

Supplementary

Table S1 Subgroup analysis of *EGFR* and *KRAS* mutations

Factors	<i>EGFR</i> mutations		P	<i>KRAS</i> mutations		P
	L858R (n=69)	exon 19del (n=55)		G12C (n=45)	non-G12C (n=46)	
Age, years						
Mean ± SD	69.7±9.2	63.9±13.0	0.005	68.9±8.7	68.8±10.6	0.92
Gender, n						
Male	18	16	0.84	19	15	0.39
Female	51	39		26	31	
Smoking, n						
Yes	20	18	0.70	43	35	0.01
No	49	37		2	11	
Tumor size, cm						
Mean ± SD	2.3±1.3	2.8±1.3	0.08	3.2±2.0	4.2±3.0	0.09
AJCC 8 th TNM stage, n						
I	53	37	0.31	27	21	0.21
II–III	16	18		18	25	
Tumor morphology in CT scan, n						
Pure solid	13	19	0.06	22	35	0.006
Part solid	55	35		20	8	
SUVmax in PET/CT*						
Mean± SD	4.4±4.1	5.2±4.2	0.34	7.0±5.7	8.8±6.5	0.19

*, n=190.

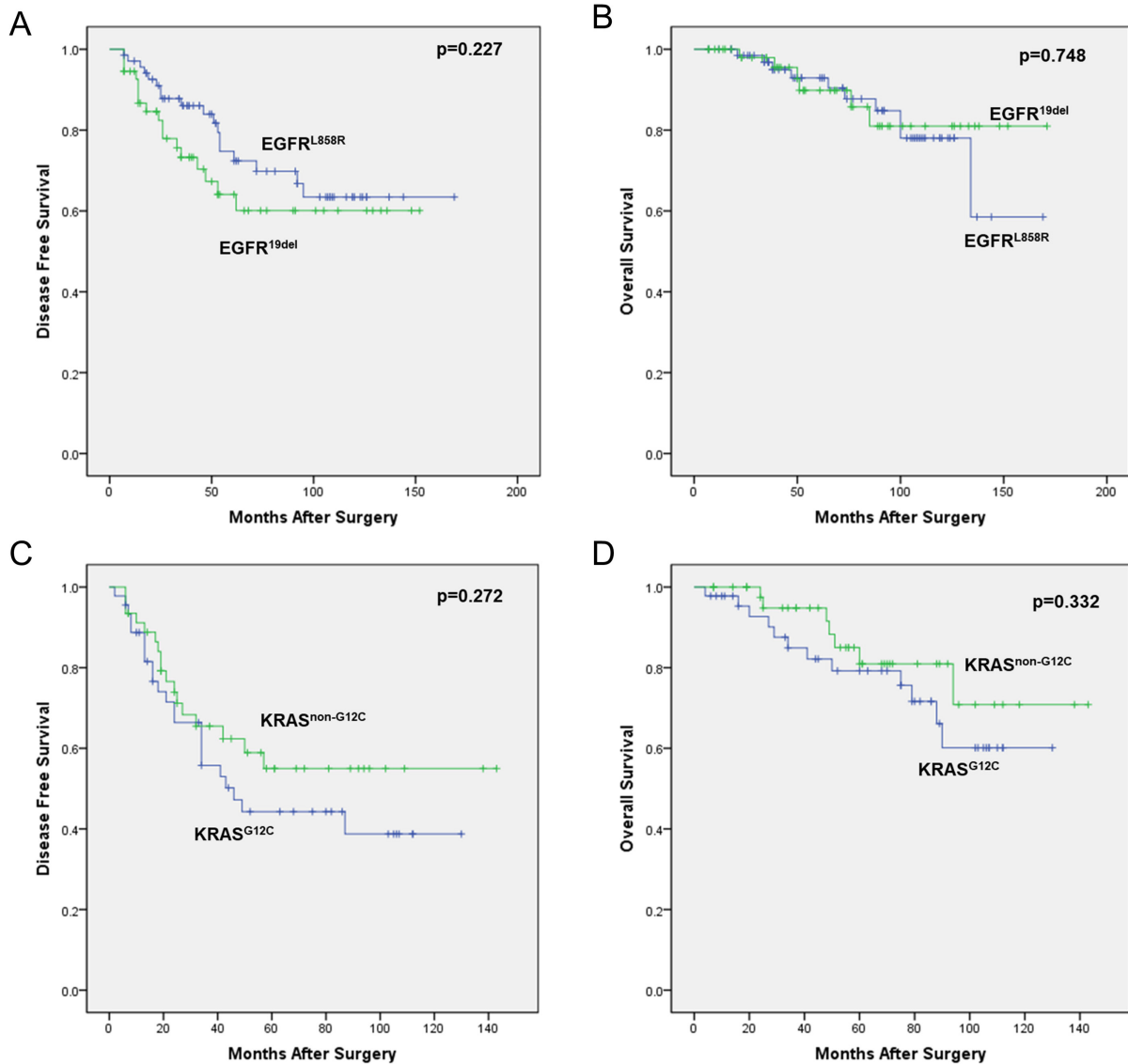


Figure S1 Survival analysis of subgroups of *EGFR* and *KRAS* mutations. Neither DFS (A) nor OS (B) were significantly different between groups with *EGFR* L858R and exon 19 deletion mutations. Neither DFS (C) nor OS (D) were significantly different between groups with *KRAS* G12C and non-G12C mutations.

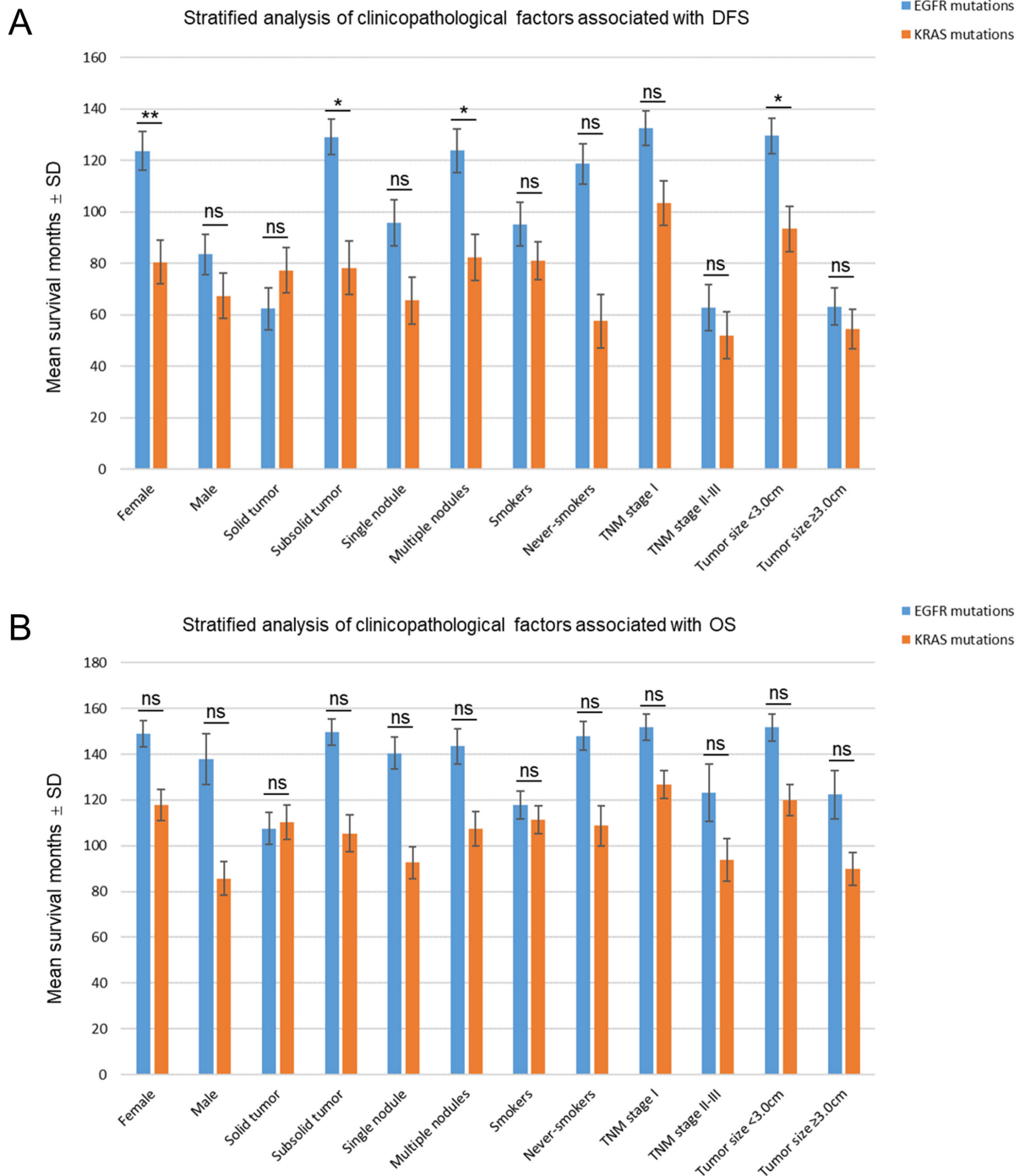


Figure S2 Stratified analysis of clinicopathological factors associated with DFS and OS. (A) Patients with *KRAS* mutations had significantly worse disease-free survival than those with *EGFR* mutations in female patients, subsolid tumor patients, multiple lung nodules patients, and smaller tumor (diameter less than 3 cm) patients. DFS was not significantly different between groups in male patients, solid tumor patients, single lung nodule patients, and larger tumor (diameter more than 3 cm) patients. (B) There was no significant difference in overall survival between groups on the stratified analysis. *, $P < 0.05$; **, $P < 0.01$; ns, not significant.

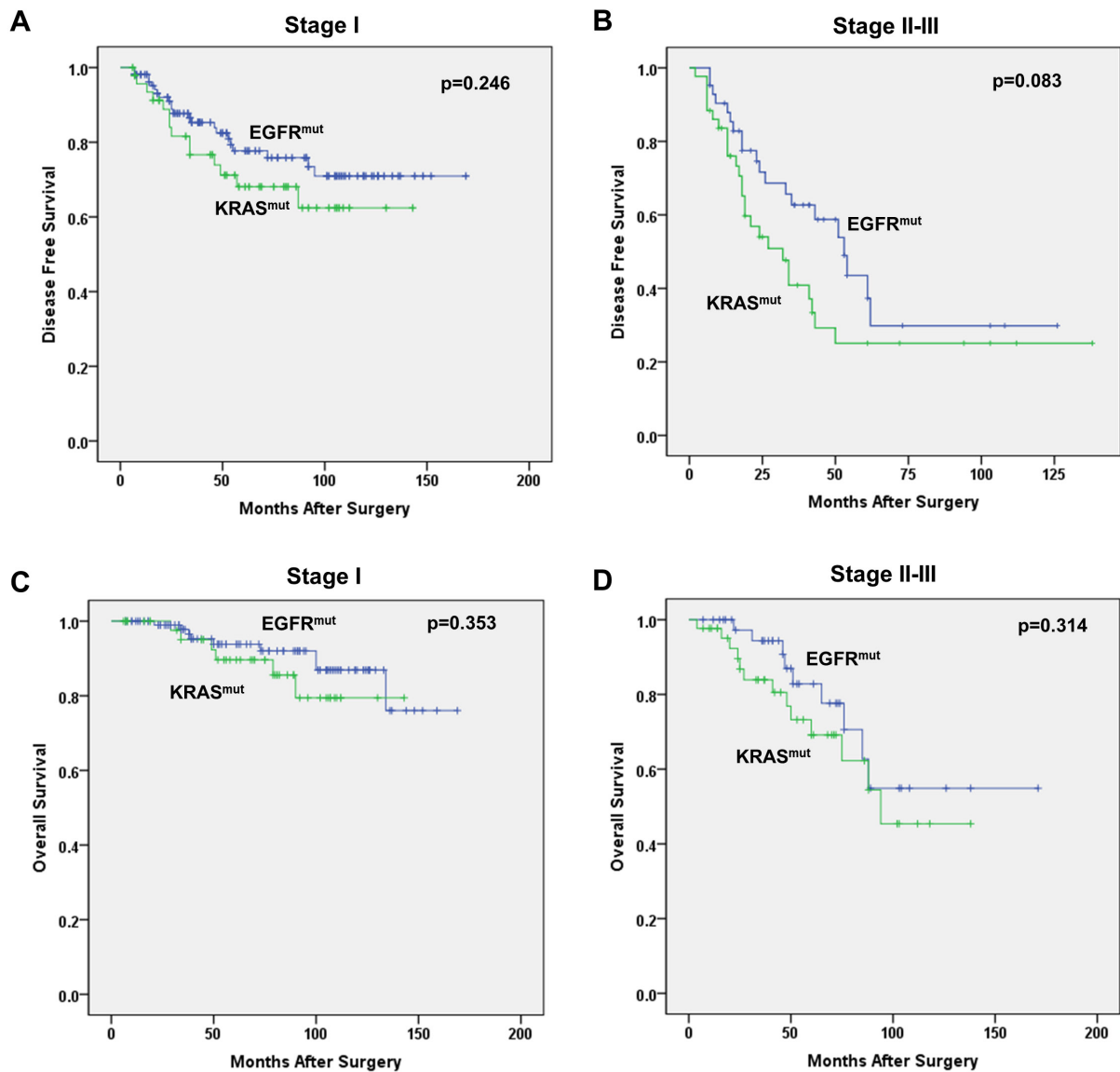


Figure S3 Survival analysis of *EGFR* vs. *KRAS* mutations in TNM stage I and II-III patients. Neither DFS (A) nor OS (C) were significantly different between groups with *EGFR* and *KRAS* mutations in stage I patients. Neither DFS (B) nor OS (D) were significantly different between groups with *EGFR* and *KRAS* mutations in stage II-III patients.