

SUPPLEMENTARY MATERIALS FOR:

**DUAL OXIDASE-INDUCED SUSTAINED GENERATION OF HYDROGEN
PEROXIDE CONTRIBUTES TO PHARMACOLOGICAL ASCORBATE-INDUCED
CYTOTOXICITY**

by

Adrienne R. Gibson¹, Brianne R. O’Leary², Juan Du², Ehab H. Sarsour³, Amanda L. Kalen¹,
Brett A. Wagner¹, Jeffrey M Stolwijk¹, Kelly Falls-Hubert¹, Matthew S. Alexander², Rory S.
Carroll², Douglas R. Spitz¹, Garry R. Buettner¹, Prabhat G. Goswami¹, and Joseph J. Cullen^{1,2}

¹From the Free Radical and Radiation Biology Division, Department of Radiation Oncology

²Department of Surgery, The University of Iowa Carver College of Medicine, Iowa City, Iowa

³Kansas City University of Medicine and Biosciences³, Kansas City, MO

*Corresponding Author

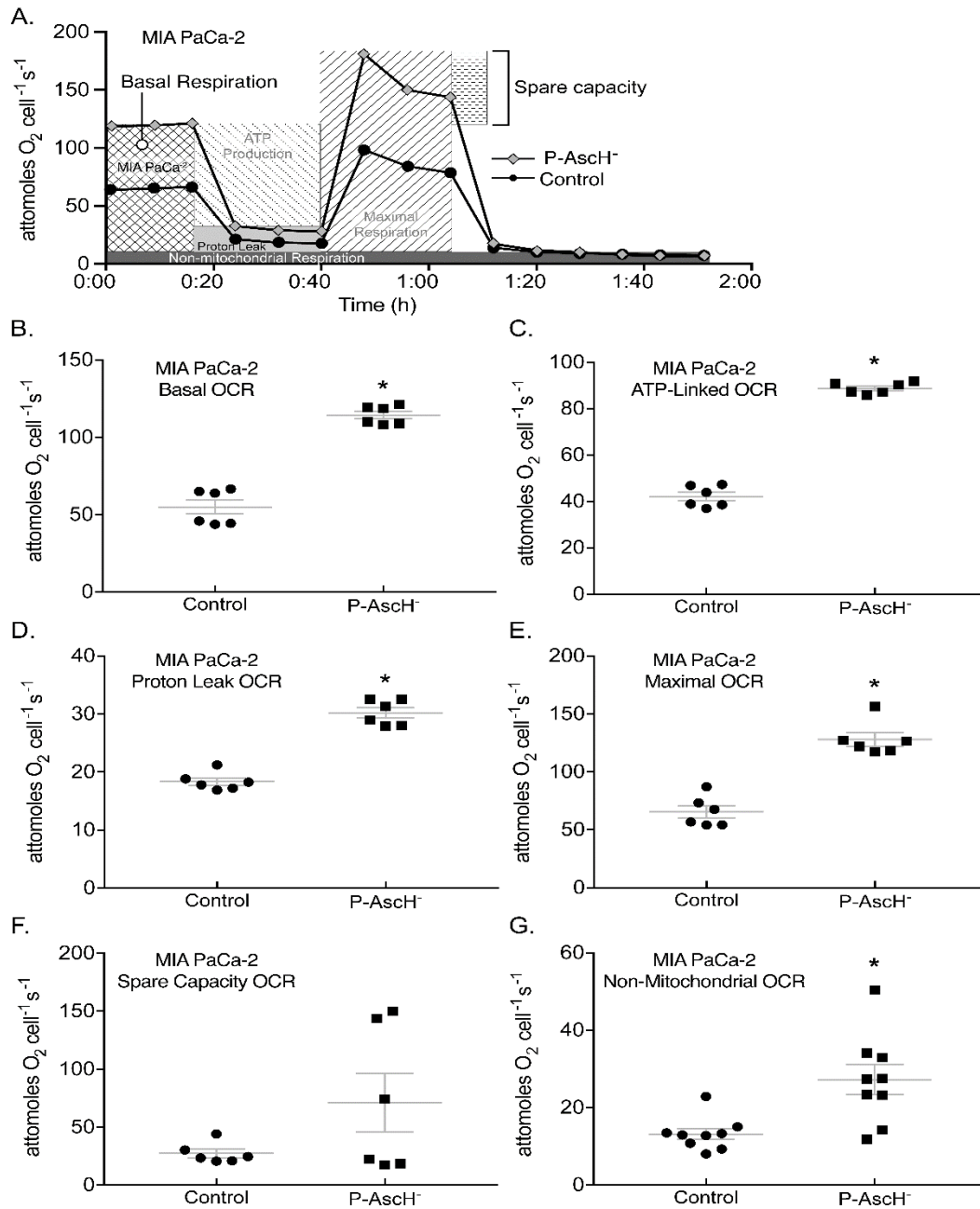
Joseph J. Cullen, M.D.

1528 JCP, Univ. of Iowa Hospitals and Clinics

Iowa City, IA 52242. Joseph-cullen@uiowa.edu

W: (319) 353-8297, Fax: (319) 356-8378.

Supplemental Figure 1



Supplemental Figure 1. Basal oxygen consumption rate is sustained after exposure to P-AscH⁻ in MIA PaCa-2 cells.

A. MIA PaCa-2 cells treated with 1 mM P-AscH⁻ demonstrate alterations in the mitochondrial stress test curves using the Seahorse XF96 analyzer 48 h after treatment.

B-G. MIA PaCa-2 cells demonstrate an increase in: basal respiration; C. ATP production; D. proton leak; E. maximal respiration; F. spare capacity and G. non-mitochondrial respiration 48 h after treatment (Means ± SEM, *n* = 6 C-G and *n* = 9 H, *p* < 0.05 vs. control, 2-tailed student's *t*-test).