Title: Phosphoproteomics Reveals HMGA1, a CK2 Substrate, as a Drug-Resistant Target in Non-Small Cell Lung Cancer

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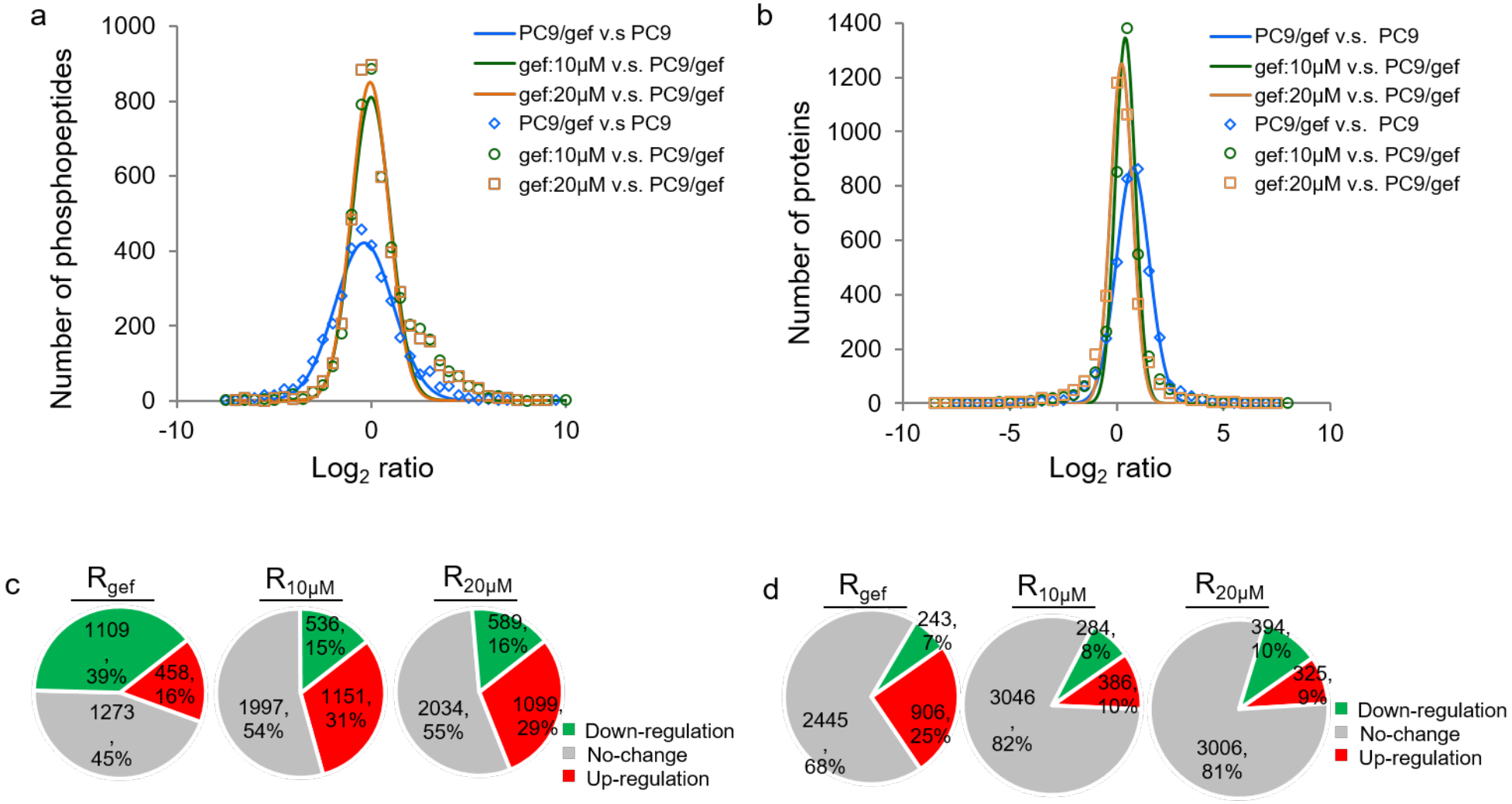
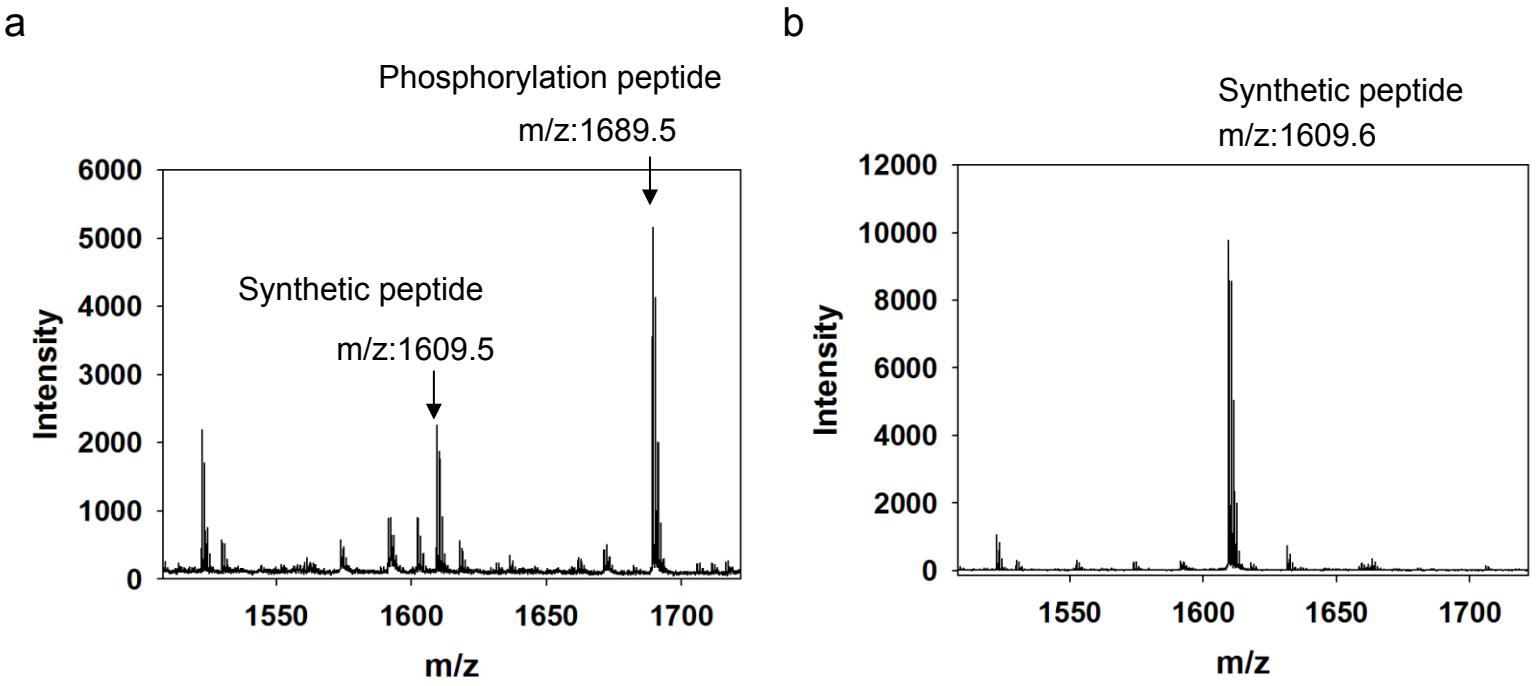
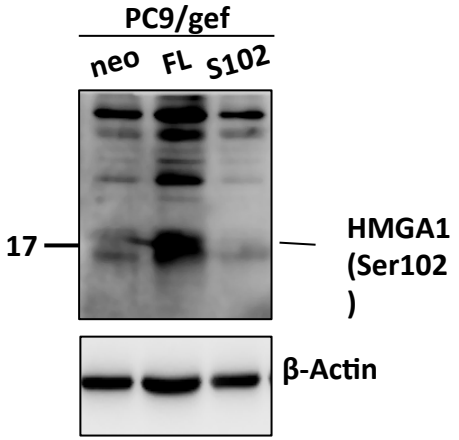


Supplementary Figure S1. **Phosphoproteomics and proteomics analysis of PC9/gef versus PC9, gefitinib 10μM and 20μM treatment of PC9/gef cells compare to PC9/gef.** (**a**) Log_2 ratios distribution for phosphopeptides analysis. (**b**) Log_2 ratios distribution for proteins analysis. (**c**) For phosphoproteomics analysis, pie charts reveal the percentage of 2-fold up-regulation, 2-fold down-regulation and no change of phosphopeptides. (**d**) For proteomics analysis, pie charts reveal the percentage of 2-fold up-regulation, 2-fold down-regulation and no change of proteins.

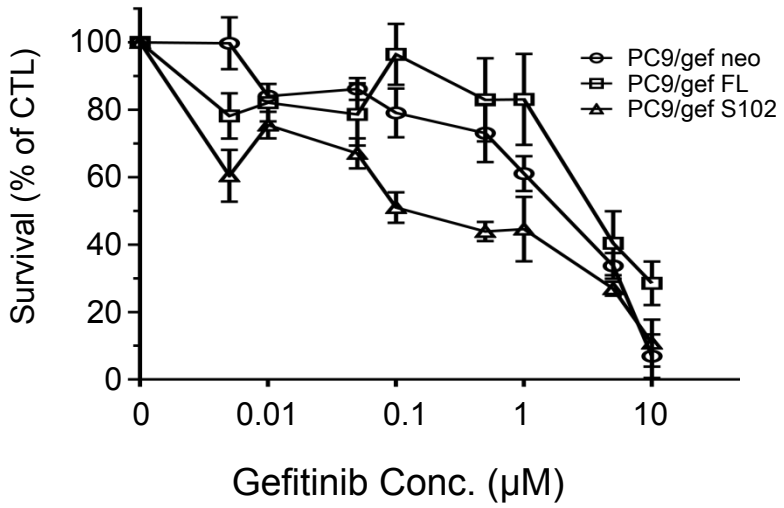


Supplementary Figure S2. *in vitro* kinase assay for synthetic peptide of HMGA1 and detect by MALDI-TOF MS. (**a**) *in vitro* kinase assay with CK2 kinase (**b**). *in vitro* kinase assay with MAP kinase.

a



b



c

Group	neo	FL	S102
IC_{50} (uM)	2.660	3.944	0.197

Supplementary Figure S3. HMGA1 phosphrylated site, S102 regulates drug resistance in NSCLC. (a) the efficiency of phosphorylation site mutation was confirmed by western blot in PC9/gef cells (neo: PC9/gef transfect pCIneo (vector only) plasmid; FL:PC9/gef transfect pCIneo-HMGA1 WT (wild type) plasmid; S102: PC9/gef transfect pCIneo- HMGA1 S102 mutant plasmid), respectively. (b) Cytotoxicity assays for various concentrations of gefitinib in HMGA1 phosphorylation mutation in PC9/gef cells in neo, FL and S102 condition. (c)The IC_{50} of PC9/gef cells treatment with gefitinib in neo, FL and S102 condition.