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RhoGDI3 and the novel role in carcinogenesis: Pancreatic Ductal Adenocarcinoma (PDAC)**Rocío Thompson Bonilla¹, Mercedes Piedad de León-Bautista^{1,2,3}, Daniel Marrero^{3,4}, Mauricio Salcedo⁴, Lorena Gorgonio-Eusebio³, Emma Vélez-Uriza³ and Miguel Vargas³**¹ISSSTE, Mexico²ACentral AND, Mexico³CINVESTAV-IPN, Mexico⁴XXI Century National Medical Center, Mexico

RhoGDI proteins have been described as small GTPases negative regulators, sequestering GTPases in cytosol avoiding their activation; nevertheless, there is evidence about their implication in cancer, particularly RhoGDI1 and RhoGDI2 but not in RhoGDI3. In our group, we have reported an imbalance among the RhoGDI3 protein and three different staging cells, from non-cancerous to highly aggressive cancerous pancreatic cells; normal and low expression levels respectively. To elucidate the possible functions of RhoGDI3 in this pathology process, we performed reconstitution assays in the cancerous pancreatic cell line (PANC-1), which let us know that, normal levels of RhoGDI3 protein produces a smaller tumor compared with the cells not reconstituted and that the different levels of RhoGDI3 regulated gene expression, such as TRIO, EPHA2, RHEB, KLF10, EGFR, all of them implicated in cancerous process and tumor maintenance. There are very few proofs about the RhoGDI3 and the correlation with cancer, specifically PDAC, and our findings open up a gap to expand knowledge, from the RhoGDI3 as a negative regulator, the classical function, to a RhoGDI3 protein with novel role, decreasing the malignant behavior in PDAC.

Biography

Rocío Thompson Bonilla has completed her Bachelor's Degree at UNAM and her MSc and PhD from ENCB-IPN in the Dept. of Immunology and PhD in Rutgers University in 2010. She served as Director of Research in ISSSTE, the second public health institution in Mexico. She won the national price of Biomedicine in 2015 and has been working with infection diseases and with developing molecular platforms to find new markers in cancer and hereditary diseases.

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