**Study of the ARHGAP21 protein in autophagy induced by glucose deprivation in prostate cancer cells**

The ARHGAP21 is a RhoGAP protein with important functions in tumorigenesis, such as adhesion, migration and cellular proliferation. We observed that ARHGAP21 silencing alters the expression of genes involved in the glycolytic pathway and autophagy. We evaluated the autophagy induced by glucose deprivation in prostate adenocarcinoma cells (PC3 and LNCaP) with inhibited ARHGAP21 expression. The cells were transfected with a siRNA for ARHGAP21 inhibition and cultured in medium with varying glucose concentrations (2000, 250, and 0 mg/L) for 72 hours. The autophagy was evaluated using acridine orange by flow cytometry. Apoptosis was also evaluated using anexin V by flow cytometry and mitochondrial activity was evaluated with a spectrophotometer. We observed that glucose-deprivation stress induced autophagy, apoptosis and mitochondrial damage in both cell lines. Interestingly, LNCaP cells presented more autophagy and less apoptosis than the ones in the control, evidencing the anti-autophagic and pro-apoptotic role of the ARHGAP21 in this type of cell. We also studied the expression of autophagy-related genes and proteins, such as p62, BECLIN, and LC3 by quantitative PCR and Western Blot. Both LC3I and LC3II isoforms were considered, owning to the importance of the LC3I conversion into LC3II during autophagy. We observed that, in LNCaP cells, the glucose deprivation increased the gene expression of p62, BECLIN, and LC3, all of which are involved in the autophagic process. In LNCaP cells, we also observed an increase in the protein expression of the LC3II and p62 when those cells had the ARHGAP21 protein inhibited. Similar modulation in the autophagic process was not observed in PC3 cells.