

Ultra-short echo time (UTE) MR imaging of the lung: Comparison between normal and emphysematous mice

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Introduction: Computed tomographic scanning is the standard method for imaging the lung parenchyma but carries potential radiation risk. MR imaging of the lung is challenging because of low proton density, severe magnetic field susceptibility, and respiratory and cardiac motion artifacts. However, the recent development of techniques along with more powerful hardware has made possible detailed, non-invasive imaging of pulmonary parenchyma, and pulmonary MRI is currently an active area of research [1]. The utility of ultra-short TE (UTE) imaging in conjunction with projection acquisition of the free inducing decay (FID) helps to acquire the MR signal from the lung parenchyma [2]. It allows us to reduce TE up to less than 100 μ sec to minimize signal decay caused by short T2 relaxation time, and brings higher SNR compared to a conventional FFT short echo image sequence. On the subtraction image between the images with an ultrashort TE and a conventional range of TE (ca. 2 msec), the biological materials/tissues which have rapid T2 decay are enhanced. In the present study, we tested the feasibility of a UTE sequence to measure T2 of the lung parenchyma in mice. The homozygous mutant klotho (Klotho^{-/-}) mouse is a mouse strain with insertional mutagenesis, which have a defect in klotho gene expression, shows growth retardation and various aging-like phenotypes including severe pulmonary emphysema [3]. Using this transgenic mouse model, we compared the signal intensity and T2 relaxation time between normal and emphysematous lung.

Materials and Methods: We used three 5-week old wild type mice for this feasibility study. UTE imaging was conducted using a 3 Tesla (T) whole-body human MRI unit (Achieva™, Philips Medical Systems, Best, Netherlands) with a small solenoid coil (I.D. 63 mm). All animals were placed supine under anesthesia with the thorax centered with respect to the center of a RF coil. For the purpose of reproducible positioning of the imaging region, a low-resolution multi-slice image, was first acquired of the entire lung in both transverse and coronal planes using a fast spin echo sequence to select a voxel of interest (VOI) encompassing the entire lung. On the selected VOI, three dimensional UTE sequence was performed with various TEs (3.5, 3, 2.5, 2, 1.5, 1, 0.8, 0.7, 0.6, 0.5, 0.4, 0.3, 0.2 and 0.11 msec) in fixed scale. The other imaging parameters were: 12 msec TR, 5x5x5 cm FOV, 84x84x84 matrix (isotropic), and 2 NEX. Signal intensities were measured in the four different regions of interests (ROIs) on the right lower pulmonary lobes on the resliced axial images (12 regions total). Intensities were also measured in air to provide an estimate of noise in each image. T2s in the 4 ROIs in each animal were separately calculated as the slope of the logarithm of the noise corrected signal intensities [4] versus the TEs in each region. The same UTE imaging and T2 measurement (8 regions total) were repeated in two age-matched (5-week old) Klotho^{-/-} mice and the results were compared to those in the WT mice.

Results: The UTE images produced more signal intensity from pulmonary parenchyma as the TE was shortened (**Fig.1**). Although neither cardiac nor respiratory gating was used, the images were not degraded by obvious motion artifact. The signal intensities were lower in the emphysematous mice (**Fig 1**) at any given TE. The signal intensities at all ROIs in the WT and emphysema mice reduced as the TE reduced and showed excellent exponential fitting ($R^2 < 0.9$) from TE of 110 μ sec to 0.2 msec or 0.1 msec, respectively (**Fig 2**). The T2 of the lung in the emphysematous mice were reduced (0.70 ± 0.07 msec) compared with that in the WT (1.16 ± 0.15 msec, **Fig.3**), which might be caused by increase of airspace (alveolar space).

Discussion: In this sequence, the readout starts immediately after the RF system is switched from transmit to receive so that the MR signal could be acquired before it decays even in the lung parenchyma where the transverse relaxation is rapid due to the severe susceptibility effect. We postulate that the amount of signal would reflect interstitial tissue density (sum of blood volume, elastic fibers in the alveolar walls and around the blood vessels, bronchi and surfactant) although short T2 effect still reduces the MRI signal, particular in emphysema. The signal is acquired using a center-out sampling scheme, which corresponds to sampling the FID. A minimal duration of the sampling window is archived using radial sampling. These might have contributed to reduce motion artifacts on the UTE images.

Conclusions: UTE imaging of the mice lung was feasible, which allowed more precise measurement of T2 of the lung and, further, discrimination of emphysematous lungs from normal lungs in this simple model. Although non-uniform disruption of lung architecture is usually assessed by computed tomography, the method may provide regional measures related to lung tissue compositions without incurring the risks of radiation exposure.

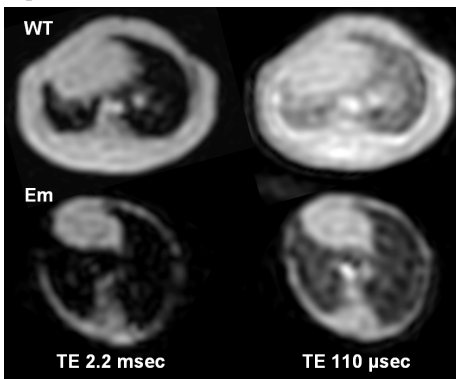


Fig. 1. Typical UTE resliced axial images of the lungs in the WT and emphysema (Em) mice at TE of 2.2 msec and 110 μ sec. Both animals were imaged simultaneously.

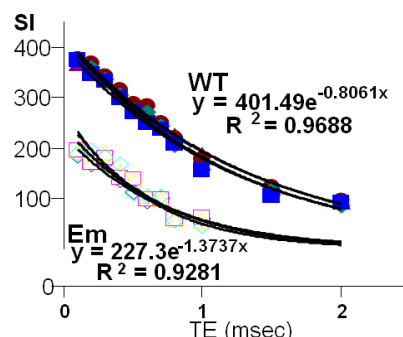


Fig. 2. T2 decay curves (SI vs. TE) in four ROIs on the right pulmonary lobes in the WT and emphysema (Em) mice in Fig.1. Representative fitting equation and R^2 values are addressed.

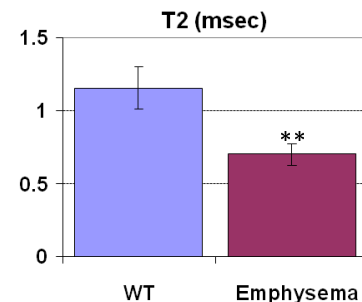


Fig. 3. T2 values (msec) in the WT and emphysema mice. Values are expressed as mean \pm SD. ** $p < .01$ by t-test.

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