

Smith Schneider Laboratory

Research Interests: *Breast cancer, Apoptosis, Immunology*

One in eight women will be diagnosed with breast cancer in their lifetime. The risk of this occurring is altered by several genetic and environmental and life style factors. For example, a full term pregnancy by the age of 20 will reduce one's risk of breast cancer by 50%. However, a family history of breast cancer, early menarche, exposure to DNA damaging agents or environmental toxicants during the ages of 12-15 or high levels of Insulin-like Growth Factor (IGF) correlate with an increased risk for breast cancer.

We are interested in signaling pathways which alter the balance of protection and increased risk in hopes of identifying important targets for preventive therapy. We are particularly interested in changes in retinoic acid signaling and how it relates to a release of p53 from its inactive state. We are also investigating how high levels of IGF may impede changes induced by pregnancy levels of hormones to impart protection. The pathways of interest all seem to relate back to the ability of the epithelium in the gland to respond to mutation through DNA repair or death. To this end a more in depth study analyzing pathways to apoptosis are underway in the lab. Signals from immune cells, fibroblasts, and adipocytes all appear to contribute to differentiation and breast cancer progression. We are trying to tease apart these signals and are investigating the regulation of the Notch and WNT pathways through these interactions.

Finally, we are interested in how nutritional elements may be responsible for imparting protection. This has been suggested from epidemiological data looking at the low rate of breast cancer in the Asian population. Interestingly, the protection is lost when these women migrate to Western countries suggesting that some dietary element(s) may be responsible for the change in susceptibility to breast cancer. We are currently looking at certain adaptogens (plant extracts with activities which allow for an easy adjustment to stressful situations) for their ability to act as chemopreventive or chemotherapeutic agents.

Selected Publications

Y Tu, B Pazik, DJ Jerry, **S Smith Schneider** (2005). Sensitivity to DNA damage is a common component of hormone based strategies for protection of the mammary gland." *Mol Cancer Res* 3(8): 435-442.

Zheng-Gang Liu*, **Sallie W. Smith***, Kelly A. McLaughlin, Lawrence M. Schwartz, Barbara A. Osborne (1994). "Apoptotic signals delivered through the T-cell receptor of a T-cell hybrid require the immediate-early gene nur77." *Nature* 367: 281-284. (*Contributed equally to this work).

Lowe, SW, Schmitt EM, **Smith SW**, Osborne BA, Jacks T (1993). "P53 is required for radiation-induced apoptosis in mouse thymocytes." *Nature* 362: 847.

Professional Highlights

1986 Donald W. Pyle Award for undergraduate thesis research

1995 Glenn Snoeyenbos Award for graduate thesis research

Member of the California Breast Cancer Research Program grant review panel



Sallie Smith Schneider, PhD

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Adjunct Research Assistant
Professor, Veterinary & Animal
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Education

B.A., Biology, Skidmore College

Ph.D. Immunology, University of
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Postdoctoral

Signaling, Harvard Medical
School, 1995-1998

Cooperative phosphorylation of
c-Fos by the ERK pathway and
its role in proliferation

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