A STUDY TO IDENTIFY THE PRESENCE OF IL-10 AND IFN-γ IN LEUKOCYTES DURING 
THEILERIA PARVA
INFECTION IN CATTLE

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A thesis submitted in partial fulfilment for the degree of Master of Science
in Biochemistry of the University of Nairobi.
Declaration

I declare that the work presented in this thesis is my original work and that it has not been presented to any other institution for the purpose of examination.

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Abstract

The gene expression of two immunoregulatory cytokines, IL-10 and IFN-γ, during infection of bovine leukocytes with *T. parva* was investigated at the single cell level. Initial results obtained from *in vitro* studies with infected cells using RT-PCR showed that both IL-10 and IFN-γ messages were present. The ability of *Theileria*-infected cells to produce IL-10 transcripts was a consistent feature. However, their ability to exhibit IFN-γ messages was less consistent. On application of *in situ* hybridisation technique using bovine IL-10 and IFN-γ-specific riboprobes, it was demonstrated that most infected lymphocytes produced IL-10 and that few of these cells displayed expression of IFN-γ transcripts. Thus, judging from the relative abundance of IL-10 and lack of IFN-γ mRNA, the expression of IL-10, a cytokine known to inhibit IFN-γ production and functions, was shown to be upregulated in leukocytes responding to *T. parva* infection in cattle. These experiments did not demonstrate the production of biologically active IL-10 during the infection. However, the accompanying relative absence of IFN-γ mRNA *in situ* may suggest that biologically active IL-10 was produced and blocked the production of IFN-γ, an important effector TH 1 cytokine. The apparent upregulated expression of IL-10 during infection with *T. parva* is indicative of a regulatory role for this cytokine in the mediation of susceptibility to acute disease. This parasite-instigated induction and upregulation of IL-10 expression may represent an important strategy by which intracellular *T. parva* eludes IFN-γ-dependent cell-mediated immune destruction. Through its induction of tissue-damaging metalloproteinase, IL-10 presence also offers a biochemical explanation to the possible mechanism of propagating the lymphoproliferative, immunopathologic and tissue destructive phenomena observed in ECF.