The ‘Fibrosis Laboratory’ is based in the Department of Pharmacology at Monash University and has established culture systems and in vivo models across various major fibrosis indications (i.e. cardiovascular, renal and lung). It can examine the anti-fibrotic efficacy and mechanism of various emerging therapies benchmarked to current front-line treatments. The laboratory is led by A/Professor Chrishan Samuel, who is a fibrosis expert and the most published (peer reviewed) author on the anti-fibrotic effects of (se)relaxin and related hormone-based agents.

Benefits of Facility:

- Access to various fibrosis culture systems and in vivo animal models of disease;
- Expertise in the analysis of efficacy and signal transduction mechanisms of various anti-fibrotic effects of anti-fibrotic agents;
- Specialisation in the anti-fibrotic effects of (se)relaxin and related agents;
- Able to correlate drug-induced changes in fibrosis with tissue function.

Background

Chronic and progressive tissue fibrosis is a condition that can affect all major organ systems and is caused by the excess accumulation of extracellular matrix components, including collagens. It is a major factor in many non-infectious diseases and there are few specific anti-fibrotic therapies available for these indications.

Activation of the renin-angiotensin system (RAS), oxidative stress and inflammation pathways are all involved in the aetiology of fibrosis. Among the mediators of these pathways, Angiotensin II (Ang II) and Transforming Growth Factor-β (TGF-β) play important roles as major drivers of tissue fibrosis in virtually all fibrotic diseases (1,2).

There is an urgent need to identify new targets and drugs that can inhibit and even reverse fibrosis without adversely disrupting collagen metabolism in other organs.

The Opportunity

The ‘Fibrosis Laboratory’ at Monash University has established various (myo)fibroblast culture systems and in vivo models across various fibrosis indications and expertise in assessing the efficacy of emerging therapies benchmarked to current front-line treatments. Leading models are listed below.

Cardiac Models
- Myocardial infarction-induced heart failure (3)
- Isoproterenol-induced cardiomyopathy (4)
- Spontaneously hypertensive rats (5)
- High salt-induced hypertension
- Type 1 diabetes (6) and
- Ageing associated with relaxin-deficiency (7)

Renal Models
- Unilateral ureteric obstruction (8)
- Ageing associated with relaxin-deficiency (9)

Lung Models
- Bleomycin-induced interstitial lung disease
- Acute and chronic models of allergic airways disease (10,11)
- Allergic airways disease incorporating epithelial damage (12)
- Ageing associated with relaxin-deficiency (13)

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