

Vascular and metabolic responses to elevated circulating PDGF-BB in mice: A multiparametric MRI study

Xiuli Yang[#], Jiekang Wang[#], Yuguo Li, Mei Wan^{*}, Zhiliang Wei^{*}

Supplementary Materials

1. Regional vulnerability to CBF impairment without normalization to PC results

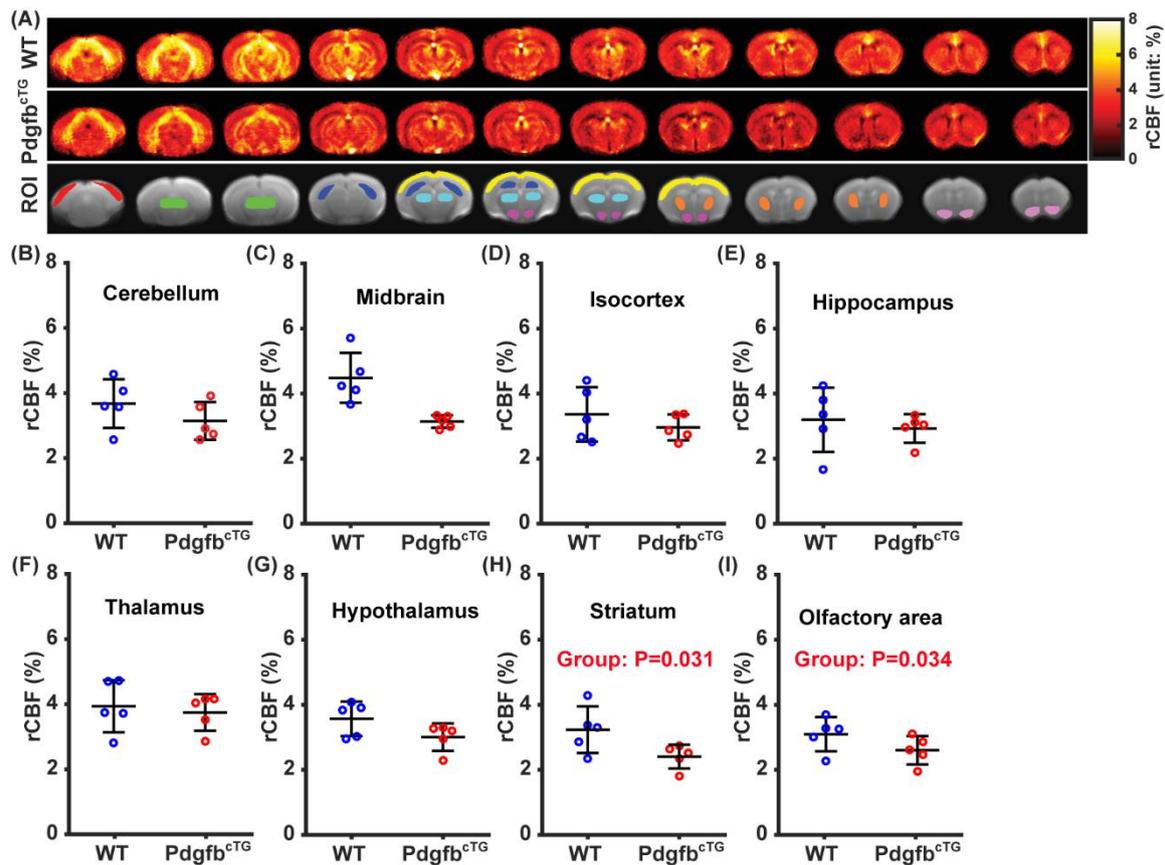


Figure S1 rCBF without normalization to PC results. (A) shows the averaged rCBF maps of WT and Pdgfb^{CTG} mice. ROIs were overlaid on the averaged control image and displayed. Without normalization, the rCBF maps are presented as percentages, representing the ratio between the difference signal (ΔM) and the equilibrium magnetization signal (M_0). (B-I) illustrate the comparisons of rCBF between WT and Pdgfb^{CTG} mice in various brain regions, including the cerebellum, midbrain, isocortex, hippocampus, thalamus, hypothalamus, striatum, and olfactory area.

Based on linear regression models, there were no significant group effects on rCBF in cerebellum (Figure S1B, coefficient = -0.75%, CI = [-1.68, 0.18], $P = 0.097$), midbrain (Figure S1C, coefficient = -1.24%, CI = [-2.58, 0.10], $P = 0.064$), isocortex (Figure S1D, coefficient = -0.87%, CI = [-1.82, 0.08], $P = 0.067$), hippocampus (Figure S1E, coefficient = -0.25%, CI = [-1.61, 1.11], $P = 0.670$), thalamus (Figure S1F, coefficient = -0.42%, CI = [-1.23, 0.38], $P = 0.247$), and hypothalamus (Figure S1G, coefficient = -0.50%, CI = [-1.31, 0.30], $P = 0.178$). In contrast, significant group effects on rCBF were observed in the



striatum (Figure S1H, coefficient = -1.07%, CI = [-2.00, -0.13], P = 0.031) and olfactory area (Figure S1I, coefficient = -0.80%, CI = [-1.52, -0.08], P = 0.034).

2. Quantitative relaxation measurements in the Pdgfb^{cTG} model

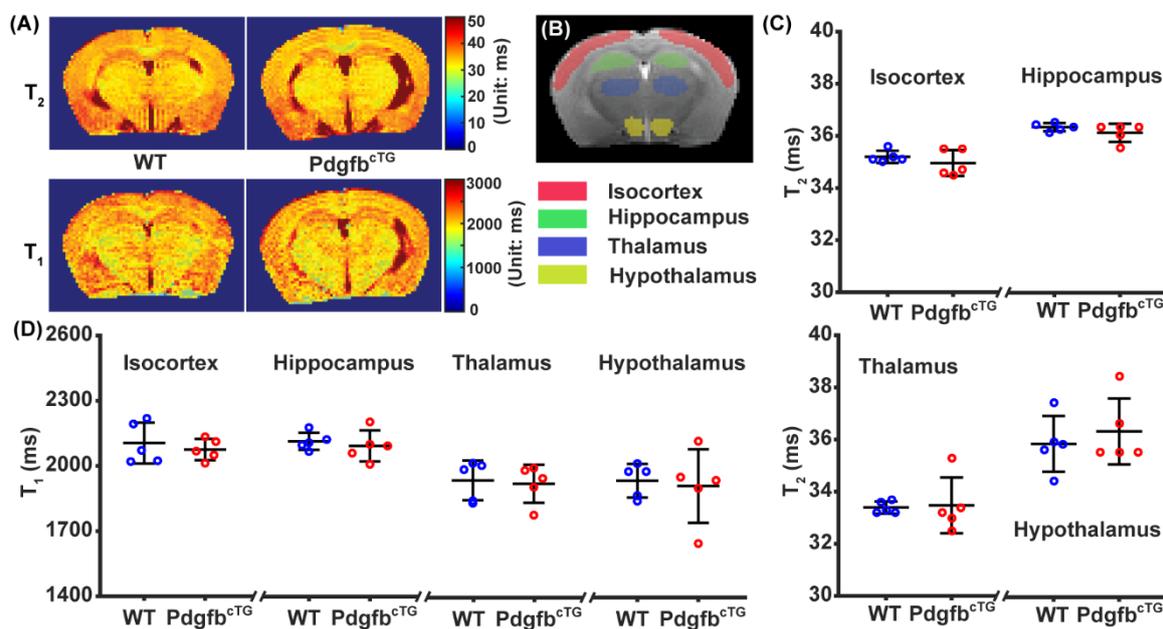


Figure S2 Quantitative relaxation measurements in the Pdgfb^{cTG} model. (A) illustrates the representative T₂/T₁ maps of WT and Pdgfb^{cTG} mice; (B) shows the ROIs used in regional T₂/T₁ quantifications; (C) and (D) present the regional comparisons of T₂ and T₁, respectively.

Quantitative transverse (T₂) and longitudinal (T₁) relaxation times were measured using multi-echo spin echo (MESE) and variable-repetition-time fast spin echo (VTR-FSE) MRI techniques, respectively. MESE was performed with the following parameters: TR = 2500 ms, 20 TE ranging from 7.5 ms to 150.0 ms with an interval of 7.5 ms, FOV = 15 mm × 15 mm, matrix size = 96 × 96, slice thickness = 0.7 mm, and scan duration = 4.0 min. VTR-FSE MRI was performed with the following parameters: TR = 5500/3000/1500/800/500/300 ms, TE = 6.5 ms, FOV = 15 mm × 15 mm, matrix size = 96 × 96, slice thickness = 0.7 mm, 8 spin echoes per scan, and scan duration = 2.3 min. Both MESE and VTR-FSE were focused on a single slice of the parietal lobe to save time while still covering major brain regions, including isocortex, hippocampus, thalamus, and hypothalamus.

Figure S2A presents the T₂ and T₁ maps of representative WT and Pdgfb^{cTG} mice. ROIs were drawn on an individual basis (Figure S2B), ensuring that regions contaminated by CSF signals were avoided. Regarding the comparisons of T₂ relaxation times (Figure S2C), there were no significant differences in the isocortex ($t(8) = 0.97$, P = 0.358), hippocampus ($t(8) = 1.28$, P = 0.235), thalamus ($t(8) = -0.16$, P = 0.874), or hypothalamus ($t(8) = -0.65$, P = 0.535), suggesting no significant hemosiderin deposits, calcification or edema (common causes affecting tissue T₂ property¹⁻³) in major regions of the parietal brain. Similar findings were observed in the T₁ measurements (Figure S2D), which showed no significant differences between WT and Pdgfb^{cTG} mice in the isocortex ($t(8) = 0.63$, P = 0.546), hippocampus ($t(8) = 0.58$, P = 0.580), thalamus ($t(8) = 0.28$, P = 0.789), or hypothalamus ($t(8) = 0.29$, P = 0.778), indicating no significant changes in water content and tissue composition of the parietal brain.⁴

References

1. Vymazal, J.; Brooks, R. A.; Baumgarner, C.; Tran, V.; Katz, D.; Bulte, J. W.; Bauminger, R.; Di Chiro, G., The relation between brain iron and NMR relaxation times: an in vitro study. *Magn Reson Med* **1996**, *35* (1), 56-61.
2. Henkelman, R. M.; Watts, J. F.; Kucharczyk, W., High signal intensity in MR images of calcified brain tissue. *Radiology* **1991**, *179* (1), 199-206.
3. Naruse, S.; Horikawa, Y.; Tanaka, C.; Hirakawa, K.; Nishikawa, H.; Yoshizaki, K., Significance of proton relaxation time measurement in brain edema, cerebral infarction and brain tumors. *Magn Reson Imaging* **1986**, *4* (4), 293-304.

4. Gaeta, M.; Galletta, K.; Cavallaro, M.; Mormina, E.; Cannizzaro, M. T.; Lanzafame, L. R. M.; D'Angelo, T.; Blandino, A.; Vinci, S. L.; Granata, F., T1 relaxation: Chemo-physical fundamentals of magnetic resonance imaging and clinical applications. *Insights Imaging* **2024**, 15 (1), 200.