

SUPPLEMENTAL INFORMATION

Supplementary Tables

Table S1: Accompanying clinicopathological information for RNA-seq data of lung adenocarcinoma samples with *MET*-amplification or *MET* Δ *ex14* mutation, downloaded from the NIH GDC Data Portal.

Table S2 – Western blot densitometry values for each visible band, quantified using ImageJ software.

Supplementary Figures

Figure S1

- (A) GSEA using positional gene sets found most genes in the Chr12Q15 region to be overexpressed in *MET* Δ *ex14* tumour samples, suggesting focal amplification.

Figure S2

- (A) WB analysis comparing effects of MET inhibition in MET-addicted H1993 and Hs746T cells shows a complete rebound in ERK1/2 phosphorylation in H1993, but not in Hs746T, cells.

Figure S3

- (A) 96-well dose response assay assessing relative sensitivity of HEK293t cells ectopically expressing wild type MET, mutant (Δ ex14, Y100F) MET, or GFP to Cabozantinib. Both mutant and wild type *MET* overexpression fails to increase sensitivity of HEK293T cells to MET inhibition in HEK293t cells under anchorage independent conditions.

Figure S4

- (A) 96-well dose response assays assessing the effects of wild type MET, mutant (Δ ex14, Y1003F) MET, or GFP overexpression in HPL1D cells on Cabozantinib sensitivity. *MET* Δ *ex14* expression confers a slight increase in sensitivity to both MET (P -val = 0.0021) and (B) MAPK inhibition (P -val = 0.0045).
- (C) The observed increased sensitivity to MET inhibition in *MET* Δ *ex14*-expressing cells was lost in the presence of MAPK inhibition (P -val = 0.3059), pointing to the MAPK pathway as the chief effector of MET-driven growth.

Figure S5

- (A) Side-by-side loading control comparison of nuclear (Histone H3) and cytosolic (Vinculin & AKT) proteins present in either fraction to evaluate the degree of cellular compartment separation.

Figure S6

- (A-L) Uncropped, original western blot images for each target included in the figures.