ON-LINE APPENDIX

Antibody Testing

After 2014, screening for onco-neuronal antibodies was performed using semiquantitative immunoblots (EUROLINE PNS 12; DL 1111–1601–7 G; Euroimmun, Lüebeck, Germany) coated with recombinant antigen or antigen fragments (dilution: serum, 1:100; cerebrospinal liquor, 1:1). In parallel, immunocytochemistry was performed using Human Embryonic Kidney 293-cells with expression of antigens on the cell surface (IIFT: Autoimmune Enzephalitis Mosaik1, Euroimmun; FA 1120–1005-1; GAD65-IIFT, Euroimmun; FA 1022–1005-50) for NMDAR, CASPR, LGI1, GABAA, GABAB, AMPAR, and GAD65 autoantibodies (dilution, serum 1:10; cerebrospinal liquor, 1:1). Before 2014, detection of GAD antibodies in serum was performed using an anti-125I-GAD radioimmunoprecipitation assay (normal values, ≤1 U/mL; Wetherall Institute of Molecular Medicine, Oxford, UK; or Euroimmun). VGKC-complex antibodies were also examined by radioimmunoprecipitation assay (normal values, <100 picomolar; Wetherall Institute of Molecular Medicine or Euroimmun). Antibodies against LGI1 and CASPR2 were detected by indirect immunofluorescence using formalin-fixed human Embryonic Kidney 293-cells containing membrane bound LGI1 or CASPR2 (normal values, <1:10; all tests performed by Euroimmun). These tests were not performed before 2010, which is why 13 patients in the early VGKC group and 14 patients in the late VGKC group have not been tested for CASPR2 or LGI1.

On-line Table 1: Patient characteristics and antibody testing—early groups[a]

Study Group	ID	Sex	Age at MR Imaging (yr)	Time between Onset and Scan (mo)	Lateralization EEG	Immunotherapy before MR Imaging Acquisition	Antibody Status (at the Time of MR Imaging Acquisition)	Clinical Imaging
Early GAD	1	F	48	23	Left temporal	–	Neg. (S. and L.)	–
Early GAD	2	F	17	11	Right temporal	–	–	–
Early GAD	3	F	25	16	Left temporal	Steroids	Pos. (S. and L.)	–
Early GAD	4	M	26	3	Left temporal	–	–	T2-FLAIR acute: mesiotemporal hyperintensities left hemisphere
Early GAD	5	F	32	24	Right temporal	–	Pos. (S. and L.)	T2-FLAIR acute: normal
Early GAD	6	F	18	1	Right temporal	–	Pos. (S. and L.)	T2-FLAIR acute: volume increase and hyperintensities right amygdala and right hippocampus
Early GAD	7	F	43	8	Right temporal	–	Pos. (S. and L.)	T2-FLAIR acute: hyperintensities left hippocampus
Early GAD	8	F	24	5	Left temporal	–	Pos. (S.; L. not tested)	T2-FLAIR acute: hyperintensities and volume increase left mesiotemporal
Early GAD	9	F	42	5	Left temporal	–	Pos. (S.; L. not tested)	T2-FLAIR acute: hyperintensities left hippocampus and left amygdala
Early GAD	10	M	43	3	Temporal	Steroids	Pos. (S.; L. not tested)	T2-FLAIR acute: hyperintensities left hippocampus and left amygdala
Early GAD	11	F	49	5	Left temporal	–	Pos. (S.; L. not tested)	T2-FLAIR acute: hyperintensities and volume increase right mesiotemporal
Early GAD	12	M	32	2	–	Steroids, plasmapheresis	Pos. (S.; L. not tested)	T2-FLAIR acute: hyperintensities and slide atrophy of right hippocampus
Early GAD	13	M	35	5	Left temporal	–	Pos. (S.; L. not tested)	T2-FLAIR acute: hyperintensities and volume increase left hippocampus and amygdala
Early GAD	14	F	27	5	Temporal	–	Pos. (S. and L.)	T2-FLAIR acute: hyperintensities and volume increase right mesiotemporal
Early GAD	15	M	58	3	Right temporal	–	Neg. (S. and L.)	T2-FLAIR acute: slide hippocampal atrophy bilateral, no signal alterations
Early GAD	16	F	43	11	Left temporal	–	Pos. (S.; L. neg.)	T2-FLAIR acute: hyperintensities and volume increase left hippocampus and amygdala
Early GAD	17	F	26	16	–	–	Pos. (S.; L. not tested)	T2-FLAIR acute: slide volume increase of right amygdala, no signal alterations
Early GAD	18	F	28	0	Left temporal	–	Pos. (S. and L.)	T2-FLAIR acute: hyperintensities and volume increase of both amygdalae
Early GAD	19	M	45	12	Left temporal	–	–	T2-FLAIR acute: hyperintensities and volume increase of left amygdala and anterior hippocampus

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On-line Table 1: Continued

Study Group	ID	Sex	Age at MR Imaging (yr)	Time between Onset and Scan (mo)	Lateralization EEG	Immunotherapy before MR Imaging Acquisition	Antibody Status (at the Time of MR Imaging Acquisition)	Clinical Imaging
Early GAD	20	M	27	16	Left temporal	–	Neg. (S. and L.)	T2-FLAIR acute: hyperintensities and volume increase of left amygdala
Early GAD	21	F	21	22	Bilateral temporal	–	Pos. (S. and L.)	T2-FLAIR acute: bilateral hyperintensities in amygdalae and hippocampi
Early GAD	22	F	23	17	Normal	–	Pos. (S.; L. neg.)	T2-FLAIR acute: volume increase left amygdala and hyperintensities in left hippocampus
Early GAD	23	F	43	6	Left temporal	–	Pos. (S.; L. not tested)	T2-FLAIR acute: hyperintensities and volume increase of left amygdala and left hippocampus
Early VGKC	24	M	55	14	Normal	Steroids	Pos. (S.; L. not tested)	T2-FLAIR acute: normal
Early VGKC	25	M	60	3	Bilateral temporal	–	Pos. (S.; L. neg.)	T2-FLAIR acute: bilateral hyperintensities increase of amygdala and anterior hippocampus
Early VGKC	26	F	48	22	Left temporal	–	Pos. (S.; L. not tested)	T2-FLAIR acute: hyperintensities and volume increase left hippocampus
Early VGKC	27	M	68	9	Bilateral temporal	Steroids, immunoadsorption	Pos. (S.; L. not tested)	T2-FLAIR acute: bilateral hippocampal hyperintensities and volume increase
Early VGKC	28	F	53	11	Right temporal	–	Pos. (S.; L. not tested)	T2-FLAIR acute: hyperintensities and volume increase of right amygdala; atrophy of the right hippocampus
Early VGKC	29	M	73	11	Left temporal	–	Pos. (S. and L.)	T2-FLAIR acute: volume-increased amygdalae (right > left)
Early VGKC	30	M	70	5	Left temporal	–	Pos. (S. and L.)	T2-FLAIR acute: hyperintensity and volume increase of left amygdala; questionable atrophy right hippocampus
Early VGKC	31	M	67	5	Right temporal	–	Neg. (S.; L. not tested)	T2-FLAIR acute: narrowed hippocampi and left mesiotemporal hyperintensities
Early VGKC	32	F	20	10	Right temporal	Steroids	Pos. (S.; L. not tested)	–
Early VGKC	33	F	61	0	Left temporal	–	Pos. (S. and L.)	T2-FLAIR acute: bilateral hyperintensities and volume increase of amygdalae and anterior hippocampi
Early VGKC	34	M	69	12	Left temporal	–	Pos. (S.; L. not tested)	T2-FLAIR acute: questionable left mesiotemporal hyperintensity and volume increase
Early VGKC	35	M	61	1	Left temporal	Steroids, plasmapheresis	Pos. (S; L. neg.)	T2-FLAIR acute: left hippocampus with subtle atrophy
Early VGKC	36	F	23	0	Left temporal	–	Pos. (S.; L. not tested)	T2-FLAIR acute: questionable bilateral hyperintensity in the pulvinar thalami

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On-line Table 1: Continued

Study Group	ID	Sex	Age at MR Imaging (yr)	Time between Onset and Scan (mo)	Lateralization EEG	Immunotherapy before MR Imaging Acquisition	Antibody Status (at the Time of MR Imaging Acquisition)	Clinical Imaging
Early VGKC (CASPR2)	37	M	38	6	Left temporal	–	Pos. (S.; L. not tested)	T2-FLAIR acute: bilateral hyperintensities of amygdalae
Early VGKC (CASPR2)	38	M	82	23	Right temporal	–	Pos. (S. and L.)	T2-FLAIR acute: right mesiotemporal hyperintensities and volume increase (especially in amygdala)
Early VGKC (LGI1)	39	F	48	4	Right temporal	–	Neg. (S. and L.)	T2-FLAIR acute: hyperintensities and volume increase of right amygdala and hippocampus
Early VGKC (LGI1)	40	F	57	2	Right temporal	–	Pos. (S. and L.)	T2-FLAIR acute enlarged mesiotemporal structures, questionable changes in signal
Early VGKC (LGI1)	41	F	72	3	Normal	Steroids	Pos. (S. and L.)	–
Early VGKC (LGI1)	42	F	71	13	Normal	Steroids	Pos. (S.; L. not tested)	T2-FLAIR acute: signs of atherosclerotic encephalopathy, no mesiotemporal hyperintensities or volume alterations
Early VGKC (LGI1)	43	M	65	7	Bilateral temporal	Steroids, immunoadsorption	Pos. (S; L. neg.)	T2-FLAIR acute: microangiopathy
Early VGKC (LGI1)	44	M	62	4	No seizures documented	Steroids	Pos. (S.; L. not tested)	T2-FLAIR acute: prominent right amygdala with subtle hyperintensity
Early VGKC (LGI1)	45	M	76	6	Normal	Steroids, immunoadsorption, intravenous immunoglobulins	Neg. (S.; L. not tested)	T2-FLAIR acute: global brain atrophy; unspecific periventricular white matter lesions on FLAIR
Early VGKC (LGI1)	46	M	74	2	Left temporal	–	Pos. (S; L neg.)	–
Early VGKC (LGI1)	47	M	54	11	Right temporal	Steroids, intravenous immunoglobulins	Neg. (S. and L.)	T2-FLAIR acute: bilateral prominent and signal-enhanced amygdalae with signal enhancement; narrowed hippocampi
Early VGKC (LGI1)	48	M	53	7	Normal	–	Neg. (S.; L. not tested)	T2-FLAIR acute: unspecific white matter lesions

Note:—S. indicates in serum; L., in liquor; not tested, not tested at the time of the respective MR imaging scan; –, not conducted; Neg., negative; Pos., positive; ID, identification.

[a] Patient characteristics of all patient groups included in the study (early GAD-LE, early VGKC-LE, late GAD-LE, late VGKC-LE).

On-line Table 2: Patient characteristics and antibody testing—late groups[a]

Study Group	ID	Sex	Age at MR Imaging (yr)	Time between Onset and Scan (mo)	Lateralization EEG	Immunotherapy before MR Imaging Acquisition	Antibody Status (at the Time of MR Imaging Acquisition)	Clinical Imaging
Late GAD	49	M	29	112	Right temporal	Steroids, azathioprine	Pos. (S.; L. not tested)	Follow-up scan: bilateral abnormal mesiotemporal structures
Late GAD	50	F	32	53	–	Steroids	Pos. (S.; L. not tested)	Follow-up scan: hyperintense left amygdala
Late GAD	51	F	26	64	Left temporal	–	Pos. (S. and L.)	Follow-up scan: volume increase and slight hyperintensity of left amygdala and hippocampus
Late GAD	52	F	24	92	Bilateral temporal	–	Pos. (S.; L. not tested)	Follow-up scan: volume increase and hyperintensity of both amygdalae; hyperintensity of both hippocampi (left > right)
Late GAD	53	F	29	87	Bilateral temporal	–	Pos. (S.; L. not tested)	Follow-up scan: normal mesiotemporal structures
Late GAD	54	F	48	110	–	–	Not tested	–
Late GAD	55	F	31	72	Left temporal	Steroids	Neg. (S. and L.)	Follow-up scan: left mesiotemporal structures appear to be volume-increased
Late GAD	56	F	48	48	–	–	Not tested	–
Late GAD	57	F	21	42	Left temporal	–	Neg. (S. and L.)	Follow-up scan: volume-increased and hyperintense left amygdala
Late GAD	21	F	21	25	–	Steroids	Pos. (S.; L. not tested)	Follow-up scan: volume-increased amygdala (left > right)
Late GAD	2	F	23	83	Left temporal	Steroids, intravenous immunoglobulins	Pos. (S.; L. not tested)	Follow-up scan: atrophy right hippocampus
Late GAD	58	F	27	91	Bilateral temporal	Steroids	Not tested	Follow-up scan: bilateral hippocampal atrophy
Late GAD	3	F	26	27	Left temporal	Steroids	Pos. (S. ; L. not tested)	–
Late GAD	4	M	29	42	Bilateral temporal	Steroids, intravenous immunoglobulins, natalizumab	Pos. (S.; L. not tested)	–
Late GAD	5	F	34	56	–	Steroids, intravenous immunoglobulins	Not tested	Follow-up scan: normal
Late GAD	59	F	30	115	Left temporal	Steroids, intravenous immunoglobulins, immunoadsorption, plasmapheresis, cyclophosphamide	Not tested	Follow-up scan: hyperintensity and volume increase of right amygdala and hippocampus; hyperintensity left hippocampus
Late GAD	6	F	21	35	–	Steroids, immunoadsorption, cyclophosphamide, mycophenolate mofetil	Neg. (S.; L. not tested)	Follow-up scan: subtle bilateral hippocampal atrophy

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On-line Table 2: Continued

Study Group	ID	Sex	Age at MR Imaging (yr)	Time between Onset and Scan (mo)	Lateralization EEG	Immunotherapy before MR Imaging Acquisition	Antibody Status (at the Time of MR Imaging Acquisition)	Clinical Imaging
Late GAD	8	F	26	21	Left temporal	Steroids, immunoadsorption, mycophenolate mofetil	Not tested	Follow-up scan: hyperintense left mesiotemporal structures; no atrophy
Late GAD	60	M	17	55	–	–	Pos. (S.; L. not tested)	Follow-up scan: questionable signal abnormality left hippocampus
Late GAD	61	M	35	69	–	–	Not tested	Follow-up scan: normal
Late GAD	10	M	48	63	–	Steroids, basiliximab	Not tested	Follow-up scan: left hippocampal atrophy and enlarged left amygdala
Late GAD	11	F	52	50	–	Steroids, basiliximab	Not tested	Follow-up scan: bilateral volume increase of amygdalae
Late GAD	62	F	24	100	–	–	Not tested	Follow-up scan: unclear lesion frontal lobe; all other areas normal
Late GAD	63	M	27	70	–	Steroids	Not tested	Follow-up scan: left hippocampal sclerosis
Late GAD	64	M	24	65	Left temporal	–	Pos. (S. and L.)	Follow-up scan: normal mesiotemporal structures
Late GAD	14	F	31	44	–	Steroids	Pos. (S.; L. not tested)	Follow-up scan: mesiotemporal volume increase and hyperintensity
Late GAD	65	M	51	44	Right temporal	Steroids	Not tested	Follow-up scan: hyperintensity and subtle volume increase of right amygdala and hippocampus
Late GAD	66	F	61	79	Bilateral temporal	Steroids	Pos. (S. and L.)	Follow-up scan: hyperintensity and subtle volume increase of right amygdala and hippocampus and of the anterior left hippocampus
Late GAD	67	M	44	45	Normal	–	Not tested	Follow-up scan: subtle hyperintensity of right hippocampus
Late GAD	68	F	24	31	Right temporal	–	Pos. (S. and L.)	Follow-up scan: hyperintensity of right hippocampus and amygdala
Late GAD	69	M	63	27	Right temporal	–	Pos. (S. and L.)	Follow-up scan: hyperintensity and volume increase right amygdala
Late GAD	70	F	30	75	Left temporal	Steroids, immunoadsorption plasmapheresis	Not tested	Follow-up scan: declining hyperintensity right hippocampus

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On-line Table 2: Continued

Study Group	ID	Sex	Age at MR Imaging (yr)	Time between Onset and Scan (mo)	Lateralization EEG	Immunotherapy before MR Imaging Acquisition	Antibody Status (at the Time of MR Imaging Acquisition)	Clinical Imaging
Late GAD	71	F	47	69	Left temporal	Steroids	Pos. (S. and L.)	Follow-up scan: hyperintensity and volume increase of the left amygdala
Late VGKC	2	M	64	59	–	Steroids	Not tested	Follow-up scan: bilateral hippocampal atrophy
Late VGKC	3	F	53	73	–	Steroids, tacrolimus	Neg. (S. and L.)	Follow-up scan: normal
Late VGKC	5	F	56	40	–	Steroids, azathioprine	Neg. (S.; L. not tested)	Follow-up scan: left hippocampal sclerosis
Late VGKC	6	M	79	87	Right temporal	Steroids	Pos. (S.; L. not tested)	Follow-up scan: subtle bilateral hippocampal atrophy
Late VGKC	7	M	78	110	Left temporal	Steroids	Pos. (S. and L.)	Follow-up scan: hyperintensity and volume increase of the left amygdala
LateVGKC (CASPR2)	14	M	40	29	–	Steroids, plasmapheresis, mycophenolate mofetil	Not tested	–
Late VGKC	9	F	23	43	–	Steroids	Neg. (S.; L. not tested)	Follow-up scan: subtle mesiotemporal hyperintensity in the left hemisphere
Late VGKC	10	F	68	90	Right temporal	Steroids	Neg. (S.; L. not tested)	Follow-up scan: right hippocampal sclerosis
Late VGKC	11	M	75	84	Right temporal	Steroids, intravenous immunoglobulins	Neg. (S.; L. not tested)	Follow-up scan: questionable left mesiotemporal hyperintensity and swelling
Late VGKC (LGI1)	16	F	52	47	Left temporal	Steroids, intravenous immunoglobulins	Neg. (S.; L. not tested)	Follow-up scan: hyperintensity of the right mesiotemporal structures
Late VGKC (LGI1)	18	F	74	27	Left temporal	Steroids, immunoadsorption	Not tested	–
Late VGKC	13	F	26	37	–	Steroids	Not tested	Follow-up scan: normal
Late VGKC (LGI1)	19	F	73	33	Right temporal	Steroids	Neg. (S.; L. not tested)	Follow-up scan: signs of subcortical arteriosclerotic encephalopathy; normal mesiotemporal structures
LateVGKC	72	F	47	45	–	Steroids	Neg. (S.; L. not tested)	–
Late VGKC	73	F	64	82	Left temporal	Steroids	Neg. (S.; L. not tested)	Follow-up scan: bilateral mesiotemporal hyperintensity (especially left amygdala); no hippocampal sclerosis
Late VGKC (CASPR2)	74	M	52	61	Bilateral temporal	Steroids	Pos. (S. and L..)	Follow-up scan: bilateral mesiotemporal hyperintensity and volume increase

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On-line Table 2: Continued

Study Group	ID	Sex	Age at MR Imaging (yr)	Time between Onset and Scan (mo)	Lateralization EEG	Immunotherapy before MR Imaging Acquisition	Antibody Status (at the Time of MR Imaging Acquisition)	Clinical Imaging
Late VGKC	75	M	31	91	Left temporal	Steroids	Not tested	Follow-up scan: bilateral hyperintensity and volume increase of amygdalae (left > right)
Late VGKC (CASPR2)	76	M	64	86	–	Steroids	Pos. (S.; L. not tested)	Follow-up scan: global brain atrophy
Late VGKC	77	M	52	51	Left temporal	Steroids	Pos. (S.; L. not tested)	Follow-up scan: hyperintensity and volume increase of left mesiotemporal
Late VGKC (CASPR2)	78	M	53	65	Left temporal	Steroids, immunoadsorption, azathioprine	Pos. (S. and L.)	Follow-up scan: hyperintense mesiotemporal structures right hemisphere
Late VGKC (CASPR2)	79	F	45	47	Left temporal	Steroids	Pos. (S.; L. not tested)	Follow-up scan: cortical atrophy; declining swelling of left amygdala; no hippocampal sclerosis
Late VGKC	80	F	76	67	–	Steroids	Not tested	Follow-up scan: global brain atrophy; microangiopathic white matter disease

Note:—–indicates not conducted; S., in serum; L., in liquor; not tested, not tested at the time of the respective MR imaging scan; Neg., negative; Pos., positive; ID, identification.

[a] Characteristics of all patient groups included in the study (early GAD-LE, early VGKC-LE, late GAD-LE, late VGKC-LE).

On-line Table 3: Volumetry of hippocampal subfields in patients and controls—multivariate linear models in early GAD group[a]

Multivariate Linear Model, $F(11, 44) = 2.33$, Prob > F = 0.023 ($n = 46$)[b]				
Post Hoc *T* Tests[c]	Coefficient	Standard Error	*T* Test	$P > \|t\|$
Hippocampal tail	–9.1	22.6	–0.40	.690
Subiculum	–11.6	15.8	–0.73	.466
CA1	–64.3	26.2	–2.46	.018[b]
Presubiculum	0.8	12.0	0.07	.947
Parasubiculum	–7.7	3.7	–2.07	.045[b]
Molecular layer	–29.9	21.4	–1.40	.168
GC-ML-DG	–12.1	12.6	–0.96	.340
CA3	–17.1	11.2	–1.61	.114
CA4	–12.4	11.4	–1.09	.284
Fimbria	–0.4	4.6	–0.10	.925
HATA	–9.9	3.3	–3.01	.004[b]

Note:—GC-ML-DG indicates granule cell layer of the dentate gyrus; HATA, hippocampus-amygdala transition area; Prob, probability.

[a] On-line Tables 3 to 8 show multivariate linear models including post hoc *t* tests of hippocampal subfield volumes between the different patient groups and their matched controls (as shown in Fig 2).

[b] Significant values.

[c] Protected by the Fisher least-significant difference.

On-line Table 4: Early VGKC group

Multivariate Linear Model, $F(11, 48) = 2.48$, Prob > F = 0.015[a] ($n = 50$)				
Post Hoc *T* Tests[b]	Coefficient	Standard Error	*T* Test	$P > \|t\|$
Hippocampal tail	1.2	21.9	0.05	.957
Subiculum	–16.2	17.7	–0.91	.365
CA1	–19.5	24.0	–0.81	.422
Presubiculum	–2.5	14.4	–0.18	.861
Parasubiculum	–10.0	3.7	–2.68	.010[a]
Molecular layer	–10.5	21.7	–0.48	.632
GC-ML-DG	–3.1	11.6	–0.27	.791
CA3	–8.7	8.3	–1.05	.301
CA4	–5.2	9.4	–0.56	.581
Fimbria	7.5	7.9	0.95	.347
HATA	–12.8	3.7	–3.43	.001[a]

Note:—GC-ML-DG indicates granule cell layer of the dentate gyrus; HATA, hippocampus-amygdala transition area; Prob, probability.

[a] Significant.

[b] Protected by the Fisher least-significant difference.

On-line Table 5: Early LGI1 group

Multivariate Linear Model, $F(11, 18) = 3.44$, Prob > F = 0.001[a] ($n = 20$)				
Post Hoc *T* Tests[b]	Coefficient	Standard Error	*T* Test	*P* >|t|
Hippocampal tail	21.5	35.0	0.62	.546
Subiculum	21.4	23.6	0.91	.377
CA1	5.5	44.7	0.12	.904
Presubiculum	20.6	18.3	1.13	.275
Parasubiculum	–9.9	5.2	–1.91	.072
Molecular layer	19.9	34.5	0.58	.572
GC-ML-DG	11.8	19.1	0.62	.545
CA3	–5.0	16.0	–0.32	.752
CA4	4.3	16.0	0.27	.787
Fimbria	29.1	8.4	3.47	.003[a]
HATA	–5.5	6.6	–0.83	.418

Note:—GC-ML-DG indicates granule cell layer of the dentate gyrus; HATA, hippocampus-amygdala transition area; Prob, probability.
[a] Significant.
[b] Protected by the Fisher least-significant difference.

On-line Table 6: Early VGKC group without LGI1

Multivariate Linear Model, $F(11, 28) = 2.75$, Prob > F = 0.015[a] ($n = 30$)				
Post Hoc *T* Tests[b]	Coefficient	Standard Error	*T* Test	*P* >|t|
Hippocampal tail	–17.4	26.9	–0.65	.523
Subiculum	–41.7	23.8	–1.75	.091
CA1	–49.1	29.0	–1.70	.101
Presubiculum	–16.9	19.6	–0.86	.398
Parasubiculum	–11.0	5.3	–2.07	.048[a]
Molecular layer	–38.7	27.5	–1.41	.169
GC-ML-DG	–16.5	13.9	–1.18	.247
CA3	–15.4	9.4	–1.64	.113
CA4	–15.1	11.5	–1.31	.200
Fimbria	–5.0	11.1	–0.45	.655
HATA	–17.2	4.3	–3.99	<.001[a]

Note:—GC-ML-DG indicates granule cell layer of the dentate gyrus; HATA, hippocampus-amygdala transition area; Prob, probability.
[a] Significant.
[b] Protected by the Fisher least-significant difference.

On-line Table 7: Late GAD group

Multivariate Linear Model, $F(11, 64) = 3.65$, Prob > F = 0.001[a] ($n = 66$)				
Post Hoc *T* Tests[b]	Coefficient	Standard Error	*T* Test	*P* >|t|
Hippocampal tail	–3.5	16.0	–0.22	.826
Subiculum	28.4	11.5	2.46	.016[a]
CA1	29.4	19.2	1.53	.131
Presubiculum	4.1	9.1	0.45	.657
Parasubiculum	2.7	2.3	1.18	.242
Molecular layer	22.4	14.7	1.53	.132
GC-ML-DG	8.1	8.4	0.97	.334
CA3	5.5	8.8	0.62	.538
CA4	6.1	7.5	0.82	.418
Fimbria	5.2	3.9	1.34	.184
HATA	10.1	2.2	4.58	<.001[a]

Note:—GC-ML-DG indicates granule cell layer of the dentate gyrus; HATA, hippocampus-amygdala transition area; Prob, probability.
[a] Significant.
[b] Protected by the Fisher least-significant difference.

On-line Table 8: Late VGKC group

Multivariate Linear Model, $F(11, 42) = 1.62$, Prob > F = 0.128 ($n = 44$)				
Post Hoc *T* Tests[a]	Coefficient	Standard Error	*T* Test	*P* >|t|
Hippocampal tail	–0.5	29.0	–0.02	.986
Subiculum	16.8	19.5	0.86	.395
CA1	1.8	28.7	0.06	.951
Presubiculum	7.1	13.3	0.53	.599
Parasubiculum	–6.0	3.7	–1.62	.114
Molecular layer	10.7	23.4	0.46	.651
GC-ML-DG	5.0	11.5	0.43	.667
CA3	0.2	9.3	0.02	.986
CA4	0.8	9.7	0.08	.938
Fimbria	11.0	6.4	1.72	.092
HATA	1.5	4.3	0.34	.734

Note:—GC-ML-DG indicates granule cell layer of the dentate gyrus; HATA, hippocampus-amygdala transition area; Prob, probability.
[a] Protected by the Fisher least-significant difference.

On-line Table 9: Volumetry of amygdala and hippocampus in patients and controls (1-tailed, 2-sample *t* tests) in early groups[a]

23 Early GAD-LE Group + 23 Controls ($n = 46$) and 25 Early VGKC-LE Group + 25 Controls ($n = 50$)			
	DOF	*T* Test	*P* >|t|
Early GAD-LE			
Amygdala affected	44	1.92	.031[b]
Hippocampus affected	44	1.35	
Amygdala unaffected	44	–0.21	>.05
Hippocampus unaffected	44	–0.74	
Early VGKC-LE			
Amygdala affected	48	2.82	.004[b]
Hippocampus affected	48	0.67	
Amygdala unaffected	48	1.19	>.05
Hippocampus unaffected	48	–0.22	

Note:—DOF indicates degrees of freedom; Pr, probability.
[a] One-tailed, 2-sample *t* tests of the amygdala and hippocampus in the affected and unaffected hemispheres between patients with LE and matched controls (as shown in Fig 1).
[b] Significant values.

On-line Table 10: Volumetry of amygdala and hippocampus in patients and controls (1-tailed, 2-sample *t* tests) in late groups[a]

33 late GAD-LE Group + 33 Controls ($n = 66$) and 22 Late VGKC-LE Group + 22 Controls ($n = 44$)			
	DOF	*T* Test	*P* >|t|
Late GAD-LE			
Amygdala affected	32	2.19	.036[b]
Hippocampus affected	32	1.62	
Amygdala unaffected	32	1.56	>.05
Hippocampus unaffected	32	0.99	
Late VGKC-LE			
Amygdala affected	21	1.33	
Hippocampus affected	21	–0.32	
Amygdala unaffected	21	0.08	>.05
Hippocampus unaffected	21	–0.57	

[a] One-tailed, 2-sample *t* tests of the amygdala and hippocampus in the affected and unaffected hemispheres between patients with LE and matched controls (as shown in Fig 1).
[b] Significant values.