Abstract
Visceral leishmaniasis is a fatal disease caused by the protozoan parasite *Leishmania donovani*. T cells are known to mediate resistance or susceptibility against this ‘neglected disease’ by the intricate cytokine milieu created by these cells during infection with *L. donovani*. Progression of visceral leishmaniasis is characterized by suppression of anti-leishmanial T cell responses, which was thought to be due to deficiency of interleukin-2, a potent T cell growth factor. The present study defies this to some extent, showing that during the first week of *L. donovani* infection, IL-2 induces IL-10 from antigen-primed T cells which suppresses the host-protective functions of infiltrating naïve T cells, in the later phase of infection. Host protection could be achieved by neutralizing IL-2/IL-2R and IL-10 at different time points after infection, which demonstrates their distinct roles at the priming and effector phases. This establishes a novel strategy of using kinetic modulation of an ongoing immune response as a principle of phase-specific immunotherapy for visceral leishmaniasis.