Tryptophan metabolism and vitamin B6 nutritional status in patients with schistosomiasis mansoni and in infected mice.

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Abstract:

Patients infected with Schistosoma mansoni showed an abnormal response to a test dose of tryptophan, with little increase in the urinary excretion of kynurenine, hydroxykynurenine, xanthurenic and kynurenic acids, N1-methyl nicotinamide, methyl pyridone carboxamide, 5-hydroxytryptamine or 5-hydroxyindoleacetic acid. In contrast to previous reports, this is different from the pattern of tryptophan metabolism seen in vitamin B6 deficiency. Furthermore, the patients' plasma concentrations of pyridoxal phosphate were within the reference range, and supplementation for 5 days with 20 mg vitamin B6/day did not affect tryptophan metabolism. Treatment with a single dose of Praziquantel resulted in a substantial restoration of normal tryptophan metabolism. In mice infected with S. mansoni there was a similar impairment of tryptophan metabolism, as shown by considerably reduced formation of 14CO2 after the administration of a tracer dose of [14C]tryptophan. Again, the administration of vitamin B6 supplements did not correct tryptophan metabolism in the mice. Treatment with Praziquantel resulted in substantial restoration of the production of 14CO2 from [14C]tryptophan. There was no evidence of vitamin B6 deficiency (as determined by erythrocyte aspartate aminotransferase activation coefficient) associated with infection in the mice, although there was a redistribution of pyridoxal phosphate between tissues, with a reduction in the concentration of liver, spleen and kidney, and an increase in skeletal muscle.