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INTRODUCTION & AIM

Glioblastoma multiforme (GBM) is the most prevalent brain tumor with high mortality rates and poor prognosis (1). Various intracellular pathways including the mitogen-activated protein kinase (MAPK) and phosphatidylinositol 3-kinase (PI3K)/AKT/mechanistic target of rapamycin (mTOR) pathways are frequently deregulated in GBM (2, 3). Melatonin (secreted by the pineal gland) has been shown to have anti-cancer effects (4). The aim of this study was to investigate the effects of melatonin and mTOR inhibitor rapamycin on the regulation of MAPK and AKT/mTOR pathways *in vitro*.

METHODS

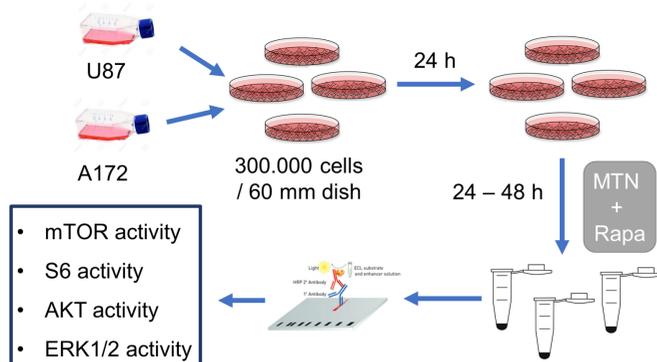


Figure 1. Experimental setup and flow diagram of the experiments.

Statistical analysis: Two-way analysis of variance followed by Tukey's multiple comparison test and Boferroni's multiple comparison test was used to compare the statistical differences between treatments within cell lines and between cell lines, respectively.

RESULTS

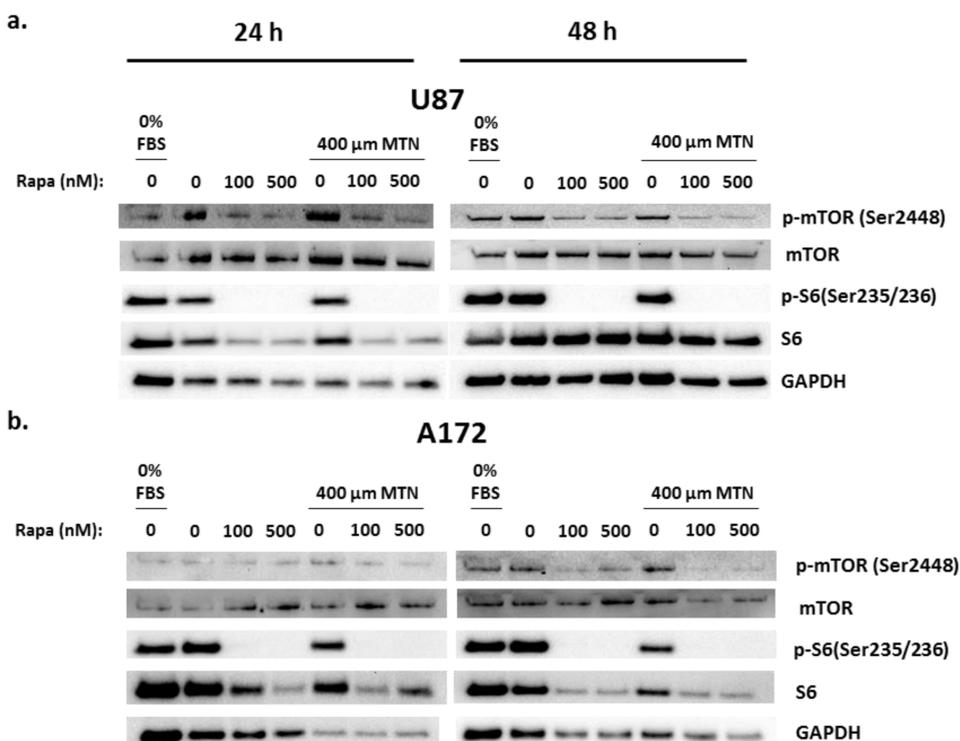


Figure 2. Representative western blot image of mTOR pathway activity in (a) U87 and (b) A172 cell lines at 24 and 48 h.

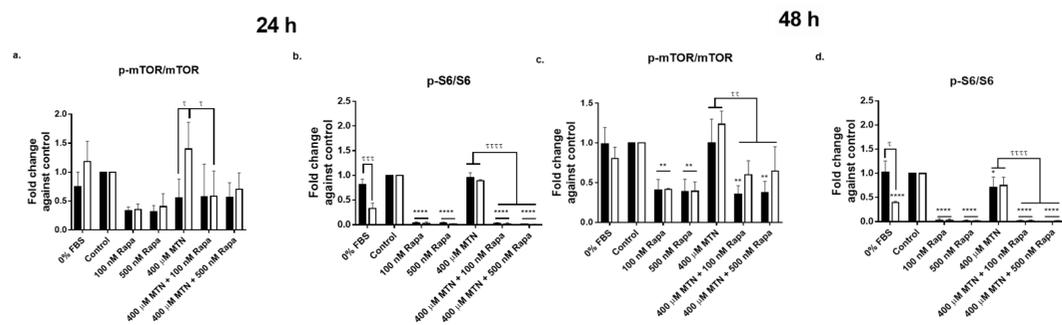


Figure 3. Relative band intensity of p-mTOR/mTOR and p-S6/S6 fold change against respective control groups in U87 and A172 cells at (a) 24 and (b) 48 h. Black bars belong to U87 cell line, while white bars belong to A172 cell line. * $p < 0.05$, ** $p < 0.01$ and **** $p < 0.0001$ against respective control groups and $T_p < 0.05$, $TT_p < 0.01$ and $TTTT_p < 0.0001$.

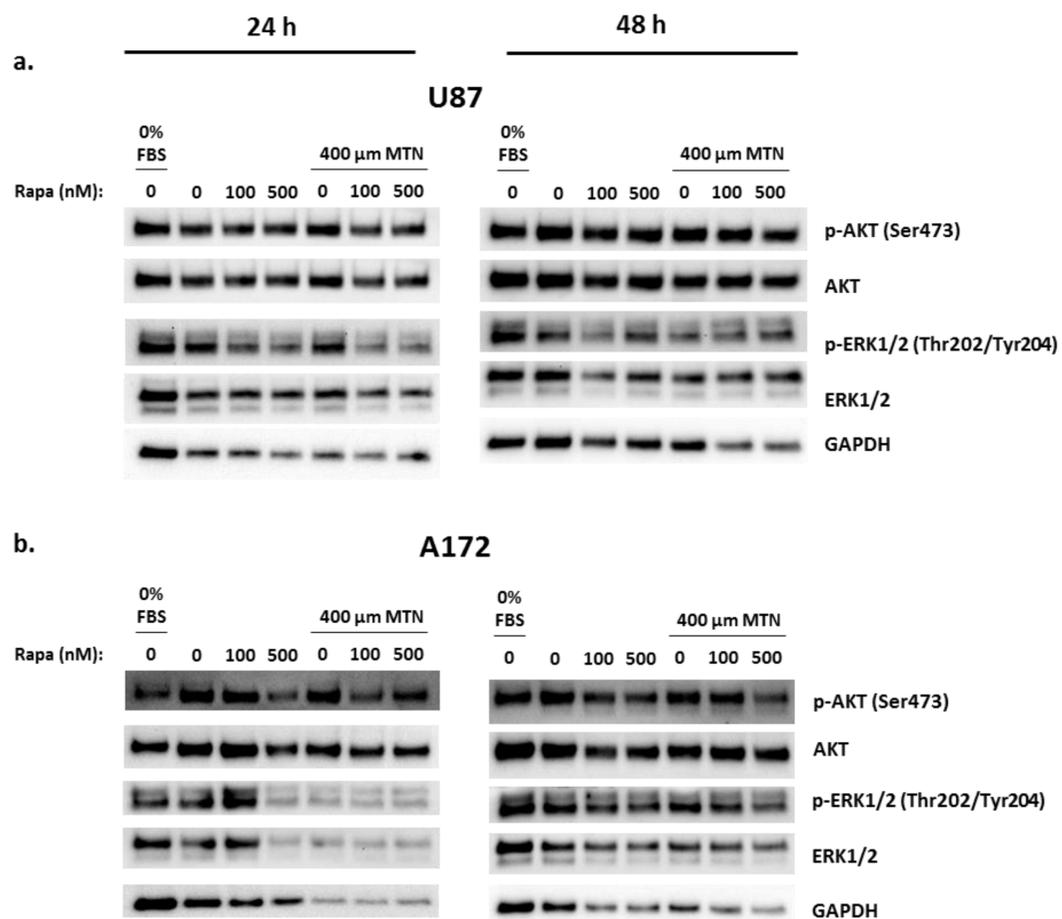


Figure 4. Representative western blot image of AKT and ERK1/2 (P44/42) activity in (a) U87 and (b) A172 cell lines at 24 and 48 h.

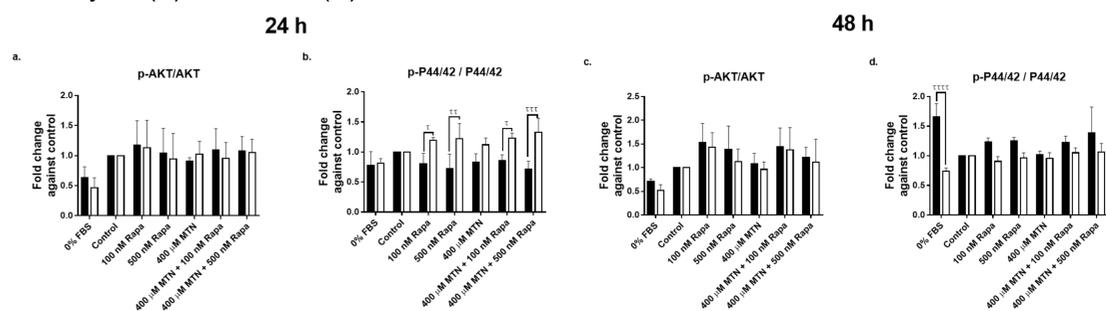


Figure 5. Relative band intensity of p-AKT/AKT and p-P44/42/P44/42 (p-ERK1/2 / ERK1/2) fold change against respective control groups in U87 and A172 cells at (a) 24 and (b) 48 h. Black bars belong to U87 cell line, while white bars belong to A172 cell line. $T_p < 0.05$, $TT_p < 0.01$, $TTTT_p < 0.0001$.

CONCLUSION

Our results suggest that AKT/mTOR and MAPK pathways may be differentially regulated by melatonin and serum deprivation in GBM cells.