# INTRODUCTION

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Neonatal sepsis is still a major and frequent cause of morbidity and mortality in neonatal period *[Nicholas and Ann, 1998].* Although the advances in neonatal intensive care have led to improved survival of very low birth weight infants, the late onset neonatal sepsis continues to be an important cause of morbidity and death among these infants, the risk of late onset infection increases with decreasing birth weight, decreasing gestational age, prolonged intravascular catheterization and with prolonged hospital stay *[Barbara et al., 1996].* Endotracheal tubes, alteration in the skin and mucus membrane barriers and frequent use of broad – spectrum antibiotics also predispose to neonatal sepsis *[Samuel, 2000].*

The human neonate may be considered an immuno-compromized host with incomplete development of multiple component of immune system *[Nicholas and Ann, 1998].* The preterm infants have lower levels of complement components and complement activity than fulterm newborns *[Laurence, 2000].* Polymorphnuclear leucocytes function and opsonic capacity are significantly impaired in newborn child. Preterm

neonates have a decreased phagocytic capacity compared with term neonates and adults *[Jan Kallman et al., 1998].* Preterm received less maternal immunoglobulin G by time of birth than fulterm infants and also susceptible to infection with gram negative organisms as they were not receive immunoglobulin M antibodies *[Laurance, 2000].*

Gram negative bacterial infection stimulate development of sepsis, shock and multiple organ failure. Lipopolysacchariedes of gram negative bacilli act as a potent activator of transcription of several inducible genes in monocytes including interleukin 1, interleukin 6 and tissue necrosis factor. The release of such cytokines may be important in mediating the various inflammatory and coagulopathic response in vivo. Cytokines also cause recureitment of leucocytes to endothelium and activate oxidative metabolism of macrophage to release reactive oxygen species *[Michael and Jeffrey, 1997].*

Antioxidants include vitamin A, E and C have been reported to modulate several functions of phagocytes such as cytokin production, tumorocidal activity, phagocytosis and procoagulant activity *[Shronts, 1993].*

***The aim of this work*** is to evaluate the role of antioxidants in the form of vitamin A, E and C in modulating preterm sepsis.