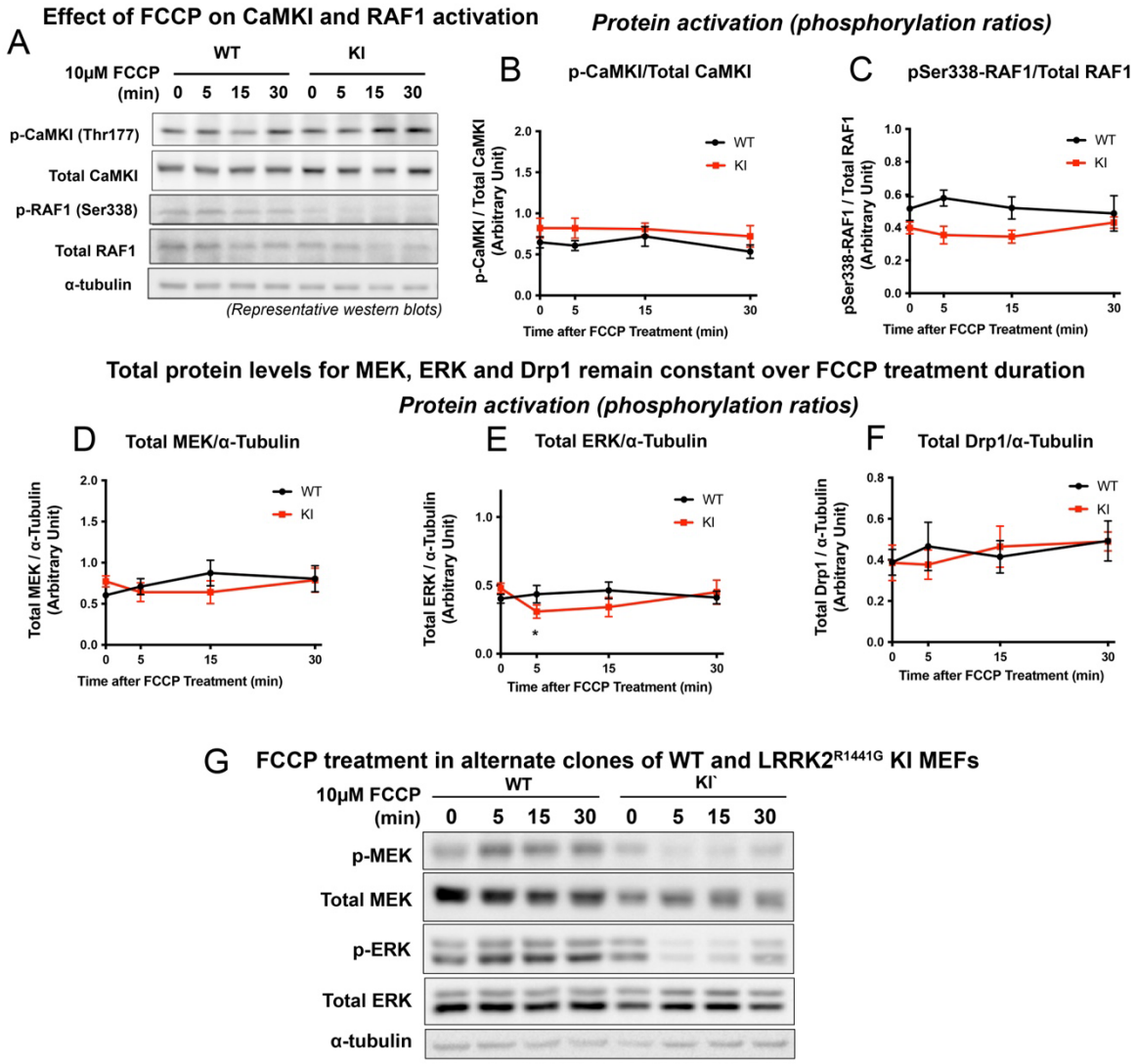
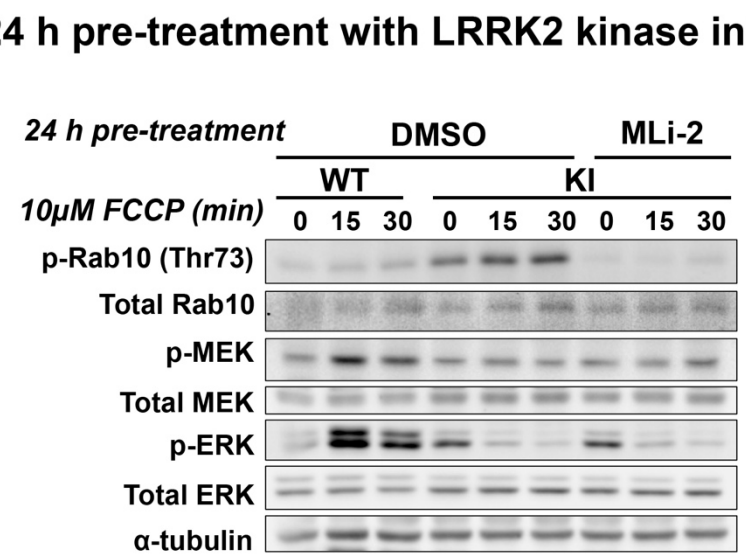
Supplementary Information



Supplementary Fig. S1

(**A**) Representative immunoblots of WT and KI MEFs treated with FCCP for p-CaMKI (Thr177), total CaMKI, p-RAF1 (Ser338) and total RAF1, and α-tubulin as a loading control. (**B, C**) FCCP did not induce phosphorylation of CaMKI and RAF1 in both cell lines. (**D-F**) Quantified total protein levels for MEK, ERK and Drp1 remain constant over the duration of FCCP treatment in both cell lines. (**G**) Representative western blots of alternate clones of WT and LRRK2^{R1441G} KI MEFs treated with FCCP (10 μM) for 0, 5, 15 and 30 min.



Supplementary Fig. S2

Representative immunoblots of KI MEFs pre-treated with either LRRK2 kinase inhibitor, MLi-2 (30 nM), or vehicle, DMSO, for 24 h prior to FCCP treatment (10 μM) for 0, 15 and 30 min (N=1). WT MEFs were pre-treated with DMSO as a positive control, followed by FCCP treatment of same duration. LRRK2 kinase inhibition by MLi-2 was confirmed by the reduction in p-Rab10 (Thr73) level. Both DMSO and MLi-2-treated KI MEFs lacked phosphorylation of MEK and ERK, unlike WT MEFs which showed clear phosphorylation of MEK and ERK at 15 and 30 min post FCCP treatment.