

Abstract

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Inflammation causes tissue-specific depletion of vitamin B6.

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BACKGROUND: Previously we observed strong and consistent associations between vitamin B6 status and several indicators of inflammation in patients with rheumatoid arthritis. Clinical indicators, including the disability score, the length of morning stiffness, and the degree of pain, and biochemical markers, including the erythrocyte sedimentation rate and C-reactive protein levels, were found to be inversely correlated with circulating vitamin B6 levels. Such strong associations imply that impaired vitamin B6 status in these patients results from inflammation.

METHODS: In the present study we examined whether inflammation directly alters vitamin B6 tissue contents and its excretion in vivo. A cross-sectional case-controlled human clinical trial was performed in parallel with experiments in an animal model of inflammation. Plasma and erythrocyte and pyridoxal 5'-phosphate concentrations, urinary 4-pyridoxic acid excretion, and the activity coefficient of erythrocyte aspartate aminotransferase were compared between patients and healthy subjects. Adjuvant arthritis was induced in rats for investigating hepatic and muscle contents as well as the urinary excretion of vitamin B6 during acute and chronic inflammation.

RESULTS: Patients with rheumatoid arthritis had low plasma pyridoxal 5'-phosphate compared with healthy control subjects, but normal erythrocyte pyridoxal 5'-phosphate and urinary 4-pyridoxic acid excretion. Adjuvant arthritis in rats did not affect 4-pyridoxic acid excretion or muscle storage of pyridoxal 5'-phosphate, but it resulted in significantly lower pyridoxal 5'-phosphate levels in circulation and in liver during inflammation.

CONCLUSION: Inflammation induced a tissue-specific depletion of vitamin B6. The low plasma pyridoxal 5'-phosphate levels seen in inflammation are unlikely to be due to insufficient intake or excessive vitamin B6 excretion. Possible causes of decreased levels of vitamin B6 are discussed.

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