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based on genetic deletion studies in mice of PAR4 and the BK receptor in which both PAR4 and the BK receptor confer both cardiorenal protection and thrombotic phenotype. This proposal will test this working hypothesis in sepsis and acute kidney injury. I will examine the role of a recently identified G protein coupled receptor (GPCR) termed PAR4 in thrombotic and cardiorenal response to experimental acute kidney injury and sepsis. I will use a bradykinin 2 (B2) receptor knockout mice which is genetically deficient in the expression of PAR4 to assess the relative importance of the PAR4-BK2 receptor system in the response to acute kidney injury and sepsis. In addition, I will use a PAR4 deficiency in the bradykinin 1 receptor knockout mice (PAR4D) to evaluate the relative importance of PAR4 and the BK receptors in the cardiac and vascular response to acute kidney injury and sepsis. I propose three specific aims: 1) To characterize the response of septic PAR4D mice to cardiorenal injury. 2) To characterize the response of septic B2 receptor knockout mice to cardiorenal injury. 3) To characterize the response of PAR4D mice to acute kidney injury. A better understanding of

these pathways and their interaction may lead to more effective therapies for cardiorenal disease. [unreadable] [unreadable] [unreadable]Innovation and

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