A complex cascade of events, known as the acute phase response is immediately initiated by mammals on infection or injury. There are local and systemic components to this response, both of which cause physiological changes that range from coagulation & cell migration to fever & increased hormone secretion. While some information regarding acute phase response during bacterial & viral infections has been reported by earlier workers, knowledge related to induction of acute phase response due to parasitic infection is fragmentary. Experiments presented in this dissertation were aimed at studying the effect of experimental malaria & filarial infections on the hepatic acute phase response of the host. During the course of this investigation involving *Plasmodium berghei* (experimental malaria) or *Acanthobothriolongana vitreae* (experimental filariasis) infections of *Mastomys natalensis*, unequivocal evidence suggestive of acute phase response in the host during parasitic infections was demonstrated.

**Experimental malaria:**

Metabolic changes in the host suggestive of induction of acute phase response with increase in *P. berghei* erythrocytic parasitaemia of mastomys were demonstrated as under:

1. Increase in wet weight of liver (hepatomegaly) & concomitant loss of body weight.
2. Dramatic fall in liver glycogen & total carbohydrate content.
3. A significant increase in hepatic lipid content & fall in
cholesterol.

4. Increase in liver lipid peroxide levels, & fall in hepatic GOT, GPT & total protein content indicating hepatocyte plasma membrane damage.

5. Induction of acute phase proteins (alpha-2 macroglobulin & alpha-1 acid glycoprotein) which was clearly demonstrated at >40% parasitaemia & suppression of synthesis of albumin, known to be a negative acute phase marker.

6. The acute phase response is cytokine mediated for, splenocyte product(s) from the infected animals induced acute phase proteins.

Experimental filariasis:

Like human filarial infection, *A. vitae*

infection in mastomys manifests prepatent, patent & latent stages of infection. Active microfilaraemia is characteristic of patent stage of the infection. Like experimental malaria, experimental filariasis was also observed to evoke acute phase response in the host. Some of the important observations are:

1. Analysis of the sera from the infected animals by crossed immunoelectrophoresis revealed peak levels of the acute phase proteins (alpha-2 macroglobulin & alpha-1 acid glycoprotein) during patent stage of infection.

2. Rise in acute phase proteins was observed to coincide with fall in hepatic cytochrome P-450, a negative acute phase marker.
3. Increase in acute phase protein levels was observed to be associated with increased synthesis of these proteins by hepatocytes.

4. Filarial infection stimulated acute phase protein induction was observed to be cytokine mediated.

From the foregoing, it appears that cytokine mediated response is also generated during *P. berghei* & *A. vitaela* infections. Further studies on the physiological role of acute phase response in pathology of parasitic diseases may help in identifying link between cytokines & morbidity. Such studies will also hopefully help in identifying factors responsible for susceptibility to or resistance against parasitic infections or carrier status.