

Is the T_2^* relaxivity of gadolinium in brain microvasculature linear with concentration?

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Introduction: Dynamic, susceptibility-weighted, contrast-enhanced (DSC) MRI perfusion measurements depend on estimating intravascular contrast agent (CA) concentration (C) from signal intensity changes in T_2^* -weighted images after bolus injection (Eq. [1]). Generally, linearity is assumed between relaxation and C , but it is well known that compartmentalization of CA and secondary magnetic field perturbations generate deviations from linearity *in vivo*. In this study we test the reliability of both linear (Eq. [2]) and non-linear (Eq. [3]) relaxivity expressions based on the static dephasing regime model (1) in estimating C and cerebral blood volume (CBV) in tissue.

Methods: This retrospective study was approved by the Institutional Review Board of this institution. 1.5T (T_R 1s; T_E 40ms; FA 90°; matrix 128×128; FOV 228×228 mm; 7×5mm slices) and 3T (T_R 1s; T_E 32ms; FA 30°; matrix 128×128; FOV 230×230 mm; 10×5mm slices) DSC data was acquired from 5 subjects at each field using a gradient echo EPI sequence during injection of 0.1mmol/kg Gd-DTPA. Signal measurements were taken in frontal white matter (WM), caudate nucleus (grey matter, GM) and 10 arterial pixels selected using an automated method (2). Signal measurements in arteries were converted to estimates of C using the quadratic relaxivity relationship found empirically by van Osch et al. (3) and Akbudak et al. (4) at 1.5 and 3T respectively. WM and GM C was estimated using Eqs. [2 and 3] where b_{lin} (32) and b_{non} (120) were determined empirically by previously described methods (5), J_0 is the zeroth order Bessel function and ζ is the blood volume fraction in tissue (2.5% in WM and 4% in GM). CBV was calculated using Eq. [4] where k_H is the difference in hematocrit between small and large vessels (.83) and ρ is the density of tissue.

$$S \propto e^{-\Lambda(C)} \quad [1]$$

$$\Lambda(C) = \zeta b_{lin} C B_0 T_E \quad [2]$$

$$\Lambda(C) = \zeta \frac{1}{3} \int_0^1 (2+u) \sqrt{1-u} \frac{1-J_0\left(\frac{2}{3} b_{non} C B_0 T_E\right)}{u^2} du \quad [3]$$

$$CBV = \frac{k_H}{\rho} \frac{\int C_{\text{tissue}} dt}{\int C_{\text{artery}} dt} \quad [4]$$

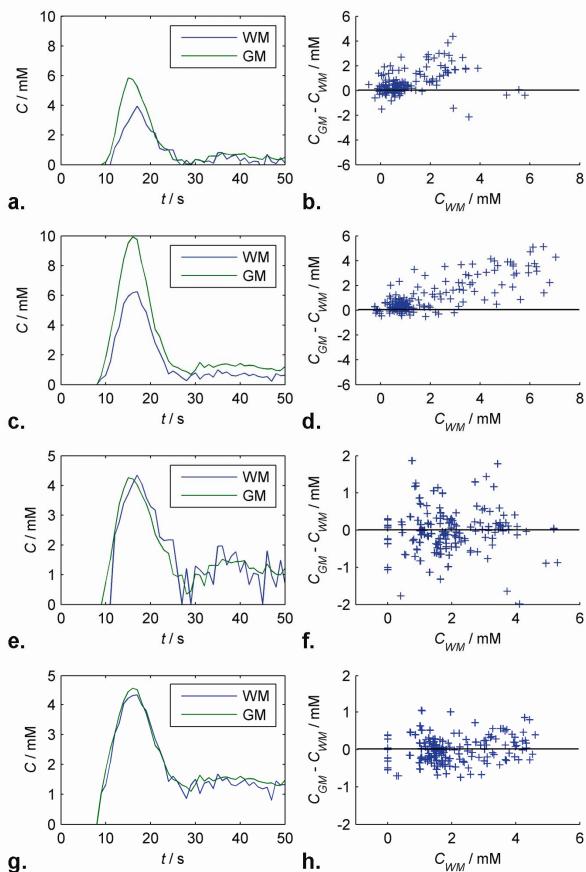


Figure 1. Comparison of WM and GM C at 1.5 and 3T.

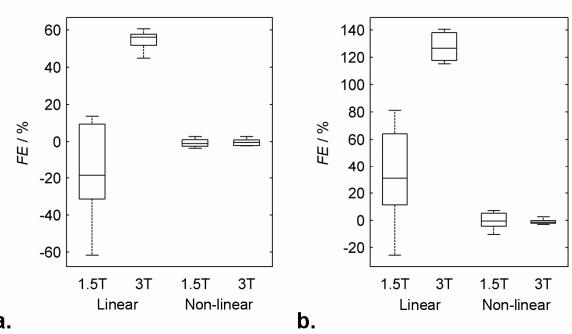


Figure 2. Fractional errors of CBV measurements in WM (a) and GM (b) using linear and non-linear relaxivity models.

Results: Figure 1 shows C curves calculated using the linear model in WM (blue) and GM (green) from a single test patient at 1.5T (a) and 3T (c) and using the non-linear model 1.5T (e) and 3T (g). Differences between GM and WM curves from the beginning of the bolus to 30 seconds after are plotted against WM concentration over all (5) test patients using the linear model at 1.5T (b) and 3T (d) and non-linear model at 1.5T (f) and 3T (h). Figure 2 gives box and whisker plots of fractional errors of measured CBV from subjects at both 1.5 and 3T and for both linear and non-linear formulations in WM (a) and GM (b). With the linear model, the mean differences between expected and measured CBV are -16% at 1.5T and 45% at 3T for WM 34% at 1.5T and 128% at 3T for GM. With the non-linear model, the mean % difference is less than $\pm 1\%$.

Discussion: The remarkable agreement between the two tissue curves at both field strengths suggests that the non-linear model is an acceptable functional form for brain tissue relaxivity. The systematic overestimation of CBV at 3T relative to 1.5T emphasizes the problems in finding a single linear relaxation relationship that fits multiple field strengths.

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