Introduction

Stress causes activation of the hypothalamo–pituitary–adrenal (HPA) axis and this mediated through the neuropeptides corticotrophin-releasing hormone (CRH) and arginine vasopressin (AVP) which are synthesised in the parvocellular subdivision of the hypothalamic paraventricular nucleus (PVN) **(Buckingham et al. ,1997)**. The involvement of NO in the modulation of endocrine systems has been reported, particularly in the control of the hypothalamo-pituitary axis**,** and pancreatic -islets**.** NO is also involved in the regulation of steroid biosynthesis. It has been shown that NO inhibits steroidogenesis in granuloser and luteal cells. Several NO donors inhibit both basal and ACTH induced corticosterone synthesis in rat adenal zona fasiculata cells **(Cymeryng et al., 1998)**

But **Tsuchiya et al., 1997 and Weidenfeld et al., 1999** show that changes in nitric oxide levels have no effect on the basal corticosterone level, but only modulate the response to stress, in different directions: NO donors decrease stress related corticosterone and ACTH release, while administration of NO synthase blockers increases release. However, intracerebroventricular administration of NO donor leads to increase in plasma corticosterone concentration, supporting the suggestion that there are differences in the central and peripheral effects of treatments applied to the nitric oxide system **(Okada et al., 2002).**

So there is a conflicting data about the role of nitric oxide in corticosterone secretion in response to stress.

The aim of the present work

Study the role of nitric oxide in modulating the level of corticosterone in response to different types of stress as cold stress , acute immobilization stress and chronic immobilization stress. We also demonstrate the level of blood glucose and lipid profile levels in blood , since corticosterone involved in their metabolism.