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My research interests include 1) mechanisms involved in sepsis-induced myocardial inflammation and dysfunction, 2) intracellular signaling pathway(s) leads to conversion of the cardiac myocytes to a proinflammatory phenotype when the myocytes conditioned with an anoxia/reoxygenation (A/R, in vivo counter part to ischemia/reperfusion) and try to understand mechanisms(s) involved in ischemia/reperfusion induced myocardial dysfunction, and 3) explore the role(s) of high-mobility group box-1 protein (HMGB1) in cardiovascular pathology. Both in vitro and in vivo approaches are employed in my laboratory.

Selected publications

Yang M, Wu J, Martin CM, Kvietys PR, **Rui T**. Important role of p38 MAP kinase/NF-kappaB signaling pathway in the sepsis-induced conversion of cardiac myocytes to a proinflammatory phenotype. *Am J Physiol Heart Circ Physiol*. 2008;294(2):H994-1001.

Rui T, Kvietys PR. NFkappaB and AP-1 differentially contribute to the induction of Mn-SOD and eNOS during the development of oxidant tolerance. *FASEB J*. 2005;19(13):1908-10.

Rui T, Feng Q, Lei M, Peng T, Zhang J, Xu M, Abel ED, Xenocostas A, Kvietys PR. Erythropoietin prevents the acute myocardial inflammatory response induced by ischemia/reperfusion via induction of AP-1. *Cardiovasc Res*. 2005;65(3):719-27.

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