**Abstract**

The main cells involved in wound repair are fibroblasts due to their ability to produce extracellular matrix proteins such as collagen 1 and contract surmounting tissue to reduce wound size. The downregulation of many critical points for fibroblast function suggests that these cells may have an aberrant phenotype in disease. TGFβ1 plays many roles in the wound repair process and is a pivotal activator of fibroblast function. Due to its pleiotropic effects, TGFβ1 is regulated by many factors including TLRs, however, by other groups have shown a close relationship between the two signalling pathways that can form a positive feedback loop to perpetuate a proinflammatory response in lung parenchymal fibroblasts (3).

**Methods**

We hypothesise that dysregulation of ANG II-TGFβ1 crosstalk within the lung fibroblasts of COPD patients disrupts normal wound repair leading to disease.

**Results**

**Figure 2. TGFβ1 signalling is preserved in parenchymal fibroblasts derived from healthy ex-smokers and COPD patients**

**Figure 3. ANG II does not augment TGFβ1 production of ex-smoker parenchymal fibroblasts**

**Figure 4. Collagen 1 production is augmented in COPD**

**Figure 5. TGFβ1 expression is dysregulated in COPD**

**Figure 6. TGFβ1 signalling is intact in healthy and COPD ex-smoker parenchymal fibroblasts**

**Figure 7. ANG II does not augment TGFβ1 production of ex-smoker parenchymal fibroblasts**

**Figure 8. Collagen 1 production is augmented in COPD**

**Summary**

- TGFβ1 downregulates AT1R protein expression in ex-smoker parenchymal-derived fibroblasts, but this response is blunted in COPD parenchymal fibroblasts.

- TGFβ1 signalling was shown to be intact in both healthy and COPD-derived parenchymal fibroblasts, thereby suggesting that TGFβ1 may mediate downregulation of AT1R by regulating receptor degradation.

- There is greater collagen 1 production in COPD lung tissues and parenchymal fibroblasts, which may be due to an over-exuberant response to TGFβ1.

**Future Directions**

- Examine the effect of TGFβ1 on AT1R receptor degradation.

- Evaluate the impact of the aberrant ANG II-TGFβ1 crosstalk on COPD parenchymal fibroblast repair functions.