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da Costa, KA, et al. Choline deficiency in mice and humans is associated with increased plasma homocystine concentration after a methionine load. *AJCN* 2005; 81: 440-444.

**Background:** Elevated concentrations of homocysteine in blood may be an independent risk factor for the development of atherosclerosis. Elevated homocysteine concentrations can be caused by decreased methylation of homocysteine to form methionine, as occurs in folate deficiency. A parallel pathway exists for methylation of homocysteine, in which choline, by way of betaine, is the methyl donor.

**Objective:** Our goal was to determine whether choline deficiency results in a decreased capacity to methylate homocysteine.

**Design:** C57BL/6J mice were fed diets containing 0, 10, or 35 mmol choline/kg diet for 3 wk. We then administered an oral methionine load to the animals and measured plasma homocysteine concentrations. Also, in a pilot study, we examined 8 men who were fed a diet providing 550 mg choline/d per 70 kg body weight for 10 d, followed by a diet providing almost no choline, until the subjects were clinically judged to be choline deficient or for  $\leq 42$  d. A methionine load was administered at the end of each dietary phase.

**Results:** Two hours after the methionine load, choline-deficient mice had plasma homocysteine concentrations twice those of choline-fed mice. Four hours after the methionine load, clinically choline-depleted men had plasma homocysteine concentrations that were 35% greater than those in men not choline depleted.

**Conclusion:** These results suggest that choline, like folate, plays an important role in the metabolism of homocysteine in humans and that response to a methionine load may be useful when assessing choline nutriture.