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Adenovirus-mediated gene therapy of SOCS-1 protects against acute lung injury

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Suppressor of Cytokine Signaling–1 (SOCS–1) is a member of the suppressor of cytokine signaling family of proteins and an inhibitor of Interleukin–6 signaling. SOCS–1 has been shown to protect from cellular damage and apoptosis induced by tumor necrosis factor, lipopolysaccharide, and interferon gamma. Whether increased SOCS–1 is protective during pulmonary oxidative stress is not known. We therefore hypothesized that SOCS–1 increased SOCS–1 in the lungs of mice would be protective in the setting of hyperoxic lung injury. We administered SOCS–1 adenovirus (Ad–SOCS–1) into the lung and exposed mice to 100%O2. Mice infected with AdGFP were used as controls. Mice treated with Ad–SOCS–1 had enhanced survival in 100% oxygen showed 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 injury as indicated by lower lung wet/dry weight, alveolar–capillary protein leakage and reduced infiltration of inflammatory cells in bronchoalveolar lavage fluid, and lower content of thiobarbituric acid–reactive substances in lung homogenate. 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 through adenovirus–mediated in the lungs of mice significantly protects from hyperoxic lung injury.

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Biography

Narasaiah Kolliputi is an assistant professor at the University of South Florida. He graduated from Osmania University, India, where he received doctoral degree in biochemistry. He received his postdoctoral training in MGH at Harvard Medical School. His present work at USF involves translational gene therapy strategies to attenuate acute lung injury (ALI) and acute respiratory distress syndrome (ARDS). Dr. Kolliputi's research is funded by NIH RO1 and American Heart Association Scientist Developmental grants.

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