INTRODUCTION

The cardiovascular system can develop adaptive supersensitivity *in vivo* to catecholamines after repeated stress (Barney *et al.* 1980, Esler & Ammon 1993, Fregly *et al.* 1977). However, the *in vitro* evidences for this phenomenon under various conditions are also reported previously (Chess-Williams 1993, Spadari *et al.* 1988). Many reports have been published concerning an alteration in the number of β-adrenoceptors in the central nervous system, for example, an increase in the density of β-adrenergic receptors, as measured by the binding of the selective antagonist [^{3}H]dihydroalprenolol ([^{3}H]DHA), in the cerebral cortex of chronic morphine treated rats has been reported (Hamburg & Tallman 1981, Llorens *et al.* 1978, Moises & Smith 1985, 1987). It has also been reported that the number of cortical β-adrenergic receptors, mainly β_1-type adrenergic receptors, are increased after the precipitation of withdrawal in morphine-dependent rats (Kuriyama *et al.* 1981). Furthermore, β-adrenergic receptors in hippocampus are up-regulated during the development of morphine dependence (19% increase) and down-regulated during morphine withdrawal (27% decrease) (Moises & Smith 1989). These changes in hippocampal β-adrenergic receptor density are likely to be of functional relevance since they are manifested in a corresponding increase and decrease, respectively, in electrophysiological responsiveness to an exogenously administered β-adrenergic receptor agonist. Since the previous results were only reported an alteration of the number of β-adrenergic receptors in some areas of rat brain (central nervous system) after chronic morphine treatment and morphine withdrawal, therefore, the main purposes of this study were extended to determine whether the alterations in β-adrenergic receptor responsiveness are also elicited in the

peripheral nervous system using isolated rat atrial preparations and anesthetized rats of morphine dependence and morphine withdrawal in comparison with saline-treated rats.