-Text should be written in English (make sure it does not contain spelling or grammar mistakes);  
-Limit text to 250 words;  
-Abstract should contain background, aims, methods, results, conclusion and information on ethical approval and funding support;  
-Use arial font 10. The system does not accept tables;  
-Title, authors names and affiliations, contact details and disclosures are not included in word counting. This information should be sent only in the attached file (doc or docx) which should also contain de abstract (attention to format: font: arial 10, line spacing: 1.5; justified paragraph)

The ARHGAP21 protein is a RhoGAP with important tumorigenesis functions, such as formation, migration and cellular proliferation. We observed that by inhibiting the ARHGAP21 expression, the glycolytic pathway and the genes involved in autophagy are altered. Autophagy is a catabolic for degrading and recycling macromolecules, damaged organelles and other cellular components. Because of the strategic interplay between cellular death and survival, defective autophagy is related to many different cancer types. We studied the autophagy in prostate adenocarcinoma cells (PC3 and LNCaP) with and without inhibited ARHGAP21 expression. We induced autophagy by glucose deprivation. The cells were grown in cultures with varying glucose concentrations and transfected with a specific interfering RNA (siRNA) for ARHGAP21 inhibition. The autophagy was evaluated using flow cytometry. Apoptosis was also evaluated with flow cytometry, mitochondrial activity with an oxygraph apparatus, and autophagy-related protein expression with real-time PCR and Western Blotting. In both ARHGAP21 inhibited cells and in the control sample, the glucose deprivation stress induced autophagy, apoptosis and mitochondrial damage in the PC3 and LNCap cells. We observed more autophagy and less apoptosis in the ARHGAP21 inhibited LNCaP cells when compared to the control sample, evidencing the anti-autophagic and pro-apoptotic role of the ARHGAP21 in this type of cell. Similar modulation in the autophagic expression was not observed in PC3 cells.