PREFACE
Oxygen is essential for the survival of aerobic cells but has been shown to be toxic at higher concentrations imposing a sort of oxidant stress. Oxidant stress in any form mediates its effect through the generation of free radicals. The free radicals, short lived chemical species, include mainly superoxide anion radical (\(\ddot{O}_2\)), hydroxyl radical (OH), and other reactive oxygen metabolites such as hydrogen peroxide (\(H_2O_2\)) and lipid hydroperoxides. They have been implicated as the initial toxic agents leading to the cell damage in many pathophysiological conditions. Free radicals are generated during normal cellular reactions and the cell has all the machinery needed to detoxify the free radicals generated. However, these cellular defense mechanisms are far from perfect, more so during oxidant stress where uncontrolled production of free radicals takes place. Thus all cells seem to produce free radicals which are essential for normal cellular functions, but the uncontrolled production of free radicals results in oxidant stress, or cytotoxic oxidative damage. Lipid peroxidation of polyunsaturated fatty acids (PUFAs) leads to the formation of aldehydes and alkenals which are implicated in a variety of pathophysiological states including DNA damage.

Guanidines, the catabolic products of proteins (Mikami et al., 1982) are commonly present in plants as well as in animals (Robin and Marescau, 1985).
Guanidine hydrochloride has been employed in the treatment of several neuromuscular diseases and was found to be effective in correcting electrophysiological defects in infantile and juvenile atrophy. In contrast to their beneficial role, the guanidines were found to accumulate during several pathophysiological states and act as uremic toxins. Guanidines are retained in the tissues and serum of uremics due to renal failure (Bonal et al., 1963) and hyperguanadinemia is a characteristic feature of uremics (Cohen, 1989). Earlier studies in this laboratory on rats showed that this compound interferes with energy metabolism and creatine synthesis and breakdown in tissues (Reddy et al., 1992; Raman Rao and Indira., 1993; Ramanjaneyulu et al., 1993a).

Though the metabolic effects of guanidines in relation to carbohydrate, protein and lipid metabolism have been studied in detail, their effects on antioxidant enzymes still remain unclear. In the present study an attempt has been made to study the effect of guanidine hydrochloride (which will induce uremic symptoms) on the activity levels of selected detoxifying enzymes. GuHCl induced alterations in the detoxification mechanisms which combat oxidative stress have been studied by taking albino rat as an experimental model.
However, the author is aware of the fact that more extensive studies are required for a thorough understanding of the antioxidant defense mechanism during guanidine stress. Limitations in the availability of chemicals and equipment have precluded the author from bridging some of the lacunae that emerged during the study. However, the present findings may throw some light on guanidine-induced free radical toxicity and the role of antioxidant defense mechanisms in countering toxic stress.