INTRODUCTION
Sleep-wakefulness is a complex behavioral phenomenon, regulated by an assemblage of factors, viz., neural, humoral and genetic (Krueger et al., 1998; Hamet and Tremblay, 2006; Szymusiak et al., 2007). Sleep has been classified into two separate states viz., Non Rapid Eye Movement (NREM) and Rapid Eye Movement (REM) sleep, each with its own distinctive electrophysiological parameters (EEG, EOG and EMG). REM sleep is characterized by desynchronized EEG, muscle atonia and rapid eye movements. It plays a significant role in the central nervous system development and sustenance as evidenced by its presence across evolution from birds to mammals and its decrease with age.

Various theories have been forwarded to explain the mechanism and function of REM sleep. Classical electrophysiological and anatomical studies have outlined the mechanism of REM sleep generation. Most of the functions of REM sleep has been elucidated by its deprivation studies. Loss of REM sleep causes a change in the normal physiological processes making the subject more irritable, aggressive and forgetful with a concomitant loss in concentration ability. The threshold to seizures and electroconvulsive shocks decrease following REM sleep loss (Kushida et al., 1989; Vogel, 1999; Gulyani et al., 2000). Thus, it was postulated that one of the functions of REM sleep is to maintain brain and neuronal excitability (Mallick et al., 1994, 1999b). Various disorders and diseases like narcolepsy, restless leg syndrome, alzheimer’s, parkinson’s, etc are manifested due to alterations in REM sleep frequency, duration and characteristic features.

Recent studies have postulated that the REM sleep deprivation-induced increase in neuronal excitability is due to the increased Na-K-ATPase activity mediated by the action of noradrenaline through the alpha1-adrenoceptors. The Na-K-ATPase is an integral membrane bound protein activated by dephosphorylation. As it is a membrane protein its activity is likely to be modulated by the organization of membrane lipids. Thus, in the present study, the relationship between REM sleep deprivation induced increased Na-K-ATPase activity and its regulation by its dephosphorylation as well as membrane lipid peroxidation with particular reference to the role of calcium has been studied. Moreover, the signaling pathway of membrane lipid peroxidation modulated by deprivation-induced elevated noradrenaline has also been investigated.
The thesis has been divided into the following parts: review of literature, lacunae and objectives, materials and methodology, results, discussion, significance of the study, conclusion and bibliography.