## Adiponectin deficiency: a model of pulmonary hypertension associated with pulmonary vascular disease

Ross Summer<sup>1</sup>, Christopher A. Fiack<sup>1</sup>, Yasumasa Ikeda<sup>2</sup>, Kaori Sato<sup>2</sup>, Daniel Dwyer<sup>1</sup>,
Noriyuki Ouchi<sup>2</sup>, Alan Fine<sup>1</sup>, Harrison W. Farber<sup>1</sup>, Kenneth Walsh<sup>2</sup>

<sup>1</sup> The Pulmonary Center, R-304 Boston University School of Medicine 80 East Concord St. Boston, MA 02118

 Whitaker Cardiovascular Institute/Molecular Cardiology Boston University School of Medicine 715 Albany Street, W611 Boston, MA 02118

\*Correspondence: Ross Summer

Running title: Adiponectin and pulmonary hypertension Supplemental table and figures

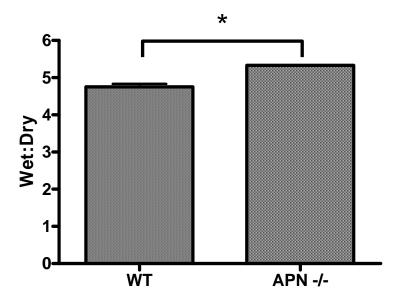
This work was supported by National Institute of Health Grants K08 HL077138, AG15052, HL-59215, and Gilead Sciences Research Scholars Program in Pulmonary Arterial Hypertension.

## Supplemental table 1:

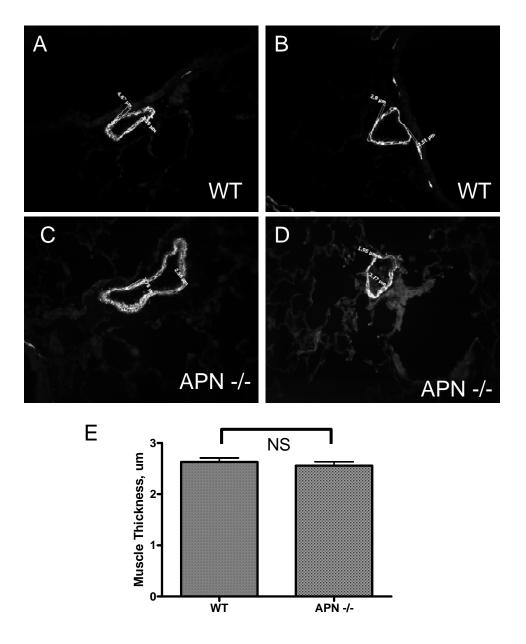
## Right ventricle cardiac echocardiographic measurements

	WT 3 M	APN -/- 3M
Number of mice	5	5
RVDd (mm)	1.73+/-0.05	1.708 +/- 0.05
PA ACT (msec)	18±1.1	20±0.9

RVDd Right ventricular dimensions, PA ACT Pulmonary artery acceleration time



Supplemental figure 1: Wet to dry ratio was slightly, but significantly increased in lungs of APN deficient mice at 3 months of age.



Supplemental figure 2: Vascular smooth muscle thickness was not statistically different in wild-type, WT (A, B) and adiponectin (APN) deficient (C, D) lungs at 1 year of age.

E) Summary of results from measuring smooth muscle thickness in lungs of WT and APN deficient mice at 1 year of age (50 blood vessels of similar size were measured in each group).