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The Relation between Regional Brain Iron and T2 Shortening

J. M. Gomori[1] and R. I. Grossman[2]

Thomas et al attempt to quantitate, using in vitro and in vivo studies, the relationship between regional brain iron and T2 shortening (1). The paper reconfirms two well-known facts: 1) iron concentration of the deep gray-matter nuclei increases during the first 3 decades of life (2–9); 2) T2 of brain tissue decreases rapidly during the first 2 years of life (10). The paper does not identify nor measure the concentration of the different forms of iron that actually contribute to the total brain iron. Furthermore, the exact contribution of iron versus all other mechanisms contributing to the T2 of the deep gray-matter nuclei are not separated and measured.

There are some problems with the use of ferric and ferrous ammonium solutions as models for brain iron. These solutions also shorten T1 almost as much as T2. This is not what occurs in vivo where there is preferential shortening of T2 (11–14). The T2 of these solutions is not sensitive to field strength unlike the T2 of the gray-matter nuclei (13) (unpublished measurements of 1/T2 at 0.5 and 2.0 T, Gomori et al, see below). Lastly, these solutions do not exhibit T2* (susceptibility heterogeneity effects), unlike the gray matter nuclei that become relatively more hypointense on long echo time gradient-echo images (14). Thus, the modeling the authors have chosen does not closely follow the clinical observations of preferential T2 shortening.

Because of these problems this study does not refute the recent conclusion by Chen et al that iron content is not the dominant moderator of brain T2 (15). However, we believe that the Chen paper is flawed by the use of 2.5-msec interecho intervals. This has been shown to significantly decrease the T2 shortening effect of hemosiderotic deposits (16, 17). Their technique may be responsible for Chen et al's observed lack of correlation between T2 and elevated brain iron content.

TABLE 1: 1/T2 (sec^{-1}) in a 37-year-old patient at 0.5 T and 2.0 T

	0.5 T	2.0 T
Genu corpus callosum	13.4 ± 1.5	14.2 ± 2.0
Frontal white matter	13.5 ± 1.2	12.9 ± 1.2
Caudate	11.5 ± 0.7	12.4 ± 1.1
Basal ganglia	11.1 ± 0.9	17.8 ± 1.4
Posterior thalamus	8.7 ± 0.7	6.5 ± 0.9

On the basis of the finding of increasing iron concentration in the deep gray matter over the first 3 decades of life and the known field-strength–dependent preferential T2 and T2* shortening of hemosiderin and ferritin, it can be concluded that iron content contributes to the T2 relaxation of the deep gray-matter nuclei.

Gomori et al measured the 1/T2 (sec^{-1}) relaxation rates of brain structures in a 37-year-old volunteer at 0.5 T and 2.0 T (Gyrex, Elscint Ltd.) using a 4-echo sequence ending at an echo time of 140 msec. The results are shown in Table 1. These preliminary measurements indicate that the field-strength–dependent contribution to a T2 relaxation rate of the adult basal ganglia is at least 40% because of field-dependent susceptibility-related mechanisms.

We agree with the conclusion of Thomas et al that brain iron appears to contribute to the decreased T2 signal seen in the deep gray-matter nuclei, although their experiment as published does not directly support this hypothesis.

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