

Supporting Information

Oxidative stress increases M₁dG, a major peroxidation DNA adduct, in mitochondrial DNA

Orrette R. Wauchope¹, Michelle M. Mitchener², William N. Beavers², James J. Galligan¹, Jeannie M. Camarillo¹, William D. Sanders¹, Philip J. Kingsley¹, Ha-Na Shim^{3,4}, Thomas Blackwell^{3,4}, Thong Luong^{3,4}, Mark deCaestecker⁵, Joshua P. Fessel^{3,4}, and Lawrence J. Marnett^{*,1,2,4}

A. B. Hancock, Jr., Memorial Laboratory for Cancer Research, Departments of ¹Biochemistry, ²Chemistry, ³Cancer Biology, ⁴Pharmacology, Vanderbilt Institute of Chemical Biology, Center in Molecular Toxicology, Vanderbilt-Ingram Cancer Center, Vanderbilt University School of Medicine, ⁵Departments of Cell and Developmental Biology, Surgery and Medicine.

Supplemental Figures:

Supplemental Figure 1. M₁dG levels in mtDNA in RKO cells after lysis and DNA isolation in the presence of 10 mM TEMPO.

Supplemental Figure 2. (A) Representative LC-MS (SRM) chromatogram of the [¹³C, ¹⁵N₂]-M₁dG internal standard co-eluting with M₁dG from a sample. **(B)** Representative LC-MS (SRM) chromatogram of the [¹⁵N₅]-6-oxo-M₁dG internal standard. No 6-oxo-M₁dG was detected in the digested mtDNA samples. **(C)** Representative CID of the M₁dG peak from a representative sample from RKO cells treated with rotenone (100 nM) showing its fragmentation pattern. **(D)** Representative CID of a synthetic M₁dG standard. In both cases, fragmentation of the M₁dG peak at 304.1 m/z with CE set 50 eV. Fragment at 188.1 corresponds to the loss of deoxyribose.

Supplemental Figure 3. M₁dG levels in mtDNA in RKO cells after treatment with antimycin A (10 μM), mitoTEMPO (10 μM), TEMPOL (μM) for 24 h.

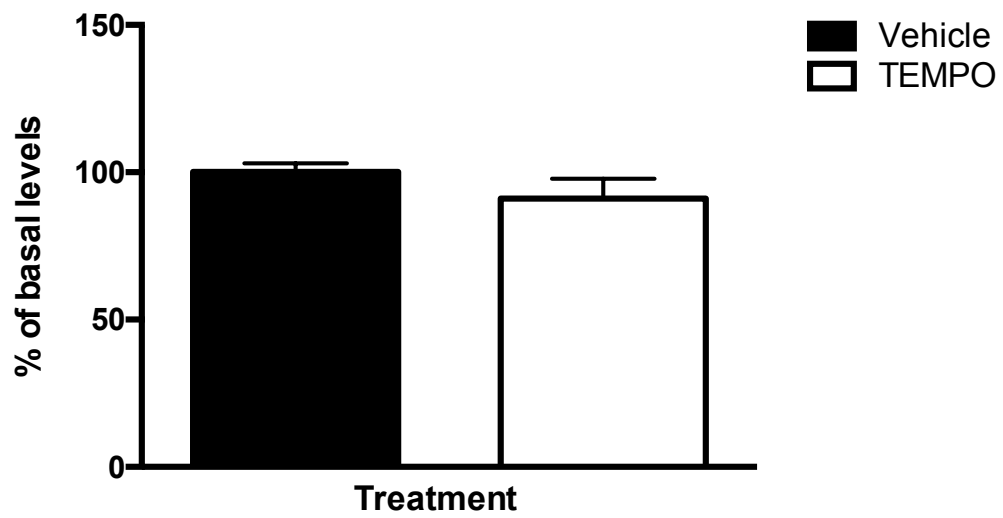
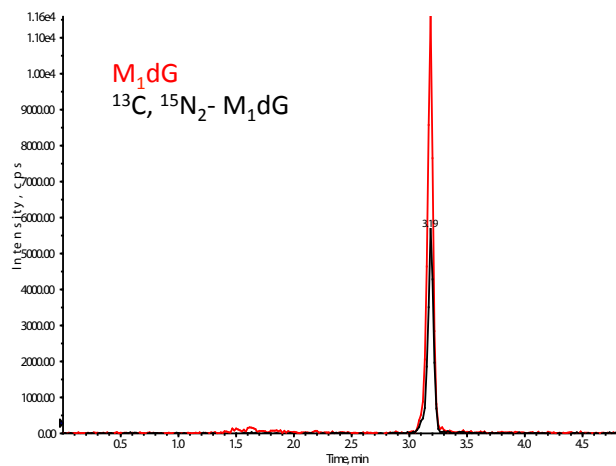
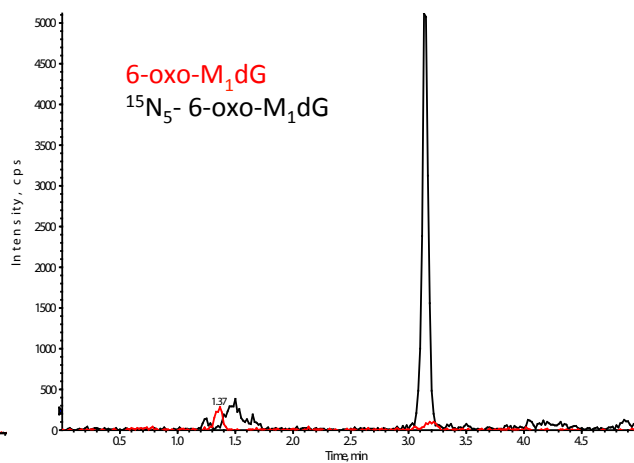
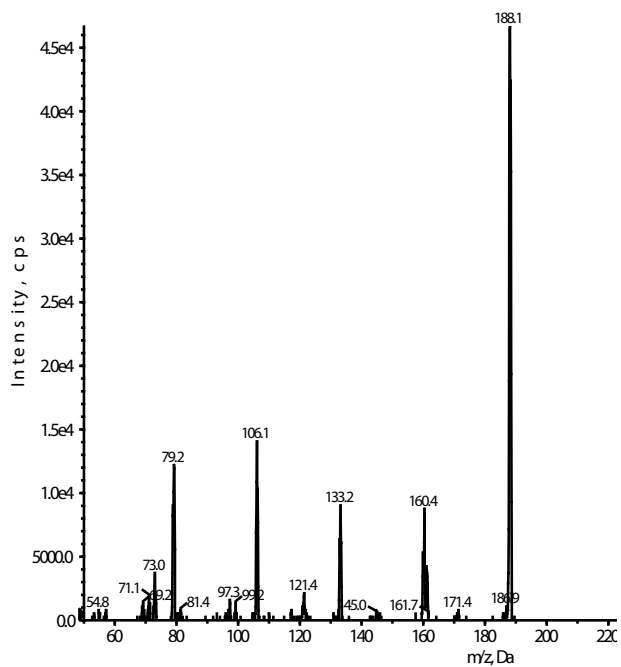
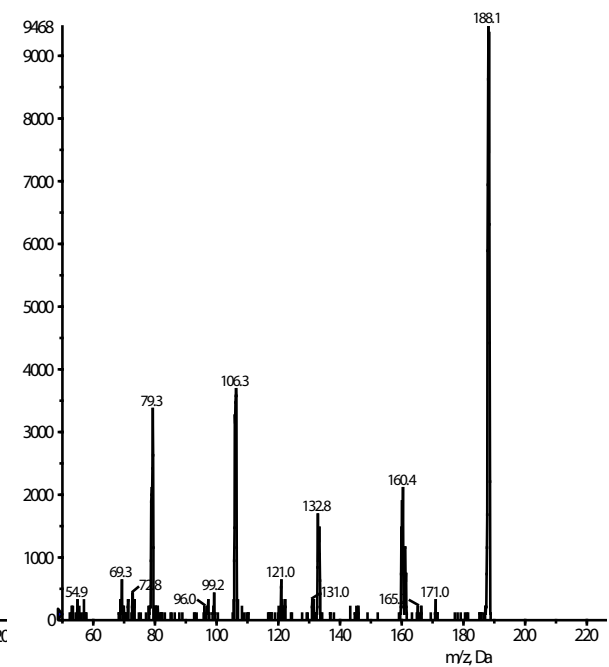


Figure S1.

A.**B.****C.****D.****Figure S2.**

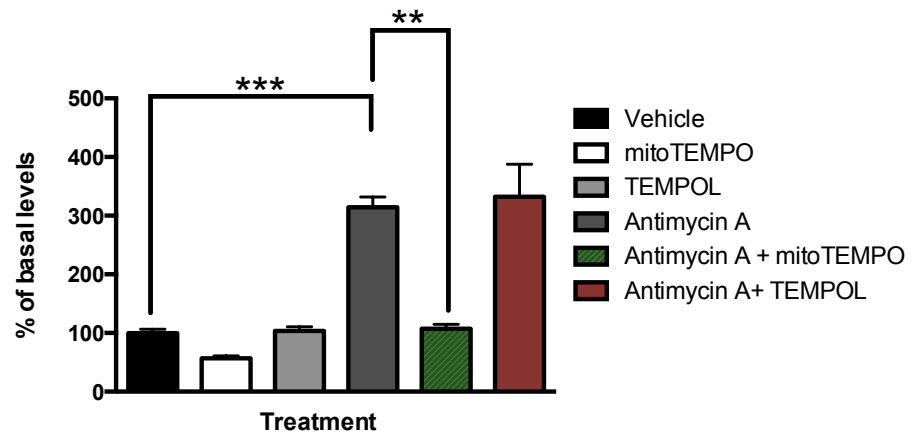


Figure S3.