enzymatic digestion with protease and collagenase. The proteins from the cardiomyocytes were extracted using RIPA buffer and western blotting was used to examine the relative protein expression levels of phosphorylated and total ERK1/2 and Akt. Data were analysed using a one-way ANOVA with a Bonferroni post-test and are presented as mean±SEM.

Results We found that the survival signalling followed a similar profile to that of the vulnerability to I/R, with the P14 ratio of phosphorylated ERK1/2 to total ERK1/2 being the highest: 1.53 ±0.16, and lower at P21: 1.26±0.13, and lower still at P28 and adult rats, 0.71±0.11 and 0.73±0.1 respectively. The difference between P14 and P28 was statistically significant (P=0.0031), as was the difference between P14 and adult (P=0.0036).

Similarly, the P14 ratio of phosphorylated Akt to total Akt was also the highest: 0.97±0.04, with a lower ratio at P21: 0.74±0.19, and still lower ratios at P28 and adult: 0.57±0.04 and 0.73±0.08 respectively. Here, the difference between the P14 and P28 ratios was statistically significant, P=0.0071.

Discussion/Conclusions Our results suggest that changes in survival signalling (ERK1/2 and Akt) during development parallel the profile of recovery from I/R injury during development (with survival signalling and functional recovery both being highest at P14, and decreasing with age). Increased survival signalling could provide an explanation for the protection against I/R injury seen at P14.

REFERENCE

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255 **SURVIVAL SIGNALLING IN CARDIOMYOCYTES & VULNERABILITY TO ISCHAEMIA REPERFUSION INJURY**

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Introduction Vulnerability of the rat myocardium to the damaging effects of ischaemia/reperfusion (I/R) changes during postnatal development. The vulnerability profile follows a bell-shaped curve, with day 14 hearts achieving the most functional recovery, and adult hearts achieving the least functional recovery following similar ischaemic insults (Modi & Suleiman 2004). The underlying mechanism for these changes is not presently known. The damaging effects of I/R are mediated by ROS production and Ca^{2+} loading, both of which trigger the opening of the mitochondrial permeability transition pore (MPTP). Cellular signalling, including ERK1/2 and Akt, has been implicated in MPTP opening. Consequently, we investigated whether the expression of these proteins change in a similar way to the vulnerability profile to I/R.

Methods Hearts from P14, P21, P28 (P=post-natal day) and adult (250g) male Wistar rats were extracted and cannulated on a modified Langendorff apparatus. Cardiomyocytes were isolated using