
BIOGRAPHICAL SKETCH

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NAME Joe William Ramos		POSITION TITLE Assistant Professor	
eRA COMMONS USER NAME joewramos			
EDUCATION/TRAINING <i>(Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)</i>			
INSTITUTION AND LOCATION	DEGREE <i>(if applicable)</i>	YEAR(s)	FIELD OF STUDY
University of St. Andrews, Scotland	NA	1988	Chemistry
University of Virginia, Charlottesville, VA	B.A.	1989	Biology
University of Virginia, Charlottesville, VA	Ph.D.	1996	Cell Biology
The Scripps Research Institute, La Jolla, CA	Postdoc	1998	Cell Signaling

A. Positions and Honors

Positions and Employment:

- 1996-1998: Research Associate (Laboratory of Mark H. Ginsberg, MD), Department of Vascular Biology, The Scripps Research Institute, La Jolla, California.
- 1999-2000: Senior Research Associate, Department of Vascular Biology, The Scripps Research Institute, La Jolla, California.
- 2000-2004: Assistant Professor, Department of Cell Biology and Neuroscience, Rutgers The State University of New Jersey, Piscataway, New Jersey.
- 2004-present: Assistant Professor, Natural Products and Cancer Biology, Cancer Research Center of Hawaii, University of Hawaii at Manoa, Honolulu, HI.

Awards and Other Professional Activities:

- 1997: National Research Service Award, awarded by the National Institutes of Health, National Cancer Institute. ID# 1 F32 CA74529-01
- 1998-2000: Leukemia Society of America Fellow Award. ID# 5561-98
- 1999: Leukemia Society of America Short-term Travel Award.

B. Selected peer-reviewed research publications (in chronological order):

1. K. Danker, H. Hacke, **J.W. Ramos**, D.W. DeSimone, and D. Wedlich. (1993). V+ Fibronectin expression and localization prior to gastrulation in *Xenopus laevis* embryos. *Mech. of Development* 44:155-165.
2. D. Alfandari, **J.W. Ramos**, L. Clavilier, D.W. DeSimone, and T. Darribere. (1996). The RGD-dependent and Hep II binding domains of fibronectin govern the adhesive behavior of amphibian embryonic cells. *Mech. of Development*. 56:83-92.
3. **J.W. Ramos** and D.W. DeSimone. (1996). *Xenopus* embryonic cell adhesion to fibronectin: position-specific activation of RGD/synergy-site-dependent migratory behavior at gastrulation. *J. Cell Biology* 134:227-240.
4. **J.W. Ramos**, C.A. Whittaker, and D.W. DeSimone. (1996). Integrin-dependent adhesive activity is spatially controlled by inductive signals at gastrulation. *Development* 122:2873-2883.
5. C.A. Fenczik, T. Sethi, **J.W. Ramos**, P.E. Hughes, and M.H. Ginsberg. (1997). Complementation of dominant suppression implicates CD98 in integrin activation. *Nature* 390:81-85.

6. **J.W. Ramos**, T.J. Kojima, P.E. Hughes, C.A. Fenczik, and M.H. Ginsberg. (1998). The Death Effector Domain of PEA-15 is involved in its regulation of integrin activation. *J. Biol. Chem.* 273(51):33897-33900.
7. **J.W. Ramos**, P.E. Hughes, M.W. Renshaw, H. Chneiweiss, and M.H. Ginsberg. (2000) The Death Effector Domain Protein, PEA-15, potentiates Ras activation of ERK by an adhesion independent mechanism. *Mol.Biol.Cell.* 11:2863-72.
8. Y. Yang, O. Redina, Y.M. Altshuller, M. Yamazaki, **J.W. Ramos**, Y. Kanaho, and M.A. Frohman. (2000) Regulation of PLD1 and PLD2 expression levels by PEA-15, a novel PLD-interacting protein. *J.Biol.Chem.* 275:35224-35232.
9. **J.W. Ramos***, E. Formstecher*, M. Fauquet, D.A. Calderwood, J. Camonis, X-T Nguyen, J-V. Barnier, B. Canton, M.H. Ginsberg, and H. Chneiweiss. (2001) PEA-15 mediates cytoplasmic sequestration of ERK MAP kinase. *Developmental Cell* 1(2); 239-250. *communicating author and co-first authors (reversed from publication order).
10. J.M. Hill, H. Vaidyanathan, **J.W. Ramos**, M.H. Ginsberg and M.H. Werner. (2002) Recognition of ERK MAP kinase by PEA-15 reveals a common docking site within the death domain and death effector domain. *EMBO J* 21(23):6494-6504.
11. H. Vaidyanathan and **J.W. Ramos**. (2003). Rsk2 activity is regulated by its interaction with PEA-15. *J.Biol. Chem.* 278(34): 32367-32372.
12. F-L Chou, J.M. Hill, J-C. Hsieh, J. Pouyssegur, A. Brunet, A. Glading, **J.W. Ramos**, M.H. Werner, and M.H. Ginsberg. (2003). PEA-15 Binding to ERK1/2 MAP Kinases is required for its Modulation of Integrin Activation. *J. Biol. Chem.* 278(52): 52587-52597.
13. R. Sur and **J.W. Ramos**. (2005). Vanishin is a novel ubiquitinated death-effector domain protein that blocks ERK activation. *Biochem. J.* 387(2):315-324.
14. H. Renganathan, H. Vaidyanathan, A. Knapinska and **J.W. Ramos**. (2005). Phosphorylation of PEA-15 Switches its Binding Specificity from ERK MAP kinase to FADD. *Biochem J.*, 390(3):729-735.
15. M.L. Matter and **J.W. Ramos**. (2006). Expression cloning of signaling proteins regulated by cell adhesion. *Cell-Cell Interactions: Methods and Protocols*. Sean P. Colgan ed., Humana Press. 341:153-164.
16. F. Renault-Mihara, F. Beuvon, X. Iturrioz, B. Canton, S. De Bouard, N. Léonard, S. Mouhamad, A. Sharif, **J.W. Ramos**, M-P. Junier, and H Chneiweiss. (2006). PEA-15 Expression Inhibits Astrocyte Migration by a PKC Delta-Dependent Mechanism. *Mol. Biol. Cell*, 17(12):5141-52.
17. H. Vaidyanathan, J. Opoku-Ansah, S. Pastorino, H. Renganathan, M.L. Matter and **J.W. Ramos**. (2007). ERK MAP kinase is targeted to RSK2 by the phosphoprotein PEA-15. *PNAS*, 105(50):19837-19842.
18. **J.W. Ramos**. The regulation of extracellular signal-regulated kinase (ERK) in mammalian Cells. (2008). *The International Journal of Biochemistry & Cell Biology*. 40(12): 2707-2719.

C. RESEARCH SUPPORT

Ongoing Research Support:

Grant # 05245002 (PI: C-W Vogel)

2006-2009

“Collaborative Cancer Research Program between Tripler Army Medical Center and the CRCH”

Role: Co-PI

Agency: Department of Defense

The Major objectives of Dr. Ramos' portion of this proposal are to 1) Perform Immunoprecipitations and pulldown experiments to determine if H-Ras or R-Ras alter cytoskeletal interaction with the integrins; 2) To measure diapedesis of cells expressing mutant integrins that preferentially bind talin or filamin. And 3) To screen a natural product library for efficacy in inhibiting inside-out integrin signaling.

Completed Research Support:

Grant# R01 CA93849 (PI: JW Ramos)

2002-2008

"Regulation of Cell Signaling and Adhesion"

Role: PI

Agency: National Cancer Institute of the National Institutes of Health.

The major objective of this proposal is to determine the functional significance of PEA-15 expression in the mouse. The specific aims are 1) determine the residues of PEA-15 that are necessary for ERK binding; 2) determine the molecular mechanism by which PEA-15 affects ERK signaling; 3) analyze the expression of PEA-15 mRNA and protein; and 4) determine the functional significance of PEA-15 using PEA-15 null mice. (Grant was extended by R56 Bridge funds to 2008)

Grant# 1N10224 (PI: JW Ramos)

1999-2001

"The Function of PEA-15, a gene overexpressed in Cancer"

Role: PI

Agency: California Cancer Research Project

The objective of this project was to 1) characterize the interaction of PEA-15 with R-Ras and ERK MAP kinase pathways using activated and dominant negative constructs of elements of these pathways; 2) Determine the residues of PEA-15 required for its effects on integrin activation; 3) Investigate the mechanism of transformation by the 3' untranslated region of PEA-15; and 4) Initiate a yeast two-hybrid screen to identify proteins that can bind PEA-15.

Grant# 4-29451 (PI: JW Ramos)

2001-2003

"The Structure of PEA-15"

Role: PI

Agency: Johnson and Johnson Foundation Discovery Grant.

The objective of this grant was to determine if mutations in PEA-15 affect PEA-15 ability to bind ERK and alter ERK function.

Grant# RSG TBE-103363 (PI: JW Ramos)

2002-2002

"Cell Signaling and Adhesion"

Role: PI

Agency: American Cancer Society

The objective of this proposal is to determine the expression pattern and functional significance of PEA-15 using PEA-15 null mice. The Specific aims were to 1) perform a mutational analysis of PEA-15; 2) determine the molecular mechanism of PEA-15 effects on ERK; 3) investigate the expression pattern of PEA-15; 4) analyze the function of PEA-15 using PEA-15 null mice.

(This grant was replaced by the NIH R01 CA93849-01 with similar aims after 6 months).