SUMMARY AND CONCLUSIONS
The present study was undertaken to investigate the effect of various pharmacological interventions, targeted against renal oxidative damage in the ischemia/reperfusion and iron-induced (glycerol-induced myoglobinuric ARF and ferric-nitrilotriacetate-induced renal damage) renal failure in rats. The endogenous protection of the kidney by ischemic preconditioning was also studied, which was further extended to remote organ and chemical preconditioning.

On the basis of the results, the salient findings of the study can be summarized as:

1. ROS play a pivotal role in renal ischemia/reperfusion injury as is evident from marked increase in lipid peroxidation, significant decline in antioxidant enzyme levels and resultant deterioration in renal function and morphology. Trimetazidine, carvedilol, quercetin, catechin and naringin ameliorated the renal oxidative stress and demonstrated a marked renoprotective activity.

2. Iron-induced oxidative stress contributes to the renal dysfunction as is evident in the glycerol-induced myoglobinuric ARF and ferric nitrilotriacetate-induced oxidative renal damage. The pharmacological modalities used in this study counterbalanced the deleterious effect of glycerol and ferric nitrilotriacetate-induced oxidative renal injury to a great extent and they may find application in the prevention and treatment of oxidant-induced renal injury.

3. Three repetitive cycles of 2 minutes renal ischemia separated by 5 minutes reperfusion period precondition the rat kidney and ameliorates the functional disturbances, altered morphology and renal oxidative stress which are observed after prolonged ischemia and reperfusion. The beneficial effects of ischemic preconditioning in the kidney are speculated to be mediated by $K_{\text{ATP}}$ channels and PKC.
4. Brief episodes of renal ischemia separated by reperfusion precondition the rat myocardium and thus the renal remote preconditioning of the myocardium is feasible in in-vivo rat model. Furthermore, a delayed phase of cardioprotection exists in the same settings.

5. Low-dose Cyclosporine A pretreatment also preconditions the rat kidneys against subsequent ischemia/reperfusion injury. This pharmacological preconditioning with Cyclosporine A may be associated with either the inhibition of mitochondrial permeability transition pore or induction of heat shock proteins.

The findings of our study strongly emphasize the role of oxidative stress in acute renal failure and the use of antioxidant supplements may be beneficial in the settings of renal diseases. The provocation of the kidney with brief episodes of ischemia and reperfusion or with a low dose of Cyclosporine A prepares the kidney to withstand a prolonged ischemic insult. The same phenomenon of renal preconditioning can be extended to the myocardium or even to other organs.
LIST OF PUBLICATIONS FROM THIS STUDY:


