INTRODUCTION AND AIM OF THE WORK

In healthy subjects, only minute amounts of endotoxins, a lipopolysaccharide derived from Gram negative bacteria present in the gut, enter the portal blood stream but none of it is detected in the peripheral blood, presumably due to clearance by reticuloendothelial system ***(Prytz et al., 1987).***

In liver cirrhosis endotoxins have been found in the peripheral blood ***(Wilkinson et al., 1976)***, probably due to increased endotoxin release from the gut together with impaired hepatic clearance. The liver is one of the major organs damaged during endotoxaemia. Hepatic response to endotoxaemia primarily involve activation of reticuloendothelial cells followed by degenerative changes in hepatocytes ***(Hirata et al., 1980).*** Little is known about the mechanisms underlying these effects.

Overproduction of nitric oxide (NO) by cells in the liver has been implicated as an important event in endotoxaemic shock which known to alter cellular function ***(Curran et al., 1991).***