Wallace H. Coulter Foundation Lecture Series

Cadherin-11 Mechanobiology in Cardiac Fibrosis and Disease

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Lecture: Friday, February 23, 2018
9:00AM-10:00AM
Room EC 1116
10555 West Flagler Street
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FIU Engineering & Computing
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Biography

W. David Merryman, PhD is an Associate Professor in the Departments of Biomedical Engineering, Pharmacology, Medicine, and Pediatrics at Vanderbilt University. He is also Associate Chair of the Department of Biomedical Engineering. His research interests are cardiovascular and pulmonary mechanobiology, cell and soft tissue biomechanics, tissue engineering, and bioengineering ethics. Prior to his arrival at Vanderbilt, Dave was an Assistant Professor of Biomedical Engineering at the University of Alabama at Birmingham and prior to that, a Research Associate of the McGowan Institute for Regenerative Medicine and Bioengineering at the University of Pittsburgh, where he was an American Heart Association Pre-doctoral Fellow. Dave has been awarded the Early Career Award from the Wallace H. Coulter Foundation, the Scientist Development Grant from the American Heart Association, the NSF CAREER Award, the K Award from the National Institutes of Health (NHLBI), the Y.C. Fung Young Investigator Award from the American Society of Mechanical Engineers, and in 2017, he was awarded the NHLBI's Emerging Investigator Award (R35), which funds the most promising cardiovascular researchers for seven years.

Abstract

Cardiac fibrosis can affect both the heart valves and the myocardium and is characterized by fibroblast activation and extracellular matrix accumulation. Valvular interstitial cells and cardiac fibroblasts, the cell types responsible for maintenance of cardiac extracellular matrix, are sensitive to changing mechanical environments, and their ability to sense and respond to mechanical forces determines both normal development and the progression of disease. Recent studies have uncovered specific adhesion proteins and mechano-sensitive signaling pathways that contribute to fibrosis progression. Cadherins mechanically link neighboring fibroblasts and likely contribute to fibrotic disease propagation. Primarily, we believe that they do this by speeding the transition of quiescent fibroblasts to the active myofibroblast phenotype, which leads to maladaptive tissue remodeling and enhanced mechanotransductive signaling, forming a positive feedback loop that contributes to heart failure. In this brief talk, I will describe some of the recent data that my laboratory has generated which are focused on a little-known, but very important, cadherin that appears to be heavily involved in cardiac fibrosis – cadherin-11.

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