episodic pain disorder.⁴ The positive response to riboflavin in patients with a distinctive DNA haplogroup may indicate correction of an underlying metabolic abnormality that could potentially lead to a chronic disabling state.

In addition to pain relief and recurrence reduction, migraine treatment should include preventing the progression to chronic migraine, which is poorly responsive to therapy. Developing an understanding of the basic pathophysiology of the disease such as determination of mitochondrial metabolism contributing to migraine may lead to this type of preventative measure. Those having this metabolic abnormality may need lifelong treatment to prevent migraine progression.

Steven R. Brenner, St. Louis, MO

Disclosure: The author reports no disclosures.

Reply from the Author: I thank Dr. Brenner for his comments on our article. However, pending further research on metabolic enhancers and other preventive antimigraine treatments, it is not possible to comment on his assertions.

The reduction of nonspecific white matter lesions (WML), typical in the migraineur brain, might be considered as another possible outcome for high-dose riboflavin prolonged treatment. WML have to be distinguished from mitochondrial encephalopathy with lactic acidosis and stroke-like episodes or other

forms of leukoencephalopathy and vasculitis.⁵ WLM could have a mitochondrial pathogenesis, and treatment with metabolic enhancers could be effective to reduce them, confirming the theory regarding their pathogenesis.

Our experience shows that long-term treatment with high-dose riboflavin has been safe. We have treated patients for both mitochondriopathy and migraine without major complications. These observations are consistent with Brenner's suggestion regarding lifelong treatment to prevent chronic migraine.

Cherubino Di Lorenzo, Latina, Italy

Disclosure: The author reports no disclosures.

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CORRECTION

From stargazing chicks to seizing infants: Thiamine deficiency redux

In the editorial "From stargazing chicks to seizing infants: Thiamine deficiency redux" by M.C. Patterson (*Neurology*® 2009;73:824–825), the author incorrectly referred to Dr. Denis Leigh as John Leigh. The author regrets the error.

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