

### **POSTER PRESENTATION**

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# 0738. Mortality is associated with early tachycardia and cardiac troponin release in a fluid-resuscitated rat model of sepsis

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#### Introduction

Tachycardia and high troponin levels prognosticate for poor outcomes in human sepsis [1]. Reducing cardiac stress with beta-blockade has been proposed as an important therapeutic strategy as high catecholamine levels are injurious [2]. We have characterized a 72h fluid-resuscitated rat model of faecal peritonitis where prognostication can be made with high sensitivity and specificity at 6h from heart rate and stroke volume [3].

#### **Objectives**

To determine whether non-survival is associated with early changes in troponin release and circulating catecholamine levels.

#### **Methods**

Male Wistar rats (325  $\pm$  15g) underwent insertion of tunneled carotid arterial and jugular venous lines under isoflurane anaesthesia, followed by immediate i.p. injection of  $4\mu l/g$  faecal slurry. Control animals were treated identically but without i.p. injection of slurry. Once awake, attachment to a swivel-tether system allowed

animals to move freely and access food and water *ad libitum*. Fluid resuscitation (50:50 mixture of 5% dextrose/Hartmann's; 10ml/kg/h) was commenced at 2h. At 6h, echocardiography was used to measure heart rate and stroke volume. Animals were observed until 72h to assess survival. In a second experiment septic animals underwent echocardiography at 6h followed by sacrifice and blood and tissue sampling. We here report plasma catecholamine and troponin T levels (measured by ELISA) in predicted survivors and non-survivors, and sham-operated controls.

#### Results

Septic animals (n = 16) had a mortality rate of 56%, with death occurring between 18-36h. A heart rate cut point of 460/min measured at 6h prognosticated 3-day survival with sensitivity of 0.88 and specificity of 0.92. Clinical features of illness at this timepoint were however mild. Table 1 shows significant differences in haemodynamics and troponin levels between predicted survivors and non-survivors. Catecholamine levels, while elevated over non-septic controls, were similar.

Table 1

390 ± 21	Predicted survival (n=6) 442 ± 17	Predicted non-survival (n=6) 488 ± 18*
	442 ± 17	488 ± 18*
2.40 . 0.02		
$0.40 \pm 0.03$	$0.25 \pm 0.02$	$0.18 \pm 0.02*$
8.56 ± 0.42	9.44 ± 0.23	10.3 ± 0.18
1.60 ± 0.25	3.21 ± 0.23	2.98 ± 0.27
171 ± 24	168 ± 15	311 ± 47*
8	8.56 ± 0.42 1.60 ± 0.25	$3.56 \pm 0.42$ $9.44 \pm 0.23$ $1.60 \pm 0.25$ $3.21 \pm 0.23$

Data shown as median  $\pm$  SE; \* p<0.05 ANOVA

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#### **Conclusions**

An association was seen between eventual non-survival and tachycardia, low stroke volume and myocardial injury (denoted by high troponin) at 6h after induction of sepsis. The impact of modulating cardiac stress on outcome merits further study.

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#### References

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