Learning to Care for Those in Harm's Way

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Heart Failure Biomarker Linked to Rare Genetic Tumor-Causing Diseases
Scientists tie Galectin-3 to tuberous sclerosis, lung disease lymphangioleiomyomatosis

Bethesda, Md. – Galectin-3, a protein that promotes cancer cell growth and is used as a biomarker for heart failure, has been linked to tumors observed in two rare genetic diseases, according to a study published July 11, 2017, in eLife (https://doi.org/10.7554/eLife.23202) by Klover, et al.

Researchers at the Uniformed Services University (USU) and the National Heart, Lung, and Blood Institute (NHLBI)/National Institutes of Health have discovered that galectin-3 is produced by tumor cells in tuberous sclerosis complex (TSC) and lymphangioleiomyomatosis (LAM).

According to the Tuberous Sclerosis Alliance, "TSC is a genetic disorder that causes tumors to form in many different organs, primarily in the brain, eyes, heart, kidney, skin and lungs." Women with TSC are highly susceptible to developing LAM, and LAM also occurs rarely in those without TSC.

LAM is a lung disease that typically attacks women "during the prime of their lives and is characterized by an abnormal growth of smooth muscle cells, especially in the lungs, lymphatic system and kidneys. Unregulated growth of these cells can lead to loss of lung function, accumulation of lymph rich-fluid in the chest and abdomen and growth of tumors in the kidneys," according to The LAM Foundation.

Both TSC and LAM are caused by mutations in the TSC1 or TSC2 genes.

Dr. Thomas Darling, professor and chair of Dermatology at USU, and Dr. Joel Moss, deputy branch chief of the Cardiovascular and Pulmonary Branch at NHLBI, and their team of researchers, worked with The American Genome Center at USU to closely examine molecular pathways affected by TSC2 mutation, and found that galectin-3 was elevated in tumors from patients with TSC or LAM. They found that serum levels of galectin-3 correlated with lung disease severity and with the presence of renal tumors. The researchers now believe this could help to assess treatment response in TSC and LAM and to detect cancers with mutations in TSC1 or TSC2.

First author Dr. Peter Klover, a senior research associate in USU's Department of Dermatology, also examined mesenchymal cells – cells that form connective tissue and skin – and found that those which did not have the TSC2 gene reproduced aspects of TSC disease. This would be useful for finding new treatment modalities and markers of disease severity.

Ultimately, these findings have important implications for precision medicine approaches to treating other diseases associated with the loss of the TSC2 gene, helping to discover new treatments and other markers for disease diagnosis and prognosis.

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About USU

The Uniformed Services University of the Health Sciences, founded by an act of Congress in 1972, is the nation's federal health sciences university and the academic heart of the Military Health System. USU students are primarily active duty uniformed officers in the Army, Navy, Air Force and Public Health Service who have received specialized education in tropical and infectious diseases, TBI and PTSD, disaster response and humanitarian assistance, global health, and acute trauma care. A large percentage of the university's more than 5,800 physician and 1,000 advanced practice nursing alumni are supporting operations around the world, offering their leadership and expertise. USU also has graduate programs in biomedical sciences and public health, most open to civilian and military applicants, and oral biology, committed to excellence in research. The University's research program covers a wide range of clinical and other topics important to both the military and public health. For more information about USU and its programs, visit *www.usuhs.edu.*