Thyrotropin (TSH) is the main regulator of the thyroid hormones (THs) synthesis/secretion, which in turn exert a negative feedback mechanism on the TSHβ mRNA expression in the pituitary gland by reducing their transcription rate. Some of triiodothyronine (T3) effects are also shown in short period of time, characterizing a non genomic actions of THs. It has been shown previously that T3 acts on posttranscriptional steps of TSH synthesis and reduces its secretion when acutely administered to hypothryroid rats. The present study aimed to: 1) characterize the pathways involved in the rapid inhibition of the TSH secretion induced by T3 in primary cultures of anterior pituitary cells and; 2) evaluate the participation of T3 on calcium and magnesium intracellular mobilization in slices of pituitary of hypothryroid rats.

RESULTS AND CONCLUSION

The results showed a rapid increase of TSHβ content in intracellular extracts while the amount of TSH in extracellular media was reduced after T3 challenge. The treatment with RGD and wortmannin abolished T3 effects. No alteration on TSH secretion induced by T3 was observed in the presence of EGTA in the culture media neither in calcium intracellular mobilization, while intracellular concentration of magnesium was increased after T3 treatment.