Supplementary information for

Lysophosphatidic Acid Receptor 3 Promotes Mitochondrial Homeostasis against Oxidative Stress: Potential Therapeutic Approaches for Hutchinson–Gilford Progeria Syndrome

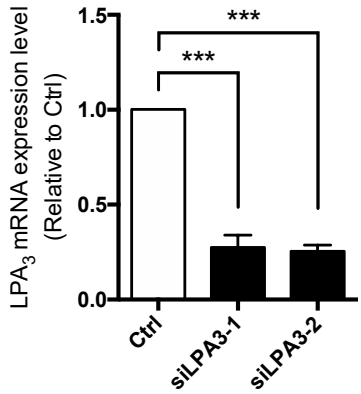

Figure S1. The knockdown efficiency of LPA_3 siRNA. LPA_3 mRNA expression level was measured using real-time PCR and relative to siRNA control. The expression level of GAPDH was used as a loading control. The bar graph was generated from three independent analyses. ***, $P < 0.001$.

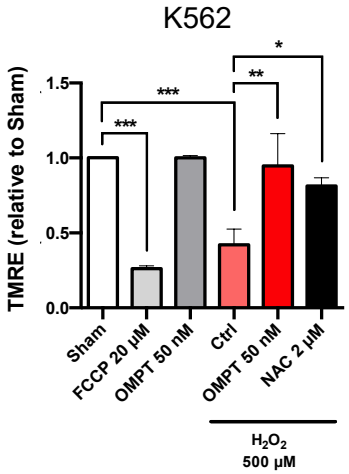

Figure S2. LPA_3 protected against oxidative stress-induced mitochondrial ΔΨm loss in K562 cells**.** K562 cells showed decreased mitochondria ΔΨm under 500 μM of H_2O_2 treatment. 50 nM of OMPT and 2 μM of NAC protected cells against H_2O_2-induced mitochondrial ΔΨm loss. FCCP was used as a positive control for loss of mitochondrial ΔΨm. The bar graphs were generated from three independent analyses. *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$.

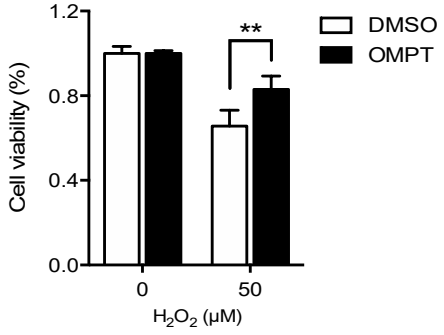

Figure S3. LPA_3 improved cell viability from H_2O_2 induced oxidative stress. HSF cells were subjected to a cell proliferation assay under 50 μM of H_2O_2 for 72 hr. 50 nM of OMPT showed protective effects against H_2O_2. The bar graphs were generated from three independent analyses. **, $P < 0.01$.