

Mary Helen Barcellos-Hoff

Mary Helen Barcellos-Hoff, Ph.D.



**Professor, Vice Chair, Research,
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Professional Focus

Dr. Barcellos-Hoff's research focuses on how tissues integrate information across scales of organization and uses this information to identify critical events in terms of effects on cell phenotype and tissue interaction during radiation carcinogenesis. To assess the relevance of these effects, Dr. Barcellos-Hoff developed a novel model in which irradiated mice with transplanted, unirradiated oncogenically primed mammary epithelial cells. Host radiation both accelerates tumorigenesis and affects the type of tumor, promoting those that are more aggressive. This model offers novel insight into the carcinogenic process. Identifying which

specific tissue alterations contribute to the action of IR may provide means to inhibit its carcinogenic potential, plus a better understanding of how tissues suppress carcinogenesis.

A main focus has been to understand biology of transforming growth factor β (TGF β), which is activated in irradiated cells and tissues, in normal mammary gland development and its contribution to radiation responses of tissues and tumors. In normal epithelial cells, TGF β controls proliferation and mammary lineage commitment. In irradiated epithelial cells, TGF β affects epithelial phenotypic stability, centrosome regulation and genomic stability. Dr. Barcellos-Hoff's laboratory demonstrated that TGF β is essential for epithelial cells to mount the canonical DNA damage response. The requirement for TGF β in the genotoxic stress program provides a link between cell fate and tissue integrity and underscores interaction between target cells and other cell types.

The requirement for TGF β in the genotoxic stress program also provided a previously unsuspected avenue to modulate radiotherapy by using concurrent treatment with TGF β inhibitors and radiation in preclinical breast, brain and lung cancer models to increase radiation sensitivity and tumor control.

Education

1978	The University of Chicago	BA	Biopsychology
1986	University of California, San Francisco	PhD	Experimental Pathology
1986-88	University of California, Berkeley	Postdoctoral	Cell Biology

Professional Experience

2015-present	University of California, San Francisco	Professor, Vice Chair, Research Dir. of Radiation Biology	Department of Radiation Oncology
2010-2015	New York University, School of Medicine	Professor (with tenure)	Departments of Radiation Oncology and Cell Biology
2008-2015	New York University, School of Medicine	Dir. of Radiation Biology	Department of Radiation Oncology
2008-2010	New York University, School of Medicine	Associate Professor	Department of Radiation Oncology
2005-2008	Lawrence Berkeley National Laboratory, University of California, Berkeley	Deputy Division Director	Life Sciences Division
2004-2008	Lawrence Berkeley National Laboratory, University of California, Berkeley	Department Head	Cancer and Systems Biology

2004-2008	Lawrence Berkeley National Laboratory, University of California, Berkeley	Senior Scientist	Life Sciences Division
2001-2003	Lawrence Berkeley National Laboratory, University of California, Berkeley	Department Head (acting)	Cells and Molecular Biology
1999-2003	Lawrence Berkeley National Laboratory, University of California, Berkeley	Group Leader	Cancer and Tissue Biology
1988-2003	Lawrence Berkeley National Laboratory, University of California, Berkeley	Staff Biologist	Life Sciences Division
1988	School of Medicine, University of California, Davis	Postgraduate Researcher	Department of Anatomy
1986-1988	University of California, Berkeley	Postdoctoral Fellow	Biophysics Training Program
1982-1986	University of California, San Francisco	Research Assistant	Department of Neurological Surgery

Recent Significant Publications :

Kleinberg DL, **Barcellos-Hoff MH**. The pivotal role of insulin-like growth factor I in normal mammary development. [2] *Endocrinol Metab Clin North Am*. 2011 Sep;40(3):461-71, vii. PMID: 21889714

Barcellos-Hoff MH. What is the use of systems biology approaches in radiation biology? [3] *Health Phys*. 2011 Mar;100(3):272-3. PMID: 21595065

Nguyen DH, Martinez-Ruiz H, **Barcellos-Hoff MH**. Consequences of epithelial or stromal TGF β 1 depletion in the mammary gland. [4] *J Mammary Gland Biol Neoplasia*. 2011 Jun;16(2):147-55. Epub 2011 May 17. PMID: 21590374

Nguyen DH, Oketch-Rabah HA, Illa-Bochaca I, Geyer FC, Reis-Filho JS, Mao JH, Ravani SA, Zavadil J, Borowsky AD, Jerry DJ, Dunphy KA, Seo JH, Haslam S, Medina D, **Barcellos-Hoff MH**. Radiation acts on the microenvironment to affect breast carcinogenesis by distinct mechanisms that decrease cancer latency and affect tumor type. [5] *Cancer Cell*. 2011 May 17;19(5):640-51. PMID: 21575864

Barcellos-Hoff MH. TGF β biology in breast: 15 years on. [6] *J Mammary Gland Biol Neoplasia*. 2011 Jun;16(2):65-6. PMID: 21534008

Barcellos-Hoff MH, Brenner DJ, Brooks AL, Formenti S, Hlatky L, Locke PA, Shore R, Tenforde T, Travis EL, Williams J. Low-dose radiation knowledge worth the cost. [7] *Science*. 2011 Apr 15;332(6027):305-6. PMID: 21493843

Andarawewa KL, Costes SV, Fernandez-Garcia I, Chou WS, Ravani SA, Park H, **Barcellos-Hoff MH.** Lack of radiation dose or quality dependence of epithelial-to-mesenchymal transition (EMT) mediated by transforming growth factor β . [8] *Int J Radiat Oncol Biol Phys*. 2011 Apr 1;79(5):1523-31. PMID: 21310544

Fernandez-Gonzalez, R., Illa-Bochaca, I., Welm, B. E., Fleisch, M. C., Werb, Z., Ortiz-de-Solorzano, C. and **Barcellos-Hoff, M. H.** (2009). Mapping mammary gland architecture using multi-scale in situ analysis. [9] *Integr Biol* 1, 80 - 89. PMID: 20023794

Maxwell, C. A., Fleisch, M. C., Costes, S. V., Erickson, A. C., Boissiere, A., Gupta, R., Ravani, S. A., Parvin, B. and **Barcellos-Hoff, M. H.** (2008). Targeted and nontargeted effects of ionizing radiation that impact genomic instability. [10] *Cancer Res* 68, 8304-8311. PMID: 18922902

Kirshner, J., Jobling, M. F., Pajares, M. J., Ravani, S. A., Glick, A., Lavin, M., Koslov, S., Shiloh, Y. and **Barcellos-Hoff, M. H.** (2006). Inhibition of TGF β 1 signaling attenuates ATM activity in response to genotoxic stress. [11] *Cancer Res* 66, 10861-68. PMID: 17090522

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