Supplementary Data:

Figure S1: HMC1.2 cell line has constitutively actived NFAT2, NFAT2 and NFAT4 species. Fractionated cell lysates from HMC1.2 cells treated for 2 hours with 1µM CSA or 100nM TPA + 1µM IonM. β-Tubulin shown as a cytoplasmic loading control, Lamin A/C shown as a nuclear loading control.

Figure S2: RT-PCR of transcripts modulated by 24 hour treatment with CSA. P815 cells were treated for 24 hours with 1uM CSA, before transcripts were quantified. Expression results were analyzed using the comparative C_T method (also known as the $2^{-\Delta\Delta CT}$ method, as described in the methods section. Results are plotted as the percent expression compared with untreated cells.

Figure S3: CNPIs do not modulate KIT signaling and KIT inhibitors do not affect NFAT localization. A) Cytoplasmic lysates from P815 cells treated with CNPIs, CSA or FK506 for 3 hours. **B)** Cytoplasmic lysates from P815 cells treated with KIT inhibitors, dasatinib or imatinib (STI) for 3 hours.β-actin is shown as a loading control.

Figure S4: Combination therapy does not enhance the effect of KIT inhibitors on KIT or downstream signaling. Whole cell extracts from P815 cells treated for 3 hours with single agents or combination.

Table S1: Summary of KIT-mutant mast cell characteristics

Table S2: Summary of antibodies used in immunoblotting experiments

Table S3: qRT-PCR analysis of NFAT expression in KIT-mutant mast cell lines