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1. Supplementary Methods

1.1. Assessment for regional associations between predominant epileptic zone and impaired neuronal synchrony

The regional distribution of an epileptiform events in each AD-EPI+ patient was localized to one of three hemispheric locations: (1) frontal: frontal lobe regions located anterior to the central sulcus; (2) temporal: temporal lobe regions anterior to the temporo-parietal-junction; (3) parietaloccipital: parietal and occipital lobe regions located posterior to the central sulcus. In each AD-Epi+ patient, we determined the region with the most prominent epileptic activity reported either LTM-EEG and/or in MEG-EEG and was identified it as the predominant epileptic zone (Extended Data Fig.3). One AD-EPI+ patient was identified with generalized subclinical epileptiform spikes in LTM-EEG and without a specific predominant zone. We excluded this subject from our regional analyses described below. To examine the regional associations between the predominant epileptic zone and the neuronal synchrony deficits we first defined whether a given AD-EPI+ patient (n=19, after excluding one patient) falls within the 1st quartile of alpha-hyposynchrony and the 4th quartile of delta-theta hypersynchrony, based on the same regional classification of three zones. Identifying the quartiles that are most deviant from the normal distribution in alpha hyposynchrony and deltatheta hypersynchrony allowed us to identify the subjects who may show the largest abnormality of frequency-specific synchrony deficit in each ROI. Specifically, we first defined the zone (i.e. frontal, temporal, or parietal-occipital, in each hemisphere) to which each of the 10 voxel-level ROIs as defined by the most affected alpha and delta-theta imaginary coherence patterns (Supplementary table 2). We examined the distribution of alpha and delta-theta imaginary coherence estimations for each of the 10 ROIs and identified the patients who falls in the 4th quartile of this distribution. Next, for each regional level (out of 6 zones), we determined whether a given subject is 'abnormal' based on the criteria whether they fell into the 4th quartile in any of the ROIs categorized into that particular zone as shown in Supplementary table 3. Next, we generated frequency tables for each predominant epileptic zone marking the subjects who identify with it as 'the predominant epileptic zone' and the subjects who show an 'abnormal' neuronal synchrony within that zone. We used Fisher's exact test to examine the statistical significance of these regional level associations (Supplementary table 5).

1.2. Neuropsychological assessments

Executive function: Set shifting or mental flexibility was assessed by modified Trail Making test.¹ The modified Trail Making test requires the patient to draw lines linking items marked on paper and serially alternate between numbers and days of the week for a period of 120 seconds. The number of correct connections and time taken for the task were recorded. To adjust for the fact that some patients do not complete the task within the required time window of 120 seconds, the dependent measure was calculated as the number of correct connections made per second. Cognitive control was assessed by the Stroop tests ²⁻³. lexical fluency, was assessed with 'D-words', in which patients generate as many words as possible that are not proper nouns within 60 seconds beginning with the letter 'D'^{4.5}. A nonverbal counterpart of fluency comprises design fluency ¹, in which patients are required to use 4 lines to connect the dots within boxes each containing five dots, creating a unique pattern each time. We recorded the number of D-words, animals and patterns patients generated, within 60 seconds. Phonological short-term memory was assessed by digit span forward, and verbal working memory was assessed by digit span backward.

Memory: Verbal episodic memory was evaluated with the California Verbal Learning Test–Short Form (CVLT), which includes a list of 9-item words, presented over 4 learning trials ⁶. Immediate (30 seconds) and delayed (10 minutes) CVLT were assessed by free recall of the list at 30-seconds and 10-minutes intervals respectively. The correct number of items recalled, out of 9 were recorded. Visual memory was assessed by asking the patients to draw the Benson figure2 from memory after a 10-minute delay, and scored on a 17 point scale².

Language: Confrontation naming was assessed with a 15-item short form of the Boston Naming Test ^{8.9}. The number of correctly named items was recorded out of a total score of 15. Repetition was assessed by having participants repeat 3 phonemically complex sentences. Verbal agility was evaluated by having participants rapidly articulate a multi-syllabic word and was measured as the number of repetitions completed correctly within 5 seconds. Category fluency, was assessed with the ability to generate a list of items within a given category, in which patients generated as many as possible names of animals within 60 seconds ^{4.5}. Surface dyslexia was tested by having subjects read 6 irregular words and measured as the number correct out of 6. Syntax comprehension was measured using a subset of 5 items from the Boston Diagnostic Aphasia Evaluation for which the examiner read a sentence aloud, and the participant had to select from among 4 options the picture that best matched the sentence.

Visuospatial: Subjects were asked to copy a complex figure (Benson figure) as the object of visual construction and the accuracy was scored on a 17 point scale². The Number Location subtest of the Visual Object Space Perception (VOSP)¹⁰ test required the participant to precisely locate a stimulus on a two-dimensional plane, requiring dorsal-stream ("where") visual processing and scored out of 10. The face matching subtest of the Comprehensive Affect Testing System (CATS)¹¹ is a ventral-stream task involving 12 trials where the participant determined whether two faces are the same or different.

Emotion naming: The affect matching subtest of the CATS¹¹ contained 16 trials where the participant was shown a photo of an emotional face and required to select the correct label from a list (i.e. 'happy', 'sad', 'angry', 'frightened', 'surprised', 'disgusted' or 'neutral').

1.3. Magnetic Resonance image acquisition and analysis

Structural brain images were acquired from all participants using a unified MRI protocol on a 3 Tesla Siemens MRI scanner at the Neuroscience Imaging Center at UCSF and were used to generate invidualized head models for source space reconstruction of MEG sensor data. The structural MRI scans were also used in the clinical evaluations of patients with AD to identify the pattern of grey matter volume loss to support the diagnosis of AD.

2. Supplementary Figures

2.1. Supplementary figure.1



Subclinical epileptiform activity in patients with AD: Distribution of regional patterns of subclinical epileptiform activity detected from the two modalities of LTM-EEG and MEG-EEG for each AD-EPI+ patient (A). Patients 6, 8 and 19 had bilateral localization of epileptiform activity. Patients 6-9 had epileptiform activity detected in both modalities. The number of subjects detected for positive subclinical epileptiform activity for six zones defined across both hemispheres, from each modality (B). One AD-EPI+ patient (patient 20) was identified with generalized subclinical epileptiform activity without a specific predominant region and is not represented here. Abbreviations: AD, Alzheimer's disease; AD-EPI+, AD patients with epileptiform activity; L, left; LTM-EEG, extended/long-term electroencephalography; MEG-EEG, magnetoencephalography with simultaneous EEG; R, right.

2.2. Supplementary figure. 2



Frequency specific functional connectivity patterns in patients with AD vs. controls: The strength of functional connectivity is depicted in global imaginary coherence (IC), within alpha (8-12 Hz) and delta-theta (2-8 Hz) frequency oscillation bands, as group averages in the full cohort of AD patients (A and C). In a direct voxelwise comparison against age-matched controls, AD patients showed reduced strength of IC within alpha band (B) and enhanced strength of IC within delta-theta band (D). Brain renderings in subplots a and c depict the average IC scores. Brain renderings in subplots b and d depict the t-maps derived from voxelwise comparisons. All images are thresholded with a cluster correction of 30 voxels (P<0.01) and at 5% FDR. (n=50, patients with AD; n=35 age-matched controls). Abbreviations: AD, Alzheimer's disease; IC, imaginary coherence.



Regional patterns of neuronal synchrony deficits in AD-EPI+ patients categorized according to the modality of epileptiform activity detection. Brain renderings show statistical comparisons of IC between AD-EPI+ patients vs. age-matched controls. From left to right, the columns represent subsets of AD-EPI+ patients depending on whether their epileptiform events were detected in LTM-EEG-only or in M/EEG-only or in both LTM-EEG and M/EEG, respectively. Subplot A shows that reductions in alpha synchrony can be detected in comparable distribution of posterior temporoparietal and occipital cortices in each subset of patients against controls. Subplot B shows that enhanced delta-theta synchrony is detected in a comparable distribution of bilateral frontal and parietal cortices in each of the subset of AD-EPI+ patients. Each brain rendering depicts the t-maps from voxelwise comparison of global imaginary coherence. The color maps are thresholded with a cluster correction of 20 voxels (P<0.05) and at 5% FDR. Abbreviations: AD, Alzheimer's disease; AD-EPI-, AD patients without epileptiform activity; AD-EPI+, AD patients with epileptiform activity.

2.4. Supplementary figure. 4



Frequency specific functional connectivity patterns AD-EPI+ vs. AD-EPI-: In a direct voxelwise comparison, AD-EPI+ patients showed reduced strength of IC within alpha band (A) and enhanced strength of IC within delta-theta band (B), compared to AD-EPI- patients. Brain renderings depict the t-maps derived from voxelwise comparisons. All images are thresholded with a cluster correction of 30 voxels (P<0.01) and at 5% FDR. (n=50, patients with AD; n=35 age-matched controls). Abbreviations: AD, Alzheimer's disease; IC, imaginary coherence.

2.5. Supplementary figure. 5







Regional associations between the predominant epileptiform activity zone and neural synchronization deficits in AD-EPI+ patients: The predominant epileptiform zone of subclinical epileptiform activity in each AD-EPI+ patient was identified from LTM-EEG and/or MEG-EEG recordings as falling into one of three hemispheric regions as frontal, temporal, or parietal-occipital (A). The subplot (B) depicts the anatomical locations of the most affected five ROIs in each frequency band (numbered locations as 1-5 on brain renderings for alpha and delta-theta). Around

each of these locations, we illustrate the distribution of subjects in the extrema (the 1st quartile in alpha and the 4th quartile in delta-theta synchrony) also depicting each subject's predominant epileptic zone identified with the same colored symbols in subplot-(a). For example, the lowest ROI region of reduced alpha synchrony was left inferior temporal cortex (number 1 in alpha brain renderings), and among the subjects who belonged to the lowest quartile of this distribution, one was identified with left temporal lobe as the predominant epileptic zone (open blue circle), while the others were identified with left frontal (2, open purple circles), right frontal (1, shaded purple) and right parietal-occipital (1, shaded green) regions as their predominant epileptic zone. Collectively, this overlap illustrated no specific spatial relationships between predominant epileptic zone and neuronal synchrony deficits. One AD-EPI+ patient (patient 20), identified with generalized subclinical epileptiform activity without a specific predominant region is not included in these analyses. The placements of subjects on the brain renderings represent symbolic regional depictions and do not repent exact coordinates of epileptiform spikes. The placements of ROIs on brain renderings are approximate representations and do not depict the exact brain coordinates which are given in Supplementary table 2. Abbreviations: AD, Alzheimer's disease; AD-EPI+, AD patients with epileptiform activity; L, left; R, right; ROI, region-of-interest.



Longitudinal changes in MMSE and PCA components of imaginary coherence predicting MMSE slopes: Estimates from a linear mixed-effects model of longitudinal change in MMSE showed a significantly steeper slope in AD-EPI+ patients (A). These associations were significant after including age and education as covariates into the models (F=4.38, P=0.04). Shaded areas are 95% confidence intervals. The first seven components from a principal component analysis of imaginary coherence (IC) data matrix combining the alpha and delta-theta IC in the full cohort of patients with AD and controls B). Residuals from the multiple regression model using the 1st two principal components of the IC matrix predicting the MMSE slopes (C). Abbreviations: AD, Alzheimer's disease; AD-EPI–, AD patients without epileptiform activity; AD-EPI+, AD patients with epileptiform activity; IC, imaginary coherence; MMSE, Mini Mental State Examination; PCA, principal component analysis.

3. Supplementary Tables

Patient number	Autopsy [‡]	CSF	Amyloid§	FDG¶
1	Confirmed	-	-	-
2	Confirmed	-	-	-
3	Confirmed	-	-	-
4	Confirmed	-	-	-
5	Confirmed	-	Positive	Positive
6	Confirmed	-	Positive	Positive
7	Confirmed	-	Positive	Positive
8	Confirmed	-	Positive	Positive
9	Confirmed	-	Positive	Positive
10	Confirmed	-	Positive	Positive
11	Confirmed	-	Positive	Positive
12	Confirmed	-	Positive	Positive
13	Confirmed	-	Positive	Positive
14	Confirmed	-	Positive	Positive
15	-	Aβ42=125.0 t-Tau=559.4 p-Tau= 82.0 [†] Aβ42-Tau Index=0.14 [†]		
16	-	Aβ42=240.3 t-Tau=1090.55 p-Tau=142.1 [†] Aβ42-Tau Index=0.16 [†]		
17	-	Aβ42=182.7 t-Tau=528.5 p-Tau=90.0 [†] Aβ42-Tau Index=0.21 [†]	Positive	Positive

3.1. Supplementary table 1: Biomarkers of Alzheimer's disease patients

Patient number	Autopsy [‡]	CSF	Amyloid§	FDG¶
18	-	Aβ42=294 t-Tau=833.65 p-Tau=113.55 [†] Aβ42-Tau Index=0.24 [†]	Positive	Positive
19	-	Aβ42=210.3 t-Tau=504.3 p-Tau=79.8 [†] Aβ42-Tau Index=0.25 [†]		
20	-	Aβ42=399.5 t-Tau=527.6 p-Tau=70.5 [†] Aβ42-Tau Index=0.46 [†]		
21	-	Aβ42=399.3 t-Tau=441.8 p-Tau=68.6 [†] Aβ42-Tau Index=0.52 [†]		
22	-	Aβ42=473.7 t-Tau=366.6 p-Tau=74.75 [†] Aβ42-Tau Index=0.70 [†]		
23	-	Aβ42=129 [¥] t-Tau=243 [¥] p-Tau=55 [¥]	Positive	Positive
24	-	Aβ42=338.75 t-Tau=348.5 p-Tau=66.7 [†] Aβ42-Tau Index=0.52 [†]	-	-
25	-	Aβ42=379.4 t-Tau=604.5 p-Tau=91.5 [†] Aβ42-Tau Index=0.4 [†]	-	-
26	-	Abeta42 low, p-tau high. Interpretation: consistent with AD ^{\$}	-	-
27	-	Abeta42 borderline low, p-tau high. Interpretation: consistent with AD ^{\$}	-	-
28	-	-	Positive	Positive
29	-	-	Positive	Positive
30	-	-	Positive	Positive
31	-	-	Positive	Positive
32	-	-	Positive	Positive
33	-	-	Positive	Positive
34	-	-	Positive	Positive

Patient number	Autopsy [‡]	CSF	Amyloid§	FDG¶
35	-	-	Positive	Positive
36	-	-	Positive	Positive
37	-	-	Positive	Positive
38	-	-	Positive	Positive
39	-	-	Positive	Positive
40	-	-	Positive	Positive
41	-	-	Positive	Positive
42	-	-	Positive	Positive
43	-	-	Positive	Positive
44	-	-	Positive	Positive
45	-	-	Positive	Positive
46	-	-	Positive	Positive
47	-	-	Positive	Positive
48	-	-	Positive	Positive
49	-	-	Positive	Positive
50	-	-	Positive	Positive

Abbreviations: $A\beta 42 = amyloid-\beta$ peptide ending in amino acid residue 42; CSF = cerebrospinal fluid; L = left; MRI = magnetic resonance imaging; p-Tau = tau phosphorylated at threonine 181; R = right; t-Tau = total tau.

‡ Alzheimer's disease was confirmed by autopsy according to National Institute on Aging– Reagan Institute criteria. [†]Values supporting a diagnosis of Alzheimer's disease are p-Tau level >61 pg/ml and Aβ42-Tau Index <1.0 (Athena Diagnostics).

¥ Values supporting a diagnosis of Alzheimer's disease are Aβ42 level <192 pg/ml, t-Tau level >93 pg/ml, and p-Tau level >23 pg/ml (Alzheimer's Disease Neuroimaging Initiative Biomarker Core at the University of Pennsylvania).

\$ Values supporting a diagnosis of Alzheimer's disease are indicated in the comments. (Alzheimer's Disease Neuroimaging Initiative Biomarker Core at the University of San Diego).

§ Positron emission tomography agent was ¹⁸F-AV-45 for patients 17, 18, 33, 34, 36, 37, 40 and 41, and ¹¹C-Pittsburgh compound B for the remainder of the patients.

¶ Positron emission tomography imaging with ¹⁸F-fluorodeoxyglucose (¹⁸F-FDG) showed patterns of hypometabolism consistent with Alzheimer's disease.

	Anatomic region	MNI coordinate	Zone category* (defined for regional epileptiform activity)
Alpha (8-12Hz) frequency ROIs			
1	L inferior temporal cortex	-60 -35 -25	L Temporal
2	R posterior superior parietal cortex	10 -40 75	R Parietal/occipital
3	L occipito-parietal cortex	-40 -85 15	L Parietal/occipital
4	L posterior superior parietal cortex	-30 -70 60	L Parietal/occipital
5	R occipito-parietal cortex	50 -75 15	R Parietal/occipital
Delta-theta (2-8 Hz) frequency ROIs			
1	R middle temporal cortex	65 -5 -25	R Temporal
2	Right posterior superior parietal cortex	45 -80 5	R Parietal/occipital
3	Left angular gyrus	-45 -75 35	L Parietal/occipital
4	Left dorsolateral prefrontal cortex	-30 35 45	L Frontal
5	Right dorsomedial frontal cortex	5 35 55	R Frontal

3.2. Supplementary table 2: MNI coordinates of the voxel-level ROIs

*Zone category indicates one of the three zones per hemisphere identified as frontal, temporal and parietal/occipital which were defined to localize subclinical epileptiform activity in AD-EPI+ patients.

Variable	AD-EPI-	AD-EPI+	Р
Episodic memory function			
Visual free recall (Benson 10 minutes)	4.6 ± 4.2	4.6 ± 3.8	0.997
Short delay verbal memory (CVLT 30 seconds)	3.7 ± 2.3	3.6 ± 2.4	0.977
Verbal free recall (CVLT 10 minutes)	2.0 ± 2.3	2.2 ± 3.0	0.788
Executive function & working memory			
Design Fluency	5.5 ± 2.8	6.4 ± 4.7	0.462
Information processing speed (Stroop color naming)	43.9 ± 23.9	51.2 ± 22.0	0.405
Cognitive control (Stroop Inhibition)	19.6 ± 13.9	20.6 ± 17.4	0.853
Verbal working memory (Digit span forward)	5 (4 – 5)	5 (4-7)	0.934
Attention (Digit span backward)	3 (2 – 3)	3 (3 – 4)	0.114
Set shifting (Modified trails – speed)	0.2 ± 0.2	0.2 ± 0.2	0.755
Verbal learning (CVLT total score)	16.9 ± 6.1	17.0 ± 5.4	0.949
Language function			
Reading irregular words	6 (6 – 6)	6 (5 – 6)	0.065
Syntax comprehension	3.3 ± 1.4	3.3 ± 1.2	0.925
Verbal Agility	4.2 ± 1.6	4.1 ± 2.1	0.802
Boston Naming Test	13 (11 -14)	13 (10 – 14)	0.627
Lexical Fluency (D words/1 minute)	8.6 ± 4.6	10.9 ± 5.8	0.147
Category Fluency (Animals/1 minute)	11.5 ± 5.9	11.1 ± 5.5	0.789
Repetition	3.5 ± 1.3	3.4 ± 1.7	0.903
Visuospatial function			
Face discrimination (CATS – face matching)	11.1 ± 1.1	10.4 ± 1.4	0.091
Visuoconstruction (Benson copy)	10.4 ± 4.7	10.1 ± 5.2	0.873
Location discrimination (VOSP number location)	6.1 ± 2.6	5.9 ± 2.7	0.822
Calculations	2.6 ± 1.5	2.9 ± 1.3	0.585
<i>Emotion naming</i> (CATS – affect matching)	12.4 ± 1.6	11.0 ± 2.4	0.038

3.3. Supplementary table 3: Neuropsychological test performance in patients with AD*

* Plus-minus values are means ±SD and remainder of values are medians with interquartile ranges in parentheses. MMSE=Mini-Mental State Examination; CDR=Clinical Dementia Rating; CDR-SOB=CDR Sum of Boxes; CVLT=California Verbal Learning Test containing 9 items; CATS=Comprehensive Affect Testing System; VOSP=Visual Object and Space Perception.

Cognitive function	AD-EPI- (LS means and 95% confidence interval)	AD-EPI+ (LS means and 95% confidence interval)	F value	Р
MMSE	21.62 (20.23 – 23.01)	$ 18.81 \\ (17.15 - 20.47) $	4.38	0.040
Executive (composite)	-2.57 (-2.922.21)	-3.40 (-3.822.98)	6.80	0.013
Memory (composite)	3.24 (2.50 - 3.98)	3.37 (2.37 - 4.37)	0.84	0.365
Visuospatial (modified Rey)	10.63 (9.29 - 11.97)	9.37 (7.55 - 11.18)	0.11	0.744
Language (repetition)	3.42 (2.98 - 3.87)	3.22 (2.64 - 3.80)	0.25	0.621

3.4. Supplementary table 4: Longitudinal cognitive functions in AD-EPI+ and AD-EPI-

The lease square means (LS means) and confidence intervals are estimated from models including age and education as covariates. Executive composite was computed as average Z-score of lexical fluency, category fluency, digit span forwards and digit span backwards and CVLT learning. Memory composite was computed as the average score of CVLT short delay (30 s) recall and long delay (10 minutes) recall. Abbreviations: CVLT=California Verbal Learning Test containing 9 items; CATS=Comprehensive Affect Testing System; VOSP=Visual Object and Space Perception.

3.5. Supplementary table 5: Regional associations between predominant epileptic zone and most affected ROIs detected in neuronal synchrony estimations

No. (%) of subjects in each predominant epileptic zone	No. (%) of subjects with abnormal neuronal synchrony within the predominant epileptic zone	No. (%) of subjects with abnormal neuronal synchrony external to the predominant epileptic zone	P value*
Left Temporal 8 (42.1 %)	1 (20.0 %)	4 (80.0 %)	0.227
Left Frontal 2 (10.5 %)	2 (33.3 %)	4 (44.7 %)	0.088
Right Temporal 4 (21.1 %)	2 (40.0 %)	3 (60.0 %)	0.235
Right Frontal 3 (15.8 %)	1 (20.0 %)	4 (80.0 %)	0.470
Right Parietal/Occipital 4 (21.0 %)	2 (16.7 %)	10 (83.3 %)	0.358

*P values are from Fisher's exact test.

Number of subjects and % estimates are calculated for the n=19 AD-EPI+ patients. One AD-EPI+ patient who was identified with generalized epileptiform activity was excluded from this analysis.

REFERENCES

- Delis DC, Kramer JH, Kaplan E, Holdnack J. Reliability and validity of the Delis-Kaplan Executive Function System: an update. *J Int Neuropsychol Soc*. Mar 2004;10(2):301-3. doi:10.1017/S1355617704102191
- Golden CJ. Stroop Color and Word Test: A manual for clinical and experimental uses. Stoelting Co; 1978.
- 3. Golden CJ. Stroop Color and Word Test: Revised examiner's manual. Stoelting Co; 2002.
- Benton AL, Hamsher Kd, Sivan AB. *Multilingual Aphasia Examination : Third Edition*. The Psychological Corporation; 1994.
- Spreen O, Benton AL. Neurosensory Center Comprehensive Examination for Aphasia. Neuropsychology Laboratory, University of Victoria; 1977.
- 6. Delis DC, Kramer JH, Kaplan E, Ober BA. *California Verbal Learning Test Second Edition, Adult Version.* The Psychological Corporation; 2000.
- Possin KL, Laluz VR, Alcantar OZ, Miller BL, Kramer JH. Distinct neuroanatomical substrates and cognitive mechanisms of figure copy performance in Alzheimer's disease and behavioral variant frontotemporal dementia. Research Support, N.I.H., Extramural Research Support, Non-U.S. Gov't. *Neuropsychologia*. Jan 2011;49(1):43-8.

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doi:10.1016/j.neuropsychologia.2010.10.026
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- Kaplan EF, Goodglass H, Weintraub S. *The Boston Naming Test : Second Edition*. Lippincott Williams and Wilkins; 2001.
- Mack WJ, Freed DM, Williams BW, Henderson VW. Boston Naming Test: shortened versions for use in Alzheimer's disease. Research Support, U.S. Gov't, P.H.S. *J Gerontol*. May 1992;47(3):P154-8.

- 10. Warrington EK, James M. *The Visual Object and Space Perception Battery*. Thames Valley Test Company; 1991.
- 11. Froming K, Levy M, Schaffer S, Ekman P. *The comprehensive affect testing system*. psychology software, Inc; 2006.