## **Legend to Supplementary Figures:**

**Supp. Fig. 1:** Serotonin 100  $\mu$ M increased O<sub>2</sub> in human aorta and mitral valve. The increase in superoxide after incubation with serotonin was attenuated by an MAO A and B inhibitor (TC=tranylcypromine 10uM) and an MAO-A inhibitor (Clorg = Clorgyline 1  $\mu$ M). Vehicle (Veh) was PBS at pH 7.4. (\*=P<0.05 vs control, #=P<0.05 vs 5-HT only-Veh; n=6-8 mitral valves, n= 4 aortas).

**Supp Fig. 2:** Serotonin 100 μM (veh) significantly increased superoxide in the tricuspid valve and pulmonary artery when compared with controls. A cyclooxygenase inhibitor (indo) did not attenuate the increase in superoxide induced by serotonin (Veh). Inhibition of MAO with transplayment 1 μM (TC) attenuated the response to serotonin (Veh). Vehicle (Veh) was PBS at pH 7.4. (\*=P<0.01 vs control; #=P<0.01 vs 5HT only-Veh, &=P<0.01 vs Indo; n=4 valves, n= 6 samples of pulmonary artery).

Supp. Fig. 3: NADPH 100  $\mu$ M increased superoxide levels in heart valves and pulmonary artery homogenates (Veh) vs control tissue. NAD(P)H oxidase inhibition with DPI significantly attenuated the increase in superoxide induced by NADPH vs Veh. MAO inhibitors (Clorg=Clorgyline 1 $\mu$ M; TC=tranylcypromine 10 $\mu$ M) did not attenuate the increase in NADPH stimulated superoxide. Vehicle (Veh) was PBS (\*=P<0.01 vs control; #=P<0.05 vs Veh; &=P<0.01 vs Clorg/TC. n=4 pulmonary valves, n= 7 tricuspid valves, n=4 pulmonary arteries).