Abstract: Ectopic calcification is the abnormal deposition of calcium salts (apatite) in soft tissues including valves, blood vessels, joints, muscle and the brain. Typically a result of disease and aging, it is also a frequent complication of severe trauma, including spinal cord and brain injury, and a major mode of failure for bioprosthetic valves. Normally, the body prevents ectopic calcification via systemic or locally–derived calcification inhibitory factors, such as osteopontin. In injury and disease, inhibitory factors are often deficient, leaving calcification inductive processes unopposed, and allowing mineral-formative processes to predominate. Ameliorating anticalcific factor deficiency and/or promoting mineral resorption will be discussed as potential strategies to treat and potentially regress ectopic calcification.

Biography: Dr. Giachelli received a BS in Biochemistry from UC Davis and PhD in Pharmacology from the University of Washington, where she is currently Professor of Bioengineering and Adjunct Professor of Pathology and Oral Health Sciences. Dr. Giachelli is internationally recognized for her work investigating the molecular mechanisms of ectopic calcification and extracellular matrix control of cell function. Her studies have led to the discovery of key inducers and inhibitors that contribute to ectopic calcification in the setting of chronic kidney disease, valve disease, atherosclerosis and medical devices. These discoveries are currently being translated to therapeutic strategies to block inappropriate calcification in disease and biomaterials development. Other key areas of research include control of inflammation and foreign body reaction, regenerative medicine, cell and tissue engineering. Dr. Giachelli is an elected fellow of the American Institute for Medical and Biological Engineering, and recipient of the American Heart Association Established Investigator Award, the Advances in Mineral Metabolism Investigator Award, and the American Society of Nephrology’s Jack W. Coburn Lectureship.