

Growth Retardation Induced by Protein and Indispensable Amino Acid Deficiencies Can Not Be Catch Up by a Supplementation in Growing Rats

Gaëtan Roisné-Hamelin, Catherine Chaumontet, Juliane Calvez, Claire Gaudichon, Daniel Tomé, and Dalila Azzout-Marniche

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Objectives: In developing countries, children are exposed to a risk of growth retardations due to their low protein (LP) intake or poor quality of protein sources with unbalanced indispensable amino acid (IAA) composition. However, the specificity of each IAA and the ability of children to catch-up their growth retardation remains unclear. The aim of this study was to assess the supplementation efficiency following a protein or IAA (lysine, threonine, and methionine) deficiencies in growing rats, and to identify the specific IAA deficiency effect.

Methods: Sixty growing rats were fed by a control (20% of proteins; P20), a LP (5% of proteins; LP) or IAA deficient (25% of the rat's requirement in lysine, threonine, or methionine; L25, T25 and M25) diets for 3w. Thereafter, all rats were supplemented by the control (P20) or a control-equivalent diet containing free AA. Body weight (BW) and food intake were daily recorded. Naso-anal length (NAL), bone mineral density (BMD) and body composition were measured at the end of each period.

Results: During the deficiency, IAA as LP diets reduced BW gain from day 2 for LP, L25 and M25 and from day 1 for T25. At the end of the deficiency, BW was reduced by 30% for L25 and M25, 50% for LP and 60% for T25. NAL was also reduced by 9, 18, 25% for L25/M25, LP and T25. At the end of the deficiency, all groups had less lean body mass (LBM), whereas only LP and T25 had a decreased BMD. Furthermore, the fat mass was only decreased for LP and T25 groups. During the supplementation, growth resumes and the weight's gap between each group was reduced, but remains after supplementation. The BW were reduced by 15, 25 and 35% for L25/M25, LP and T25, respectively. For NAL and LBM, the gap slightly reduced too, but the difference remains after the supplementation. Indeed, the NAL was reduced by 5% for L25 and M25, 8% for LP and 10% for T25. All groups had reduced kidney, muscle and carcass weight, and LP and T25 had a reduced liver weight and BMD. The T25 group was the most affected by the deficiency, even more than LP.

Conclusions: A single IAA deficiency as LP induced growth retardation, and the LBM is highly affected. A supplementation allows growth resume, but the growth retardation cannot be catch up. The stronger effect observed for threonine deficiency, could be due to a wrong estimation of threonine requirement.

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