

Online Supplementary Data for

Genetically induced moderate inhibition of 20S proteasomes in cardiomyocytes facilitates heart failure in mice during systolic overload

Mark J. Ranek^{*†}, PhD; Hanqiao Zheng^{*†}, MD, PhD; Wei Huang^{*†}, MD, PhD; Asangi R. Kumarapeli^{*†}, MD, PhD; Jie Li^{*}, MD, PhD; Jinbao Liu^{*†}, MD, PhD; and Xuejun Wang^{*}, MD, PhD

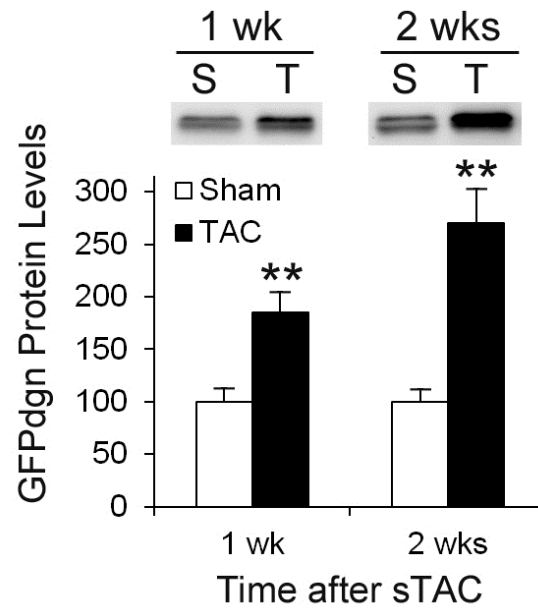
^{*}Division of Basic Biomedical Sciences, Sanford School of Medicine of the University of South Dakota, Vermillion, SD 57069 USA; [†]State Key Lab of Respiratory Disease, Protein Modification and Degradation Lab, Department of Pathophysiology, Guangzhou Medical University, Guangdong 510182, China

Brief title: Cardiac proteasome in systolic overload

[†]These authors contributed equally.

Current address: Division of Cardiology, Johns Hopkins Medical Institutions, Baltimore, MD, USA (M. J. R.); Harvard University School of Public Health, Cambridge, MA, USA (H. Z.); Department of Cardiology, Affiliated Drum Tower Hospital, Nanjing University School of Medicine, Nanjing, Jiangsu, China (W.H.); Department of Pathology, Summa Health System Summa Akron City Hospital, 525 East Market St., Akron, Ohio (A. R. K.); Vascular Biology Center, Medical College of Georgia at Georgia Reagent University, Augusta, GA, USA (J. L.)

Address for correspondence: Dr. Xuejun Wang, Sanford School of Medicine of the University of South Dakota, 414 E. Clark Street, Vermillion, SD 57069, USA Tel. 605-677-5132, Fax. 605-677-6381, Email: xuejun.wang@usd.edu



Online Supplementary Figure 1. Western blot analyses of myocardial GFPdgn protein levels in mice at 1 and 2 weeks after severe transverse aortic constriction (sTAC). GFPdgn transgenic mice were subject to sTAC at ~10 weeks of age. The magnitude of systolic overload was controlled using a 29G needle as the TAC template, which generates a pressure gradient of ~60mmHg. For each time point, n=4 mice/group. **p<0.01 vs. Sham; unpaired Student's *t*-test. T=TAC; S= sham.