**CV**

Dr. Rodriguez-Blanco obtained her PhD at University of Oviedo (Asturias, Spain) on 2009 in Neurosciences. The research group of her thesis mentors Dr. Rodriguez and Dr. Antolin was focused on the relationship between redox status, neurodegeneration and cancer. During her graduate studies she was conferred several competitive fellowships, and published 18 papers in per review journals, including 2 first author ones. Soon after her thesis dissertation, she joined the laboratory of Dr. Greene at Columbia University (New York, USA) to conclude her Parkinson's disease project during this time. Currently, she is a postdoctoral fellow at University of Miami working in Hedgehog pathway and brain tumors. She has been working in Dr. Robbins laboratory at Surgery department since 2010. Her work in Hedgehog and cancer has been awarded with a European Postdoctoral Fellowship (FICYT) and the Childhood Brain Tumor Foundation (CBTF) grant. She focuses her research in finding alternative chemotherapy able to prevent tumor reparse in infants caring Hedgehog driven tumors.

**Abstract**

Medulloblastoma (MB) is the most common malignant pediatric brain cancer. While the bulk of patients respond to multimodal therapy (surgery, radiation and cytotoxic chemotherapy), significant treatment induced morbidity, and relapse (which almost always results in death) remain significant barriers in the clinical management of these patients. Thus, significant effort has gone into identifying novel targeted therapies for this cancer that both shrink the primary tumor and prevent relapse. One of these compounds, vismodegib, which is FDA-approved for basal cell carcinoma, has shown promise in various clinical trials for MB patients. Unfortunately, clinical relapses and cross-resistance to Smoothened (SMO) inhibitors have illustrated the necessity to develop new therapeutic strategies. Our laboratory has found a population of MB stem cells refractory to vismodegib, an observation likely to lead to tumor recurrence in patients treated with this drug. In our effort to find alternative targets to SMO for MB cure, we have found that pyrvinium, a previously FDA-approved anthelmintic agent, is a nanomolar agonist of CK1α, kinase involved in HH pathway regulation. Pyrvinium negatively regulates Hedgehog signaling by affecting GLI1 stability downstream to SMO, bypassing the mechanisms of resistance associate to SMO inhibition. Encouraging, we have seen that pyrvinium is able to reduce tumor growth in murine models of MB, successfully targeting CSC reservoir.