# Pericardial adipose accumulation and cardiac pathology - mechanistic insights <br> J.R. Bell, Department of Physiology, University of Melbourne, VIC 3010, Australia. 

Obesity has long been recognized as a risk factor for cardiovascular disease, increasing the likelihood of myocardial infarction, heart failure, arrhythmias and premature death. Approximately $60 \%$ of Australian adults are overweight or obese, with rates continuing to rise. While there is clearly a systemic influence on the heart in obesity, increasing evidence supports a direct action of pericardial adipose (combined epicardial and paracardial adipose depots) on the myocardium. Pericardial adipose content is known to increase in both obesity and aging, and adiposity has been linked with cardiac-specific diseases, particularly atrial fibrillation (AF).

Currently, the underlying cellular mechanisms linking pericardial adipose and cardiac pathologies are poorly understood. Studies to date have focused solely on structural remodelling of the myocardium, without assessing electro-mechanical influence. Adipocyte infiltration will undoubtedly cause a physical disruption to inter-myocyte conduction that may augment heterogeneity and arrhythmogenesis. Limited evidence also suggests pro-inflammatory/fibrotic mediators produced locally in the pericardial adipose exert paracrine actions on the myocardium to cause fibrosis. We have recently provided evidence to suggest that pericardial adiposederived estrogens may also be involved. We showed capacity of pericardial adipose to synthesise estrogens correlates with atrial arrhythmia vulnerability, and that exogenous estrogens increase atrial arrhythmias. We now extend these findings to identify the pericardial adipose-derived factors that lead to electro-mechanical dysfunction, and demonstrate that modulation of local cardiac sex steroid levels through aromatase action plays an important role in cardiac disease etiology.

