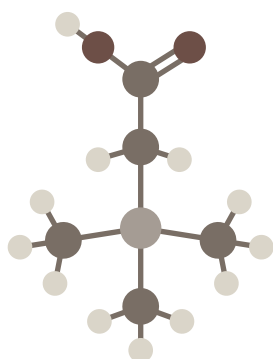
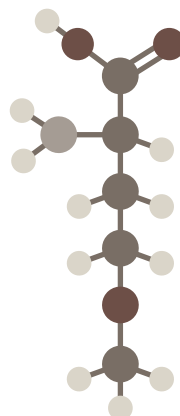


The science is clear: Betaine cannot replace methionine

BY ANDREAS LEMME



Betaine 



Methionine 

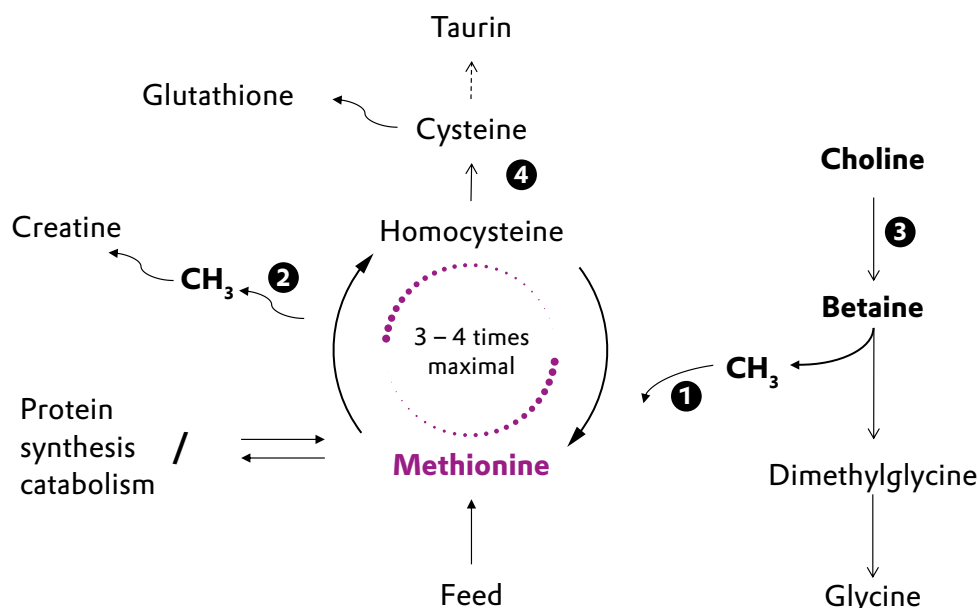
BETAINE CAN NOT REPLACE METHIONINE

NUTRITIONISTS CONSTANTLY SEEK OPPORTUNITIES TO REDUCE DIET COSTS WHILE MAINTAINING OPTIMAL ANIMAL PERFORMANCE. WHEN INGREDIENT PRICES RISE, NUTRITIONISTS SEARCH FOR WAYS TO REDUCE THEIR USE OR REPLACE THEM WITH ALTERNATIVE INGREDIENTS.

With respect to methionine (**Met**), it is sometimes claimed that supplemental betaine (**BET**) has the potential to replace a substantial portion of supplemental DL-Met. Indeed, BET and Met are connected, as BET can donate a methyl group (CH_3 ; 1) to remethylate homocysteine to form Met. Met, in turn, can donate a methyl group towards creatine formation (2), releasing homocysteine. This pathway is known as the homocysteine cycle. BET cannot directly participate in creatine synthesis. Although BET can be

formed out of choline (3), choline itself cannot donate a methyl group directly into the homocysteine cycle. By definition, BET cannot replace Met, but it may increase the number of homocysteine remethylation cycles. It has been reported that, under adequate Met supply, homocysteine undergoes remethylation 1.5 to 2 times until it is transformed into cysteine (4) or further degraded [1]. When only half of dietary Met is given, the number of remethylation cycles only doubled [1].

Metabolic pathways

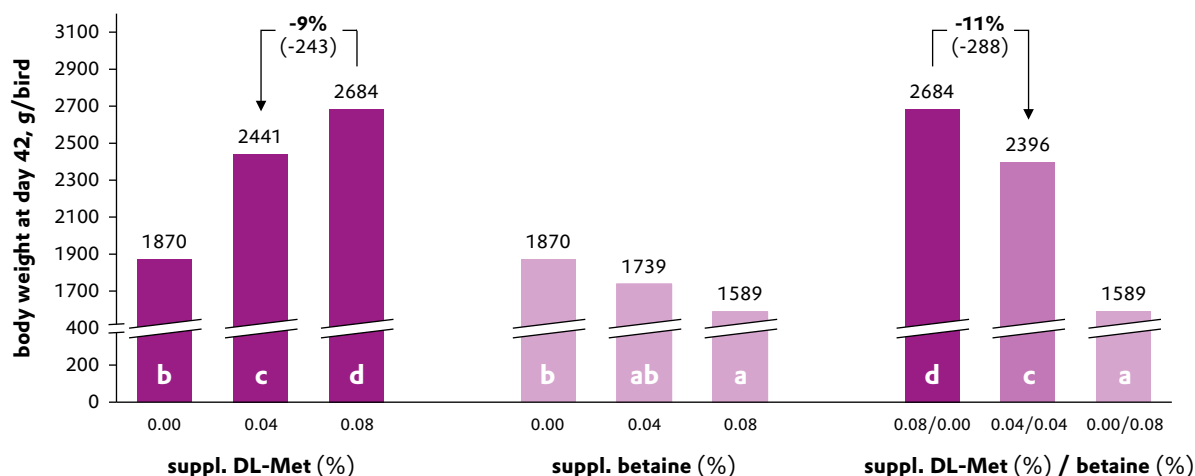


Consequently, BET has limited potential to improve Met metabolism via the homocysteine cycle, and only under Met deficient conditions. Such conditions do not occur in commercial diets. In fact, the opposite is often true. Dietary Met often exceeds the optimal concentration as the exact requirement is difficult to estimate for specific production conditions due

to the involvement of Met in numerous metabolic pathways [2]. In addition, between-batch variations of the nutritional profile of macro feed ingredients force nutritionists to implement safety margins, often resulting in luxury rather than marginal Met supply. If Met is oversupplied, the surplus can be replaced with any other compound, including BET, or simply reduced.

A trial with male broilers (130 birds/pen; 6 pens/trt), in which equimolar levels of DL-Met or BET were added to Met+Cys deficient starter (0-10d), grower (11-27d) and finisher (28-42d) diets, demonstrated that BET had neither a beneficial impact on growth performance nor the potential to replace DL-Met [3]. Accordingly, addition of 0.04 or 0.08% DL-Met significantly increased final body weights by 31 or

44%, respectively, whereas equimolar additions of 0.04 or 0.08% BET had no positive impact. Moreover, gradual replacement of DL-Met with BET did not suggest any Met-sparing potential for BET. The significant reduction in body weight with increasing BET supplementation does not indicate more efficient metabolic utilization of Met.



The growth study provided evidence that dietary addition of BET cannot replace supplemental DL-Met at or below Met+Cys requirement. This finding is substantiated by the mode of action of the homocysteine cycle. If replacing DL-Met with BET does

not compromise animal performance, then only surplus Met above the required amount has been replaced. Therefore, adjusting the dietary Met+Cys specification is advisable in order to minimize diet costs.

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