Supplementary Information

Quantifying the area-at-risk of myocardial infarction *in-vivo* using arterial spin labeling cardiac magnetic resonance

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SUPPLEMENTARY TABLES

Supplementary Table 1

Table 1: IS/LV% values

IS/LV% from histological staining compared to LGE and ASL (2SD threshold). The number of histological slices used for analysis matches the

spatial extent of each CMR method

	Hist s	olog	y (7 5)		Т	2-mapp	bing	Hist s	olog	y (6	AS	L pe	erfusion	mapping	T1-mapping					
Control	64.3	±	6.1	60.3	±	5.8	(p = 0.43)	64.3	±	9.6	65.9	±	5.6	(p = 0.71)	59.3	±	9.2	(p = 0.40)		
IPC	59.8	±	7.9	50.7	±	5.4	(p = 0.02)	59.8	±	7.9	53.9	±	14.3	(p = 0.33)	47.3	±	13.8	(p = 0.04)		
Vehicle	65.4	±	7.0	65.4	±	10.1	(p = 0.32)	61.4	±	7.0	57.9	±	5.0	(p = 0.79)	58.3	±	13.8	(p = 0.90)		
CsA	58.0	±	12.6	62.8	±	13.1	(p = 0.32)	57.3	±	12.1	61.5	±	12.6	(p = 0.10)	47.2	±	16.6	(p = 0.12)		

Supplementary Table 2

Table 2: AAR/LV% values

AAR/LV% from histological staining compared to T_2 mapping, ASL (1SD threshold) and T_1 -mapping. The number of histological slices used for analysis matches the spatial extent of each CMR methods.

	Histology								olog	IÝ								
	(/	slice	es)			(6 5	slices	5)	AS	L pei	rfusion i	mapping						
Control	32.3	±	3.7	31.7	±	5.8	(p = 0.74)	31.5	±	4.8	24.8	±	11.2	(p = 0.28)				
IPC	16.8	±	3.0	17.8	±	4.9	(p = 0.77)	15.6	±	3.3	26.6	±	17.9	(p = 0.45)				
Vehicle	34.9	±	9.6	29.2	±	5.8	(p = 0.07)	29.0	±	8.3	25.1	±	11.8	(p = 0.14)				
	His	stolo	gy					Hi	stol	ogy								
	(7	slice	es)		-	F₂ -map	ping	(6	slic	es)	А	SL p	erfusior	n mapping		T	1 -mapp	bing
Control	64.3	±	6.1	60.3	±	5.8	(p = 0.43)	64.3	±	9.6	65.9	±	5.6	(p = 0.71)	59.3	±	9.2	(p = 0.40)
IPC	59.8	±	7.9	50.7	±	5.4	(p = 0.02)	59.8	±	7.9	53.9	±	14.3	(p = 0.33)	47.3	±	13.8	(p = 0.04)
Vehicle	65.4	±	7.0	65.4	±	10.1	(p = 0.32)	61.4	±	7.0	57.9	±	5.0	(p = 0.79)	58.3	±	13.8	(p = 0.90)
CsA	58.0	±	12.6	62.8	±	13.1	(p = 0.32)	57.3	±	12.1	61.5	±	12.6	(p = 0.10)	47.2	±	16.6	(p = 0.12)
CsA	21.9	±	4.5	19.2	±	6.9	(p = 0.44)	20.0	±	4.7	23.6	±	20.3	(p = 0.69)				

Supplementary Table 3

Table 3: Quantitative values from T₂ mapping, perfusion mapping and T₁ mapping

Quantitative normal and elevated or reduced values for T₂ mapping, perfusion mapping (1SD and 2SD thresholds) and T₁ mapping. p values from one-way ANOVA with Bonferroni correction are given to compare Control vs. IPC groups and Vehicle vs. CsA groups.

	T2 [ms]										Perfusion (1std threshold) [ml/g/min]									
			Nor	mal			Elevat	ed		Ν	lormal (1std)	Reduced (1std)							
Control	17.8	±	2.3		21.5	±	3.0		14.0	±	3.4		4.3	±	1.0					
IPC	19.6	±	3.4	(p > 0.9999)	26.8	±	5.4	(p = 0.07)	21.0	±	5.0	(p = 0.47)	5.4	±	2.8	(p > 0.9999)				
Vehicle	17.3	±	1.9		22.7	±	2.4		17.0	±	0.8		1.6	±	1.6					
CsA	17.7	±	1.9	(p > 0.9999)	23.8	±	2.5	(p = 0.95)	28.9	±	11.1	(p = 0.02)	7.8	±	4.4	(p = 0.41)				

	Perfusion (2 SD threshold) [ml/g/min]										T1 [s]									
		N	ormal	(2std)	Reduced (2std)						No	ormal	Elevated							
Control	8.2	±	2.3		2.3	±	0.9		1.7	±	0.1		1.9	±	0.1					
IPC	13.7	±	6.1	(p = 0.47)	0.6	±	3.6	(p > 0.9999)	1.7	±	0.3	(p > 0.9999)	2.0	±	0.3	(p > 0.9999)				
Vehicle	12.7	±	1.6		1.2	±	3.3		1.5	±	0.5		2.0	±	0.1					
CsA	19.8	±	7.6	(p = 0.13)	2.3	±	4.4	(p > 0.9999)	1.6	±	0.4	(p > 0.9999)	2.0	±	0.1	(p > 0.9999)				

SUPPLEMENTARY FIGURES

Supplementary Figure 1: Treatment groups for murine in vivo IRI

Mice were randomized to control or treatment groups for each study.

A. IPC study: control group received standard IRI protocol of 15 minutes stabilization, 30 minutes index ischemia and 72 hours reperfusion. IPC group received 5 minutes stabilization, one cycle of IPC consisting of 5 minutes ischemia and 5 minutes reperfusion, followed by 30 minutes index ischemia and 72 hours reperfusion.

B. CsA study: both groups received standard IRI protocol of 15 minutes stabilization, 30 minutes index ischemia and 72 hours reperfusion. Control group received a matched volume dose of vehicle (cremophor / ethanol-94%). CsA group received CsA 10mg/kg. Vehicle and CsA were administered as single intravenous dose delivered 5 minutes prior to the onset of reperfusion.

