FSGS: Podocytes, Cytokines and Signaling in Progressive Glomerular Injury

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The podocyte has assumed a prominent role in the understanding of the glomerular filtration barrier since the identification of nephrin as the site of the defect in congenital Finnish nephropathy. Since then there has been an explosion of studies directed at the detailed description of podocyte structural proteins and their interactions as they relate to the filtration barrier and to the development of renal disease. These proteins and the metabolic responses of podocytes have been studied in cell culture and in preparations of isolated glomeruli as well is in animal models and human tissues. The physiology of glomerular filtration has been of particular interest to our group and we have focused on the acute responses of the glomerulus to physiologic and pathologic stimuli. We have confirmed the importance of several signaling pathways in modulating barrier function and are currently studying the responses to cardiotrophin-like cytokine 1, which we have identified as a candidate for the circulating permeability factor in FSGS. We propose that cell signaling, as well as later changes in morphology, is essential to proteinuria and to progressive to the glomerulus and the remainder of the kidney. Current information regarding podocytes, proteinuria and the cellular responses and injury will be required in order to permit us to develop rational therapies to prevent progressive renal failure. Participants will have the opportunity to review current advances in podocyte biology, to review the role of cytokines and other circulating substances in FSGS and to focus on the role of the podocytes inprogressive renal disease.