ABSTRACT
The initial functional indication of the enhancement of SERCA pumping ability in ER stressed cells is presented by these findings. As three distinct and unrelated mechanisms elicited the functional upregulation of Ca2+ transport into the ED, these results provide evidence that the regulation of at least one SERP pump isoform is involved in the induction of UPR.

INTRODUCTION

The study of the effects of stress on the expression of genes is an important topic in cell biology, and the aim of this study was to investigate whether the upregulation of the Ca2+ pump activity of the endoplasmic reticulum (ER) is regulated by chronic stress.

Materials and Methods:

The study was carried out in male Wistar rats (Charles River Laboratories, USA). The rats were treated with a single dose of the selective serotonin reuptake inhibitor (SSRI) fluoxetine (0,250 mg/kg) or saline (0.5 mg/kg) for 30 days and then subjected to a series of stress tests: (1) stress test 1, (2) stress test 2, A significant portion of the ER is responsible for both protein synthesis and homeostasis in cells, while other compartments like the secretory pathway are located within the body. The resulting post-translational processes include folding, glycosylation (with the aid of chaperone proteins) and transfer to the Golgi compartment. ER-specific stress responses, including Endoplasmic Reticulum Overload Response (EOR), are activated by specific conditions such as glucose deprivation or glycosylation inhibition. The folding and maturation processes within the ER are dependent on the presence of Ca2+ ions in the compartment, which is essential for the survival of many cells. However, the upregulation of SERCA pumping capacity can also be mediated by reducing the amount of calcium in culture. Our findings provide preliminary evidence that depletion of ca2 + during cell culture leads to an increase in SERP activity.

CONCLUSION

Remarkable conclusions A new mechanism for signaling SERCA activity can be proposed, as it shows that the UPR pathway is likely to account for the increase in SERP activity resulting from a change in the internal ER environment caused by various agents. We have reported a recent study that revealed SERCAb immunoreactive protein and mRNA increased by 3- or 4-folds after treating PC12 cells with EGTA, tunicamycin, DTT, or brefeldin A; however, this work did not provide any functional evidence of the stress-induced increase in SERC2b activity due to technical difficulties, and it was necessary to demonstrate that ER stress may be responsible for impaired function.