Scope & Objectives
1.6. Scope and objectives

Male reproductive functions such as spermatogenesis and steroidogenesis are known to be impaired by illness, infection, and chronic inflammatory disease (Adamopoulous et al., 1978; Cutolo et al., 1988; Buch and Havlovec, 1991). Inflammation associated with infections, reproduced in vivo by the administration of bacterial LPS, was shown to inhibit testicular steroidogenesis and disrupt spermatogenesis (Wallgren et al., 1993; O’Bryan et al., 2000b). Studies in mice show that the administration of LPS causes inhibition of Leydig cell steroidogenesis via reduced synthesis of StAR protein and also other steroidogenic enzymes (Bosmann et al., 1996). Also, significant damage to the seminiferous epithelium was reported through sloughing and apoptosis of germ cells in animals treated with LPS (O’ Bryan et al., 2000b). These studies thus reveal that localized or systemic microbial infections result in impaired steroidogenesis and spermatogenesis and essentially leading to male infertility. Also, the studies indicate that LPS-induced acute inflammation closely resembles that of microbial infections in altering testicular functions. However, the precise molecular mechanisms involved in the infection/acute endotoxemia induced alterations in male reproduction are largely unknown.

Compared to the large number of in vitro studies on the effects of inflammation on male reproductive system, the in vivo effects are very limited. The mechanisms involved in inflammation-induced alterations in male reproductive functions remain poorly understood. Further there are no in vivo studies that demonstrate the role of inflammatory mediators, including
**Scope & Objectives**

interleukins, prostaglandins, nitric oxide and oxygen free radicals as well as apoptosis on the male reproductive function. The specific studies on testicular marker enzymes are lacking. It is in this connection that the present study was undertaken to analyze the effects of acute inflammation on male reproductive functions of steroidogenesis and spermatogenesis.

The **specific objectives** of the present study are:

- To standardize a rat model of acute inflammation.
- To study the effects of acute inflammation on the testicular functions:
  - Steroidogenesis
  - Spermatogenesis
- To understand the mechanisms involved in acute-inflammation induced testicular derangement by studying the relative contribution of the following factors on impaired steroidogenesis and spermatogenesis during testicular inflammation:
  - Role of inflammatory mediators
  - Role of reactive oxygen species and oxidative stress
  - Role of cell death mediators