

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, *N*¹-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.