

1 Online only supplementary material

2 **Supplementary Table 1: Neuropsychological Tests**

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Domain	Test
General intelligence, semantic memory, and language	National Adult Reading Test (NART) [1]
	Camel and Cactus Test (C&CT) [2]
	Graded Naming Test (GNT) [3]
	Wechsler Abbreviate Scale of Intelligence (WASI/WASI-II) Vocabulary and Similarities [4]
executive function	Wechsler Memory Scale III (WMS-III) digit span [5]
	Delis-Kaplan Executive Function System (DKEFS) [6] Trail Making Test and Verbal Fluency
visuospatial and motor function	Rey-Osterrieth Complex Figure Test (ROCFT) [7] copy
	DKEFS visual scanning; motor speed [6]
	Visual Object and Space Perception (VOSP) cube analysis; dot counting; position discrimination [8]
episodic memory	WMS-III (immediate and delayed verbal recall: Logical Memory I; II; Word List I; II; recognition) [5]
	ROCFT [7] (immediate; delayed visual recall)
	Warrington Recognition Memory Test (faces; words) (RMT) [9]
	Warrington Topographical Memory test (scenes) (RMT) [10]
	Doors and People test (D&P) Names: verbal recognition; People: verbal recall; Doors: visual recognition; Shapes: visual recall [11]
Depression and anxiety	Hospital Anxiety and Depression Scale (HADS) [12]

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1 **S1. Acute clinical MRI ratings**

2 Acute clinical scans were available for 23/24 patients. T2 signal intensity and volume were
3 evaluated in these MRIs. Diffusion was evaluated in 17 patients. The consultant
4 neuroradiologist (FS) graded T2 signal intensity (1 = normal, 2 = high, 3 = very high), volume
5 (1 = very atrophic, 2 = mildly atrophic, 3 = normal, 4 = enlarged, 5 = very enlarged) and
6 diffusion (1 = normal, 2 = facilitated, 3 = restricted) of both left and right hippocampi.

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1 **S2. Research Scan Procedures**

2 Image acquisition for research scans was conducted on a Siemens 3T Trio system using a 32-
3 channel head coil (University of Oxford Centre for Clinical Magnetic Resonance Research).

4 We acquired 3D T1-weighted structural MRIs (MPRAGE sequence; echo time = 4.7ms,
5 repetition time = 2040ms, 8° flip angle, field of view = 192mm, voxel size = 1 x 1 x 1mm)
6 and resting-state BOLD-weighted fMRI data (Gradient Echo EPI sequence; 180 volumes;
7 slice thickness = 3.5mm, echo time = 28ms, repetition time = 2410ms, 89° flip angle, field of
8 view = 192mm, voxel size = 3 x 3 x 3.5mm). Subjects were instructed to lay still, watch a
9 fixation cross on the in-scanner projector and not to fall asleep.

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1 **S3. Volumetry**

2 Manual delineation of MTL structures was conducted by a trained researcher (ARF) using ITK-
3 SNAP [13] (protocol:
4 https://www.ndcn.ox.ac.uk/files/research/segmentation_protocol_medial_temporal_lobes.pdf
5). Segmentation procedures were based on established and published atlases and protocols
6 [14,15]. Intra-rater reliability was measured using intra-class coefficient correlations on a
7 random selection of 24 hemispheres (12 patients; 12 controls) segmented twice by ARF with
8 an interval of at least 4 months between segmentations. The intra-rater reliability results were
9 at least as good those reported in the literature for manual segmentations of the MTL [16] for
10 the hippocampus (0.982), amygdala (0.803), and the temporopolar (0.96), perirhinal (0.904),
11 entorhinal (0.816), and parahippocampal cortices (0.93). Automated segmentation was used
12 for the brainstem, thalamus, caudate nucleus, putamen, and pallidum. Volumes were
13 segmented in each hemisphere and corrected for total intra-cranial volume (TIV) derived from
14 the unified segmentation procedure [17] in SPM12. For each patient, volumes were expressed
15 as z scores based on the mean volumes and SDs of individually age-matched (+/- 10 years of
16 age) controls (number of controls per patient: mean = 22; SD = 7).

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1 **S4. VBM**

2 To identify GM volume discrepancies between groups at a whole-brain level, the T1-weighted
3 images were analyzed with VBM ([18]) using the Statistical Parametric Mapping software
4 (SPM12 v7219; <http://www.fil.ion.ucl.ac.uk/spm/software/spm12>) running in Matlab R2017b.
5 Images were examined for scanner artefacts, reoriented to have the same point of origin
6 (anterior commissure) and spatial orientation, bias-corrected to remove intensity non-
7 uniformities, and segmented into GM, white matter (WM), and cerebrospinal fluid (CSF),
8 using the unified segmentation procedure [17]. The diffeomorphic anatomical registration
9 through the exponentiated lie algebra (DARTEL) toolbox was applied to all participants' GM,
10 WM, and CSF, to refine inter-subject registration, and study-specific GM templates were
11 generated [19]. After affine registration of the GM DARTEL templates to the tissue probability
12 maps in MNI (Montreal Neurological Institute, Quebec, Canada) space, non-linear warping of
13 GM images was performed to the DARTEL GM template in MNI space. Voxel values in the
14 tissue maps were modulated by the Jacobian determinant (calculated during spatial
15 normalization), with modulated GM images reflecting tissue volume. These images (voxel
16 size: 1 mm³) were smoothed using a standard Gaussian filter (8 mm FWHM). Analysis was
17 also conducted on images smoothed with a lower Gaussian filter (4 mm FWHM) to increase
18 spatial specificity within MTL structures. We compared GM volume between groups,
19 including age, sex, and TIV as second-level covariates. We report clusters surviving FWE-
20 correction ($p < .05$) at peak-voxel level over $p < .001$, as well as clusters surviving correction
21 for non-stationary smoothness [20] and FWE-correction for cluster size ($p < .05$). The average
22 volumes of these clusters were extracted for all participants using the MarsBaR toolbox [21]
23 (<http://marsbar.sourceforge.net/>), were residualized for age, sex, and TIV, and entered in
24 bivariate correlation analyses with composite memory scores.

1 **S5. Resting-state fMRI preprocessing**

2 Resting-state fMRI preprocessing and connectivity analyses were conducted using the CONN
3 toolbox version 17.f (<https://www.nitrc.org/projects/conn>) [22] for 23/24 patients and 30/33
4 controls (4 datasets were discarded due to acquisition errors and/or movement). Functional
5 scans were spatially realigned to correct for interscan movement and were slice time-corrected.
6 The structural MRIs and the manually-delineated MTL structures were coregistered to the
7 functional images. Structural images were segmented and normalized along with functional
8 images to the MNI reference brain, followed by motion outlier detection (ART-based
9 scrubbing). Denoising including the anatomical component-based correction method
10 (CompCor) [23] was employed to remove sources of noise in the BOLD time series data,
11 deriving the principal components from the WM and CSF. WM, CSF and the six movement
12 parameters were included as first-level nuisance covariates. A temporal band pass filter (0.01-
13 0.1 Hz) was applied to this residual BOLD signal to remove motion artefacts, physiological
14 and other artefactual effects from the fMRI-signal. Images were smoothed using a 8 mm
15 Gaussian kernel.

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1 **S6. Resting-state functional connectivity (MVPA)**

2 MVPA assesses the multivariate pattern of pairwise connections between all voxels across
3 the whole brain by performing a principal component analysis (PCA) separately for each
4 voxel that characterizes its functional connectivity with the rest of the brain. In the first PCA
5 step, separately for each subject, a default number of 64 PCA components were retained
6 while characterizing each subject's voxel-to-voxel correlation structure. The resulting
7 component scores were then stored as first-level voxel-to-voxel covariance matrices for each
8 subject. In the second PCA step, separately for each voxel and jointly across subjects, the 5
9 strongest components were retained from a principal component decomposition of the
10 between-subjects variability in seed-to-voxel connectivity maps between this voxel and the rest
11 of the brain, in keeping with a conventionally used conservative 1:10 ratio between the
12 number of components extracted and the number of subjects ($n = 53$). Subsequently, second-
13 level analyses were performed to test for group differences in whole-brain connectivity by
14 means of an F-test across all MVPA components, comparing for each voxel the component
15 scores across the two groups (covariates: age, sex). The results for each voxel thus reflected
16 between-group differences in functional connectivity between this voxel and the rest of the
17 brain.

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1 **S7. Statistical Analysis**

2 Non-imaging statistical analyses were performed using SPSS (v.25.0, SPSS Inc). Variance
3 homogeneity was assessed using Levene's test, and normal distribution using the Shapiro-
4 Wilk test. Parametric (Student t-test; Welch t-test used when the assumption of homogeneity
5 of variances was violated) and non-parametric tests (when the assumption of normal
6 distribution was not met in a group: Mann-Whitney U for controls vs. patients, and Kruskal-
7 Wallis H for patients with bilateral vs. unilateral vs. no hippocampal atrophy) were used
8 appropriately. To compare neuropsychological test performance between groups, age-scaled,
9 standardized scores were used for independent samples comparisons. The relationship of
10 composite memory scores with GM volumes and functional connectivity measures was
11 investigated with bivariate correlation analyses, using Pearson r and Spearman's ρ (when
12 variables were not normally distributed). Correction for multiple testing for non-imaging data
13 was applied using the Holm-Bonferroni sequential correction method [24].

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1 **Supplementary Table 2: Patients' clinical details**

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Clinical Variables		Patients (n)	M	IQR
Acute clinical T2-weighted MRI abnormalities	HPC high T2 signal	19/23		
	HPC enlargement	8/23		
	abnormal diffusion	9/17 (facilitated)		
	extra-HPC MTL abnormalities	5/24 (ERC, AMG)		
	extra-MTL abnormalities	1/24 (high T2 signal in caudate nucleus) 4/24 (mild microangiopathic changes)		
Seizures	Seizure-free for over a year at time of research study	17/24		
	FBDS (acute)	4/19		
	GTCS	9/19		
	SPS	1/19		
	CPS	13/19		
	MCS	4/19		
	PCS	1/19		
Autoantibodies	LG11 only	14/24		
	LG11 + CASPR2	4/24		
	VGKCC	6/24		
	VGKCC-Ab titre at initial clinical presentation (pmol/L)		1353	1452
	acute MRI - antibody testing delay (days)		21	940
Immunosuppressive therapy	Oral steroids	22/24		
	PLEX	9/24		
	IVIG	18/24		
	symptom onset - immunosuppressive treatment onset delay (months)		3	6

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AMG: amygdala; CASPR2: anti-contactin-associated protein-like 2; CPS: Complex Partial Seizures; ERC: entorhinal cortex; FBDS: Faciobrachial dystonic seizures; GTCS: generalised tonic-clonic seizures; HPC: hippocampus; IQR: Interquartile Range; IVIG: intravenous immunoglobulin; LG11: anti-leucine-rich glioma-inactivated1; M: Median; MCS: myoclonic seizures; MTL: medial temporal lobe; PLEX: plasma exchange; SPS: Simple Partial Seizures; VGKCC: anti-voltage-gated potassium channel complex (the 6 patients that had positive VGKCC antibody titres presented prior to the availability of LG11 or CASPR2 antibody assays).

1 **S8. VGKCC patients**

2 The 6/24 patients that had not been tested for LGI1 or CASPR2 antibodies did not differ from
3 the other 18/24 patients in their acute clinical T2-weighted MRIs (signal intensity, volume,
4 diffusion of left and right hippocampus: all Us, $51 \geq U \geq 22$; all ps, p-corr > 0.999; occurrence
5 of extra-HPC abnormalities: $\chi^2 = 0.641$, $p = 0.423$) or post-acutely in any of the
6 neuropsychological tests administered (all Us, $54 \geq U \geq 14$; all ps, p-corr ≥ 0.231), in their
7 MTL and subcortical volumes (all Us, $53 \geq U \geq 24$; all ps, p-corr ≥ 0.999), in the mean GM
8 volumes of the clusters identified by VBM (residualized for age, sex, and TIV; all Us, $49 \geq U$
9 ≥ 36 ; all ps, p-corr > 0.999), or in the mean FC values of the clusters and ROIs identified in the
10 FC analyses (residualized for age, sex, and seed ROI volume; all Us, $48 \geq U \geq 32$; all ps, p-
11 corr > 0.999).

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1 **S9. Patients with ongoing seizures vs seizure-free patients**

2 Seizure history was recorded and the patient group was dichotomized into those with ongoing
3 seizures (< 1 year since last seizure) and those that had remained seizure-free for over 1 year
4 before participation. The group of 7 patients with ongoing seizures did not differ from the 17
5 patients that had been seizure-free for over a year in their acute clinical T2-weighted MRIs
6 (signal intensity, volume, diffusion of left and right hippocampus: all Us, $51 \geq U \geq 21$; all ps,
7 $p\text{-corr} > 0.999$; occurrence of extra-HPC abnormalities: $\chi^2 = 0.123$, $p = 0.726$) or post-acutely
8 in any of the neuropsychological tests administered (all Us, $59 \geq U \geq 21$; all ps, $p\text{-corr} \geq 0.561$),
9 in their MTL and subcortical volumes (all Us, $58 \geq U \geq 31$; all ps, $p\text{-corr} \geq 0.999$), in the mean
10 GM volumes of the clusters identified by VBM (residualized for age, sex, and TIV; all Us, 55
11 $\geq U \geq 39$; all ps, $p\text{-corr} > 0.999$), or in the mean FC values of the clusters or ROIs identified in
12 the FC analyses (residualized for age, sex, and seed ROI volume; all Us, $49 \geq U \geq 35$; all ps,
13 $p\text{-corr} > 0.999$).

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1 **S10. Patients with faciobrachial dystonic seizures**

2 The 4/24 patients that had presented with faciobrachial dystonic seizures in the acute phase of
3 the disease did not differ from the other 20/24 patients in their acute clinical T2-weighted MRIs
4 (signal intensity, volume, diffusion of left and right hippocampus: all Us, $34 \geq U \geq 19$; all ps,
5 $p\text{-corr} > 0.999$; occurrence of extra-HPC abnormalities: $\chi^2 = 2.273$, $p = 0.132$) or post-acutely
6 in any of the neuropsychological tests administered (all Us, $38 \geq U \geq 17$; all ps, $p\text{-corr} > 0.999$),
7 in their MTL and subcortical volumes (all Us, $40 \geq U \geq 13$; all ps, $p\text{-corr} \geq 0.925$), in the mean
8 GM volumes of the clusters identified by VBM (residualized for age, sex, and TIV; all Us, 40
9 $\geq U \geq 38$; all ps, $p\text{-corr} > 0.999$), or in the mean FC values of the clusters or ROIs identified in
10 the FC analyses (residualized for age, sex, and seed ROI volume; all Us, $34 \geq U \geq 20$; all ps,
11 $p\text{-corr} > 0.999$).

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1 **Supplementary Table 3: Correlations of memory scores with**
 2 **hippocampal and thalamic volumes (volumetry)**

Structure	Correlations with Memory Composite Scores					
	Visual Recall		Verbal Recall		Verbal Recognition	
	r	p	r	p	r	p
R HPC head	0.139	0.527	-0.072	0.737	-0.183	0.392
L HPC head	0.157	0.474	0.216	0.310	0.048	0.822
R HPC body	-0.065	0.767	0.079	0.712	0.186	0.385
R Thalamus	0.078	0.724	0.262	0.217	0.087	0.685
L Thalamus	0.047	0.832	0.245	0.249	0.015	0.943

3 Volumes in which patients showed significant volume reduction were entered in a series of bivariate correlation
 4 analyses with memory composite scores (averaged age-scaled standardized scores across subtests in which
 5 patients showed impaired performance at group level as compared with controls) for visual recall (ROCFT
 6 Immediate and Delayed Recall), verbal recall (WMS-III Logical Memory I and II, Word List I and II, D&P
 7 People), and verbal recognition (WMS-III Word List Recognition, RMT Words, D&P Names) across patients; r:
 8 Pearson correlation coefficient; p: significance values are presented at uncorrected levels for display purposes.
 9 All volumes were TIV-corrected and expressed as z-scores, based on the mean and SD of patients' individually
 10 age-matched controls (+/- 10 years of age).

1 **Supplementary Table 4: Correlations of memory scores with GM**
 2 **volumes (VBM clusters)**

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Smoothing (FWHM)	FWE-correction level	Structure	Correlations with Memory Composite Scores					
			Visual Recall		Verbal Recall		Verbal Recognition	
			r	p	r	p	r	p
4mm	peak	L HPC head	0.243	0.264	0.448	0.028	0.211	0.323
		R HPC body	0.051	0.816	0.180	0.401	0.020	0.926
		R HPC head	0.083	0.705	0.171	0.425	0.207	0.332
	cluster size	L HPC head/body	0.188	0.389	0.427	0.038	0.154	0.472
		R HPC head/body	0.028	0.899	0.122	0.569	0.018	0.935
		R Thal	0.286	0.186	0.370	0.075	0.273	0.197
		L/R PCC/PrCu	-0.117	0.594	0.073	0.736	-0.057	0.790
8 mm	peak	R HPC head/body	0.059	0.789	0.173	0.419	0.064	0.767
		L HPC head/body	0.227	0.298	0.438	0.032	0.161	0.454
		R MD Thal	0.250	0.250	0.360	0.084	0.262	0.217
	cluster size	L/R HPC/Thal	0.213	0.330	0.369	0.076	0.145	0.498
		L/R PCC/PrCu	-0.116	0.598	0.089	0.678	-0.084	0.696

4 The average GM volume from each VBM cluster (in which patients showed significant GM volume reduction)
 5 was extracted from each participant, residualised against age, sex, and TIV, and entered in a series of bivariate
 6 correlation analyses with memory composite scores (averaged age-scaled standardized scores across subtests in
 7 which patients showed impaired performance at group level as compared with controls) for visual recall (ROCFT
 8 Immediate and Delayed Recall), verbal recall (WMS-III Logical Memory I and II, Word List I and II, D&P
 9 People), and verbal recognition (WMS-III Word List Recognition, RMT Words, D&P Names) across patients.
 10 Left hippocampal volumes correlated at uncorrected levels with the verbal recall memory composite scores across
 11 patients; r: Pearson correlation coefficient; p: significance values of the bivariate correlations conducted are
 12 presented at uncorrected levels; HPC: Hippocampus; L,R: Left, Right; PCC: Posterior Cingulate Cortex; PrCu:
 13 Precuneus; MD: Mediodorsal; Thal: Thalamus.

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1 **Supplementary Table 5: Multiple step-wise linear regression analyses**

2 In an exploratory fashion, we assessed the proportion of the variance of patients' verbal
3 recall, verbal recognition, and visual recall memory composite scores that was explained by
4 the patient group's structural and functional abnormalities. We thus entered measures of these
5 abnormalities in a series of multiple step-wise linear regression analyses as independent
6 variables (default alpha level of 0.05 for entry to model and 0.1 for removal). Independent
7 variables were measures of patients' reduced FC between the right hippocampus and the
8 medial prefrontal cortex, the posteromedial cortex, the left hippocampus, the right
9 parahippocampal cortex, and the left temporoparietal cortex, as well as between the right
10 parahippocampal cortex and the left perirhinal cortex; they also included measures of volume
11 reduction in the right, left hippocampus, right and left thalamus, and the posteromedial
12 cortex. In order to examine all different measures of volume reduction in the aforementioned
13 regions (volumes segmented in native space, GM volumes of VBM clusters disclosed at
14 different levels of smoothing and FWE-correction), and in order to minimize
15 multicollinearity, 5 different analyses were conducted for each of those 3 scores, including
16 measures of functional abnormalities, along with measures of volume reduction disclosed by:
17 i) either volumetric measures (table 3); ii) or VBM clusters (cluster-size FWE correction,
18 smoothing at 4 mm FWHM; table 4); iii) or VBM clusters (cluster-size FWE-correction,
19 smoothing at 8 mm FWHM; table 4); iv) or VBM clusters (peak-level FWE-correction,
20 smoothing at 4 mm FWHM; table 4); v) or VBM clusters (peak-level FWE-correction,
21 smoothing at 8 mm FWHM; table 4). All 5 analyses for each of those 3 memory scores
22 showed that patients' reduced hippocampal FC explained the variance of their memory
23 scores.
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Memory Composite Score (Dependent Variable)		Model 1	Model 2
Verbal Recall (z)		R-L HPC FC (z-res)	R-L HPC FC (z-res), R HPC – R PHC FC (z-res)
	R ²	0.214	0.356
	F	5.71	5.52
	p	0.026	0.012
Verbal Recognition (z)		R-L HPC FC (z-res)	-
	R ²	0.466	
	F	18.3	
	p	< 0.0005	
Visual recall (z)		R HPC – Posteromedial Cortex FC (z-res)	-
	R ²	0.299	
	F	8.53	
	p	0.008	

1 z: age-scaled, standardized scores in memory tests in which patients showed impaired performance at group level
2 as compared with healthy controls; z-res: measures of functional abnormalities (reduced FC between the R HPC
3 and other regions) were residualized across participants against age, sex, and seed ROI volume; FC: functional
4 connectivity; R: right; L: left; HPC: hippocampus; PHC: parahippocampal cortex; -: the step-wise regression was
5 terminated in a single step.
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