Correlates of thalamic and hippocampal injury in temporal lobe epilepsy by NAA spectroscopic imaging

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Introduction

Recent FDG-PET and ^{31}P MRS studies in patients with intractable temporal lobe epilepsy (TLE) found that glucose uptake and bioenergetics of the thalamus is correlated with neuronal loss and gliosis in the ipsilateral hippocampus. The functional and energetic impairment in the thalamus is believed to be the result of recurrent seizures and propagation of seizure induced damage through a network of involved structures. Despite these neuropathologic and imaging relationships between the hippocampus and thalamus, FDG PET did not detect any significant relationship between them. ^{31}P spectroscopy has evaluated this; however the spatial overlap in the ^{31}P data (~15%) precluded definitive correlations between the ipsilateral thalamus and hippocampus. In this study we evaluated the relationship between neuronal injury/loss in the hippocampus as measured by NAA and its effect on the thalamus in TLE patients.

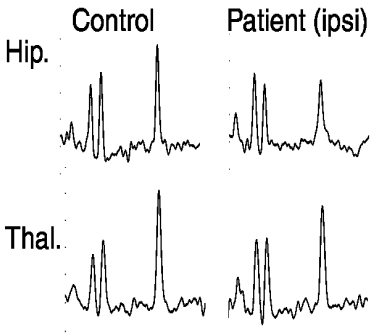
Methods

^{1}H spectroscopic images from n=18 controls and n=21 patients with TLE was acquired at 4T with a Varian INOVA system using a quadrature TEM head coil. Hippocampal data was collected using a modified LASER sequence (10mm thickness, 80x100mm in-plane FOV selection) in combination with two dimensions of phase encoding (24x24, FOV=192x192mm, 19.2 min) angulating the plane along the temporal pole. Thalamic data was collected using the same sequence but the plane was angulated along the AC-PC line and selected at the level of the thalamus. Voxels spanning each hippocampus (n=4) and each thalamus (n=5) were selected; fit in the spectral domain and the mean NAA/Cr ratio was calculated.

Results

As expected there were large reductions in NAA/Cr in the ipsilateral hippocampus in TLE patients (Table). In the thalamus, we found significant reductions in NAA/Cr in both the ipsilateral (1.35±0.16, $p<2x10^{-4}$) and contralateral thalamus (1.43±0.15, $p<0.02$) in comparison to controls (1.52±0.16). A paired T-test comparison of the ipsi-and contralateral thalami revealed a highly significant reduction in the ipsilateral thalamus ($p<6x10^{-4}$). To evaluate the extent to which the neuronal injury seen in the hippocampi reflected propagation along the network to the thalami we correlated the NAA/Cr values between the hippocampus and thalamus, finding a highly significant linear relationship ($r=+0.43, p<0.005$) in TLE patients.

NAA/Cr		Hippocampus		Thalamus	
	n	Mean	SD	Mean	SD
Ipsi	21	1.12	0.19	1.35	0.16
Contra	21	1.20	0.20	1.43	0.15
Controls	18	1.35	0.13	1.52	0.16



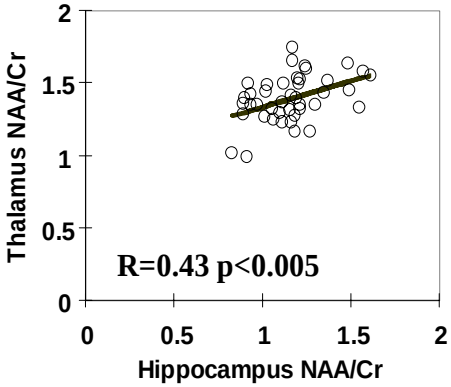


Figure 1: Spectra (0.64cc) from the hippocampus and thalamus of a control patient. **Figure 2:** Regression of NAA/CR in the thalamus and hippocampus from TLE patients, comparing data from the same hemisphere, either ipsi- or contralateral.

Conclusions

We find significant neuronal injury/loss in both the ipsilateral and contralateral thalami in TLE patients, with greater damage in the ipsilateral thalamus. The degree of neuronal injury/loss in the thalamus is directly correlated with that seen in the hippocampus from the same hemisphere. This data supports previous observations by PET and ^{31}P MR studies that thalamic dysfunction in TLE is associated with histological changes in the hippocampus. Thus these data indicate that the impairment and damage associated with recurrent seizures as measured by NAA originating in the hippocampus propagates through the network of involved structures and results in corresponding impairment/damage in the thalamus.