

Jerry Laboratory

Research Interests: *Breast cancer, p53, Genetics, Tumor suppressors*

Tumor Suppressor Genes and the Cellular Basis for Susceptibility to Breast Cancer

Reproductive factors and family history of breast cancer are the most important predictors of an individual's risk of developing breast cancer. These observations emphasize the important contributions of both genetic background and hormonal exposures in determining breast cancer risk. Our laboratory has demonstrated an association between activity of the p53 tumor suppressor protein and incidence of mammary tumors. Expression and activity of p53 protein are responsive to hormonal stimuli and vary across different stages of mammary gland development. Therefore, a major focus of the laboratory is to discover the normal cellular mechanisms that regulate p53 function and determine whether sustained elevation in p53 activity prevents mammary tumors in response to physiologic stimuli. We also use genetic mapping strategies in mice to identify low-penetrance modifiers of mammary tumor susceptibility. Genes that regulate p53 function would provide novel targets for prevention and treatment of breast cancer. Through the use of contemporary techniques in molecular and cellular biology and animal models, we are defining the developmental biology of the breast epithelium itself, while identifying both the genetic and cellular basis for susceptibility to breast cancer.

Selected Publications

Blackburn, A.C., Hill, L.Z., Roberts, A.L., Wang, J., Aud, D., Jung, J., Nikolcheva, T., Allard, J., Peltz, G. Otis, C.N., Cao, Q. J., Ricketts, R. St. J., Naber, S.P., Mollenhauer, J., Poustka, A., Malamud, D., and **Jerry, D.J.** 2007. Genetic mapping in mice identifies *DMBT1* as a candidate modifier of breast cancer risk. *Am. J. Pathol.* 170:2030-41

Koch, J.G., Gu, X., Han, Y., El-Naggar, A.K., Olson, M.V., Medina, D., **Jerry, D.J.**, Blackburn, A.C., Peltz, G., Amos, C.I., and Lozano, G. 2007. Mammary tumor modifiers in BALB/cJ mice heterozygous for p53. *Mamm. Genome Jun 8*; Epub ahead of print.

Becker, K.A., Lu, S.L., Dickinson, E.S., Dunphy, K.A., Mathews, L., Schneider, S.S. and **Jerry, D.J.** 2005. Estrogen and progesterone regulate radiation-induced p53 activity through TGF- β dependent pathways. *Oncogene* 24:6345-6353.

Tu, Yifan, **Jerry, D.J.**, Pazik, B. And Schneider, S.S. 2005. Sensitivity to DNA damage is a common component of hormone based strategies for protection of the mammary gland. *Mol. Cancer Res.* 3:435-442.

Blackburn, A.C., McLary, S.C., Naeem, R., Luszczyk, J., Stockton, D.W., Donehower, L.A., Mohammed, M., Mailhes, J.B., Soferr, T., Naber, S.P., Otis, C.N., and **Jerry, D.J.** 2004. Loss of heterozygosity occurs via mitotic recombination in *Trp53^{+/-}* mice and associates with mammary tumor susceptibility of the BALB/c strain. *Cancer Res.* 64:5140-5147.

Jerry, D.J., Kittrell, F.S., Kuperwasser, C., Laucirica, R., Dickinson, E.S., Bonilla, P.J., Butel, J.S., and Medina, D. 2000. A mammary-specific model demonstrates the role of the *p53* tumor suppressor gene in tumor development. *Oncogene* 19:1052-1058.

Kuperwasser, C. Hurlbut, G.D., Kittrell, F.S., Medina, D., Dickinson, E.S., Naber, S.P. and **Jerry, D.J.** 2000. Development of spontaneous mammary tumors in BALB/c *p53*-heterozygous mice: A model for Li-Fraumeni syndrome. *Am. J. Pathol.* 157:2151-2159.

Professional Highlights

Milestone Award for 5th Patent, University Of Massachusetts Amherst



D. Joseph Jerry, PhD

Chair, Breast Cancer Working Group, PVLSI

Associate Professor, Veterinary & Animal Science, University of Massachusetts Amherst
Adjunct Faculty in Pathology, Baystate Medical Center

Education

B.S., Animal Science, University of Vermont

M.S., Nutrition, Purdue University

Ph.D., Nutrition, Pennsylvania State University

Postdoctoral

Molecular Genetics, Jackson Laboratory, Bar Harbor, Maine, 1987-1990

Molecular Virology, Baylor College of Medicine, 1991-1993

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