

Abstract

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Nutritional genomics: defining the dietary requirement and effects of choline.

Zeisel SH.

Nutrition Research Institute, School of Public Health and School of Medicine, The University of North Carolina at Chapel Hill, Kannapolis, NC 28081.

BACKGROUND: As it becomes evident that single nucleotide polymorphisms (SNPs) in humans can create metabolic inefficiencies, it is reasonable to ask if such SNPs influence dietary requirements. Epidemiologic studies that examine SNPs relative to risks for diseases are common, but there are few examples of clinically sized nutrition studies that examine how SNPs influence metabolism.

FINDINGS: Studies on how SNPs influence the dietary requirement for choline provide a model for how we might begin examining the effects of SNPs on nutritional phenotypes using clinically sized studies (clinical nutrigenomics). Most men and postmenopausal women develop liver or muscle dysfunction when deprived of dietary choline. More than one-half of premenopausal women may be resistant to choline deficiency-induced organ dysfunction, because estrogen induces the gene [phosphatidylethanolamine-N-methyltransferase (PEMT)] that catalyzes endogenous synthesis of phosphatidylcholine, which can subsequently yield choline. Those premenopausal women that do require a dietary source of choline have a SNP in PEMT, making them unresponsive to estrogen induction of PEMT.

CONCLUSIONS: It is important to recognize differences in dietary requirements for choline in women, because during pregnancy, maternal dietary choline modulates fetal brain development in rodent models. Because choline metabolism and folate metabolism intersect at the methylation of homocysteine, manipulations that limit folate availability also increase the use of choline as a methyl donor. People with a SNPs in MTHFD1 (a gene of folate metabolism that controls the use of folate as a methyl donor) are more likely to develop organ dysfunction when deprived of choline; their dietary requirement is increased because of increased need for choline as a methyl donor.

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