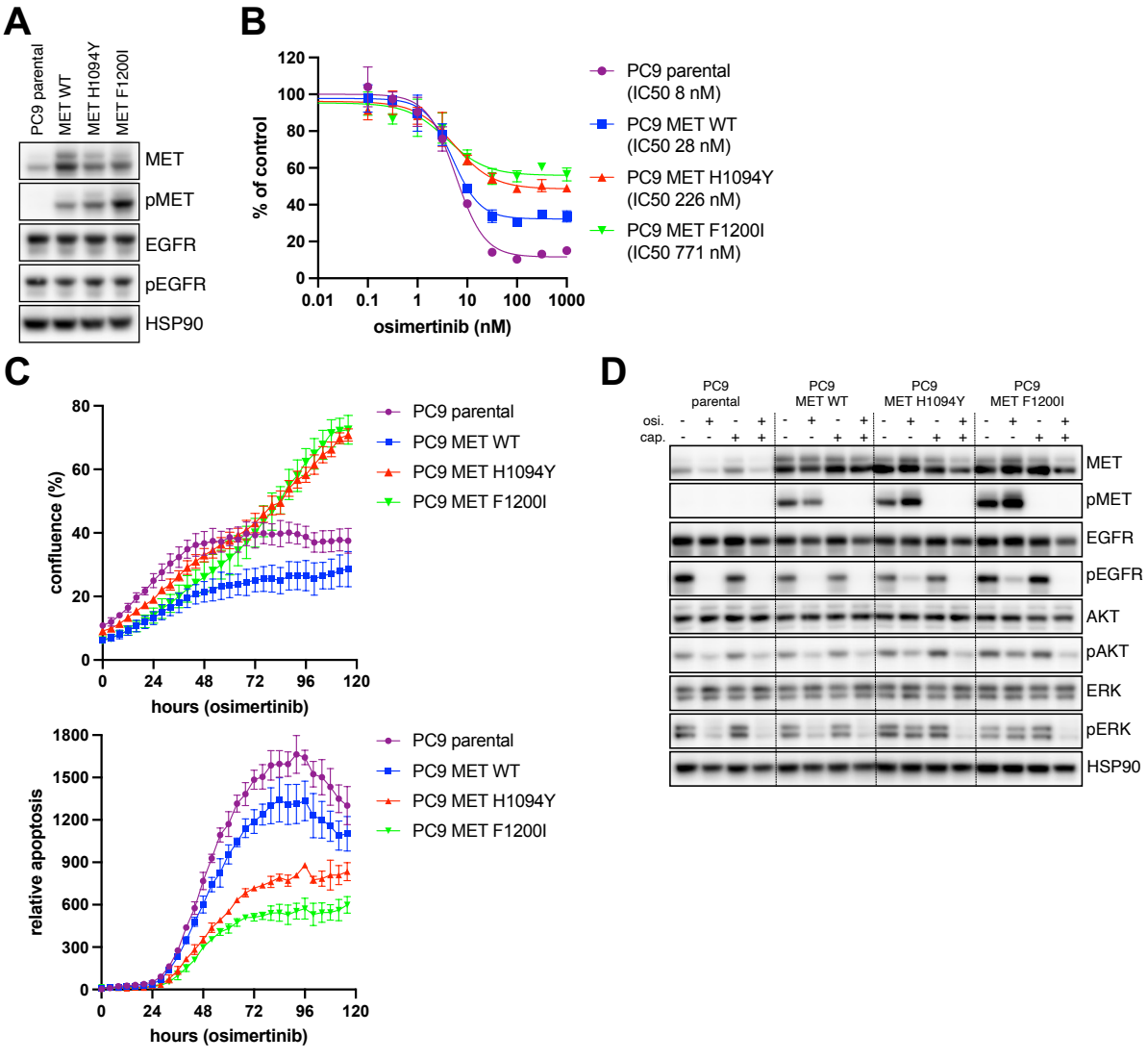
Supplementary Figure 15



E

		Type Ia	Type Ib			Type II
	Mutation	crizotinib	capmatinib	tepotinib	elzovantinib	cabozantinib
PC9 MET TKIs + osi. 100 nM	parental	3397	>10000	>10000	735.9	3025
	H1094Y	7.8	0.0	1.1	3.1	15.4
	F1200I	59.8	9.1	41.4	9.8	500

1nM≥IC50
10nM≥IC50>1nM
200nM≥IC50>10nM
1000nM≥IC50>200nM
IC50>1000nM

Adapted from Figure 5	Mutation	Type Ia crizotinib	Type Ib capmatinib	Type Ib tepotinib	Type Ib elzovantinib	Type II cabozantinib
Ba/F3	WT (+IL3)	2348.0	4978.0	>10000	>10000	2367.0
	H1094Y	5.5	0.05	0.1	1.8	13.0
	F1200I	32.1	3.3	7.9	3.9	371.1

Supplementary Figure 15*. In vitro* analysis of *MET* H1094Y and F1200I mutations in PC9 cells. (**A**) Establishment of NSCLC cancer cell lines expressing *MET* TKD mutations. PC9 cells were transduced with wild-type (WT) *MET* (as control) and *MET* H1094Y and F1200I. (**B**) Growth inhibition assay of PC9 cells in response to EGFR inhibition with osimertinib. (**C**) Cell proliferation and relative apoptosis were monitored under EGFR inhibition with osimertinib (300 nM). (**D**) Phosphorylation of MET, EGFR, AKT, and ERK was analyzed in PC9 cells expressing *MET* TKD mutants. PC9 cells were treated with osimertinib (1 μM) with or without capmatinib (1 μM) for 48 hrs. (**E**) IC50 values of PC9 cells (upper table) and Ba/F3 cells (lower table) expressing *MET* TKD mutants in response to MET TKIs. PC9 cells were treated with gradient dose of MET TKI combined with a fixed dose of osimertinib (100 nM) for 72 hrs.