Thyroid disorders are associated with impaired fertility in women and these abnormalities are improved by restoring euthyroid homeostasis. The exact mechanisms are not well known; however, it is conceivable that thyroid hormones might act on ovarian physiology via receptors in granulosa cells. We evaluated the effect of thyroid hormone T3 treatment on proliferation, apoptosis and survival of non tumoral rat granulosa cells (rGROV) and freshly isolated rat follicles. Cells and follicles were treated with T3 (100nM). Cell growth and viability were evaluated by cell counting and MTT assay, whether follicles growth was evaluated by volume measurement. T3 induced a 40% increase in the cell number and in cell metabolism in rGROV (48h) and a 40% increase in follicle volumes (after 7 days). Also, cytofluorimetry revealed T3 ability to induce cell cycling in rGROV, and cell cycle molecules were regulated, as shown by Western Blot analysis. As demonstrated by Tunel assay, in serum-free condition, T3 decreased the cell apoptotic rate by 40%; according with this observation the proapoptotic molecules Casp 3 and Bax were downregulated. In these animal models, PI3k was investigated and an increase in pAkt levels was observed, suggesting that this pathway may be involved in the survival effect of T3. These findings strongly suggest that THs influence cell survival of ovarian granulosa cells.