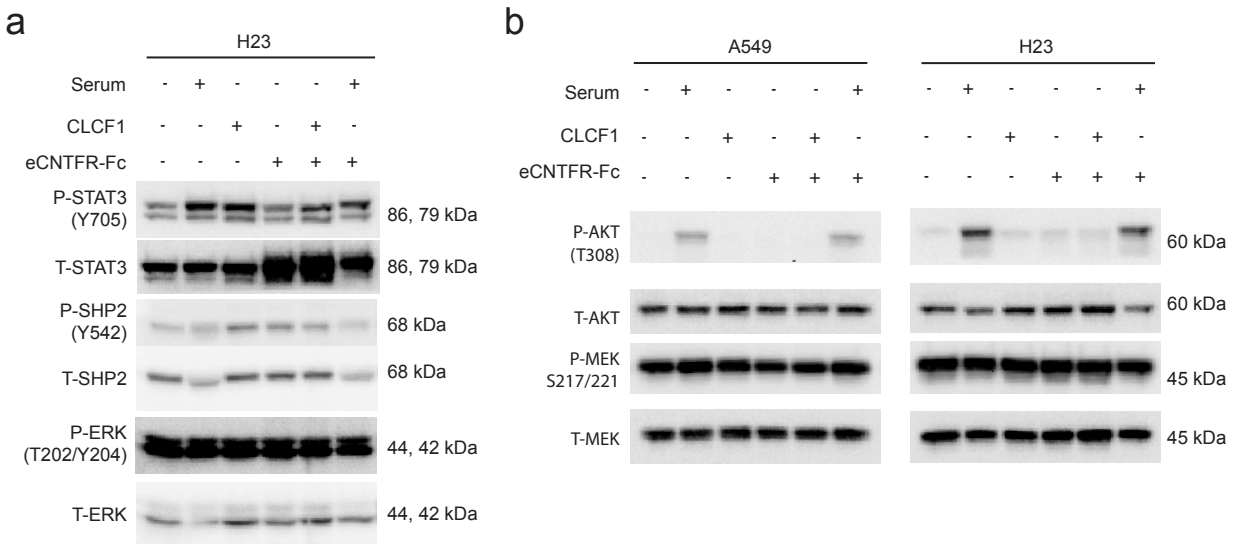
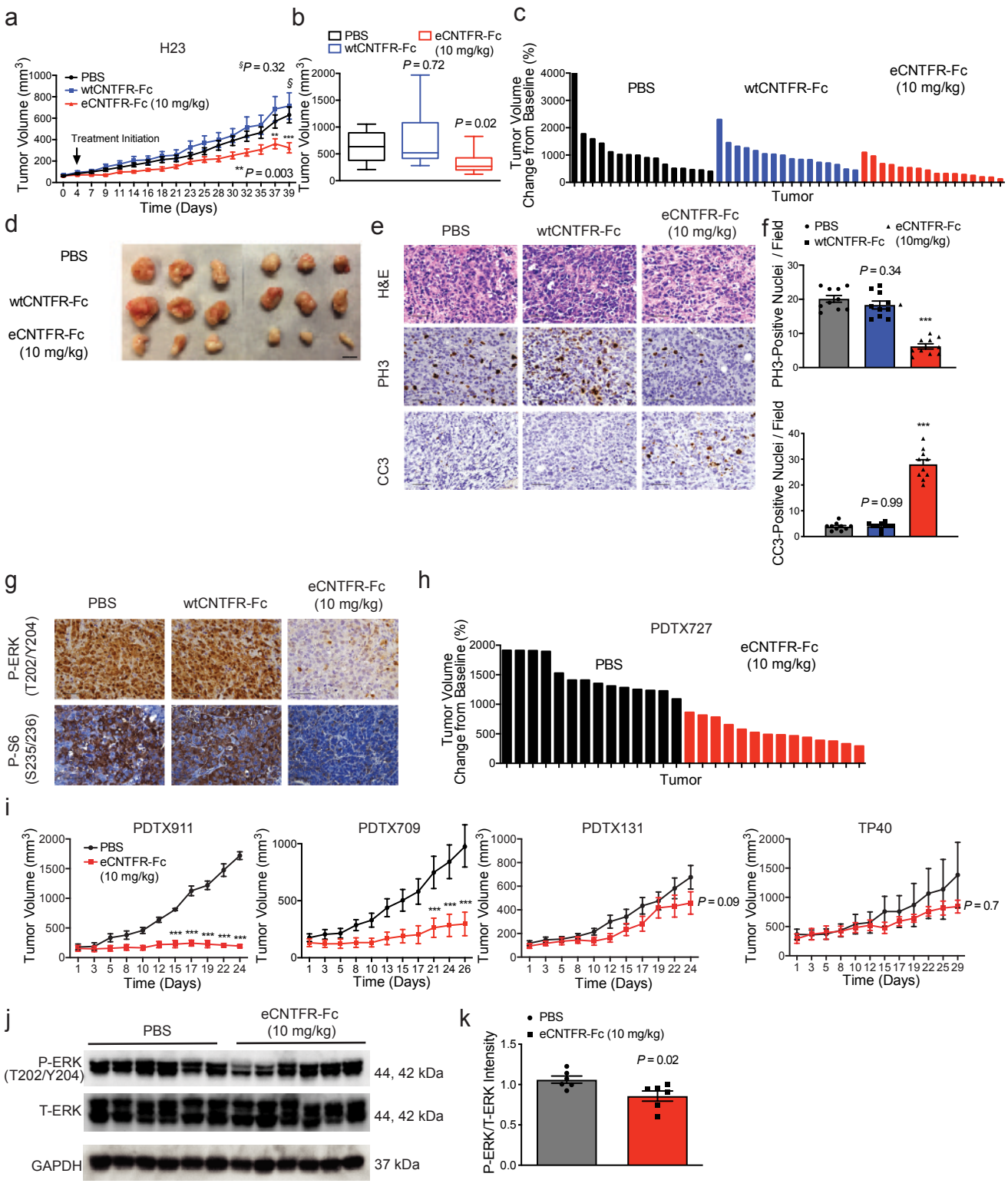
Kim and Marquez et al. Supplementary Figure 1



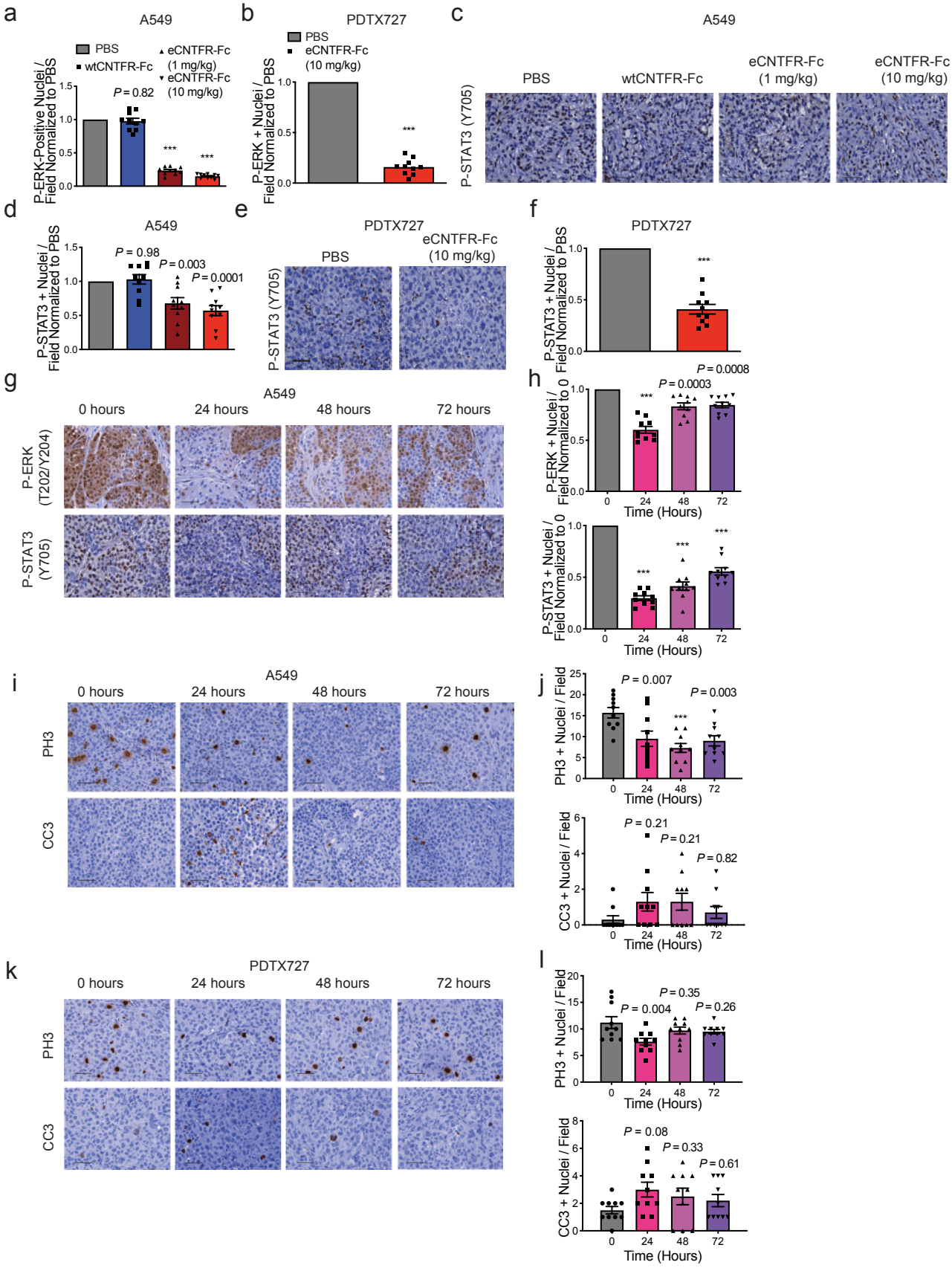
Supplementary Figure 1: CLCF1-CNTFR downstream signaling pathways. (**a**) Representative cropped western blot of H23 treated with serum, CLCF1, eCNTFR-Fc, CLCF1 + eCNTFR-Fc, or eCNTFR-Fc + serum after 24 h serum starvation (*n* = 2 independent experiments with similar results). (**b**) Representative cropped western blot of A549 and H23 treated with serum, CLCF1, eCNTFR-Fc, CLCF1 + eCNTFR-Fc, or eCNTFR-Fc + serum after 24 h serum starvation (*n* = 2 independent experiments with similar results).

Kim and Marquez et al. Supplementary Figure 2



Supplementary Figure 2: eCNTFR-Fc decreases tumor growth in xenograft and PDTX models containing a *KRAS* or *EGFR* mutation. (**a**) Tumor volume quantification of H23 xenografts (*n* = 16 biologically independent samples). *** *P* < 0.001 using two-way analysis of variance (ANOVA) and Dunnett's multiple comparison test (DCMT). Data represented as mean ± S.E.M. (**b**) Tumor volume quantification of final time point of H23 xenografts. Whiskers identify the maximum and minimum values; boxes indicate the 75th and 25th percentile and the line the median. (**c**) Waterfall plot showing tumor percent change from baseline for H23 xenografts. (**d**) Representative images of H23 xenograft tumors (*n* = 16 biologically independent samples). Scale bars, 10 mm. (**e**) Representative H&E staining and IHC for phospho-histone H3 (PH3) and cleaved caspase-3 (CC3) from H23 xenografts. Scale bars, 50 μm. (**f**) Quantification of PH3- and CC3-positive foci (*n* = 16 biologically independent samples with 10 technical replicates per group). *** *P* < 0.001 using one-way ANOVA and DCMT. Data are represented as mean ± S.E.M. (**g**) Representative IHC for phospho-ERK (P-ERK) and Phospho-S6RP (P-S6) in H23 xenografts (*n* = 3 independent experiments with similar results). (**h**) Waterfall plot showing tumor percent change from baseline for PDTX 727 model. (**i**) Tumor volume quantification of PDTXs [PDTX911 (*n* = 4), PDTX709 (PBS: *n* = 7 and eCNTFR-Fc: *n* = 8), PDTX131 (*n* = 10), and TP40 (*n* = 4) biologically independent samples]. *** *P* < 0.001 using using one-way ANOVA and DCMT. Data represented as mean ± S.E.M. (**j**) Representative cropped western blot of PDTX727 xenografts (*n* = 2 independent experiments with similar results). (**k**) Quantification of western blot (*n* = 2 independent experiments with similar results). One-way ANOVA and DMCT. Data represented as mean ± S.E.M.

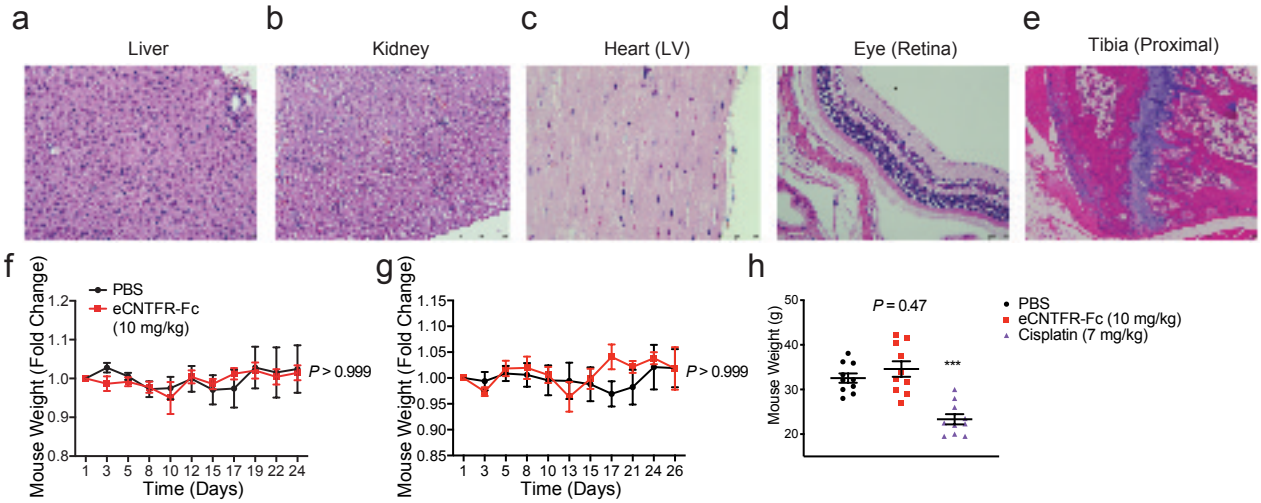
Kim and Marquez et al. Supplementary Figure 3



Supplementary Figure 3: Signaling changes mediated in a time-dependent manner by ERK and STAT3 *in vivo*. (**a**) Quantification of P-ERK data in A549 xenografts presented in **Fig. 5l** (*n* = 3 independent experiments with 10 technical replicates per group). *** *P* < 0.001 using one-way analysis of variance (ANOVA) and Dunnett's multiple comparison test (DMCT). Data represented as mean ± S.E.M. (**b**) Quantification of P-ERK data in PDTX727 presented in **Fig. 5m**. (*n* = 3 independent experiments with 10 technical replicates per group). *** *P* < 0.001 using two-tailed unpaired Student's *t*-test. Data represented as mean ± S.E.M. (**c**) Representative IHC for phospho-STAT3 (P-STAT3). (**d**) Quantification of this P-STAT3 data in A549 xenografts (*n* = 3 independent experiments with 10 technical replicates per group). One-way ANOVA and DMCT. Data represented as mean ± S.E.M. (**e**) Representative IHC for P-STAT3 data in PDTX727. (**f**) Quantification of P-STAT3 data (*n* = 3 independent experiments with 10 technical replicates per group). *** *P* < 0.001 using two-tailed unpaired Student's *t*-test. Data represented as mean ± S.E.M. (**g**) Representative IHC for P-ERK and P-STAT3 in A549 xenografts treated with eCNTFR-Fc (10 mg/kg) and harvested at time points shown. (**h**) Quantification of P-ERK and P-STAT3 data (*n* = 2 independent experiments with 10 technical replicates per group). *** *P* < 0.001 using one-way ANOVA and DMCT. Data represented as mean ± S.E.M. (**i**) Representative IHC for phospho-histone H3 (PH3) and cleaved caspase-3 (CC3) in A549 xenografts treated with eCNTFR-Fc (10 mg/kg) and harvested at time points shown. (**j**) Quantification of PH3 and CC3 data (*n* = 2 independent experiments with 10 technical replicates per group). *** *P* < 0.001 using one-way ANOVA and DMCT. Data represented as mean ± S.E.M. (**k**) Representative IHC for PH3 and CC3 in PDTX727 treated with eCNTFR-Fc (10 mg/kg) and harvested at time

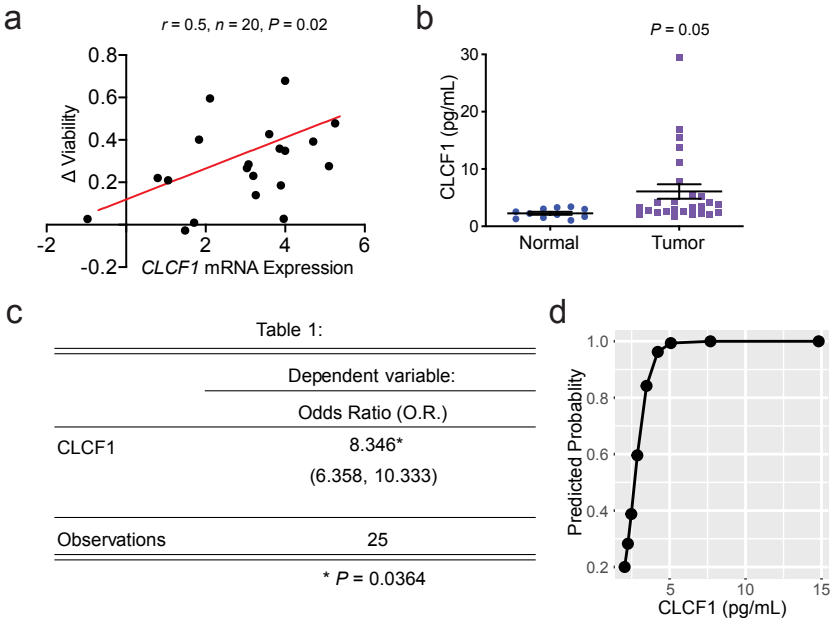
points shown. (**I**) Quantification of PH3 and CC3 data (*n* = 2 independent experiments with 10 technical replicates per group). One-way ANOVA and DMCT. Data represented as mean ± S.E.M. Scale bars, 50 μm.

Kim and Marquez et al. Supplementary Figure 4



Supplementary Figure 4: eCNTFR-Fc produces no observable adverse effects in PDTXs and GEMMs. Histologic sections and H&E staining of liver (**a**), kidney (**b**), left ventricle of the heart (**c**), retina (**d**), and proximal tibia (**e**) from a mouse that received the highest dose of CNTFR (10 mg/kg) for four weeks [*n* = 2 independent experiments (*n* = 3 mice per group) with similar results]. (**f**) Mouse weights following treatment course for PDTX911 and PDTX131 [PDTX911 (*n* = 2) and PDTX131 (*n* = 5) biologically independent samples]. (**g**) Mouse weights following treatment course for PDTX709 (*n* = 4 biologically independent samples). Two-way analysis of variance (ANOVA) and Dunnett's multiple comparison test (DMCT). Data represented as mean ± S.E.M. (**h**) Endpoint mouse weights for *Kras*$_{G12D}$/*Trp53*$_{f/f}$ mice treated 3 times per week with PBS, eCNTFR-Fc (10 mg/kg), or Cisplatin (7 mg/kg) for 4 weeks (Day 28) by intraperitoneal (i.p.) injection starting at 8 weeks post-delivery of 5 × 10_6 pfu of adenovirus-expressing Cre (Ad-Cre) (Day 0) (*n* = 10 biologically independent samples). *** *P* < 0.001 using one-way ANOVA and DMCT. Data represented as mean ± S.E.M.

Kim and Marquez et al. Supplementary Figure 5



Supplementary Figure 5: CLCF1 as a predictive biomarker in LUAD. (**a**) Scatter plot depicting Pearson's correlation coefficient *r* (two-tailed) for *CLCF1* mRNA expression obtained from the Cancer Cell Line Encyclopedia (CCLE) and the change in viability after treatment with 2.5 µM eCNTFR-Fc (in the cell line panel presented in **Figure 4a**) (*n* = 3 independent experiments). (**b**) CLCF1 ELISA performed on patient plasma samples and normal controls [Normal (*n* = 10) and Tumor (*n* = 26)]. (**c**) Odds ratio (O.R.) was calculated from a logistic regression (logit) model, and the *P*-value (two-tailed) was calculated using Wald test, *P*-value = 0.0364. Lung cancer samples were categorized as either having a 'mutation of interest' defined as *KRAS* G12C, *KRAS* G12V, or *KRAS* wild-type (wt) / *EGFR* mutant (dummy coded as 1, *n* = 17) or KRAS wt (dummy coded as 0, *n* = 8). (**d**) Graph showing the predictive probability of being in the mutation group as a function of blood plasma CLCF1 (pg/mL): y-axis is the predicted probability and the x-axis is the blood plasma CLCF1 (pg/mL).

Sample	Mutation Status	Staging Information	Induction	Pathology	Age	Sex	Race
CAF/NF1 HT141107	EGFR Exon 19	IA (T1bN0)	n/a	adenocarcinoma, 90% acinar focal lepidic, ALK/ROS1-, EGFR+exon19	55	F	white
CAF/NF2 HT141208	TP53 and BRAF G466V	n/a	n/a	n/a	n/a	n/a	n/a
CAF/NF3 HT141218	TP53	T1aN0	n/a	adenocarcinoma, CK7/TTF1/NapsinA+, p63 CK5/6-, CK20-, ALK/ROS1-	79	M	asian
CAF/NF4 HT151120	None	mucinous adenocarcinoma	breast chemo 2010-present	adenocarcinoma w/ mucinous features, EGFR/KRAS/ALK/ROS1-, TTF1/napsin-	64	F	other
CAF/NF5 HT151203	EGFR Exon 19	IIA NSCLC, stage T2a N1M0	n/a	adenocarcinoma TTF1+, napsin+, GATA-, micropapillary	65	F	other
CAF/NF6 HT151210	n/a	T2aN2	n/a	CK7/TTF-1/napsin +, p63/CK5&6 -, adenocarcinoma 50% acinar, 30% papillary 20% micropapillary	76	M	asian
CAF/NF7 HT160120	TP53	T1BN0 (stage IA) adenoCA	n/a	adenocarcinoma, CK7+, TTF1+, CK20-, napsinA+, GATA3-, acinar70%, lepidic 20%, micropapillary 5%, papillary 5%	79	F	white
CAF/NF8 HT160216	KRAS G12C, TP53	(T2a N0)IB non-small cell lung cancer, poorly differentiated adenocarcinoma	n/a	adenocarcinoma, solid 90%, acinar 10%	76	F	white

Supplementary Table 1: Detailed patient information from samples collected for the cancer-associated fibroblast (CAF) and normal lung fibroblast (NLF) pairs.

Kim and Marquez et al. Supplementary Table 2

Gene Name	Primer Sequence
	5 '- SEQUENCE - 3'
HPRT	
Forward	TGACACTGGTAAAACAATGCA
Reverse	GGTCCTTTTCACCAGCAAGCT
GAPDH	
Forward	ACACCATGTATTCCGGGTCAAT
Reverse	TGTGGGCATCAATGGATTTGG
CNTFR_1	
Forward	ACAAGGTCTCCATAAGTGTCA
Reverse	TTCTGGAGGATCAGGCTTCA
CNTFR_2	
Forward	CCTGCTGTGCTGTGCTTG
Reverse	AACATCCCCCACGAGTCC
CLCF1_1	
Forward	CCAGAAAACCTATGACCTCAC
Reverse	TGAAGTCTGGCTCGTTGAAA
CLCF1_2	
Forward	TGGCGGATGGGATTATTAAA
Reverse	CGCTCGTACTGCACATGG

Supplementary Table 2: Primer sequences used for *CLCF1* and *CNTFR* used for quantitative real-time PCR (qRT-PCR).

Kim and Marquez et al. Supplementary Table 3

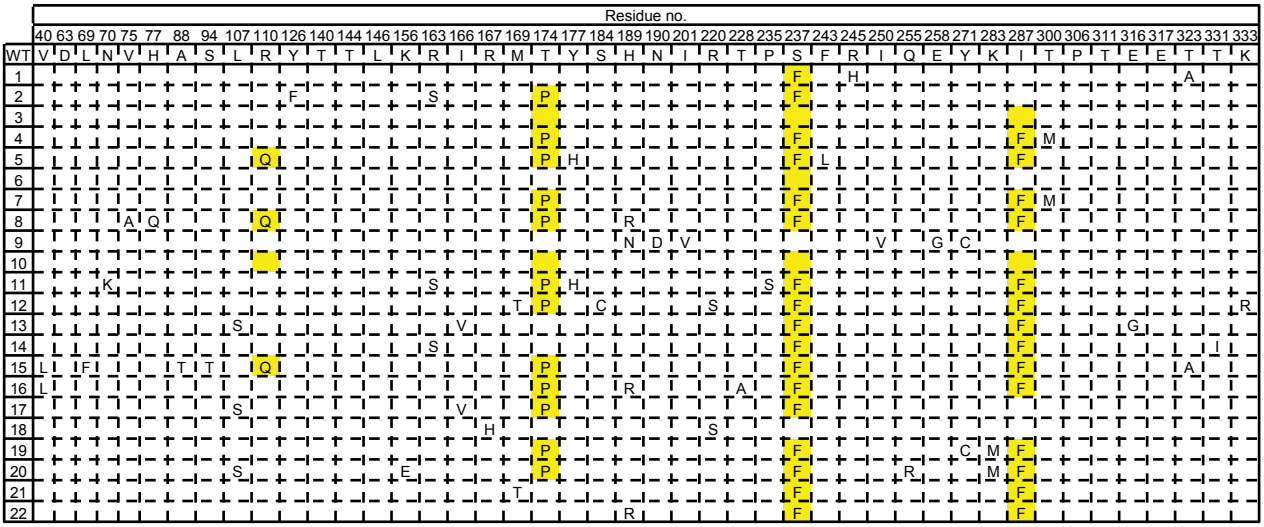
a

	Residue no.																														
	36	61	88	107	132	140	144	146	163	166	168	169	174	184	189	189	190	201	220	224	235	237	243	250	255	258	271	283	287	304	308	333
WT	E	G	A	L	P	T	T	L	R	I	T	M	T	S	H	H	N	I	R	V	P	S	F	I	Q	E	Y	K	I	E	H	K
1				S		S																										
2																																
3									S				P												L							
4									S				P												L							
5													P			R								V		G	C					R
6														C																		
7		G									C									A												
8												T		G					S													
9										V													S					M				
10																						F										
11					L			Q																					F			
12	V		T																		L								F			
13																															R	
14													P		N		D	V														
15													P																	G		
16													P			R								V		G	C					R
17							S																						F			
18													P																	G		

b

	Residue no.																				
	59	63	69	75	94	110	156	167	191	228	235	237	243	266	287	289	304	311	317	323	333
WT	V	D	L	V	S	R	K	R	A	T	P	S	F	T	I	T	E	T	E	T	K
1									E			F									
2												F									
3			F		N	Q						F									
4				A								Y				A					
5												F									
6												F									
7		N								A		F	L								
8												F									
9												F									
10			F		N	Q						F									
11	L											F							G		
12												F						A			
13												F									M
14												F		A							
15								H			S	F			F						
16												F									
17								H			S	F			F						
18							E					F					G			A	

Supplementary Table 3: Sequence of CNTFR variants isolated from sort 3 (**a**) and sort 4 (**b**) of Library 1 (error-prone CNTFR library). Eighteen clones were sequenced at random from CNTFR variants recovered from sort 3 and sort 4. Note the emergence of T274P in sort 3 and S237F in sort 4 consensus mutations.



Supplementary Table 4: Sequence of twenty-two randomly selected clones from sort 3 of Library 2, shuffled library prepared using staggered extension process (StEP). Note the emergence of different combinations of consensus mutations R110Q, T174P, S237F, and I287F.

PDTX	ID	Gender	Age	Ethnicity	Smoking	KRAS	TP53	LKB1	CDKN2A	KEAP1	ALK	EGFR	Histology	Staging
PDTX131	30131	F	80	White	yes (Former; 17-pack year)	N	Y	N	N	N	N	N	Squamous	T2a N0 MX
PDTX727	20727	M	67	White	yes (Former; 51-pack year)	G12V	Y	N	N	Y	N	N	Adenocarcinoma	T2a N2 MX
PDTX911	30911	M	80	White	yes (Former; 40-pack year)	G12C	Y	N	Y	Y	N	N	Adenocarcinoma	T2b N0 M1b
BM002	50709	M	73	Asian	n/a	N	Y	N	Y	N	N	L813R	Adenocarcinoma	brain met
TP40	TP40	n/a	n/a	n/a	n/a	N	Y	N	Y	N	Y	N	Adenocarcinoma	T2a N2 M0

Supplementary Table 5: Detailed patient information corresponding to patient-derived tumor xenografts (PDTXs).

			eCNTFR-Fc			
Hematological	PBS	wtCNTFR-Fc	1 mg/kg	10 mg/kg	Reference Range	Units
WBC	7.14 ± 0.3	6.81 ± 0	5.85 ± 0.2	6.23 ± 0.4	5.5 – 9.3	K/µl
RBC	7.32 ± 0.8	7.65 ± 0.3	7.61 ± 1.2	6.52 ± 1.3	7.0 – 8.8	M/µl
HGB	13 ± 0.3	13.3 ± 1.2	13.3 ± 0.7	13.7 ± 1.9	13.7 – 16.4	gm/dL
HCT	38.5 ± 2.0	40.3 ± 0.9	44.9 ± 1.4	39.3 ± 5.0	39.0 – 47.0	%
Platelet (estimate)	Adequate	Adequate	Adequate	Adequate	Adequate	
Chemistry Panel						
Glucose	212 ± 10.7	224 ± 12.3	200 ± 1.9	227 ± 25.3	184 – 220	mg/dL
Cholesterol	111 ± 11.5	116 ± 2.3	85 ± 22.0	114.3 ± 25.8	N/A	mg/dL
Calcium	10.5 ± 1.5	10.4 ± 0.4	9.7 ± 0.1	10.8 ± 0.9	8.9 – 9.7	mg/dL
Phosphorus	8.7 ± 0.5	7.2 ± 1.0	7.2 ± 0.7	9.2 ± 2.7	N/A	mg/dL
Sodium	155 ± 0.3	155 ± 1.1	153 ± 4.5	155.5 ± 3.1	114 – 154	mmol/L
Potassium	8.4 ± 0.4	8.7 ± 0.3	7.7 ± 0.9	9.9 ± 2.3	3.0 – 9.6	mmol/L
Chloride	115 ± 2.8	115 ± 1.6	114 ± 2.8	112.5 ± 3.9	N/A	mmol/L
Carbon Dioxide	26 ± 1.2	24.6 ± 0.6	23.3 ± 0.8	23.2 ± 1.4	N/A	mmol/L
Anion Gap	22.4 ± 1.6	24.1 ± 2.1	24.4 ± 3.9	29.7 ± 4.8	N/A	mmol/L
Hepatic						
AST	88 ± 21.2	117 ± 15.2	133.5 ± 18.9	151 ± 28.6	192 – 388	U/L
ALT	48 ± 15.1	45 ± 11.2	61 ± 17.8	64.8 ± 25.0	76 – 160	U/L
Alkaline Phosphatase	50 ± 26.4	56 ± 18.4	91 ± 22.5	52.6 ± 35.8	171 – 183	IU/L
GGT	0	0	0	0	N/A	U/L
Bilirubin (total)	0.3 ± 0	0.3 ± 0.1	0.2 ± 0	0.4 ± 0	N/A	mg/dL
Protein (total)	5.1 ± 0.1	5.2 ± 0.5	5.5 ± 0.2	5.5 ± 0.1	5.0 – 6.2	g/dL
Albumin	2.8 ± 0.2	2.4 ± 0.1	3 ± 0.4	2.9 ± 0	3.2 – 3.6	g/dL
Globulin	2.3 ± 0.3	2.4 ± 0.1	2.5 ± 0.2	2.5 ± 0.1	N/A	
Renal						
BUN	22 ± 2.5	19 ± 1.7	15 ± 4.8	18.3 ± 3.2	20.3 – 24.7	mg/dL
Creatinine	0.2 ± 0.1	0.2 ± 0.1	0.1 ± 0	0.2 ± 0	0.1 – 1.1	mg/dL

Supplementary Table 6: Toxicology studies examining complete blood counts and chemistry panels for mice treated with PBS, wtCNTFR-Fc, eCNTFR-Fc (1 mg/kg), and eCNTFR-Fc (10 mg/kg) (*n* = 3 per group). Error shows the range of values.

Kim and Marquez et al. Supplementary Table 7

Sample	Mutation Status	Staging Information	Induction	Pathology	Age	Sex	Race
1	KRAS G13C, TP53	T2aNO Adenocarcinoma	None	MODERATELY DIFFERENTIATED INVASIVE ADENOCARCINOMA, PAPILLARY PREDOMINANT papillary (50%), lepidic (40%) and acinar (10%)	81	F	white
2	EGFR Exon 19 PLS	T2aN1 Adenocarcinoma	n/a	Moderately differentiated, predominantly invasive with less than 10% lepidic	64	F	Asian
3	TP53	T1b Adenocarcinoma	marginal zone lymphoma that was treated with chemoradiation in the past.	90% solid, 10% lepidic, poorly differentiated	72	M	White
4	KRAS G12C, TP53	T2a NX Adenocarcinoma	n/a	invasive adenocarcinoma with papillary (70%), lepidic (15%) and acinar (15%) features	76	M	white
5	n/a	T1a	n/a	LYMPHOEPITHELIOMA-LIKE CARCINOMA poorly differentiated, positive for CK5/6, as well as EBV RNA by in situ hybridization. CD5 highlights T lymphocytes within the lymphocytic infiltrates.	76	M	asian
6	MET exon 14	T2a minimally invasive adenocarcinoma	n/a	well differentiated invasive adenocarcinoma with acinar (60%) and lepidic (40%) growth patterns. An EVG stain shows focal invasion of the visceral pleura (PL2)	66	M	white
7	EGFR Exon 19	IIA NSCLC, stage T2a N1M0	n/a	adenocarcinoma TTF1+, napsin+, GATA-, micropapillary	65	F	other
8	EGFR L861Q	T2aN2 adeno	n/a	MODERATELY DIFFERENTIATED INVASIVE ADENOCARCINOMA, (50% ACINAR, 30% PAPILLARY, 20% MICROPAPILLARY) There is infiltration through the elastic layer of the visceral pleura with involvement of the surface of the visceral pleura, confirmed by immunohistochemical stains (positive for MOC 31 and BerEp4; negative for calretinin and D2-40). This corresponds to PL2	75	M	asian
9	TP53	T1BN0 (stage IA) adenoCA	n/a	adenocarcinoma, CK7+, TTF1+, CK20-, napsinA+, GATA3-, acinar70%, lepidic 20%, micropapillary 5%, papillary 5%	79	F	white
10	KRAS G12C, TP53	(T2a N0)IB non-small cell lung cancer, poorly differentiated adenocarcinoma	n/a	adenocarcinoma, solid 90%, acinar 10%	76	F	white
11	KRAS G12C	T1c N2 adeno	n/a	Adenocarcinoma (60% acinar, 40% solid), strongly CK7-positive and focally positive for napsin and TTF-1	82	F	white
12	n/a	T2aNx adeno	n/a	ADENOCARCINOMA, POORLY DIFFERENTIATED, Immunohistochemistry is positive for CK7 with focal p63 and CK5/6, 95% solid, 5% acinar)	84	M	white
13	n/a	T1bN0	history of bilateral, on 2.5mg of letrozole daily breast cancer	The histological sections of the right upper lobe (Part B) demonstrate an adenocarcinoma with mixed morphological patterns including lepidic and acinar, with focal clear cells. The acinar pattern is predominant with approximately 60%, while the lepidic pattern is 30% and the clear cell component is 10%.	74	F	white

Supplementary Table 7: Detailed patient information for plasma samples collected for CLCF1 ELISA.

Kim and Marquez et al. Supplementary Table 8

Sample	Mutation Status	Staging Information	Induction	Pathology	Age	Sex	Race
14	KRAS G12A, TP53	T1a adeno	n/a	well-differentiated invasive adenocarcinoma with predominantly acinar type (95%) histology with a minority lepidic pattern (5%)	73	F	white
15	KRAS G12V	T4 N2 adeno	n/a	well-differentiated invasive adenocarcinoma with predominantly papillary type (70%) morphology and minor components of lepidic (15%), solid with mucin production (10%), and acinar (5%) growth patterns. In many areas, the neoplastic cells show goblet cell-like mucinous differentiation. Immunohistochemical stains show focal TTF-1 nuclear positivity and focal Napsin A granular cytoplasmic positivity	78	M	white
16	KRAS G12C	T1b Adenocarcinoma	n/a	The tumor cells have abundant, pale, eosinophilic cytoplasm with distinct cell borders and display acinar (90%) and solid (10%) growth patterns. Immunostains demonstrate that the tumor cells are positive for napsin and positive for nuclear TTF-1	68	M	white
17	n/a	T1a carcinoid	n/a	TYPICAL CARCINOID TUMOR (WELL DIFFERENTIATED NEUROENDOCRINE TUMOR), 1.1CM	52	F	white
18	EGFR L858R	T1b Adenocarcinoma	n/a	The vast majority of the tumor is invasive with a minor component, about 5%, of lepidic pattern.	71	F	white
19	EGFR G752A, TP53	T1b Adenocarcinoma	n/a	moderately differentiated invasive adenocarcinoma, with a predominantly papillary architecture (50%). Acinar (20%), solid (20%) and micropapillary (10%) patterns are also present.	58	M	white
20	KRAS G12V, TP53	T2a	n/a	multilobular moderately differentiated invasive adenocarcinoma with predominantly solid (90%) and acinar (10%) patterns	79	F	asian
21	KRAS G12C	T1b Adenocarcinoma	n/a	approximately 80% acinar pattern with small (approximately 20%) portions of lepidic pattern at the lesion periphery. Malignant cells are moderately differentiated, with high nuclear to cytoplasmic ratios, prominent nucleoli and coarse chromatin. Bronchovascular margins are negative.	77	F	white
22	EGFR G719A, TP53	T1b N2 Adenocarcinoma	n/a	invasive poorly differentiated adenocarcinoma measuring 2.0 cm. The majority (90%) of the adenocarcinoma shows a acinar pattern of growth and displays mucinous differentiation. About 5% of the tumor shows a lepidic growth pattern and about 5% shows a micropapillary pattern of growth.	63	F	white
23	TP53, EGFR L858R, AKT1	T3N0 adenosquamous	n/a	demonstrates a lung tumor with histological features of adenocarcinoma and squamous carcinoma. The adenocarcinoma component is positive for TTF1, positive for CK 7, focally positive for CK 20, weakly positive for Napsin A, negative for p63, and negative for CK 5/6 by immunohistochemical stain. Meanwhile, the squamous component is positive for p63, positive for CK5/6, weakly positive for CK 7, negative for TTF1, negative for CK 20, and negative for Napsin A.	77	M	asian
24	EGFR 20 insertion	T3 (2.9 cm, 0.4 cm, 0.3 cm, 0.3 cm nodules) adeno	n/a	A main mass measuring 2.9 cm as well as three separate tumor nodules measuring from 0.3 cm to 0.4 cm in greatest dimension are seen in the same lobe. CK7 and CD163 immunohistochemical stain performed on the main tumor and the satellite nodules (block F2) fail to reveal any evidence of connection of the main tumor to the satellite nodules.	64	F	Native Hawaiian
25	APC	T2a	n/a	moderately-differentiated adenocarcinoma. The tumor cells are positive for CK7, TTF-1, and napsin A. CK5/6 and p63 immunohistochemical stains are negative for the tumor cells.	76	M	other
26	n/a	T2a	n/a	invasive poorly-differentiated adenocarcinoma that includes numerous individual cells with atypical features and some cells with signet-ring like features. Elastic Van Gieson stained sections demonstrate extension of the tumor to the visceral surface, which places the tumor within the PL2 and T2 staging categories	86	M	white

Supplementary Table 8: Detailed patient information for plasma samples collected for CLCF1 ELISA.