Journal of Morphology and Anatomy

Editorial Open Access

Editorial on Anti-Fibrotic Treatment in Scleroderma/Fibrosis

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Scleroderma is a debilitating autoimmune disease characterized by immune system activation, vasculopathy, inflammation and fibrosis in the skin, lungs and other organs. The major cause of death in this disease is pulmonary fibrosis, which is characterized by excessive accumulation in the lungs of extracellular matrix proteins, particularly collagen. Scleroderma lung disease is particularly devastating due to its poor prognosis, and lacking of effective treatments. Caveolin-1 is a ubiquitously expressed integral membrane protein and essential for the formation of so-called caveolae, small invaginations of the plasma membrane. Caveolae are involved in major physiological functions of the mammalian cell, including endocytosis and transcytosis processes, signal transduction and cholesterol homeostasis. Observations on lung tissue from human patients and animal lung disease model have suggested that under-expression of caveolin-1 is a common feature of interstitial lung diseases, such as scleroderma and idiopathic pulmonary fibrosis is the. Caveolin-1 plays a central role in regulating lung injury/fibrosis. Restoring caveolin-1 expression/function in bleomycin-treated mice using either the caveolin scaffolding domain (CSD) peptide or adenovirus encoding the full-length molecule inhibits the progression of pulmonary fibrosis. Recently I developed a useful murine model for SSc in which bleomycin is delivered by implanted osmotic mini-pumps. In this model, a wide range of organ fibrosis is observed which accurately recapitulates the hallmark features of SSc. By employing this model, we observed that the caveolin-1 scaffolding domain peptide (CSD) inhibits fibrocyte infiltration in dermis and blocks dermal fibrosis. We also demonstrated that CSD inhibits human SSc monocytes hypermigration toward a group of chemokines.

Cancer biology and cancer therapy: Cancer is one of the most common diseases in the world. Today, millions of people are living with cancer. New diagnostic approaches and therapeutic strategies are urgently needed to counteract this particular epidemic. I have devoted most of my career working on cancer research, which were mainly involved in tumor markers, multidrug resistance, angiogenesis and anti-angiogenic therapy in human cancers. In addition, I was directly involved in studying the anti-cancer effect of β-elemene in human cancers.

Hypoxia regulation: Hypoxia plays essential roles in the pathobiology of human diseases, such as cancer, cardiovascular diseases, chronic pulmonary diseases, stroke, anemia, ischemia, etc. The central transcriptional response to hypoxia is mediated by the Prolyl Hydroxylase Domain protein (PHD): Hypoxia Inducible Factor (HIF) pathway. In this pathway, PHD prolyl hydroxylates and thereby negatively regulates the α-subunit of the transcription factor HIF (HIF-α). An important HIF target gene is that for Erythropoietin (EPO), which controls red cell mass. My studies were focused on the HIF: PHD2: EPO pathway to investigate the role of PHD2 in the development of polycythemia in mice, as well as the integrity of the PHD2: EPO axis in aging mice, by employing a mouse line with a globally-inducible PHD2 conditional knockout allele. My work has revealed that acute global deletion of PHD2 result in a robust erythrocytosis in both young and aging mice, with both groups showing marked extramedullary hematopoiesis in the spleen. These findings have tremendous implications for targeting this pathway to treat anemia and ischemia.

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Received September 19, 2017; **Accepted** September 19, 2017; **Published** September 28, 2017

Citation: Lee R (2017) Editorial on Anti-Fibrotic Treatment in Scleroderma/Fibrosis. J Morphol Anat 1: e102.