Supplemantary fig. 1
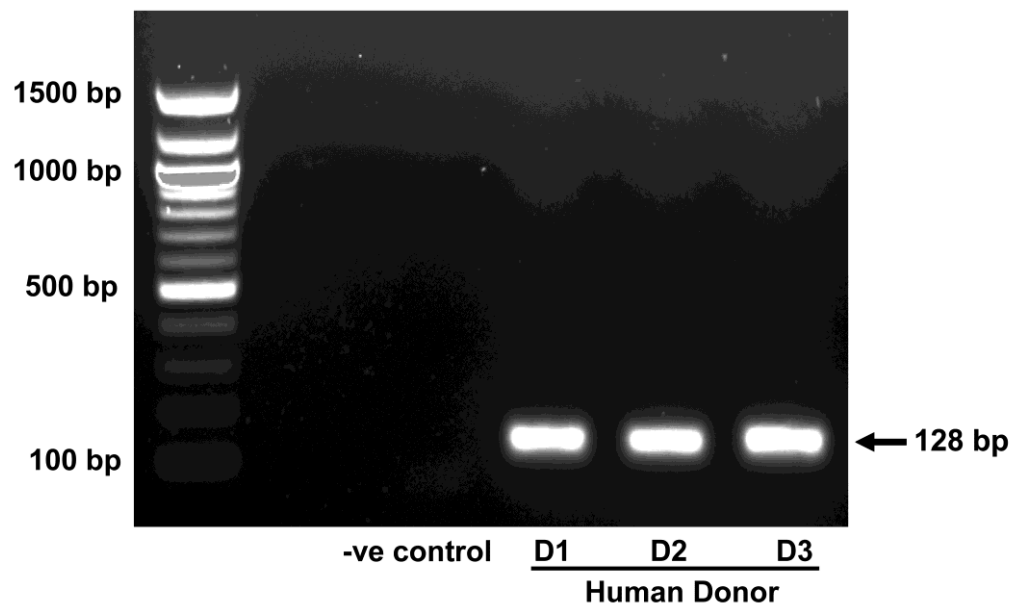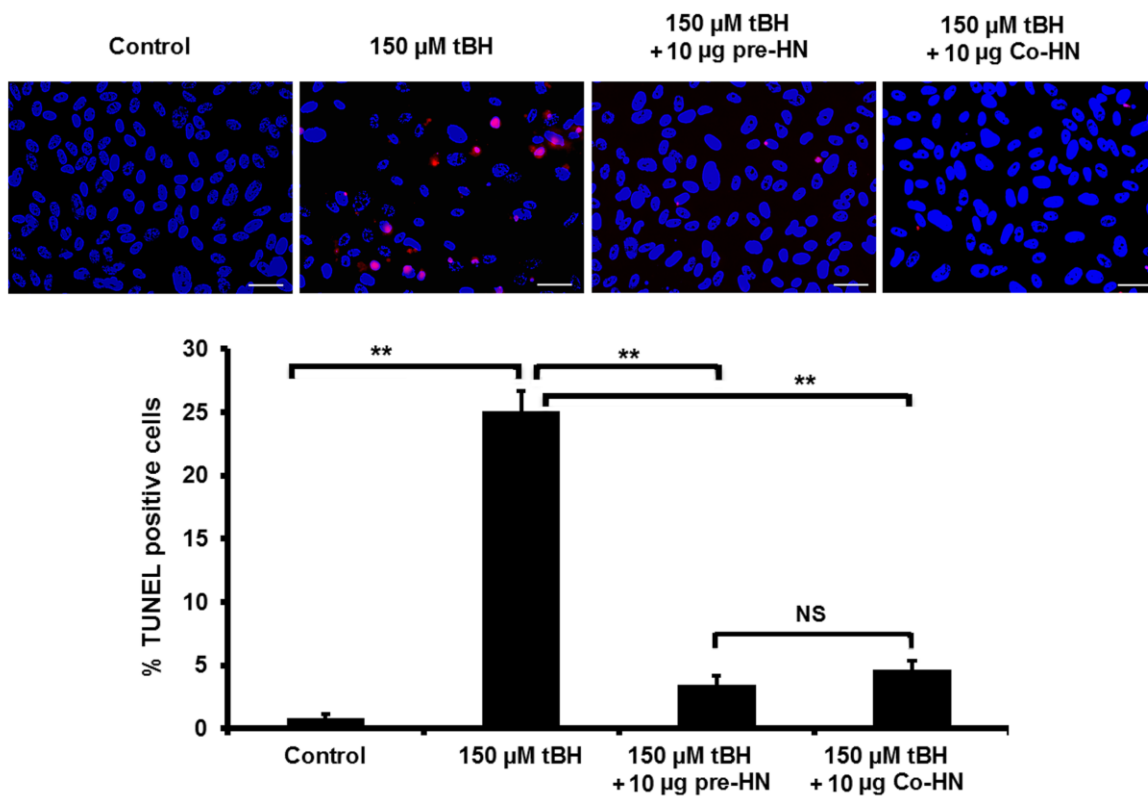

Supplementary Fig. 1. **RPE cells express mitochondrial transcripts for HN**.

PCR showing mitochondrial HN transcripts from three independent samples derived from 3 donors (D1-D3). Primer pairs used are stated in the results section.
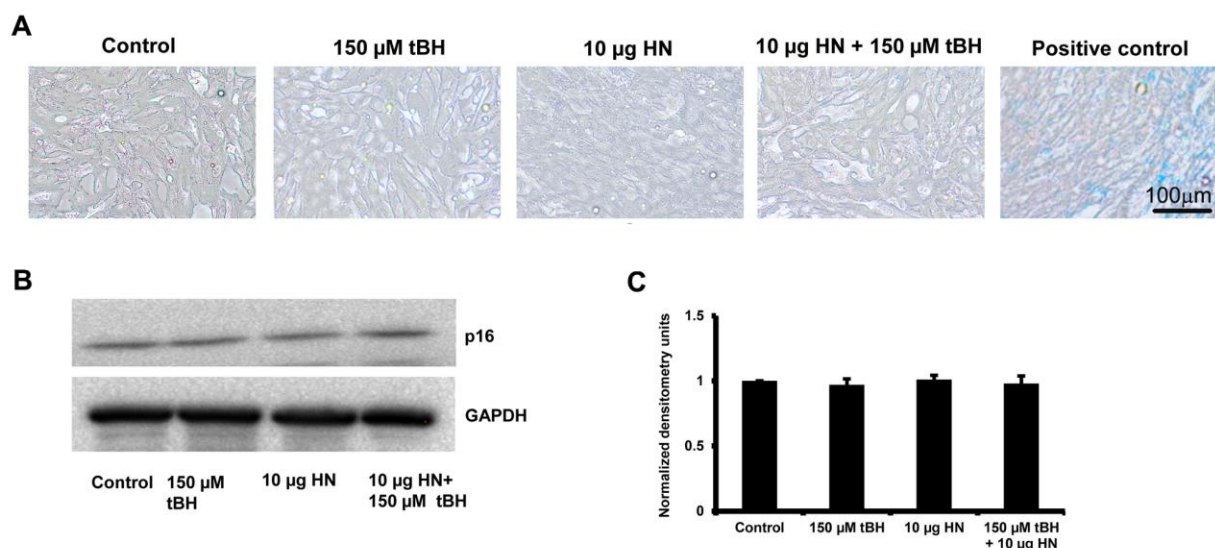
Supplementary Fig. 2

Supplementary Fig. 2. Pre- or co-treatment or RPE with HN similarly protect from oxidant stress.

Effect of pre- treatment and co-treatment of HN on RPE cell survival. Non-polarized RPE cells were either pre-treated overnight or co-treated with 10µg HN and 150 µM tBH for 24h. TUNEL staining is shown in A and B shows quantification of percentage of TUNEL positive cells from three experiments. **$P < 0.01$. NS: not significant. Scale bar = 10 µm.

Supplementary Figure 3

A

Control | 150 µM tBH | 10 µg HN | 10 µg HN + 150 µM tBH | Positive control

100µm

B

p16

GAPDH

Control | 150 µM tBH | 10 µg HN | 10 µg HN+ 150 µM tBH

C

Normalized densitometry units

Control | 150 µM tBH | 10 µg HN | 150 µM tBH + 10 µg HN

Supplementary Fig. 3.

Short-term oxidative stress does not induce senescence in RPE cells

RPE cells were treated with 150 µM tBH or 150 µM tBH plus 10 µg/ml HN for 24 h and processed for SA-β-Gal staining or P16^{INK4a} immunoblot. As a positive control, RPE cells were treated with 30 µM tBH for 2 h, allowed to recover in fresh medium with 10% fetal bovine serum for 22 h. The procedure was repeated and a complete experiment comprised of five sequential tBH treatments.[6] (**A, B**) No significant change in the number of SA-β-Gal positive cells nor of p16^{INK4a} protein regulation was found with 150 µM tBH treatment for 24 h. Sequential tBH (30 µM) treatment induced senescence in RPE cells (**A**). Scale bar = 100 µm.