depicting how scan completion and movement restrictions influenced subject N in each sample.

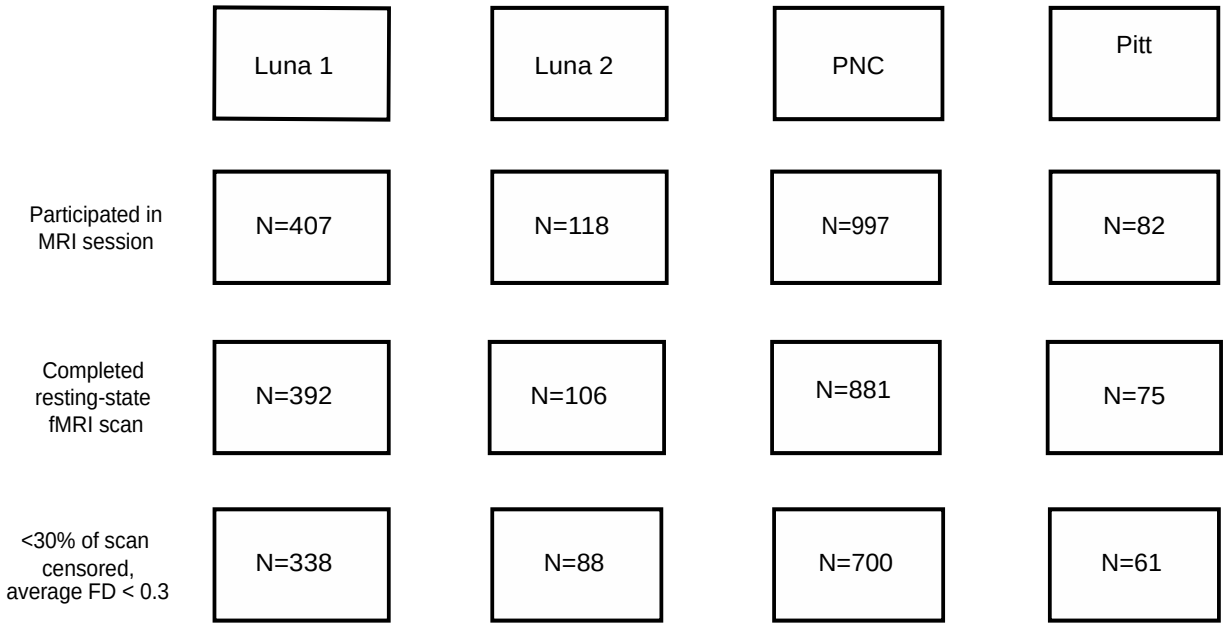

FIGURE S2. Nineteen clusters exhibited developmental decreases in connectivity with the centromedial amygdala. One cluster that exhibited developmental decreases in connectivity with the basolateral amygdala.

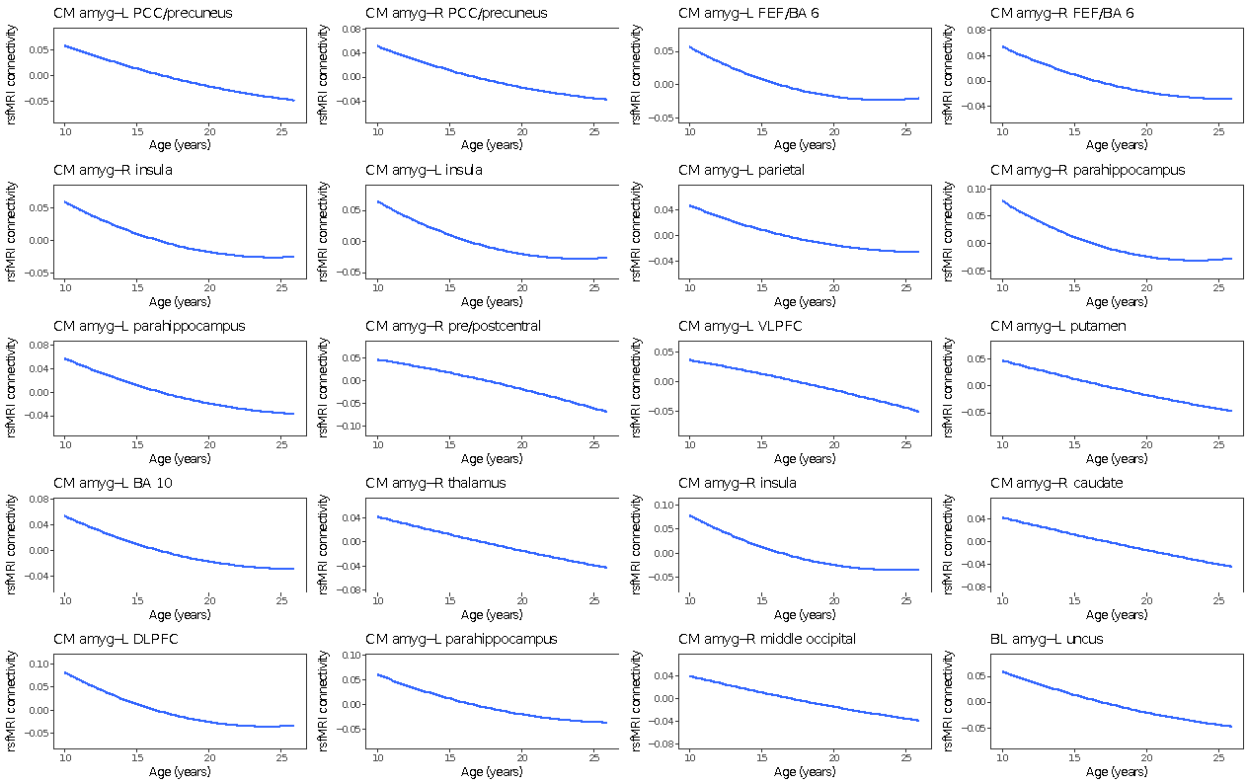

FIGURE S3. For each amygdala subregion connectivity measure that exhibited a significant developmental change in typically developing youth, we plotted the age-associated line of best fit in each protocol (Luna 1, Luna 2, PNC, and Pitt). The pattern of age-associated change is remarkably consistent across different samples.

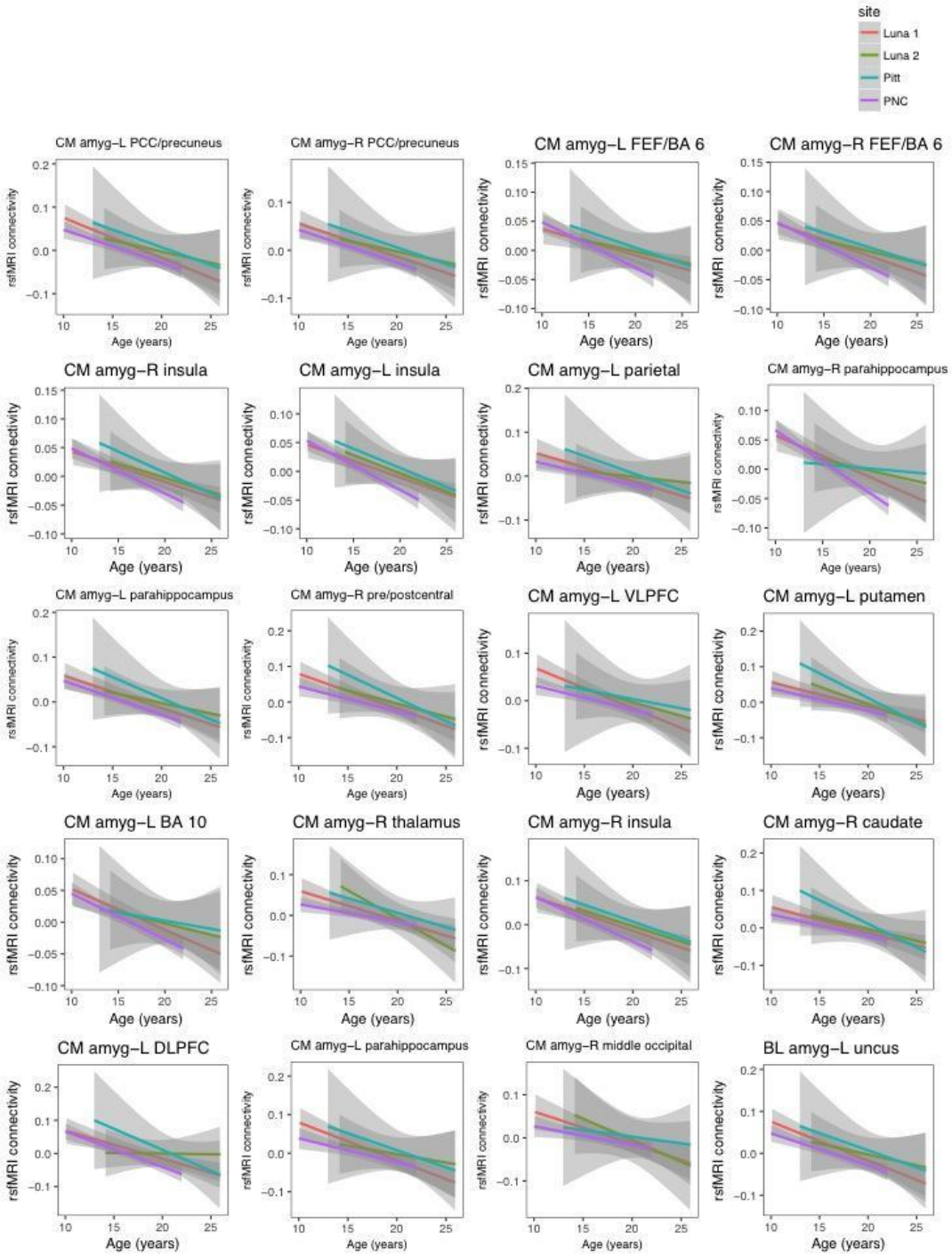

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FIGURE S4. After regressing out protocol as a covariate, we plotted the residuals in each protocol separately. In all 20 regions that exhibited age associated changes, the residuals cluster around a mean of zero and do not significantly differ from each other in each protocol. This suggests that we adequately accounted for site in our analyses.

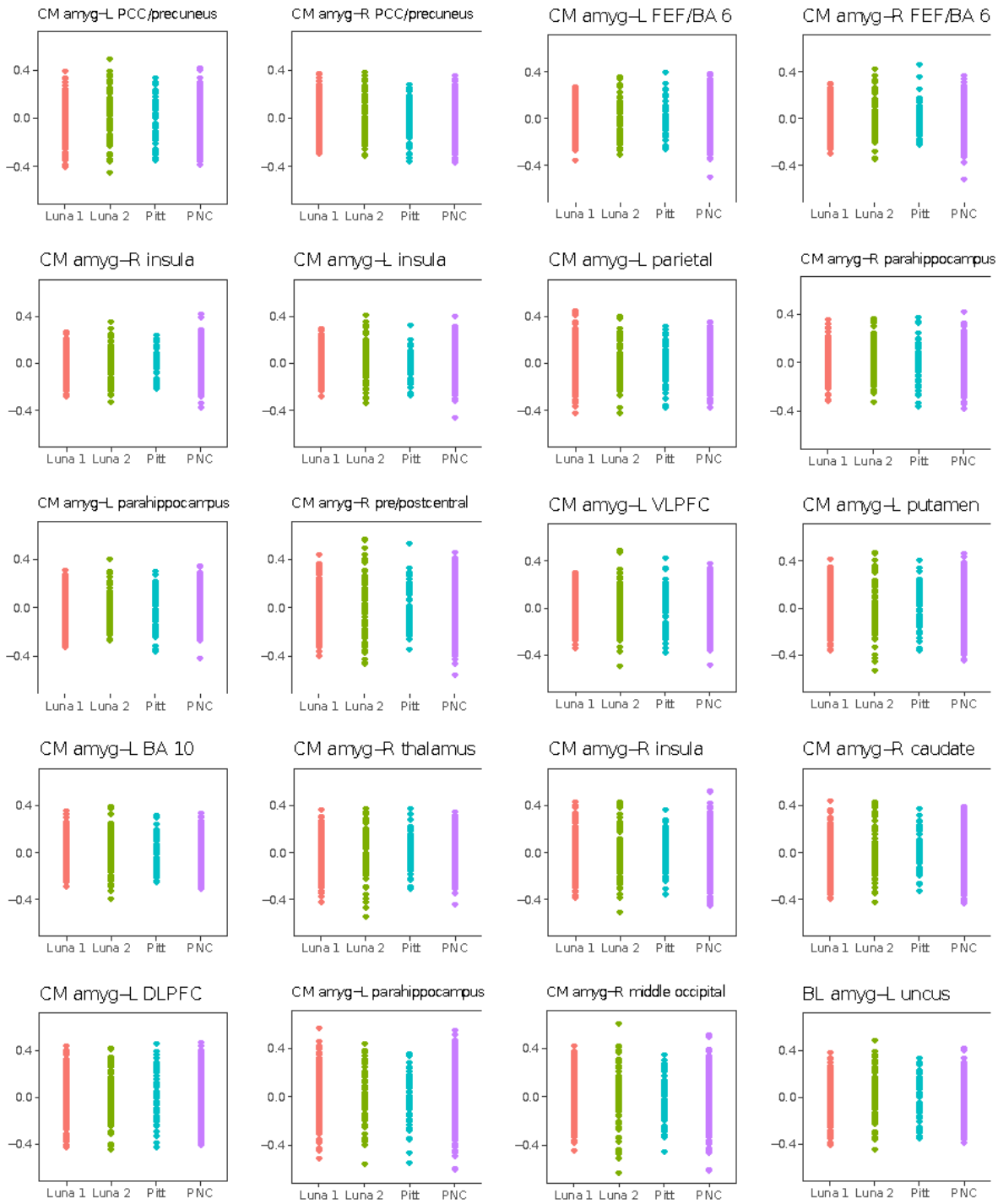
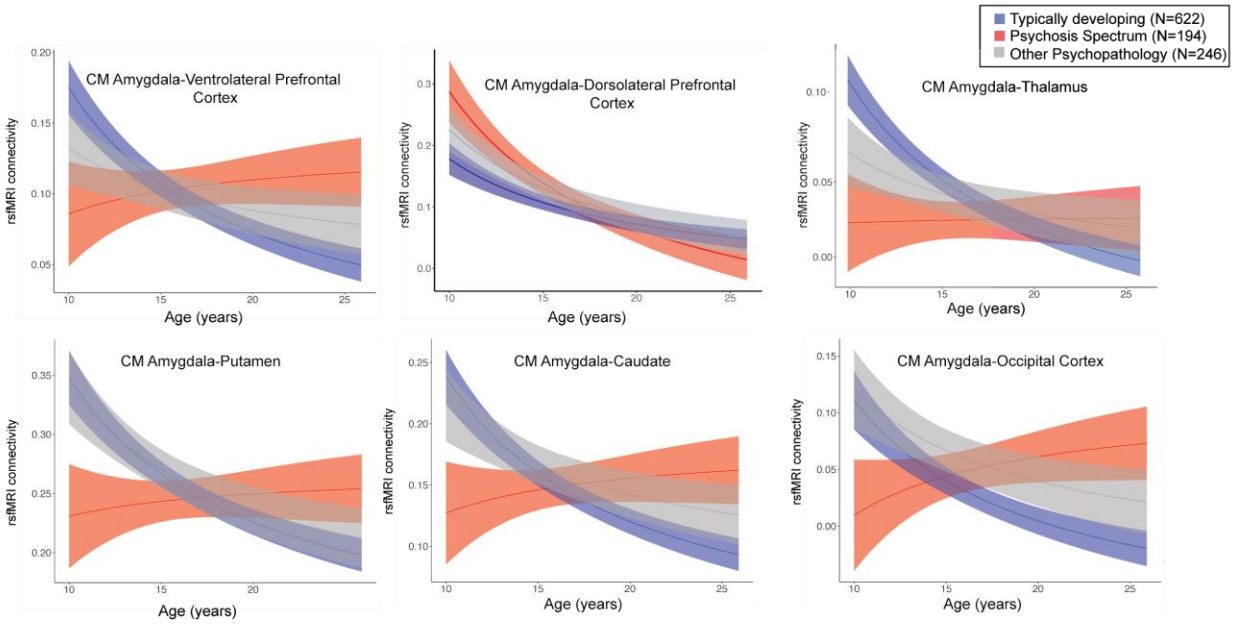


FIGURE S5. Like controls (blue), youth with other psychopathology (grey) showed significant age-related decreases with increasing age in connectivity between the following regions: CM amygdala-dorsolateral prefrontal cortex, CM amygdala-putamen, CM amygdala-caudate, and CM amygdala-occipital cortex. Like psychosis spectrum youth (red), the other psychopathology group failed to show age-associated changes in CM amygdala-ventrolateral prefrontal cortex connectivity, and CM amygdala-thalamus connectivity.



Supplemental Tables

TABLE S1. Responses to the following SIPS/PRIME Screen-Revised questionnaire were summed as a dimensional measure of A) positive and B) negative symptoms. For positive symptoms, responses were rated on a Likert scale (0=definitely disagree, 1=somewhat disagree, 2=slightly disagree, 3=not sure, 4=slightly agree, 5=somewhat agree, 6=definitely agree).

A. Positive Symptoms	
SIP003	I think that I have felt that there are odd or unusual things going on that I can't explain.
SIP004	I think that I might be able to predict the future.
SIP005	I may have felt that there could possibly be something interrupting or controlling my thoughts, feelings, or actions.
SIP006	I have had the experience of doing something differently because of my superstitions.
SIP007	I think I may get confused at times whether something I experience or perceive may be real or may be just part of my imagination or dreams.
SIP008	I have thought that it might be possible that other people can read my mind, or that I can read others' minds
SIP009	I wonder if people may be planning to hurt me or even may be about to hurt me.
SIP010	I believe that I have special natural or supernatural gifts beyond my talents and natural strengths.
SIP011	I think I might feel like my mind is "playing tricks" on me.
SIP012	I have had the experience of hearing faint or clear sounds of people or a person mumbling or talking when there is no one near me.
SIP013	I think that I may hear my own thoughts being said out loud.
SIP014	I have been concerned that I might be "going crazy."
B. Negative Symptoms	
SIP001	Trouble with focus and attention severity
SIP035	Changes in perception of self, others, or the world in general severity
SIP037	Expression of emotion severity
SIP041	Occupational functioning severity
SIP043	Avolition severity

TABLE S2. Resting state and structural scan sequences for each cohort.

	LUNA 1	LUNA 2	PNC	Pitt
head coil (# of channels)	12	32	32	32
rsfMRI parameters				
Instructions	Eyes closed, stay awake & still	Eyes open, stay awake & still, fixate on crosshair	Eyes open, stay awake & still, fixate on crosshair	Eyes open, stay awake & still, fixate on crosshair
Acquisition time (s)	300 s	360s	378 s	360s
TR/TE (ms)	1500/29 ms	1000/30 ms	3000/32 ms	1000/30
Flip angle (°)	70°	50°	90°	55°
Voxel size (mm)	3 mm	2.3 mm	3 mm	2.3 mm
gradient echo field map	no	yes	no	yes
MPRAGE parameters				
Acquisition time (s)	435	424	208	362
TI (ms)	800	1000	1100	1260
TR/TE (ms)	1570/3.4	2200/3.5	1810/3.5	2530/1.7/3.6/5.46/ 7.3
Flip angle (°)	8	9	9	7
Voxel size (mm)	1 mm	1 mm	1 mm	1 mm

TABLE S3. All significant age effects remained with inclusion average framewise displacement as an additional covariate.

cluster	predictor	χ2	*df*	*p*
Centromedial amygdala connectivity				
L posterior cingulate/precuneus	inverse age	25.7	1	3.90E-07
	site	1.7	3	0.64
	sex	0.4	1	0.51
	average framewise displacement	5.0	1	0.03
R posterior cingulate/precuneus	inverse age	22.4	1	2.16E-06
	site	5.4	3	0.14
	sex	1.4	1	0.23
	average framewise displacement	4.4	1	0.04
L FEF/BA 6 & Precentral gyrus	inverse age	23.3	1	1.37E-06
	site	19.2	3	2.49E-04
	sex	0.3	1	0.58
	average framewise displacement	0.9	1	0.34
R FEF/BA Precentral gyrus	inverse age	24.9	1	6.06E-07
	site	15.3	3	1.56E-03
	sex	0.0	1	0.83
	average framewise displacement	1.7	1	0.19
R insula/claustrum	inverse age	26.5	1	2.66E-07
	site	62.5	3	1.72E-13
	sex	1.3	1	0.25
	average framewise displacement	23.1	1	1.55E-06
L insula/claustrum	inverse age	31.2	1	2.36E-08
	site	98.9	3	2.68E-21
	sex	1.3	1	0.26
	average framewise displacement	13.6	1	2.31E-04
L parietal cortex/middle temporal gyrus	inverse age	21.6	1	3.29E-06
	site	6.0	3	0.11
	sex	0.0	1	0.94
	average framewise displacement	0.9	1	0.33
R parahippocampal gyrus	inverse age	34.6	1	4.06E-09
	site	9.0	3	0.03
	sex	0.3	1	0.60
	average framewise displacement	12.4	1	4.36E-04

(Continued)

L parahippocampal gyrus	inverse age	25.8	1	3.76E-07
	site	1.7	3	0.63
	sex	2.0	1	0.16
	average framewise displacement	11.1	1	8.69E-04
R precentral/postcentral gyrus	inverse age	16.1	1	5.97E-05
	site9	6.7	3	0.08
	sex9	0.8	1	0.36
	average framewise displacement9	1.6	1	0.20
L ventrolateral prefrontal cortex	inverse age	24.6	1	6.95E-07
	site0	2.9	3	0.41
	sex0	0.3	1	0.61
	average framewise displacement0	8.9	1	2.92E-03
L putamen	inverse age	23.1	1	1.56E-06
	site	160.8	3	1.24E-34
	sex	0.3	1	0.57
	average framewise displacement	9.5	1	2.10E-03
L BA 10/superior frontal gyrus	inverse age	23.8	1	1.09E-06
	site	33.9	3	2.03E-07
	sex	0.7	1	0.39
	average framewise displacement	9.3	1	2.31E-03
R thalamus	inverse age	29.7	1	4.96E-08
	site	4.0	3	0.27
	sex	0.8	1	0.36
	average framewise displacement	0.9	1	0.35
R insula	inverse age	20.6	1	5.62E-06
	site	16.2	3	1.03E-03
	sex	0.4	1	0.52
	average framewise displacement	13.6	1	2.31E-04
L caudate	inverse age	20.7	1	5.46E-06
	site	44.6	3	1.13E-09
	sex	5.1	1	0.02
	average framewise displacement	9.8	1	1.75E-03
L dorsolateral prefrontal cortex/BA 9	inverse age	24.0	1	9.82E-07
	site	8.4	3	0.04
	sex	0.0	1	0.92
	average framewise displacement	3.8	1	0.05

(Continued)

L parahippocampal gyrus	inverse age	18.9	1	1.38E-05
	site	12.5	3	0.01
	sex	2.5	1	0.12
	average framewise displacement	7.3	1	0.01
R middle occipital gyrus	inverse age	13.0	1	3.15E-04
	site	1.6	3	0.66
	sex	1.8	1	0.19
	average framewise displacement	4.9	1	0.03
Basolateral amygdala connectivity				
L uncus	inverse age	22.4	1	2.50E-06
	site	18.7	3	3.20E-04
	sex	2.3	1	0.13
	average framewise displacement	5.1	1	0.02

TABLE S4. All significant age effects remained when the highest motion subjects (top 25%, >0.17) were removed from the analysis.

cluster	predictor	$\chi 2$	*df*	*p*
Centromedial amygdala connectivity				
L posterior cingulate/precuneus	inverse age	16.2	1	5.84E-05
	site	2.4	3	0.49
	sex	0.3	1	0.60
	average framewise displacement	1.8	1	0.18
R posterior cingulate/precuneus	inverse age	16.1	1	6.11E-05
	site	5.8	3	0.12
	sex	1.3	1	0.26
	average framewise displacement	2.1	1	0.15
L FEF/BA 6 & Precentral gyrus	inverse age	14.3	1	1.56E-04
	site	17.9	3	4.63E-04
	sex	1.0	1	0.32
	average framewise displacement	0.9	1	0.34
R FEF/BA Precentral gyrus	inverse age	17.6	1	2.66E-05
	site	13.0	3	4.73E-03
	sex	0.9	1	0.33
	average framewise displacement	1.3	1	0.26

(Continued)

R insula/claustrum	inverse age	20.7	1	5.25E-06
	site	48.0	3	2.12E-10
	sex	2.0	1	0.16
	average framewise displacement	4.2	1	0.04
L insula/claustrum	inverse age	19.8	1	8.60E-06
	site	69.8	3	4.69E-15
	sex	1.5	1	0.23
	average framewise displacement	7.7	1	0.01
L parietal cortex/middle temporal gyrus	inverse age	14.7	1	1.24E-04
	site	2.4	3	0.49
	sex	0.5	1	0.50
	average framewise displacement	7.2	1	0.01
R parahippocampal gyrus	inverse age	26.0	1	3.48E-07
	site	10.6	3	0.01
	sex	0.0	1	0.98
	average framewise displacement	7.5	1	0.01
L parahippocampal gyrus	inverse age	14.2	1	1.67E-04
	site	5.3	3	0.15
	sex	0.0	1	0.96
	average framewise displacement	8.0	1	4.75E-03
R precentral/postcentral gyrus	inverse age	12.2	1	4.79E-04
	site9	7.1	3	0.07
	sex9	0.5	1	0.47
	average framewise displacement9	2.8	1	0.10
L ventrolateral prefrontal cortex	inverse age	15.9	1	6.51E-05
	site0	5.9	3	0.11
	sex0	0.3	1	0.61
	average framewise displacement0	4.0	1	0.05
L putamen	inverse age	16.9	1	3.97E-05
	site	127.4	3	1.98E-27
	sex	1.0	1	0.32
	average framewise displacement	10.6	1	1.15E-03
L BA 10/superior frontal gyrus	inverse age	21.0	1	4.60E-06
	site	36.5	3	5.95E-08
	sex	0.7	1	0.39
	average framewise displacement	7.6	1	0.01

(Continued)

R thalamus	inverse age	18.2	1	2.03E-05
	site	1.2	3	0.75
	sex	0.3	1	0.59
	average framewise displacement	1.8	1	0.18
R insula	inverse age	16.7	1	4.36E-05
	site	14.1	3	2.79E-03
	sex	0.2	1	0.66
	average framewise displacement	7.0	1	0.01
L caudate	inverse age	18.6	1	1.58E-05
	site	36.9	3	4.77E-08
	sex	3.7	1	0.06
	average framewise displacement	21.2	1	4.16E-06
L dorsolateral prefrontal cortex/BA 9	inverse age	10.4	1	1.26E-03
	site	5.9	3	0.11
	sex	0.1	1	0.80
	average framewise displacement	3.8	1	0.05
L parahippocampal gyrus	inverse age	12.1	1	4.92E-04
	site	5.8	3	0.12
	sex	3.3	1	0.07
	average framewise displacement	6.8	1	0.01
R middle occipital gyrus	inverse age	6.6	1	0.01
	site	3.5	3	0.32
	sex	0.9	1	0.34
	average framewise displacement	5.0	1	0.03
Basolateral amygdala connectivity				
L uncus	inverse age	18.6	1	1.70E-05
	site	21.8	3	6.90E-05
	sex	2.3	1	0.13
	average framewise displacement	4.3	1	0.04

TABLE S5. Youth with psychosis spectrum exhibited lower connectivity in comparison to controls during late childhood for the following clusters: CM amygdala-ventrolateral prefrontal cortex, CM amygdala-putamen, CM amygdala-thalamus, CM amygdala-caudate, CM amygdala-occipital cortex. Youth with psychosis spectrum exhibited lower connectivity in comparison to other psychopathology during late childhood for the following clusters: CM amygdala-putamen and CM amygdala-occipital cortex. Youth with psychosis spectrum exhibited increased connectivity in comparison to controls during adulthood in the following clusters: CM amygdala-ventrolateral prefrontal cortex, CM amygdala-putamen, CM amygdala-caudate, and CM amygdala-occipital cortex.

Amygdala connectivity measure	Psychosis spectrum vs. Typically Developing		Psychosis spectrum vs. Other Psychopathology		Other Psychopathology vs. Typically Developing	
	↓ connectivity in psychosis spectrum	↑ connectivity in psychosis spectrum	↓ connectivity in psychosis spectrum	↑ connectivity in psychosis spectrum	↓ connectivity in other psycho-pathology	↑ connectivity in other psycho-pathology
Ventrolateral Prefrontal Cortex	10–12 yrs	17.9–25.9 yrs	--	--	--	--
Dorsolateral Prefrontal Cortex	--	10–14 yrs	--	--	--	--
Putamen	10–14 yrs	24–25.9 yrs	10–14 yrs	--	--	--
Thalmaus	10–15 yrs	--	--	--	--	--
Caudate	10–13 yrs	20–25.9 yrs	--	--	--	--
Occipital Cortex	10 yrs	17–25.9 yrs	10–12 yrs	--	--	--

TABLE S6. When the other psychopathology group was added to models in which there were inverse age x group associations observed between psychosis spectrum youth and controls, the interaction term remained significant.

Cluster	rsfMRI connectivity measure	Typically developing*psychosis*other psychopathology		
		χ^2	*p*	*Q*
11	CM amygdala-ventrolateral prefrontal cortex	8.2	0.01	0.03
12	CM amygdala-putamen	6.3	0.04	0.04
14	CM amygdala-thalmaus	7.5	0.02	0.03
16	CM amygdala-caudate	7.7	0.02	0.03
19	CM amygdala-occipital cortex	6.3	0.04	0.04

TABLE S7. For all significant inverse age*group interactions, controls exhibited age-associated decreases in CM amygdala connectivity. Psychosis spectrum youth failed to show significant age-associated changes in all CM amygdala connectivity clusters. The other psychopathology group exhibited age-associated decreases in CM amygdala-putamen connectivity, CM amygdala-caudate connectivity, and CM amygdala-occipital connectivity.

rsfMRI Connectivity Measure	Group	Inverse age beta	Z-ratio	*p*
CM amygdala-ventrolateral prefrontal cortex	psychosis spectrum	-0.4	-0.4	0.66
	typically developing	2.1	5.0	5.4E-07
	other psychopathology	0.9	1.4	0.16
CM amygdala-putamen	psychosis spectrum	-0.4	-0.3	0.73
	typically developing	2.5	5.0	6.6E-07
	other psychopathology	2.1	2.8	0.005
CM amygdala-thalamus	psychosis spectrum	0.0	0.0	0.98
	typically developing	2.0	5.4	5.8E-08
	other psychopathology	0.8	1.5	0.13
CM amygdala-caudate	psychosis spectrum	-0.9	-0.9	0.37
	typically developing	2.4	5.0	5.1E-07
	other psychopathology	1.4	2.1	0.04
CM amygdala-occipital cortex	psychosis spectrum	-1.3	-1.1	0.28
	typically developing	2.1	3.8	1.2E-04
	other psychopathology	1.6	2.0	0.05

References

1. Achenbach TM, Rescorla LA: The Manual for the ASEBA School-Age Forms & Profiles. Burlington, VT, Research Center for Children, Youth, and Families., 2001

2. Reynolds CR, Kmaphaus RW: Reynolds Intellectual Assessment Scales2003;

3. Calkins ME, Merikangas KR, Moore TM, et al.: The Philadelphia Neurodevelopmental Cohort: constructing a deep phenotyping collaborative. J Child Psychol Psychiatry 2015; 56:1356–1369

4. Satterthwaite TD, Connolly JJ, Ruparel K, et al.: The Philadelphia Neurodevelopmental Cohort: A publicly available resource for the study of normal and abnormal brain development in youth. Neuroimage 2015; 124:1115–1119

5. Calkins ME, Moore TM, Merikangas KR, et al.: The psychosis spectrum in a young U.S. community sample: findings from the Philadelphia Neurodevelopmental Cohort. World Psychiatry 2014; 13:296–305

6. Kaufman J, Birmaher B, Brent D, et al.: Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (K-SADS-PL): initial reliability and validity data. J Am Acad Child Adolesc Psychiatry 1997; 36:980–988

7. Miller TJ, McGlashan TH, Rosen JL, et al.: Prodromal assessment with the structured interview for prodromal syndromes and the scale of prodromal symptoms: predictive validity, interrater reliability, and training to reliability. Schizophr Bull 2003; 29:703–715

8. Kobayashi H, Nemoto T, Koshikawa H, et al.: A self-reported instrument for prodromal symptoms of psychosis: testing the clinical validity of the PRIME Screen—Revised (PS-R) in a Japanese population. Schizophr Res 2008; 106:356–362

9. Wolf DH, Satterthwaite TD, Calkins ME, et al.: Functional neuroimaging abnormalities in youth with psychosis spectrum symptoms. JAMA Psychiatry 2015; 72:456–465

10. Satterthwaite TD, Wolf DH, Calkins ME, et al.: Structural Brain Abnormalities in Youth With Psychosis Spectrum Symptoms. JAMA Psychiatry 2016; 73:515–524

11. Wechsler D: Wechsler adult intelligence scale--Fourth Edition (WAIS--IV) [Internet]. San Antonio, Texas: Psychological Corporation 2014; Available from: https://pdfs.semanticscholar.org/2858/f906a462c4424192f80361f689bdec24c16d.pdf

12. First MB, Spitzer RL, Gibbon M, et al.: Structured clinical interview for DSM-IV-TR axis I disorders, research version, patient edition. SCID-I/P, 2002

13. Patel AX, Bullmore ET: A wavelet-based estimator of the degrees of freedom in denoised fMRI time series for probabilistic testing of functional connectivity and brain graphs [Internet]. Neuroimage 2015; Available from: http://eutils.ncbi.nlm.nih.gov/entrez/eutils/elink.fcgi?dbfrom=pubmed&id=25944610&retmode=ref&cmd=prlinks

14. Hallquist MN, Hwang K, Luna B: The nuisance of nuisance regression: spectral misspecification in a common approach to resting-state fMRI preprocessing reintroduces noise and obscures functional connectivity. Neuroimage 2013; 82:208–225

15. Fischl B, Salat DH, Busa E, et al.: Whole brain segmentation: automated labeling of neuroanatomical structures in the human brain. Neuron 2002; 33:341–355

16. Pruim RHR, Mennes M, van Rooij D, et al.: ICA-AROMA: A robust ICA-based strategy for removing motion artifacts from fMRI data. Neuroimage 2015; 112:267–277

17. Pruim RHR, Mennes M, Buitelaar JK, et al.: Evaluation of ICA-AROMA and alternative strategies for motion artifact removal in resting state fMRI. Neuroimage 2015; 112:278–287

18. Eickhoff SB, Paus T, Caspers S, et al.: Assignment of functional activations to probabilistic cytoarchitectonic areas revisited. Neuroimage 2007; 36:511–521

19. Roy AK, Shehzad Z, Margulies DS, et al.: Functional connectivity of the human amygdala using resting state fMRI. Neuroimage 2009; 45:614–626

20. Gabard-Durnam LJ, Flannery J, Goff B, et al.: The development of human amygdala functional connectivity at rest from 4 to 23 years: a cross-sectional study. Neuroimage 2014; 95:193–207

21. Jalbrzikowski M, Larsen B, Hallquist MN, et al.: Development of White Matter Microstructure and Intrinsic Functional Connectivity Between the Amygdala and Ventromedial Prefrontal Cortex: Associations With Anxiety and Depression. Biol Psychiatry 2017; 82:511–521

22. Lancaster JL, Tordesillas-Gutierrez D, Martinez M, et al.: Bias between MNI and Talairach coordinates analyzed using the ICBM-152 brain template. Hum Brain Mapp 2007; 28:1194–1205