Carotid sinus nerve electrical stimulation in *conscious* rats attenuates systemic inflammation via chemoreceptor activation

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Supplementary Figure S1

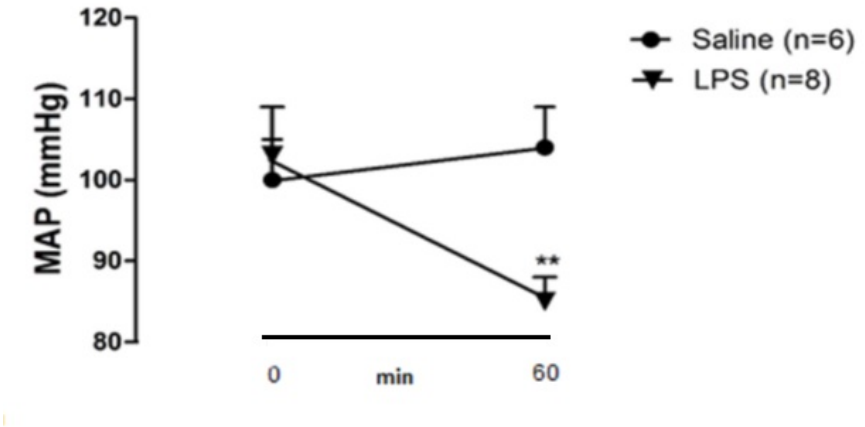

Figure S1. Mean arterial pressure (MAP) before (zero) and 60 minutes after intravenous administration of saline or Escherichia coli lipopolysaccharide (LPS). Data are expressed as the mean ± standard error of the mean; **p=0.01 compared to saline. In parentheses the number of animals.

Supplementary Figure S2

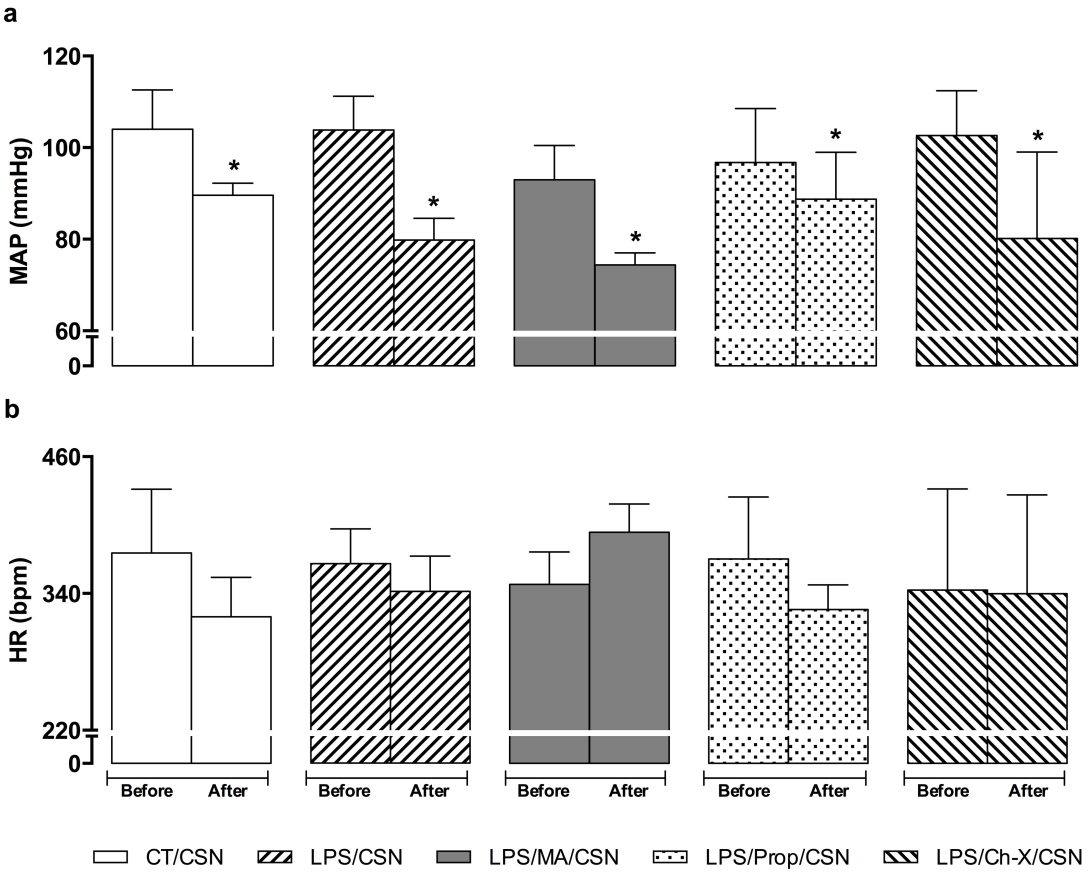

Figure S2. Hemodynamic responses [(MAP: mean arterial pressure; panel a) and HR: heart rate; panel b)] displaying the efficacy of electrical activation of the carotid sinus nerve in promoting a prompt fall in MAP in the different groups. **CT/CSN**: Control plus Carotid Sinus Nerve stimulation; n=5; **LPS/CSN**: Escherichia coli lipopolysaccharide (LPS) plus Carotid Sinus Nerve stimulation; n=7; **LPS/MA/CSN**: LPS plus Methylatropine administration and Carotid Sinus Nerve stimulation; n=5; **LPS/Prop/CSN**: LPS plus Propranolol administration and Carotid Sinus Nerve stimulation; n=5; **LPS/Ch-X/CSN**: LPS plus chemoreceptor denervation and Carotid Sinus Nerve stimulation; n=7. *Stimulation factor $p<0.0001$.

Supplementary Figure S3

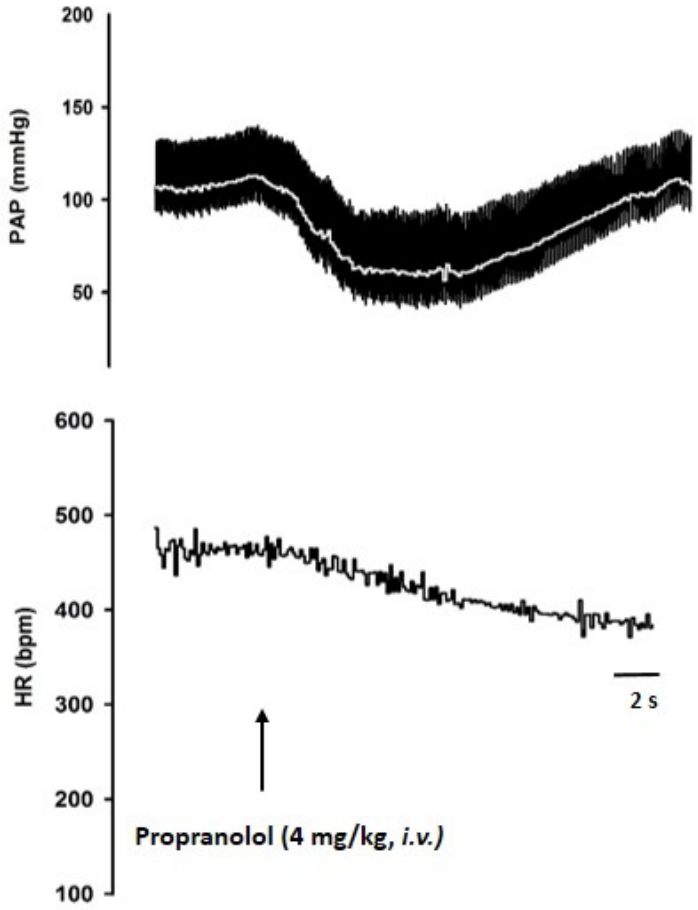

Figure S3. Tracings of pulsatile arterial pressure (PAP) [white line represents mean arterial pressure] and heart rate (HR) before and right after intravenous administration of propranolol in a conscious rat.