Variation in thermal maps during RF heating due to variation in electrical conductivity in TEM coil at 298 MHz

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Introduction: RF heating is a potential safety risk in multi-channel transmit MRI at ultra-high fields and RF thermotherapy applications [1]. RF hyperthermia, with or without MR control, is an adjuvant to radiation therapy, and involves deep region heating of human tissue with RF energy (100MHz to 433MHz). This temperature rise has to be carefully controlled to prevent inadvertent ablation of healthy tissue [2]. The relationship of local SAR induced temperature increase in tissues can be described with the Penne's bioheat equation [3]. For a given set of E-fields, local SAR is directly proportional to the electrical conductivities (σ) of tissues. However, the values of σ in tissues also determine the underlying E-field distributions. In EM modeling of RF hyperthermia and SAR applications, it is common for researchers to use a set of nominal values for σ at some desired frequency and temperature [4-5]. However, variability in reported electrical conductivity values exists due to several disparate reasons, e.g., differences in tissue compositions and measurement conditions, measurement errors, etc. A variation of up to 20% in the values of σ has been reported [6-7]. In addition, σ is a function of temperature [8], and a rise of about 2% in σ per °C has also been reported [9-10]. Accurate estimates of σ are important in EM modeling to accurately predict fields and associated current densities [11]. If the nominal values of σ deviate significantly from the true values, it could lead to under- or over- estimation of the thermal risk to the patient. In this work, we investigate the variation of SAR_{10g} and simulated temperature maps in a muscle phantom and human body model (HBM) due to incorrect assignment of values for σ. We drive a TEM coil at 298 MHz, which is a frequency that may provide a good tradeoff between adequate penetration depth and heat focal size.

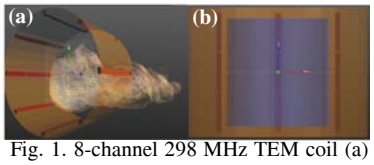

Fig. 1. 8-channel 298 MHz TEM coil (a) 'Duke' HBM (b) "Muscle" Phantom

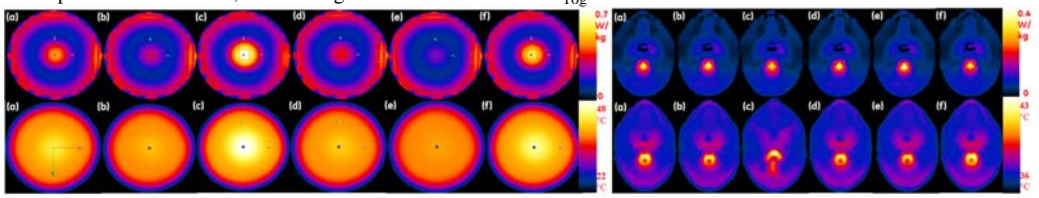
Fig. 2. SAR_{10g} (top row) and temperature maps (bottom row) for: muscle phantom (left panel), Duke HBM (right), at (**a**) nominal σ, (**b**) nominal σ+20%, (**c**) nominal σ-20%, (**d**) nominal σ+12% (due to temperature dependent σ alone), (**e**) nominal σ+20% + 12%, and (**f**) nominal σ-20% + 12% (due to uncertainty of variability of literature values of σ and temperature dependence of σ).

Methods: ***EM modeling.*** An 8-channel TEM coil (dia=33.0 cm, L=22.5 cm) was tuned to 298 MHz using SEMCAD X (SPEAG, Zurich, Switzerland). The FDTD cell sizes used were set to a maximum of 2mm×2mm×2mm. In the first set of EM simulations, a cylindrical "muscle" phantom (dia=18cm, L=20cm, σ=0.77 S/m, ε=58.82) surrounded by a 5 cm thick water bolus was heated using a total input power of 50 Watt. Next, the Duke model (76 different tissues) from the Virtual Family was heated [12] (Fig. 1), using 300 Watt of total input power. In both cases a SAR_{10g} focal spot was induced near the center of the coil when all 8 elements were driven with identical amplitudes (1V) and phases (0°).

Thermal Modeling. SAR maps for both Duke and phantom were used as inputs in thermal simulations [3]. Tissue dependent and temperature independent perfusion and metabolic heat terms were used for Duke, but ignored for the muscle phantom. A Dirichlet boundary condition was assigned to the interface between the water bolus and muscle phantom, and temperature of the water bolus was maintained at 15 °C. The temperature of free space was maintained at 22 °C. The muscle phantom was assigned an initial temperature of 37 °C. The nominal values of ε and σ for all tissues were assigned using [4].

Variation Study In adjuvant mild hyperthermia, temperature is raised to about 43 °C [13], i.e., temperature rise (ΔT) of 6 °C from 37 °C. This implies that a 12% increase in σ (2% per °C) was needed to account for the temperature dependence of σ. SAR and thermal simulations were performed for six scenarios for the muscle phantom and Duke: (**A**) nominal σ, (**B and C**) nominal σ±20%, (**D**) nominal σ+12% (temperature dependent σ alone), and (**E and F**) nominal σ±20%+12% (due to uncertainty of literature values and temperature dependence of σ). A total input power of 300 W was applied to heat Duke. Amount of time needed to get to 43 °C (17.80 min for phantom, 3 min for Duke) in using nominal σ (case **A**) was used for all cases **B** to **F**.

	Nominal σ (Case A)	Nominal σ + Inter-subject Variability (20%) (Case B)	Nominal σ - Inter-subject Variability (20%) (Case C)	Nominal σ + temperature dependent σ (12%) due to (ΔT) of 6°C (Case D)	Nominal σ + Inter-subject Variability (20%) (Case E)	Nominal σ - Inter-subject Variability (20%) (Case F)
Muscle σ (S m^{-1})	0.770	0.924	0.616	0.862	1.035	0.689
Peak SAR_{10g} (W/kg) (Phantom)	0.471	0.311	0.719	0.366	0.225	0.586
Peak SAR_{10g}(W/kg) (Duke)	0.369	0.395	0.344	0.385	0.412	0.356
Max Temp (°C) After 17.80 min (Phantom)	43.00	40.30	48.30	41.20	39.30	45.30
Max Temp (°C) after 3 min (Duke)	43.00	42.50	43.50	42.70	42.30	43.20
Time to get to 43C (min) (Phantom)	17.80	-	8.13	28.47	-	12.10
Time to get to 43°C (min) (Duke)	3.00	3.42	2.65	3.25	3.63	2.85

Table 1. Peak SAR and temperature for nominal σ (default used), nominal σ ±20% (due to inter-subject variability alone), nominal σ + 12% (due to temperature dependent σ alone), and nominal σ ±20% + 12% (due to both literature variability and temperature dependent σ).

Results and Discussion: Fig. 2 shows the axial SAR_{10g} and temperature maps for both muscle phantom and Duke for scenarios (**A**) to (**F**). Table 1 lists the peak SAR_{10g} and temperatures after 17.8 minutes of heating in the phantom, and after 3 minutes in Duke. In principle, SAR is linearly proportional to σ for a given set of E fields. However, due in part to the decreased skin depth when σ increases (higher RF energy absorption on sample surface), a higher σ does not necessarily imply a higher SAR_{10g} in the center of the muscle phantom and HBM. Indeed, when σ is 20% lower than the nominal values (case **C**), the peak SAR_{10g} values in the center of the phantom was higher than that in the nominal σ case. This leads to a peak temperature of 48.30 °C after 17.80 minutes of heating, which is 5.3 °C higher than the peak temperature of 43.0 °C in the nominal σ case. If the true temperature is higher than that predicted in the nominal σ scenario, the latter would be underestimating the thermal risk to the patient. Similarly, an increase in σ by 32% above nominal leads to a ΔT of 3.7 °C, below that for the nominal σ case. In the Duke HBM, peak temperatures of 43.50 ºC and 42.3 ºC were observed after 3 minutes of heating in cases **C** and **E**, respectively. These values were 0.5 ºC higher (case **C**), and 0.7 ºC lower (case **E**) than those for the nominal σ case. In Duke, the interaction of fields with biological tissues is more complicated due to the asymmetric shape of the head, gradients in dielectric properties, and tissue-dependent blood perfusion. However, it was still observed that a prediction error of up to 1.6ºC could arise from using the nominal value of σ in place of the range of possible values defined in Table 1.

Conclusions: Errors in temperatures of up to 5.3 °C in phantom, and 0.7 °C in Duke, were induced by the misestimating of σ. This may impact thermal risk assessment during EM model-based safety evaluation of MRI systems and RF thermotherapy treatment. These results can be also extended to understand associated errors in SAR prediction in parallel transmit 7T MR systems.

References: [1] Van der Zee J. Ann of Onc 2002;13(8):1173–84. [2] Wust P, et al. Lanc Onc 2002; 3(8):487–97. [3] Vaughan JT, et al. Wiley, 2012. [4] Gabriel S, et al. Phys Med Biol 1996;41:2271-93. [5] Gabriel C, et al. Phys Med Biol 1996;41:2231-49. [6] Keshvari J, et al. Phys Med Biol. 2006;51:1463-77. [7] Xu L, et al. IEEE TBE 2009; 56:2083-94. [8] Zheng E, et al. EEE TBE, 1984;BME-31:477–81. [9] Esrick MA, et al. Phys Med Biol 1994;36:133-144. [10] Baumann SB, et al. IEEE TBE. 1997;44:220-3. [11] Polk C, et al. CRC 1986. [12] Christ A. et al. Phys Med Biol 2010;55: N23–38. [13] Jones EL, et al. J Clin Oncol 2005;23:3079-085