

Imaging cardiac function and response to infarct in the fetus

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In 2011-12, 590,000 Australians were living with coronary heart disease and it was the largest cause of death (20,000 in 2011). In 2012, there were ~68,200 acute myocardial infarction events and the incidence of hospital admissions almost doubled between 1993 and 2007. Although the public is better equipped to recognise heart attack and seek treatment quickly, the critical problem is that the heart of the adult human does not undergo repair, leaving the patient with a poor long-term heart health outcome. Consequently, current treatments mainly involve pharmacological agents or medical devices to pace or defibrillate the heart, which only control symptoms rather than effecting repair. Remarkably, the hearts of adult zebrafish, newborn mice and fetal sheep have the capacity for repair. These animal models indicate that there are critical molecular mechanisms that can be activated to induce myocardial repair at specific times in development. We have developed the ability to use MRI to visualise cardiac function. This will allow us to 'see' the cardiac response to changes in nutrient supply to the fetus or injury caused by infarction. Understanding these processes in the fetus, where the heart is capable of repair, will lead to improved treatment of congenital heart disease and low birth weight.