

Enhanced inducible nitric oxide synthase expression and nitrotyrosine accumulation in experimental granulomatous hepatitis caused by *Toxocara canis*

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Abstract

The involvement of inducible [nitric oxide synthase](#) (iNOS) and nitrotyrosine (NT) in [pathogenesis](#) of toxocaral [granulomatous hepatitis](#) (TGH) in a murine host was quantitatively determined by biochemical, parasitological, [pathological](#), and [immunohistochemical](#) assessments in a 42-week investigation. [Mice](#) were sacrificed for serum collection and [histological](#) processing as well as acid-pepsin [digestion](#) of the [liver](#) in a [larval](#) recovery study. Significantly increased levels of total serum NO were found in the trial, indirectly suggesting iNOS [activation](#) in the [liver](#). iNOS reactivity was predominantly observed in infiltrating [leucocytes](#) in [lesions](#) and normal and apocrine-like [cholangiocytes](#); in contrast, [hepatocytes](#) and [multinucleated giant cells](#) showed negative [cytoplasmic staining](#) in TGH. Strong iNOS-like reactivity was also detected on the body wall of larvae. The locations of NT reactivity were nearly identical to those of iNOS expression; infiltrating [leucocytes](#) or [cholangiocytes](#) stained for iNOS were also stained for NT in TGH. Enhanced iNOS expression, but not invading larvae ($r = 0.256$, $P = 0.211$), seemed to play a certain role in [pathological](#) damage in TGH due to a significant [correlation](#) between iNOS expression and serum [alanine aminotransferase](#) (ALT) levels ($r = 0.593$, $P = 0.021$) in the trial. Our present results indicate a potential therapeutic strategy for treatment of GH caused by other [nematodes](#) through manipulation of iNOS expression.