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SOCS-1 gene therapy protects against hyperoxia- Induced acute lung injury and pulmonary edema

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Suppressor of Cytokine Signaling-1 (SOCS-1) is an anti-apoptotic and potent anti-inflammatory, negative regulator of the IL-6-mediated JAK-STAT signaling pathway. SOCS-1 has been shown to induce protection from cellular damage and apoptosis induced by tumor necrosis factor, lipopolysaccharide, and interferon gamma. However the protective role of SOCS-1 in pulmonary oxidative stress is unknown. We hypothesized that SOCS-1 induction though gene therapy may induce protection against hyperoxic lung injury. To test this hypothesis we administered SOCS-1 adenovirus (Ad-SOCS-1) into the lung and exposed mice to 100% O_2 (hyperoxia). Mice infected with AdGFP were used as controls. Mice treated with Ad-SOCS-1 had enhanced survival in 100% oxygen when compared to AdGFP-administered mice. After 3 days of hyperoxia, Ad-GFP mice were ill and tachypnic and died after four days. In contrast, all of the Ad-SOCS-1 mice survived for at least 6 days in hyperoxia and 80% survived beyond 7 days. SOCS-1 transfection protected mouse lungs from hyperoxia-induced injury as indicated by decreased infiltration of inflammatory cytokines, alveolar-capillary protein leakage and reduced infiltration of inflammatory cells in bronchoalveolar lavage fluid, and lower content of thiobarbituric acid-reactive substances in lung homogenates. Further, SOCS-1 treatment suppressed hyperoxia-induced pulmonary edema as indicated by reduced lung wet to dry weight ratio when compared to their controls. Our results also indicate that SOCS-1 significantly inhibits hyperoxia induced nuclear factor kappa B (NF-κB) activation which is associated with reduced p65 (a subunit NF-κB) expression. These findings show that increased expression of SOCS-1 in the lungs of mice significantly protects from hyperoxic lung injury and pulmonary edema.

Biography

Narasaiah Kolliputi is an Assistant Professor and Division Director of Research Education for Division of Allergy and Immunology at the Morsani College of Medicine. Kolliputi is working on translational strategies to attenuate oxidative stress mediated acute lung injury (ALI). He received his postdoctoral training in the Massachusetts General Hospital, Harvard Medical School. Kolliputi is currently serving as a grant reviewer for the Department of Defense and American Heart Association Grants. He is an Associate Editor for Frontiers in Respiratory Pharmacology, Frontiers in Oxidant Physiology, Frontiers in Non-coding RNA and Guest Associate Editor for Frontiers in Mitochondrial Physiology, Editorial Board Member for Translational Medicine, Virology & Mycology and Journal of Biocatalysis & Biotransformation. His research is funded by NIH RO1 and American Heart Association Scientist Developmental grants.

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