Abstract


Dietary vitamin B6 intake modulates colonic inflammation in the IL10(-/-) model of inflammatory bowel disease.


Vitamin Metabolism, Jean Mayer USDA Human Nutrition Research Center on Aging at Tufts University, Boston, MA.

BACKGROUND: Pyridoxal-5-phosphate, the biologically active form of vitamin B6, is a cofactor for over 140 biochemical reactions. Although severe vitamin B6 deficiency is rare, mild inadequacy [plasma pyridoxal 5'-phosphate (PLP) <20 nmol/L] is observed in 19-27% of the US population. Plasma PLP concentrations are inversely related to markers of inflammation such as C-reactive protein. Furthermore, plasma PLP is diminished in those with inflammatory conditions and, in the case of inflammatory bowel disease (IBD), more so in those with active versus quiescent disease.

OBJECTIVE: Restricting B6 intake attenuates IBD pathology in mice; however, the effects of supplementation are unclear. We therefore sought to determine the effects of mild inadequacy and moderate supplementation of B6 on the severity of colonic inflammation.

METHODS: Weanling IL-10(-/-) (positive for Helicobacter hepaticus) mice were fed diets containing 0.5 (deficient), 6.0 (replete) or 24 (supplemented) mg/kg pyridoxine HCl for 12 weeks and then assessed for histological and molecular markers of colonic inflammation.

RESULTS: Both low and high plasma PLP were associated with a significant suppression of molecular (TNFα, IL-6, IFN-γ, COX-2 and iNOS expression) and histological markers of inflammation in the colon. PLP is required for the breakdown of sphingosine 1-phosphate (S1P), a chemotactic lipid, by S1P lyase. Colonic concentrations of S1P and PLP were significantly and inversely correlated.

CONCLUSION: If confirmed, vitamin B6 supplementation may offer an additional tool for the management of IBD. Although B6 is required in dozens of reactions, its role in the breakdown of S1P may explain the biphasic relationship observed between PLP and inflammation.

PMID: 24183308