Investigator:	**Bum-Yong Kang** Phone: (404) 321-6111 ext. 206143 Email: bkang6@emory.edu
Primary Research Interest:	Genetics
Description of Research:	The pathogenesis of pulmonary hypertension (PH), a cardiopulmonary disorder associated with significant morbidity and mortality, involves endothelial dysfunction with increased production of vasoconstrictors, e.g. endothelin-1 (ET-1). Although existing PH therapies are designed to attenuate these derangements, the poor outcomes in PH indicate an urgent need for novel therapeutic strategies. Recent studies indicate that stimulating peroxisome proliferator-activated receptor gamma (PPARg) with thiazolidinedione (TZD) ligands attenuates PH in several experimental models, whereas reduced PPARg is associated with PH. Preliminary and published data demonstrate that PPARg ligands attenuate hypoxia-induced ET-1 levels and PH in part by suppressing NF-kB activation. Our preliminary data also indicate that hypoxia reduces miR-98 levels to increase ET-1 expression and that PPARg activation restores miR-98 levels to reduce ET-1 expression. Therefore, we hypothesize that PPARg reduces ET-1 in PH by attenuating NF-kB and enhancing miR-98 levels.
Relevance to VA:	Veterans suffer disproportionately from lung injury because of their high burden of disease leading to pulmonary hypertension (PH). New and more effective therapies for veteran patients with these disorders could have significant impact on patient outcomes and VA healthcare. The results of these pre-clinical studies can better inform future clinical trials examining PPARg activation as a novel therapeutic approach in PH.