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Shahla Masood, J Clin Exp Pathol 2017, 7:2 (Suppl)
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Shahla Masood

University of Florida, USA

Breast cancer prediction and early detection: The potential value of cytomorphology and *hTERT* gene DNA methylation

Risk assessment has become an integral part of multi-disciplinary breast care, and breast cancer risk reduction interventions, and identification of high risk individuals have received a great deal of attention. Atypical proliferative changes in breast epithelial cells are ranked high among various known breast cancer risk factors and have been the subject of several investigations. Breast tissue and fluid in the ductal system provides a rich source of cells and biomarkers that has the potential to measure short-term risk of breast cancer development, and assess responses to interventional prevention efforts. Minimally invasive procedures such as fine needle aspiration biopsy, ductal lavage, and nipple fluid aspiration are commonly used in breast cancer detection and research. We have established the "Masood Cytology Index" as a morphologic risk predictor and believe that the development of a novel malignancy-associated biomarker amplified by PCR will enhance our ability to stratify high-risk patients. DNA hypermethylation has been documented to be prominent using qualitative methylation specific PCR on DNA isolated from the tumor cell line. Using quantitative pyrosequencing technology, we demonstrated that there is significant hypermethylation in tumor cells versus low DNA methylation in normal tissue. The results of this study highlight the value of DNA hypermethylation as a potential marker for early breast cancer detection. More importantly, we believe that integration of this novel malignancy associated testing with morphology is of significant value in the accurate interpretation of breast cancer precursors obtained from minimally invasive procedures and may be used as a breast cancer risk predictor.

Biography

Shahla Masood is a Persian-born Physician, who is currently the Professor and Chair of the Department of Pathology and Laboratory Medicine and Medical Director of the UF Health Breast Center at the UF College of Medicine, Jacksonville. As an internationally recognized expert in breast cancer diagnosis and prognosis, she has lectured extensively in over 50 countries, has authored numerous publications, book chapters, and textbooks, and is the recipient of countless educational and scientific awards. She has been named as one of the Top Doctors in America, Top Doctors in Cancer, and the Top 20 Most Influential Professors in Oncology.

Shahla.Masood@jax.ufl.edu

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Dale D Tang

Albany Medical College, USA

Polo-like kinase 1 (Plk1) in smooth muscle and allergic asthma

Smooth muscle contraction and cell proliferation are critical for the pathogenesis of airway hyper-responsiveness and hyperplasia of allergic asthma. Polo-like kinase 1 (Plk1) is a serine/threonine protein kinase that has been implicated in mitosis and cytokinesis. The role of Plk1 in smooth muscle contraction and cell growth has not been previously investigated. Here, stimulation with acetylcholine induces Plk1 phosphorylation at Thr-210 (an indication of Plk1 activation) in smooth muscle. Contractile stimulation also activates Plk1 in live smooth muscle cells as evidenced by changes in fluorescence resonance energy transfer signal of a Plk1 sensor. Plk1 is necessary for smooth muscle force development. Plk1 regulates airway smooth muscle contraction by affecting vimentin phosphorylation at Ser-56, but without modulating myosin light chain phosphorylation. Plk1 phosphorylation is mediated by Ste20-like kinase (SLK), a serine/threonine protein kinase that has been implicated in spindle orientation and microtubule organization during mitosis. Moreover, Plk1 is indispensable for airway smooth muscle cell proliferation. Plk1 knockdown by lentivirus-mediated shRNA attenuates the growth factor-induced phosphorylation of MEK1/2 and ERK1/2. However, Plk1 knockdown does not affect the phosphorylation of Raf-1or AKT. Finally, smooth muscle conditional knockout of Plk1 attenuates airway resistance, airway smooth muscle hyper-reactivity and hyperplasia in a murine model of allergic asthma. Taken together, these findings suggest that Plk1 is critical for the regulation of smooth muscle contraction and cell proliferation. Plk1 regulates smooth muscle contraction by controlling vimentin phosphorylation, whereas, it orchestrates cell proliferation by modulating the MAPK pathway. Plk1 contributes to the pathogenesis of allergic asthma. Plk1 may be a pharmacological target for the development of new therapy to treat asthma.

Biography

Dale D Tang has received training at the University of Texas Southwestern Medical Center at Dallas in 1990s. He is a Professor of the Department of Molecular and Cellular Physiology at Albany Medical College, New York, USA. He is Director of Cytoskeletal Signaling and Asthma Research Program at the school. He is an Associate Editor of BMC *Respiratory Research* and an Editorial Board Member of *Nature Scientific Reports*. His research focuses on the role and mechanism of cytoskeleton-associated proteins in smooth muscle in vitro and the pathogenesis of asthma and hypertension in vivo. He has published >70 peer-reviewed articles in journals including the *Journal of Biological Chemistry and Circulation Research*.

TangD@mail.amc.edu

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Robert H Schiestl

University of California, USA

Effect of intestinal microbiota on lymphoma and longevity in Atm deficient mice

Intestinal microbiota plays a role in the nutrient metabolism, modulation of the immune system, arthritis, obesity and intestinal inflammation. In the literature, there have been huge differences in the same Atm deficient mice in different labs reported. When our lab moved from Harvard to UCLA, we found a similar difference in genetic instability and longevity. When we changed the intestinal microbiota back to conventional microbiota, we could reproduce the phenotype at Harvard. We tested Atm deficient mice for genotoxicity, genetic instability, DNA damage, inflammation markers, cancer latency and longevity, and high throughput sequencing of the intestinal microbiota. Isogenic mice from different housing facilities showed a four fold difference in life expectancy, a 4.5 fold difference in genetic instability and DNA damage. The onset of lymphomas was significantly 2 fold different. We sequenced the microbiota of both facilities and found *Lactobacillus johnsonii* 456 as dominant bacterial strain in the health beneficial microbiota. Just this bacterium by itself reduced genotoxicity, reduced inflammation and reduced levels of cytotoxic T cells in the liver and blood. We also found similar differences in Trp53 deficient and even in wild type mice. The underlying mechanisms are probably due to inflammation promotion or suppression mediated by the intestinal microbiota. The understanding of this effect may lead to a breakthrough in the understanding of the causes of carcinogenesis, which might lead to prevention of AT, a currently incurable progressive disease and possibly other cancer-prone DNA repair deficient diseases or even wild type mice and people.

Biography

Robert H Schiestl has obtained his PhD from the University of Vienna. He was a Postdoctoral Fellow at Edmonton, Alberta, Rochester, NY, and Chapel Hill, NC, before being a Professor at Harvard, where he stayed for 10 years. Since 16 years, he is working as a Professor at UCLA with 200 publications, 11 patents and 5 startup companies.

RSchiestl@mednet.ucla.edu

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