Pulmonary arterial hypertension (PAH) is a disease characterized by high blood pressure in the lungs. The pressure increase is caused by the obstruction in the small arteries or vasoconstriction in the lung. Some of these obstructions can be attributed to fibrosis.

In pulmonary fibrosis, the lung tissue becomes scarred and ultimately reduces alveoli space and oxygen consumption declines.

Deficiency of bone morphogenic protein receptor type 2 (BMPR2) is found in PAH patients.

It is believed that BMPR2 is profibrotic and promotes Epithelial to mesenchymal transition (EMT). This is a process in which epithelial cells lose their cell polarity and cell-cell adhesion and gain migratory and invasive properties to become mesenchymal cells.

To test the effects of BMPR2 knockdown, short hairpin RNA was cloned into a pLKO vector. HEK-293T cells were then transfected with the shRNA.

Post transfection, the virus was collected and Wi38 human long fibroblast cells were infected in order to knockdown BMPR2.

BMPR2 Knockout will be assessed via western blot

Immunofluorescence will be used to determine the difference between the KO and control cells

Fibrosis & EMT will be modeled by the use of different stimulants such as TGFβ and TNFα