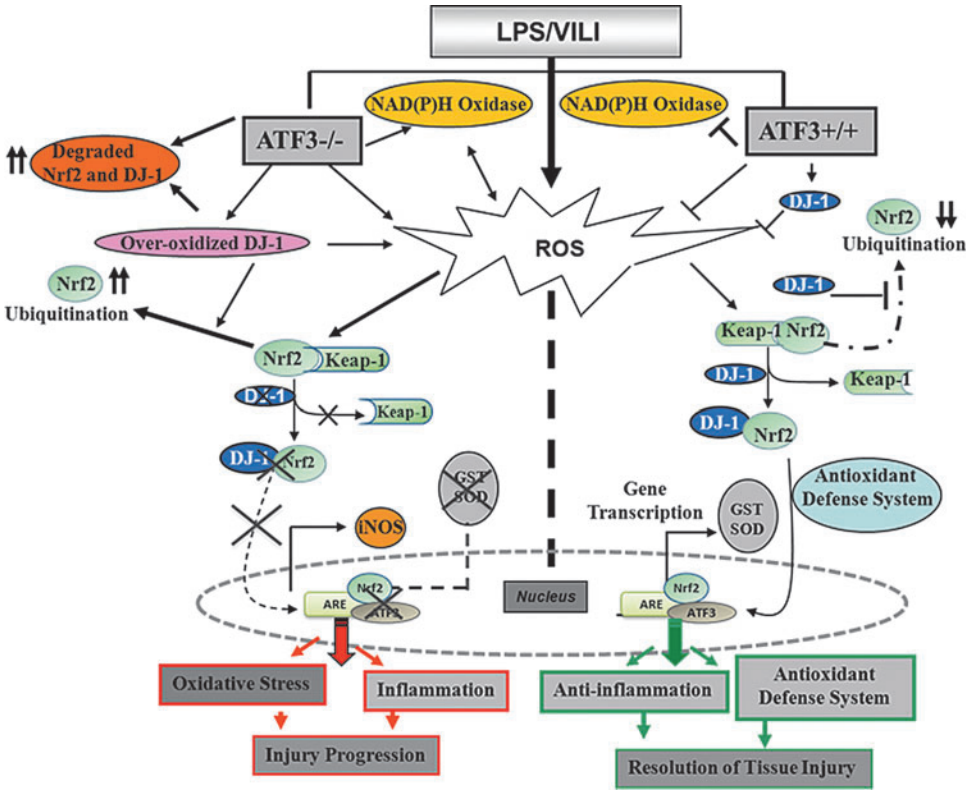

SUPPLEMENTARY FIG. S4. Schematic of proposed ATF3-dependent protection against VILI-induced oxidative stress in pulmonary cells: In wild type mice (ATF3+/+), DJ1 binds to Nrf2 protein, preventing Keap-1 from tagging it for degradation. Nrf2 is then able to drive transcription of critical antioxidant genes GST and *MnSOD*, thus protecting cells from oxidative stress. Absence of ATF3 results in DJ-1 oxidation. Dimerized DJ-1 is no longer available to bind to Nrf2 and to protect it from degradation. Inactivation of DJ-1 results in Nrf2 degradation, decreased expression of antioxidant genes, increased oxidative stress, and inflammation, resulting in loss of barrier function and progression of lung injury.