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Reply

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Reply:

On behalf of all of the authors of our study,¹ we thank Dr Renema for his interest in our work and we appreciate his comments. We are fully aware that other studies demonstrate that the cellular pools of adenosine triphosphate (ATP) are very stable during tissue activation and that they are regulated not only by the creatine kinase (CK) system but also by mitochondrial oxidative phosphorylation, glycolysis, and the adenosine monophosphate-cyclase system. Most of these studies have been performed on muscles, and only few data are available for the brain in which the control, coupling, and kinetics of the phosphocreatine/CK/ATP system are much more complex.^{2,3} Acquisition and postprocessing of our ³¹P-MR spectroscopy studies were carried out according to standard procedures for human brain studies. The increased signal intensity of the ATP peaks in the brain of the child with the guanidinoacetate methyltransferase defect (GAMT-d; Fig 2 of our original article) was unique. Due to the different arbitrary units used by Renema et al² and Schulze et al,⁴ we have probably erroneously referenced these articles as supporting our results. There-

fore, we believe that the question raised by Renema is still waiting to be confirmed by further ³¹P-MR spectroscopy studies of patients with GAMT-d aimed at measuring absolute concentration of brain ATP.

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