

Abstract

In mice, infection with 20–30 cercariae of *Schistosoma mansoni* resulted in a considerable reduction in the formation of $^{14}\text{CO}_2$ from [^{14}C]tryptophan. Infected animals excreted significantly lower amounts of kynurenine, kynurenic acid, and methyl pyridone carboxamide than did uninfected controls. There was no difference in the ability of hepatocytes isolated from infected or control animals to metabolise [^{14}C]tryptophan. Hepatocytes from infected animals synthesized less NAD(P), but more niacin and N^1 -methyl nicotinamide from tryptophan. They showed no greater accumulation of kynurenine metabolites than did cells from control animals. The hepatocyte content of pyridoxal phosphate and the erythrocyte aspartate aminotransferase activation coefficient were the same in both groups of mice, suggesting that infection with *S. mansoni* does not deplete vitamin B₆. The impairment of tryptophan metabolism *in vivo* was apparently not due to impaired hepatic metabolism. Rather, it seems likely that the parasites or their eggs take up tryptophan avidly from the host's circulation. Studies of parasite and egg metabolism of tryptophan may suggest novel approaches to the chemotherapy of bilharzia.