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American Journal of Clinical Nutrition, Vol. 85, No. 4, 946-949, April 2007 © 2007 American Society for Nutrition

## COMMENTARY

Consideration of betaine and one-carbon sources of  $N^5$ -methyltetrahydrofolate for use in homocystinuria and neural tube defects<sup>1, 2</sup>

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A major focus in attempts to ameliorate homocystinuria and neural tube defects is supplementation of the diet with B vitamins. The metabolic defect in these cases may be due in part to a deficiency of methyl groups. B vitamin supplementation supports the need for enzyme cofactors but cannot provide substrate in the form of methyl groups. L-Methionine is an essential amino acid and is required for protein synthesis, but it also plays a unique role in metabolism as S-adenosylmethionine, which is the primary methyl donor in metabolism. The observation that L-homocysteine, which is produced in the metabolism of L-methionine, is remethylated 2— 4 times before it is destroyed is key to understanding the possibility of a methyl group deficiency. This suggests that the requirement for methyl groups (ie, S-adenosylmethionine) may be 2— 4 times that for methionine in support of protein synthesis. L-Homocysteine can be remethylated to form

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L-methionine by betaine or  $N^5$ -methyl tetrahydrofolate. Betaine and one-carbon sources that lead to the production of  $N^5$ -methyl tetrahydrofolate and the remethylation of L-homocysteine to form L-methionine should be considered along with B vitamin supplementation in the treatment of homocystinuria and neural tube defects.

Key Words: Betaine • methyltetrahydrofolate • homocystinuria • neural tube defects

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R. N. Dilger, T. A. Garrow, and D. H. Baker

Betaine Can Partially Spare Choline in Chicks but Only When Added to Diets Containing a Minimal Level of Choline

J. Nutr., October 1, 2007; 137(10): 2224 - 2228.

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