Supplementary Figure 3. *Dose-response reactions of the CF10 cells treated with pathway inhibitors.* To determine the range in which inhibitors influenced intracellular ROS production in response to serum deprivation (if the inhibitor does exert an effect) without significantly reducing viability, dose-response curves were carried out using the DCFDA assay followed by determination of cellular viability at 24 hours post-treatment by MTS metabolism. Plates show the following pathway inhibitors; clathrin internalisation inhibitor Tyrphostin A-23 [T-A23] (**A, B**); Dynamin inhibitor Dynasore (**C, D**); PP2 against Src family kinases (**E, F**); DPI against NADPH oxidase (**G, H**); MEK1/2 inhibitor U0126 (**I, J**); ERK inhibitor 3-(2-Aminoethyl)-5-((4-ethoxyphenyl)methylene)-2,4-thiazolidinedione hydrochloride (**K, L**); p38 inhibitor SB203580 (**M, N**); PI3K inhibitor wortmannin (**O, P**); Balfilomycin against vacuolar ATPase (**Q, R**); Lysosomal inhibitor chloroquine (**S, T**); DMSO carrier molecule (**U, V**). DCFDA assays are shown in plates **A, C, E, G, I, K, M, O, Q, S** and **U**, with MTS assay results shown in **B, D, F, H, J, L, N, P, R, T** and **V**. MTS assays are shown normalised to the serum deprived control value and the 10% (v/v) serum condition is not included on the MTS viability plots as serum deprivation alters cellular metabolism resulting in the serum deprived condition appearing falsely viable. Significant results as determined by one-way ANOVA with Tukeys secondary analysis are indicated on graphs as *$p<0.05$, **$p<0.01$, ***$p<0.001$.

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