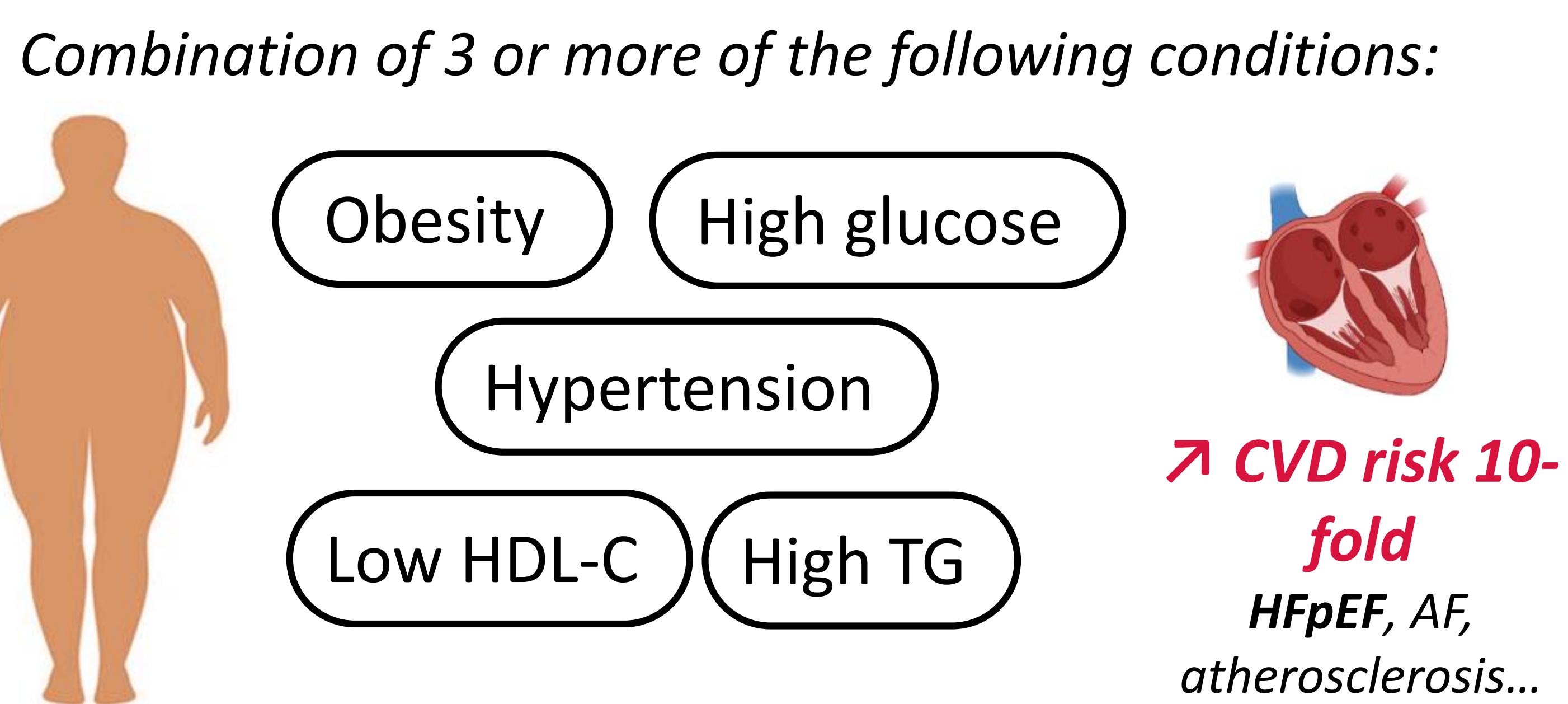


Structural and inflammatory changes in the myocardium of a porcine model of metabolic syndrome and HFpEF

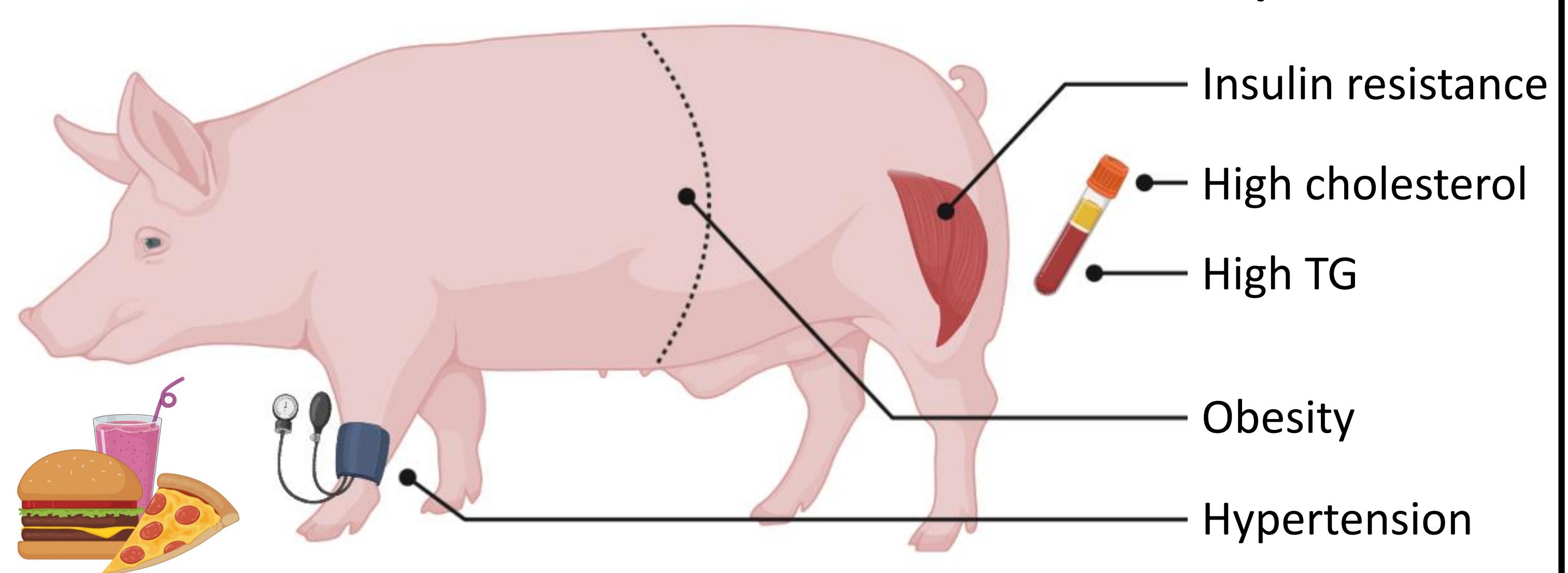
Gaston L. Cluzel^{1,2,*}, Florence M. Herisson^{1,2}, Maria Antonia Llopis-Grimalt^{1,2}, Siddhant Gawli^{1,2}, Noel M. Caplice^{1,2}

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Metabolic syndrome



Porcine model of MetS and HFpEF

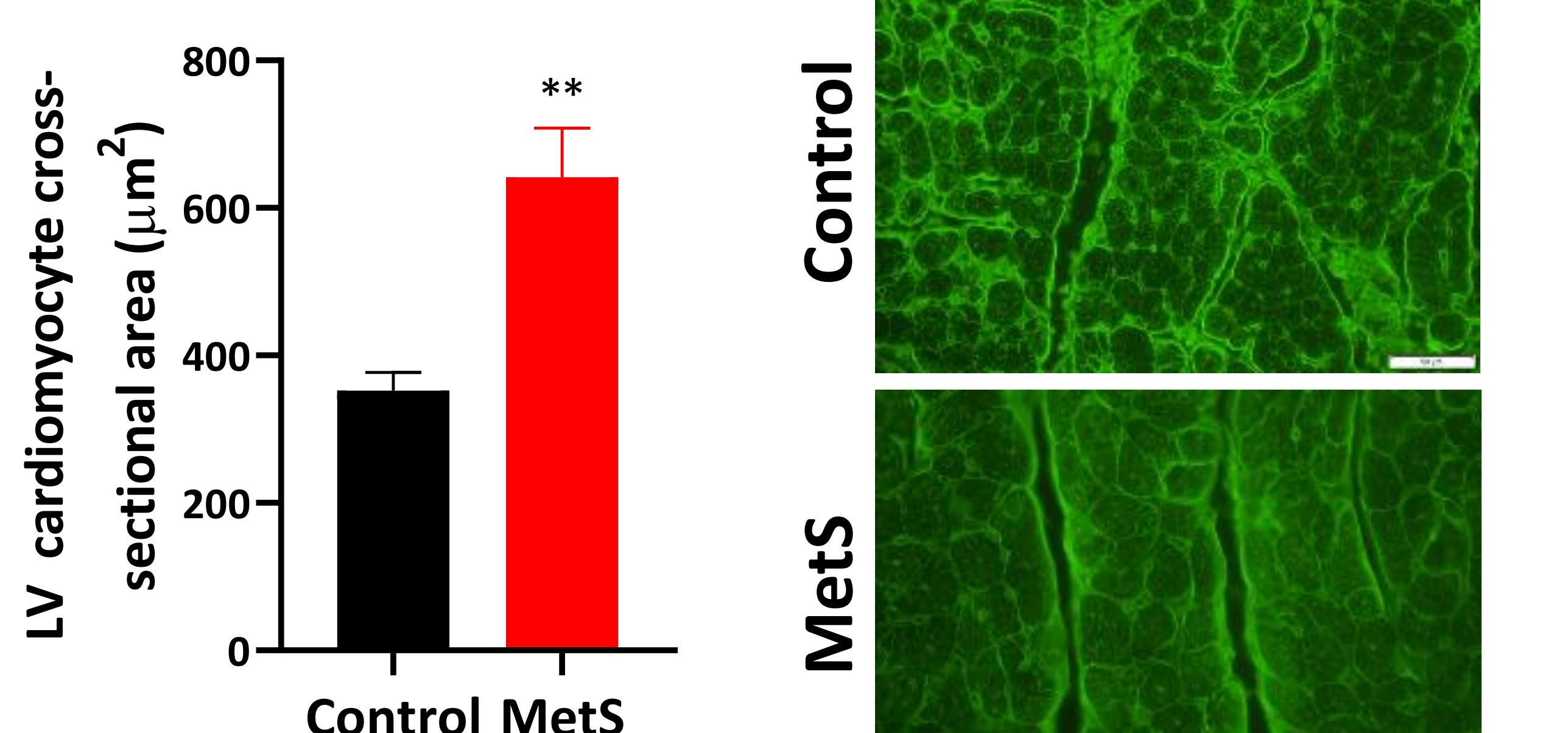
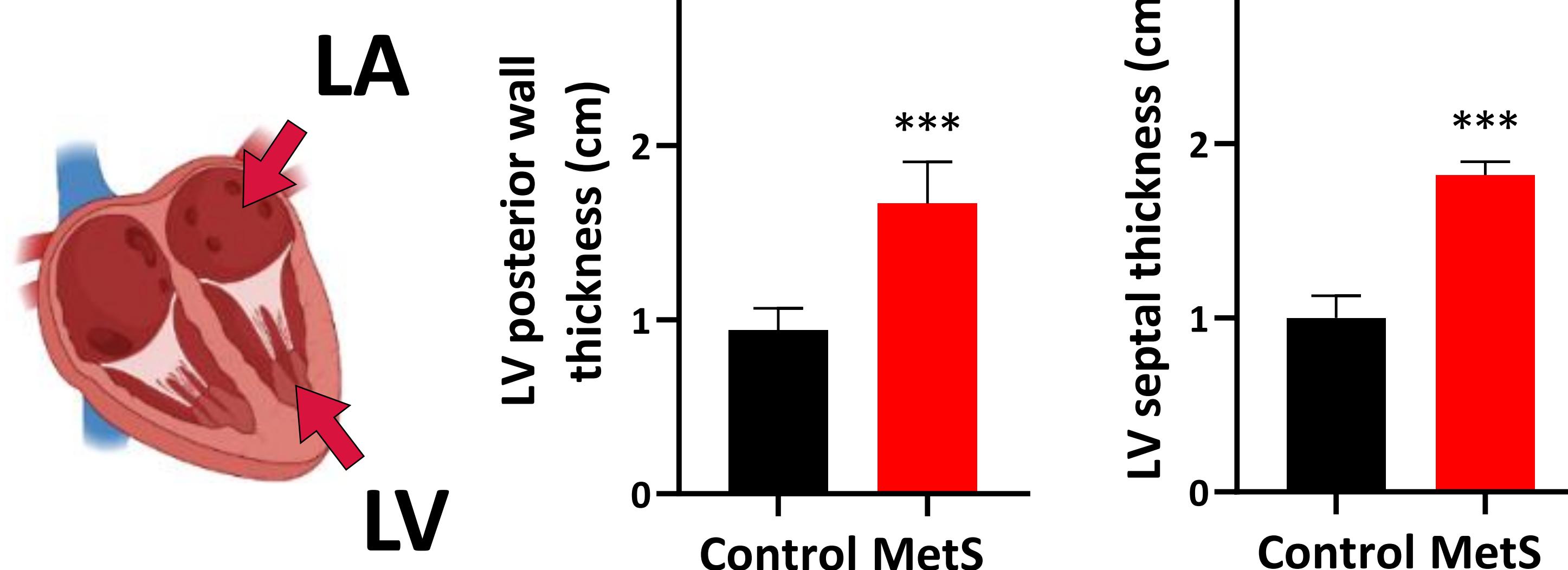
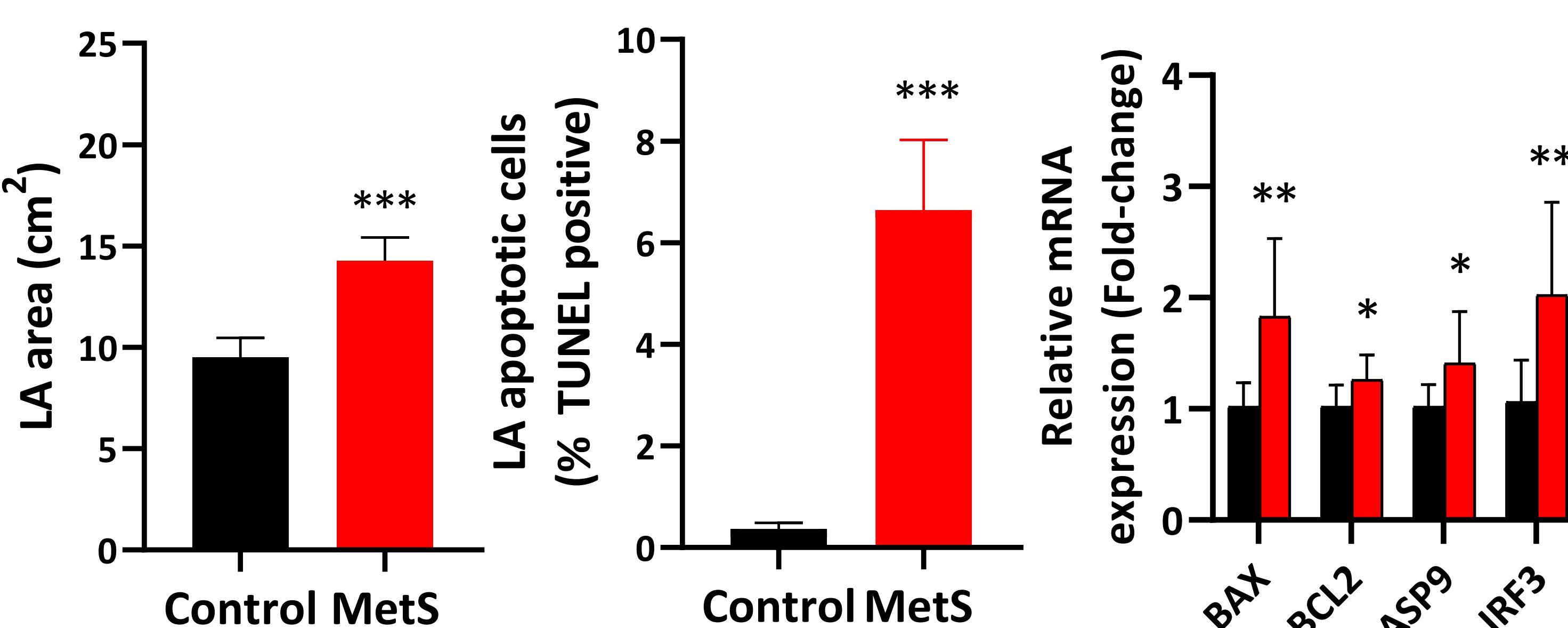


Western diet + steroid-induced hypertension for 12 weeks

→ MetS with multiple organ dysfunction

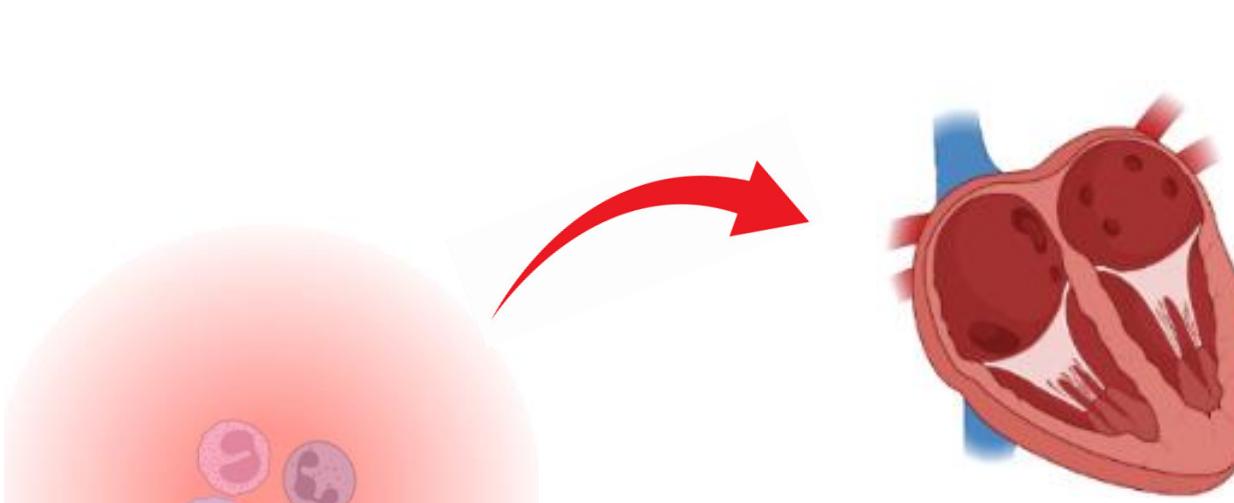
Altered gut microbiota, intestinal permeability, systemic inflammation, NASH, HFpEF

1. Cardiac structural and cellular alterations

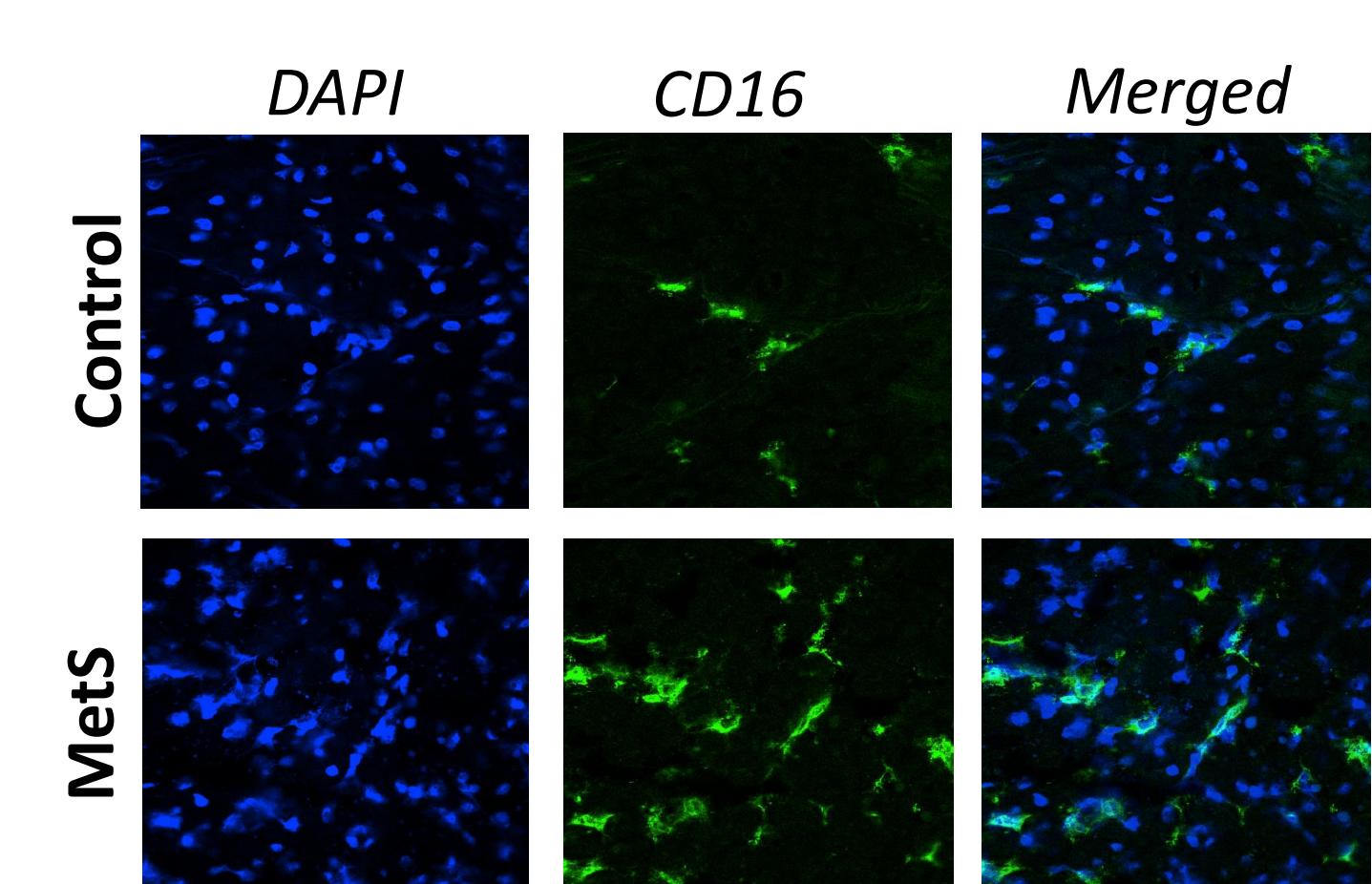
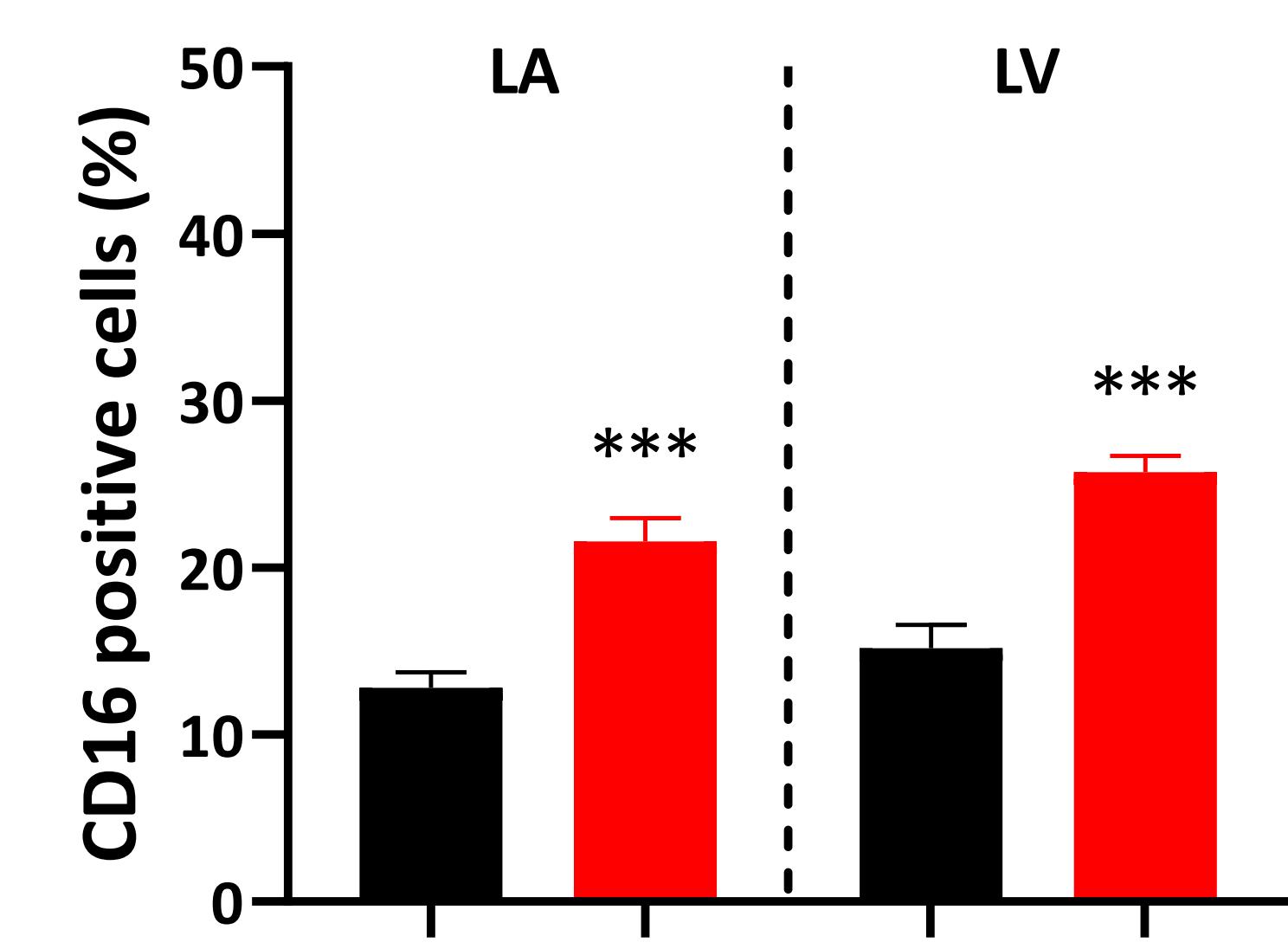
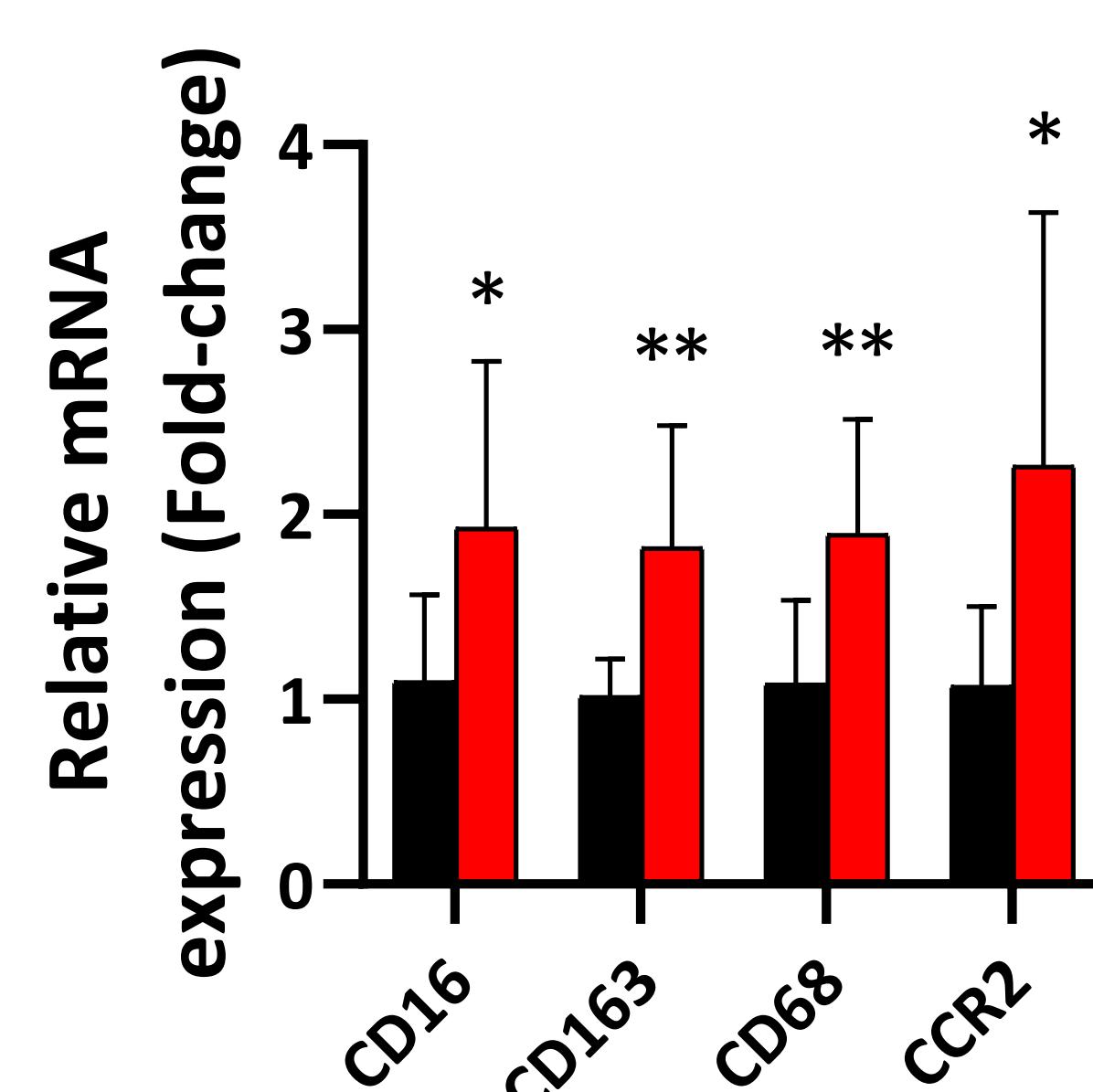


- LA dilatation and apoptosis
- LV concentric hypertrophy

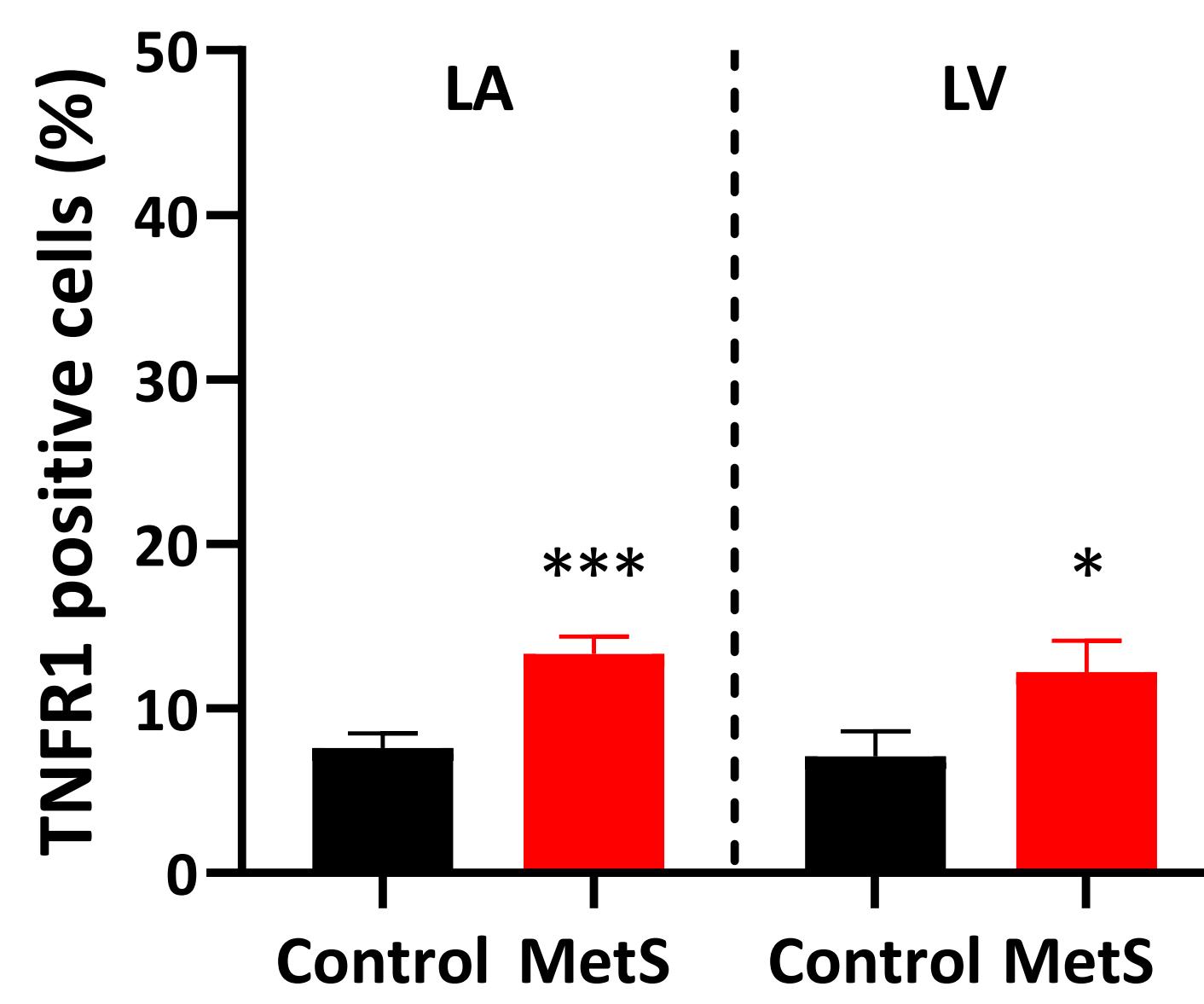
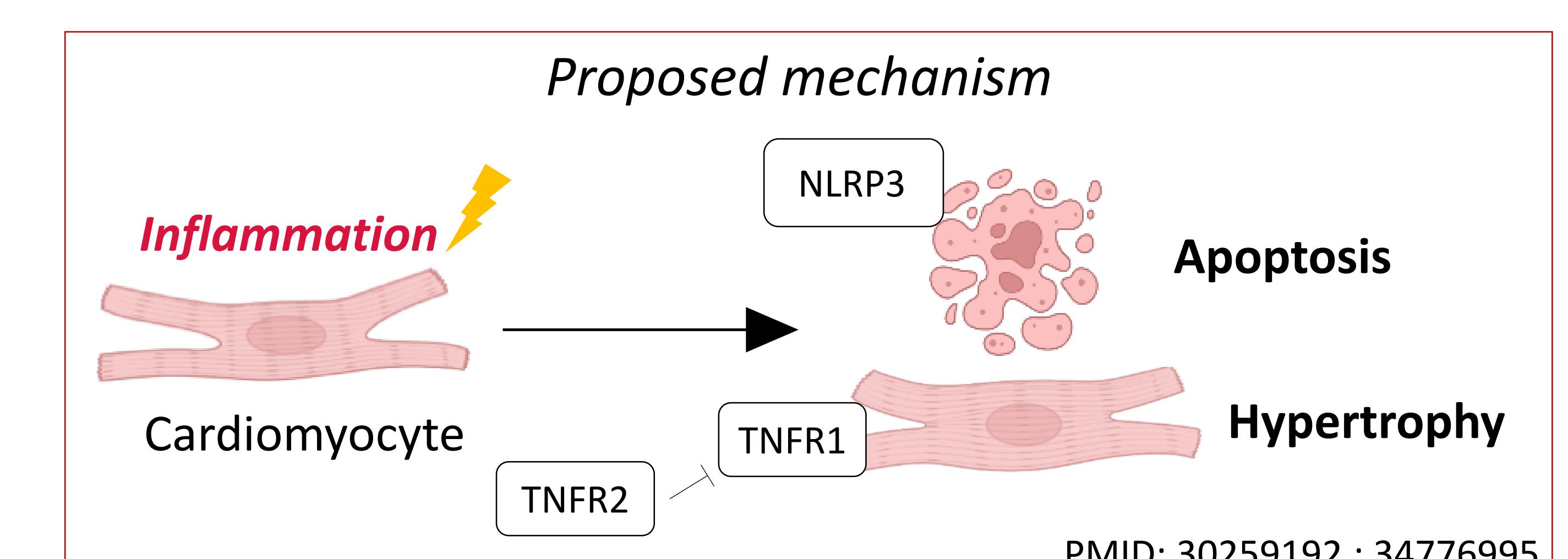
2. Cardiac macrophage infiltration



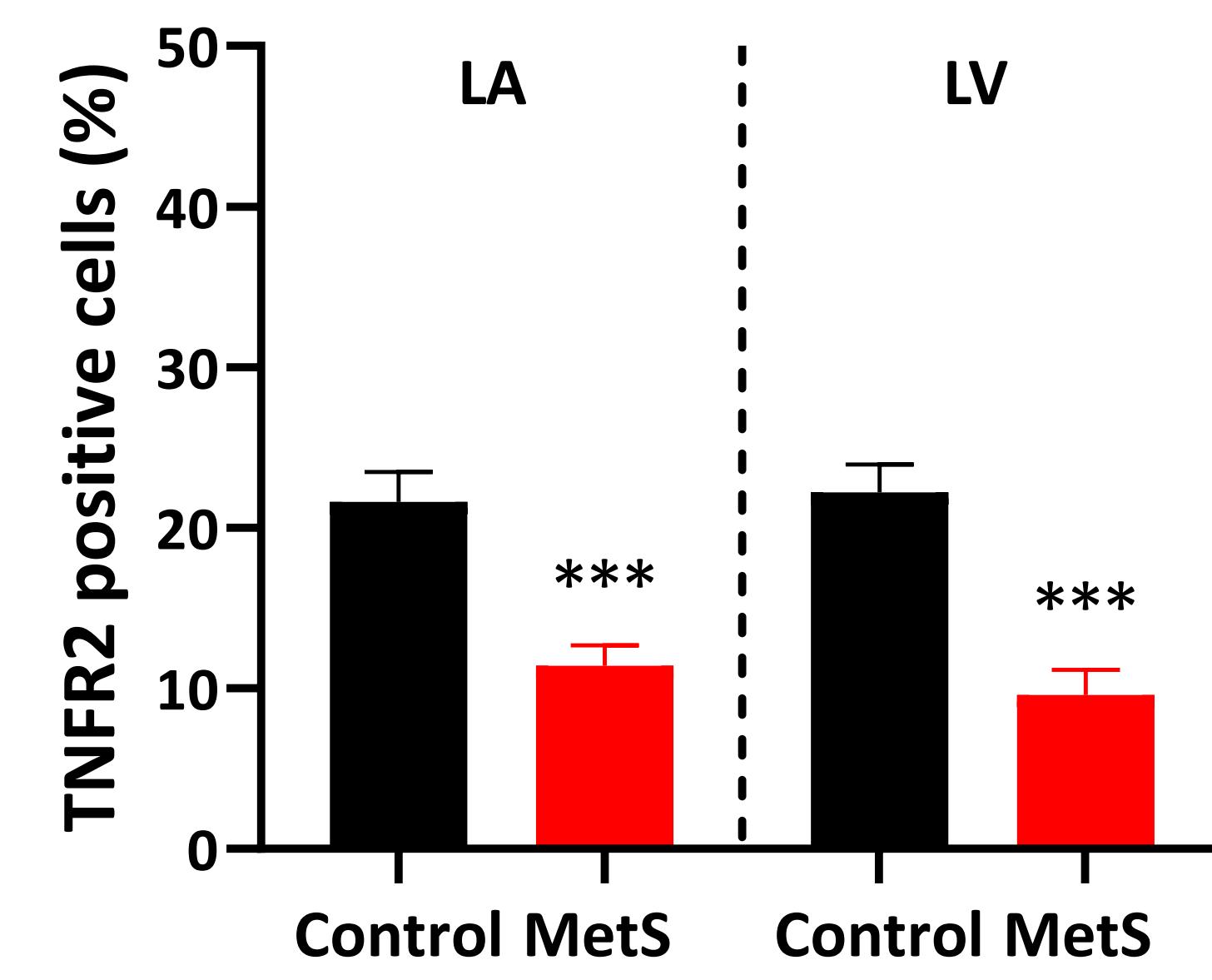
CD16⁺ macrophage



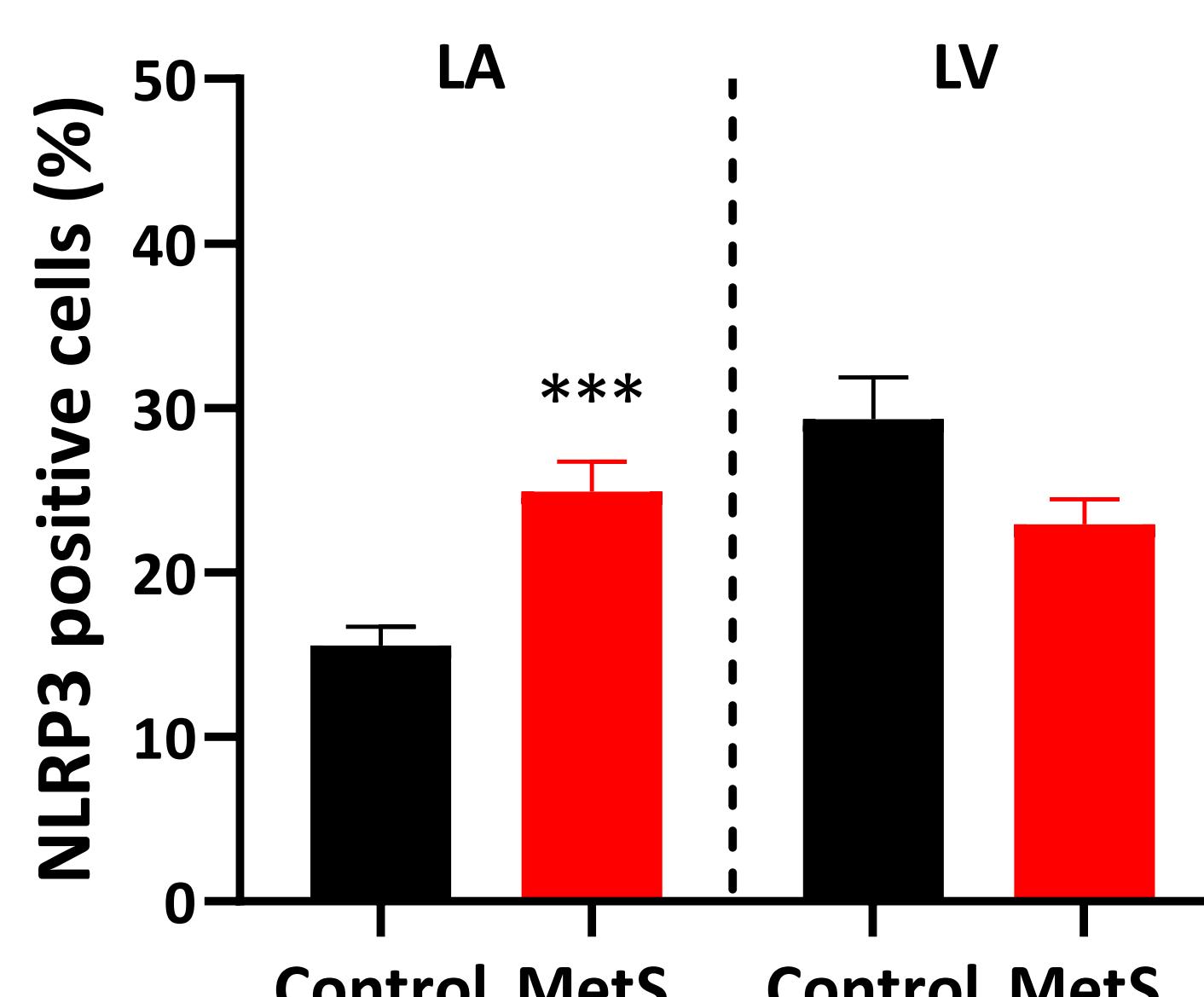
3. Inflammatory pathway activation



LA LV

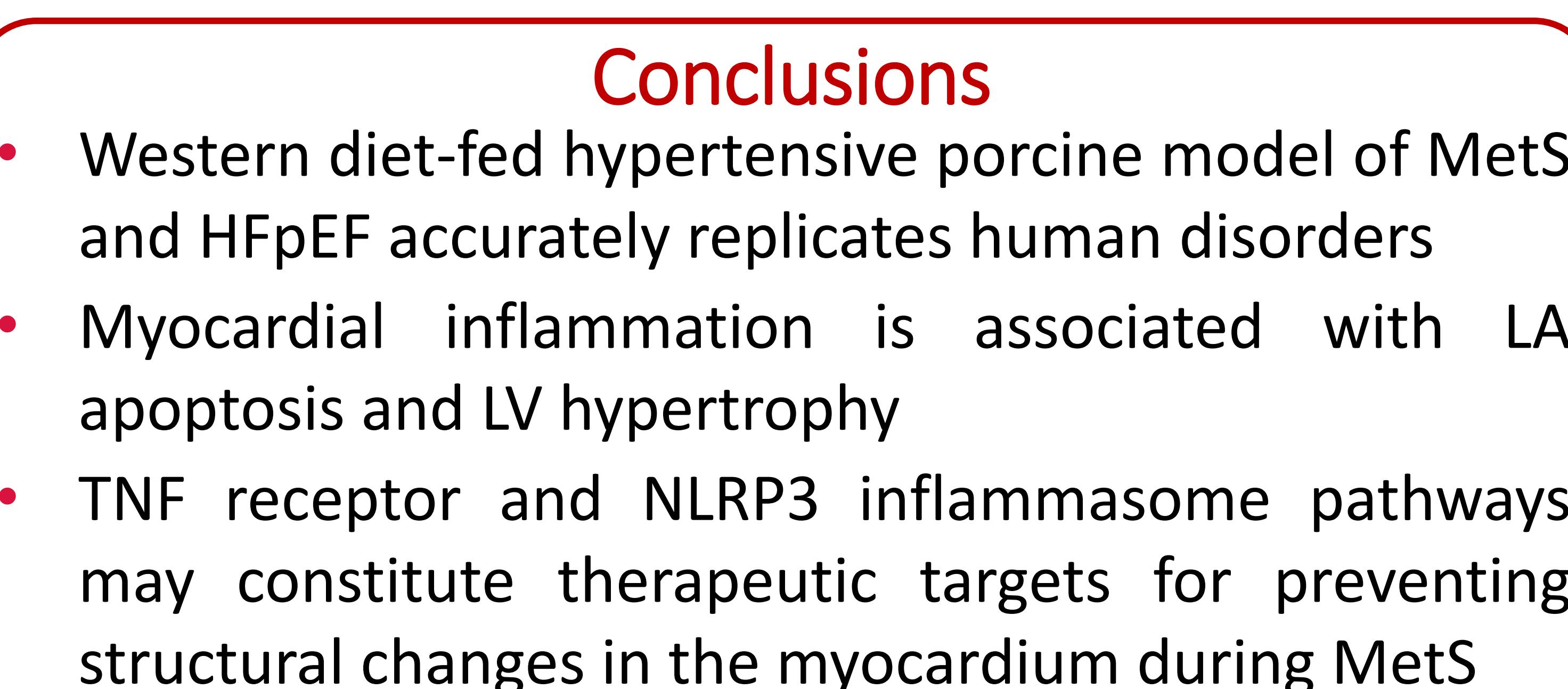


LA LV



LA LV

LA LV



Conclusions

- Western diet-fed hypertensive porcine model of MetS and HFpEF accurately replicates human disorders
- Myocardial inflammation is associated with LA apoptosis and LV hypertrophy
- TNF receptor and NLRP3 inflammasome pathways may constitute therapeutic targets for preventing structural changes in the myocardium during MetS