BIOGRAPHICAL SKETCH

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NAME: **Rodriguez, Georgialina**

eRA COMMONS USER NAME (credential, e.g., agency login): **GEORGIALINA**

POSITION TITLE: **Research Assistant Professor**

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
University of Texas at El Paso	**B.S.**	**2003**	**Biological Sciences**
University of Texas Health Science Center-Houston & MD Anderson Cancer Center	**Ph.D.**	**2009**	**Cell Signaling & Immunology**
University of Texas at El Paso	**Postdoctoral**	**2013**	**Immuno-Oncology & Drug Development**

A. Personal Statement

For the past 15 years, I have dedicated myself to investigating the regulation of Jak/Stat cell signaling proteins involved in human cancer. My early work focused on negative regulatory cross-talk between GPCR receptors and cytokine pathways in human T-lymphocytes. Using molecular biology and proteomics techniques we described how elevated levels of cAMP can negatively affect Jak3 auto-activation, kinase activity, downstream signaling and receptor association. Through this work, I gained valuable experience in investigating the molecular mechanisms that mediate immune homeostasis as well as those which drive kinase mediated diseases.

My on-going research at The University of Texas at El Paso, where I am a Research Assistant Professor, aims to further understand the drivers of hematological cancers which disproportionately affect Hispanic children in the region. It is my hope that the knowledge gained from such work can be used to develop new therapeutic drugs to effectively treat relapse and drug resistant leukemia. Toward this goal, my work investigates Jak tyrosine kinase mutations, including those found within Jak3, which are known drivers of leukemia. Using whole exome sequencing coupled to bioinformatics pipeline sorting our team has identified previously unreported SNPs within the human kinome including Jak proteins from a small cohort of Hispanic acute lymphoblastic leukemia samples. Moreover, I continue to study both positive and negative regulation of Jak3. This work has resulted in the generation of a unique set of tools and reagents including novel phospho-antibodies which are not commercially available against newly identified phospho-tyrosine and phospho-serine residues. These unique tools have been confirmed for their ability to recognize Jak3 and will be used to address the questions proposed within the current application. Lastly, through my most recent collaborative work with Dr. Chaun Xiao (UTEP, Biochemist) a full-length model of Jak3 depicting how the various functional domains fold and connect in a 3D structure was established. Using this model and the information gathered from the proposed work we hope to generate small molecule inhibitors which more selectively target Jak3 in contrast to the current pan-Jak inhibitors available .

As a Co-Investigator on the current application, my firm commitment to a life-long career in cancer research, knowledge gained over 15 years of experience and the solid partnerships cultivated with research collaborators will aid in the success of the proposed work.

B. Positions and Honors

Positions and Employment

2009-2013	Post-doctoral Fellow, Department of Biological Sciences, The University of Texas at El Paso, El Paso, Texas
2014-Present	Research Assistant Professor, Department of Biological Sciences, The University of Texas at El Paso, El Paso, Texas

Mentoring Experience

Spring 2007	UTEP Biology 4398, Special Problems, Jessica Hernandez
Spring 2009	UTEP Biology 4398, Special Problems, Juan Becerra
Spring 2009	UTEP Biology 4398, Special Problems, Hadit Morales
Summer 2009	UTEP Biology 4398, Special Problems, John L. Wilson
Summer 2009	UTEP Biology 4398, Special Problems, Jason C. Meyer
Fall 2010	UTEP Biology 4398, Special Problems, Adriana H. Saldivar
Spring 2011-13	UTEP Biology Undergraduate Student Volunteer, Tanya S. Maestas
Spring 2010-15	UTEP Biology Undergraduate Student Volunteer, Alejandra Saenz
Fall 2012-15	UTEP Biology Rise Undergraduate Student, Hanna Pena
Spring 2013	UTEP Biology 4398, Special Problems, Amanda Gonzalez
Summer 2013-2015	UTEP Undergraduate Student Volunteer, Sun Ah Yung-Reid
Summer 2013	UTEP COURI Summer Student, Victoria Riveria-Peterson
Spring 2014	UTEP Undergraduate Student Volunteer, Amy Arrieta
Spring 2014-17	UTEP Undergraduate Student Volunteer, Diana Padilla
Fall 2014-17	UTEP Undergraduate Student Volunteer, Roberto L. Garcia
Spring 2015	UTEP Undergraduate Student Volunteer, Lillian Ellis
Summer 2015-2017	UTEP Undergraduate Student Volunteer, Daniel A. Armendariz
Fall 2015-17	UTEP Marc Undergraduate Student, Rosabril Acuna
Fall 2017-Present	UTEP Biological Sciences Master Student, Roabril Acuna
Fall 2017-Present	UTEP BUILDing Scholars Undergraduate Student, Daniela F. Diaz
Summer 2017	UTEP BUILDing Scholars Undergraduate Student, Juan Hernandez
Summer 2018	UTEP BUILDing Scholars Undergraduate Student, Juan Hernandez
Fall 2018-Present	UTEP Undergraduate Volunteer, Kristen A. Ahumada
Fall 2018-Present	UTEP BUILDing Scholars Undergradute Student, Luisa F. Castillo
Fall 2018-Present	UTEP BUILDing Scholars Undergraduate Student, Johnathan Muniz-Becerra
Fall 2018-Present	UTEP ACSScellence Undergraduate Student, Karin Carmona

Honors

1999-2003	UTEP College of Science Deans List (GPA > 3.5)
1999-2003	UTEP Presidential Scholarship Recipient
2002	Golden Key International Honor Society Award Recipient
2003	UTEP *Magna Cum Luade* Recipient
2010	RCMI 2010 Travel Award Grant Recipient

C. Contributions to Science

1. *Mapped novel regulatory sites within Jak3 and Stat5*. My work continues to examine the cross-talk via G-protein coupled receptors, regulation by serine and threonine kinases, and phosphatases that include gamma cytokine receptor regulation.
    a. **Rodriguez, G**., Ross, J.A., Nagy, Z.S., and Kirken, R.A. Forskolin inducible cAMP pathway negatively regulates T-cell proliferation by uncoupling the Interleukin-2 receptor complex. J. Biol. Chem, 288: 7137-46, 2013.
    b. Mitra, A., Ross, J.A., **Rodriguez, G**., Nagy, Z.S., Wilson, H.L. and Kirken, R.A. Signal transducer and activator of transcription 5b (Stat5b) serine 193 is a novel cytokine induced phospho-regulatory

site that is constitutively activated in primary hematologic malignancies. J. Biol. Chem, 287: 16596-608, 2012.

c. Ross, JA., **Rodriguez G**, and Kirken RA. Analysis of Janus tyrosine kinase phosphorylation and activation. Methods Mol Biol. 2013; 967:3-20.

d. Rodriguez, G. Armendariz, DA., Pena, H. and Kirken, R.A. Identification and Characterization of Novel Jak3 Serine 448 and Serine 449 SH2 Domain Residues. Manuscript in Preparation.

2. *Jak3 and Stat5a/b proteins are key regulators of T-cell survival*. The lab continues to investigate downstream targets of Jak3/Stat5 which may represent targets for pharmaceutical intervention of lymphoid diseases.
    a. Nagy, ZS., Ross, JA., **Rodriguez, G.**, Balint, BL., Szeles, L., Nagy, L. and Kirken, RA. Genome wide mapping reveals PDE4B as an IL-2 induced STAT5 target gene in activated human PBMCs and lymphoid cancer cells. PLoS One. 2013;8(2)
    b. Nagy, ZS., Ross, JA., **Rodriguez, G.**, Bader, J., Dimmock, J. and Kirken, RA. Uncoupling JAK3 activation induces apoptosis in human lymphoid cancer cells via regulating critical survival pathways. FEBS Lett. 2010 Apr 16;584(8):1515-20.
3. *Development of Novel Therapeutics for the Treatment of Cancer*. The lab continues to identify small molecules and other agents that can disrupt oncogenic pathways. Our lab provided the first evidence that inhibition of imatinib-resistant T315I BCR-ABL driven Chronic Myeloid Leukemia by combinational therapy of Ponatinib and Forksolin effectively reduces viability of drug resistant CML cells.
    a. Oaxaca, DM. Yang-Reid, S.A. Ross, J.A., **Rodriguez, G.**, Staniswalis, J.G., Kirken R.A. Sensitivity of imatinib-resistant T315I BCR-ABL CML to a synergistic combination of ponatinib and forskolin treatment. Tumour Biol. 2016 Sep;37(9):12643-12654.
4. Metronomic chemotherapy has been successfully used to for the treatment of solid tumors. Separately, much has been reported on the successful use of targeting co-inhibitory receptors to activate the immune system against melanoma. Our team of collaborators, spanning two continents, seek to combine the two methods for treatment of solid tumors, including breast cancer.
    a. Parra, K., Valenzuela, P., Lerma, N., Gallegos, A., Reza, L.C., **Rodriguez, G.**, Emmenegger, U., Di Desidero, T., Bocci, G., Felder, M.S., Manciu, M., Kirken, R.A., Francia, G. Impact of CTLA-4 blockade in conjunction with metronomic chemotherapy on preclinical breast cancer growth. Br J Cancer. 2017 Jan;116(3):324-334.

D. Additional Information: Research Support and/or Scholastic Performance

<u>Completed Research Support</u>

Marsh Foundation (Co-Investigator) 05/01/14-10/31/18
Therapeutic Strategies to Treat High-Risk Relapse Childhood Cancers
Description: This application seeks to identify new molecular targets for therapeutic intervention of childhood cancer.

BBRC Pilot Program (Principal Investigator) 04/01/17-03/31/18
Generation of Novel Jak3 Therapeutics through Homologous Protein Modeling
Description: This application sought to employee protein modeling techniques to determine the structure of Jak3 protein.

Coldwell Foundation (Co-Investigator) 07/01/14-3/31/17
Validation of New Targeted Therapies for the Treatment of High-Risk Malignancies
Description: Investigates Jak3 as a molecular target for treating certain leukemias derived from minority populations.