Metabolic Networks in Temporal Lobe Epilepsy

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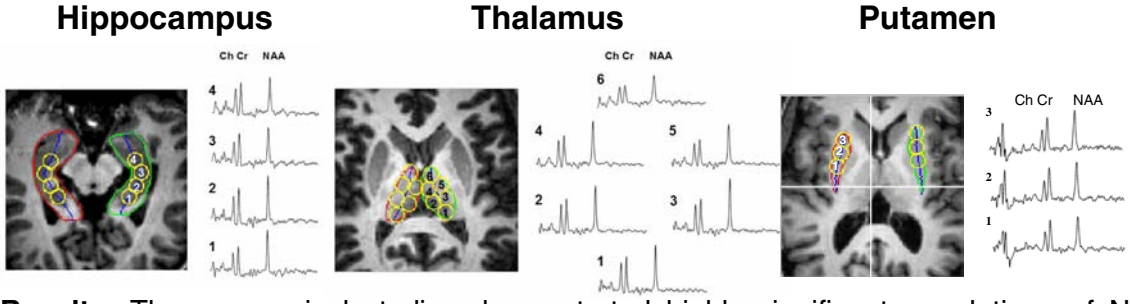
Introduction: Typically, mesial temporal lobe epilepsy (MTS) is thought of as a focal disease, confined to the hippocampus. However, recent ^{31}P and PET data has suggested the involvement of the thalami and basal ganglia. To better understand the role of the thalami and basal ganglia, and their possible role in predicting outcome, we evaluated these structures in 24 MTS surgical patients using high-resolution ^{1}H spectroscopic imaging at 4T prior to surgery and in 8 patients following surgery and correlated these data with outcome.

Methods: ^{1}H spectroscopic images of the hippocampi, thalami and basal ganglia of 24 patients with temporal lobe epilepsy and 20 controls were acquired with a modified LASER sequence at 4T and a volume TEM head coil. The SI data were acquired with a slice thickness of 10mm from a FOV of 19.2x19.2 cm^2 using 24x24-encoding steps (0.64cc nominal volume). Hippocampal (4), thalamic (6) and basal ganglia (3) volumes within each structure on each side were reconstructed automatically using coordinates derived from manually defined borders of the structures on the scout images (Figure 1). The statistical significance was determined from the Pearson correlation coefficient. Surgical outcome was assessed according to the ILAE scale.



Results: The pre-surgical studies demonstrated highly significant correlations of NAA/Cr in the ipsilateral hippocampus, the anterior and posterior ipsilateral and contralateral thalami, the ipsilateral and contralateral basal ganglia and the contralateral hippocampus (Table 1). The ipsilateral hippocampus was significantly correlated with every structure. The strongest correlations were with the bilateral anterior thalami, and bilateral basal ganglia. No such correlations were seen in the controls. Patients attaining seizure free outcomes (ILAE class I and II) demonstrated ipsilateral/contralateral NAA/Cr ratios of <1.00 in the hippocampus and anterior and posterior thalamus (Table 2); while patients continuing to experience seizures (class III and above) demonstrated ratios greater than 1.0. Those patients achieving a seizure free outcome showed improvement in the contralateral hippocampus (9% increase in NAA/Cr), while those continuing to experience seizures (class III) showed a 6% decline.

Table 1 Correlations with Ipsilateral Hippocampus

Structure Correlated with	R	P
Ipsilateral Anterior Thalamus	0.76	<0.00002
Contralateral Anterior Thalamus	0.58	<0.003
Contralateral Putamen	0.53	<0.01
Ipsilateral Putamen	0.51	<0.01
Ipsilateral Posterior Thalamus	0.50	<0.02
Contralateral Hippocampus	0.48	<0.02
Contralateral Posterior Thalamus	0.42	<0.05

Table 2 ipsi/contra NAA/Cr and Outcome

	Class I&II	Class III-V
Hippocampus	0.92	1.01
Anterior Thalamus	0.97	1.00
Posterior Thalamus	0.94	1.04

Conclusions: This work supports the involvement of the thalamus and basal ganglia in the pathophysiology of temporal lobe epilepsy, and the presence of a network of metabolically impaired structures. The pre-surgical studies suggest that measurements of the network may provide predictive power for surgical outcome. Finally, post-surgical studies indicate that the extent of damage in the contralateral hippocampus continues to progress, raising the issue of its role in ongoing seizure activity.