

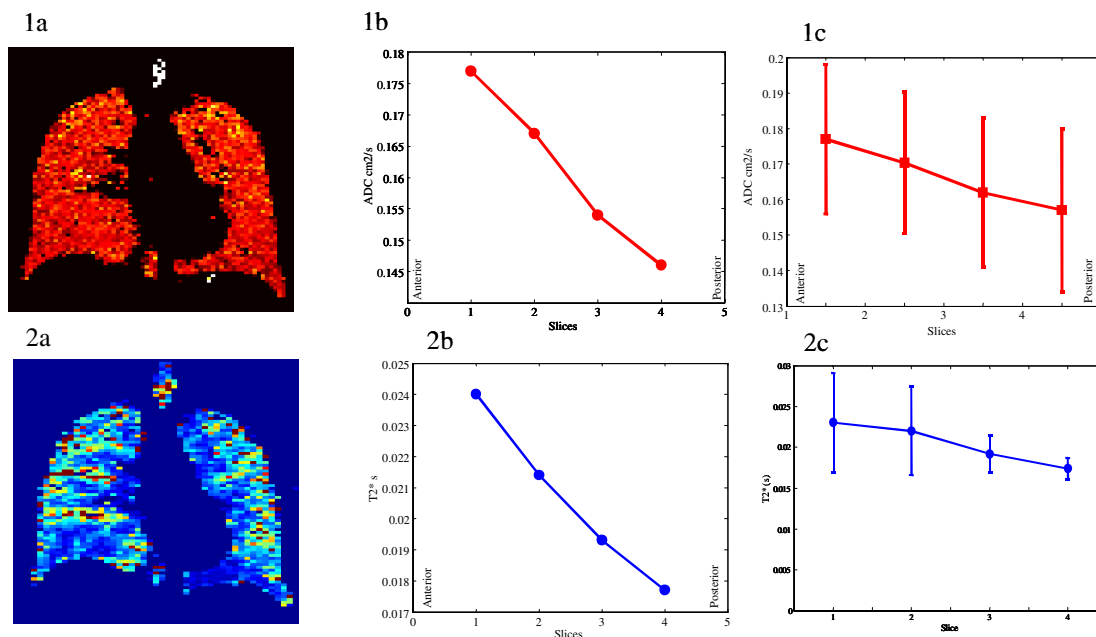
Anatomical trends in coregistered ADC and T2* maps of 3He gas in the lungs of healthy normals

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Introduction Measurement of the ³He gas ADC with pulsed gradient methods can be used to infer spatial dimensions at the alveolar level [1], and a gradient in the ³He ADC in the anterior-posterior direction has previously been reported in healthy normals [2]. The ³He T2* is a second parameter which will depend upon the alveolar dimensions as these define the microscopic field gradients in which the gas is diffusing. Field strength, macroscopic B₀ field homogeneity, adjacent blood perfusion (microscopic susceptibility difference) and degree of gas diffusion will also play a role in T2*. In previous work at 1.5T [3] the ³He T2* was found to increase with lung volume (inflation of the alveoli). In this work ³He ADC and T2* data was collected from spatially registered 2D slices in four volunteers and spatial trends within the lungs of the two parameters was investigated.

Methods ³He gas was polarized on site to 26% by optical pumping with rubidium spin exchange apparatus (GE Healthcare). All work was performed on a 1.5T whole body system, tuned to ³He frequency of 48.6 MHz (Eclipse –Philips Medical Systems). A T-R elliptical birdcage body coil was used for the volunteer studies [4]. Four healthy male non-smokers (Age: 24-34) performed two breath-hold exams each of 250 ml ³He mixed with 750 ml N₂, starting from a position of relaxed expiration in a supine posture. The gas was inhaled from a Tedlar bag and breath was held upon full inhalation of the 1 litre gas mix. The MRI sequences were: (i) **ADC** exam. This consisted of an interleaved low flip angle spoiled gradient echo (SPGR) acquisition with a reference (ventilation) scan ($b=0$) followed by diffusion-weighted acquisition ($b=2.89 \text{ scm}^{-2}$ - bipolar trapezoids of plateau strength 26.2 mTm^{-1} and duration $460 \mu\text{s}$ with $500\mu\text{s}$ ramp time –*direction in-slice*). Phase encoding was centric with 96 views, 11 slices, 15 mm thick and no gap, FOV =42 cm, TE=5.5 ms, TR=9 ms, 128 samples, BW $\pm 16\text{kHz}$, flip angle of 7° . (ii) **T2*** exam: A four interleaved SPGR acquisition with TE values : TE = (4.2, 6.2, 8.2 and 10.2 ms), 5 slices 15mm thick with a 15 mm gap (alternate ADC slices spatially registered to all five T2* slices). FOV =42 cm, phase encoding was centric with 64 views, BW $\pm 16\text{kHz}$, flip angle 6° .



Data analysis The mean SNR in the ADC and T2* images ($b=0$ and TE=4.2 ms images) was measured. The ADC and T2* images were segmented with a noise threshold $> 4*s.d.$ in the background noise. ADC maps were computed on a pixel-by-pixel basis by a 2 point exponential fit, T2* maps were computed by a 4 point exponential fit. Mean and s.d. ADC and T2* were measured in all co-registered slices from anterior to posterior. Data with SNR < 10 was discarded from the group analysis.

Results Figures 1a and 2a are example co-registered ADC and T2* maps from the same slice in one of the volunteers (34 y). Note the lower T2* around the diaphragm and major vessels. From the four subjects scanned, three had ADC and T2* images with SNR >10 in four of the five slices. The plots of Figure 1b and 2b show the mean ADC (red curve) and T2* (blue curve) averaged over the whole of the lungs in each slice for the four slices - note the A–P gradient and also note the gradient in T2* in the same subject in the A-P direction. The mean T2* values are in accordance with those measured previously in our centre and reported previously from the whole of the lungs at 1.5T [3]. Figures 1c and 2c are the pooled data (mean and s.d. error bars) for the four slices analyzed for all three subjects. A clear decreasing trend is evident in both ADC and T2* from A-P. When the ADC and T2* were analysed for correlation : Pearson correlation $r = 0.930$ (2 tailed sig. $p = 0.07$) Spearman's $r = 1.0$, $p < 0.01$.

Discussion Spatially registered ADC and T2* maps of ³He have been demonstrated in volunteers imaged supine at a point of mid-inspiration (1 litre gas inhaled) and a decreasing trend in the two parameters has been observed across the anatomical A-P gradient. This has been reported previously for the ADC [2] but to our knowledge has not been demonstrated for the T2*. The cause of the effect could be partly gravitational in that the posterior alveoli are more prone to compression causing a decrease in mean alveolar dimension, this was the explanation proposed for the ADC gradient seen in [2]. This could also contribute to a reduction in T2* due to the increased microscopic field inhomogeneity that results from a smaller alveolar gas space (lower SA/V ratio) and increase in bulk susceptibility difference due to the concomitant higher parenchymal density. Another related factor might be the increased perfusion in the posterior slices which is well known from supine lung perfusion studies [5], this would cause a localised increase in field inhomogeneity due to the susceptibility of the increased blood perfusion. Although additional physical mechanisms contribute to the T2*, evidence of a linear correlation between ADC and T2* was observed and these anatomical trends warrant further investigation and explanation. In further work we are investigating pixel-pixel spatial correlation between the two parameters and the option of collecting both ADC and T2* in one scan [6] to eliminate mis-registration errors.

References [1] Magn Res Med, 2000; 44, 174-179 [2] J Magn Reson Imaging, 2004 ;20(2):331-335. [3] Proc 11th ISMRM 2004, p2724 [4] Proc 12th ISMRM 2007, p240.[5] J Appl Physiol 2002;93:1841–1851.[6] Magn Reson Med, 2007;57(6):1185-9.

Acknowledgements. EU Phelinet Framework 6 grant, EPSRC #GR/S81834/01(P), GE Healthcare, Philips Medical Systems, Spectra Gases.