Abstract


The nutritional burden of methylation reactions.

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PURPOSE OF REVIEW: Methyl group metabolism is a metabolically demanding process that has significant nutritional implications. Methionine is required not only for protein synthesis but also as the primary source of methyl groups. However, demethylated methionine can be remethylated by methyl groups from methylneogenesis (via folate) and betaine (synthesized from choline). This review discusses the impact of methylation precursors and products on the methionine requirement.

RECENT FINDINGS: Recent evidence has clearly demonstrated that transmethylation reactions can consume a significant proportion of the flux of methionine. In particular, synthesis of creatine and phosphatidylcholine consume most methyl groups and their dietary provision could spare methionine. Importantly, methionine can become limiting for protein and phosphatidylcholine synthesis when creatine synthesis is upregulated. Other research has shown that betaine and choline seem to be more effective than folate at reducing hyperhomocysteinemia and impacting cardiovascular outcomes suggesting they may be limiting.

SUMMARY: It appears that methyl groups can become limiting when dietary supply is inadequate or if transmethylation reactions are upregulated. These situations can impact methionine availability for protein synthesis, which can reduce growth. The methionine requirement can likely be spared by methyl donor and methylated product supplementation.

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