Supplementary Figure S1. 3D movie demonstrating the normal mitochondrial network in pIRES vector control cells stained with TMRM.

Supplementary Figure S2. 3D movie demonstrating the normal mitochondrial network in pIRES PINK1-wt expressing cells stained with TMRM.

Supplementary Figure S3. 3D movie demonstrating the normal mitochondrial network in pIRES PINK1-F104A expressing cells stained with TMRM.

Supplementary Figure S4. 3D movie demonstrating loss of the mitochondrial network and presence of aggregated mitochondria in pIRES PINK1-P95A expressing cells stained with TMRM.

Supplementary Figure S5. 3D movie demonstrating the intermediate mitochondrial phenotype induced by PINK1-C92F expression in cells stained with TMRM.

Supplementary Figure S6. Endogneous PINK1 interacts with PARL in SHSY5Y cells.

PINK1 peptides identified through LC-MS-MS and emPAI scored mass spectrometry. PINK1 peptides were absent from the peptide competition control as were PARL peptides to confirm efficient inhibition of binding.

Supplementary Figure S7. PINK1 antibodies fail to detect endogenous protein in PARL MEFs.

PARL KO and WT MEFs were assessed by WB for alterations in PINK1 cleavage at the endogenous level. The PINK1 Novus 494 antibody, utilised recently in publications to detect endogenous PINK1 protein, failed to detect the endogenous protein but recognised the over-expressed PINK1-HA control (lane 3). Western blots were re-probed with an anti-HA antibody to confirm the over-expressed PINK1-HA protein.