

Abstract

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Cognitive impairment in folate-deficient rats corresponds to depleted brain phosphatidylcholine and is prevented by dietary methionine without lowering plasma homocysteine.

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BACKGROUND: Poor folate status is associated with cognitive decline and dementia in older adults. Although impaired brain methylation activity and homocysteine toxicity are widely thought to account for this association, how folate deficiency impairs cognition is uncertain.

OBJECTIVE AND METHODS: To better define the role of folate deficiency in cognitive dysfunction, we fed rats folate-deficient diets (0 mg FA/kg diet) with or without supplemental L-methionine for 10 wk, followed by cognitive testing and tissue collection for hematological and biochemical analysis.

RESULTS: Folate deficiency with normal methionine impaired spatial memory and learning; however, this impairment was prevented when the folate-deficient diet was supplemented with methionine. Under conditions of folate deficiency, brain membrane content of the methylated phospholipid phosphatidylcholine was significantly depleted, which was reversed with supplemental methionine. In contrast, neither elevated plasma homocysteine nor brain S-adenosylmethionine and S-adenosylhomocysteine concentrations predicted cognitive impairment and its prevention by methionine.

CONCLUSION: The correspondence of cognitive outcomes to changes in brain membrane phosphatidylcholine content suggests that altered phosphatidylcholine and possibly choline metabolism might contribute to the manifestation of folate deficiency-related cognitive dysfunction.

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