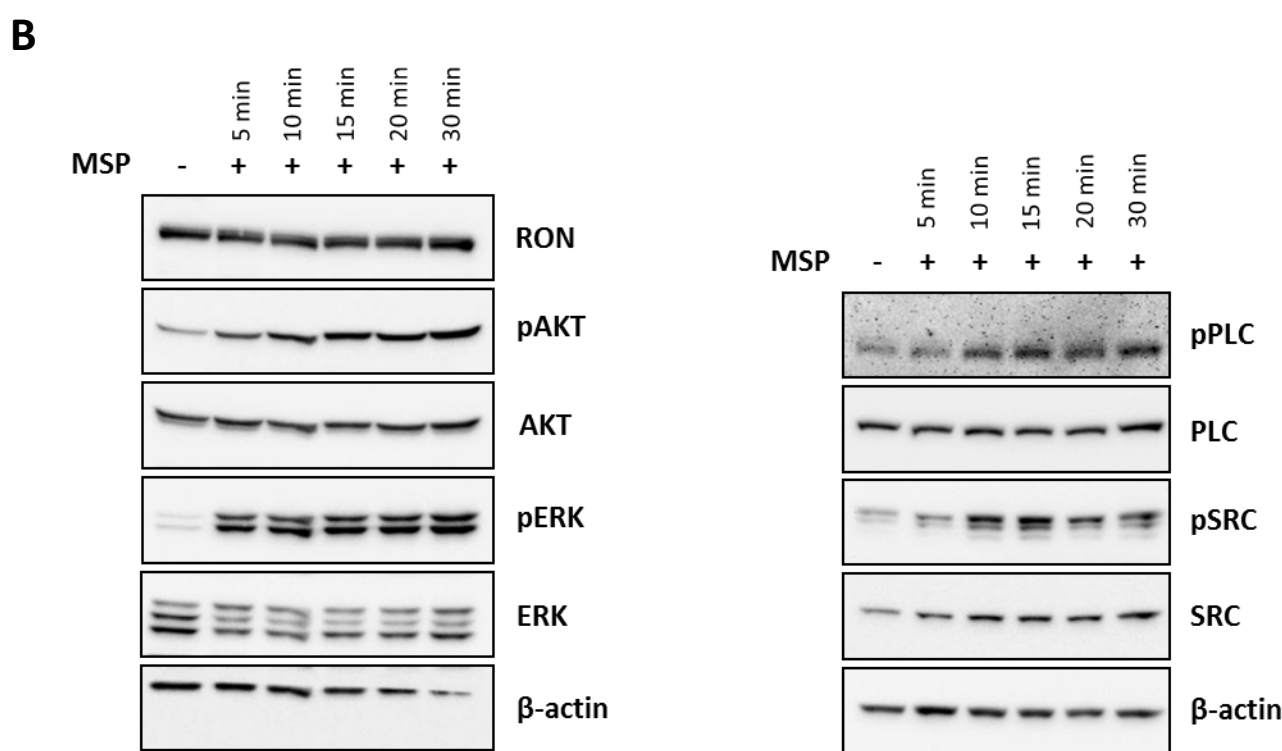
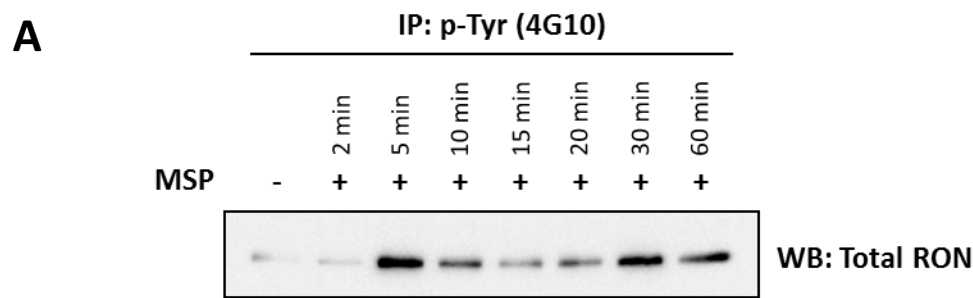
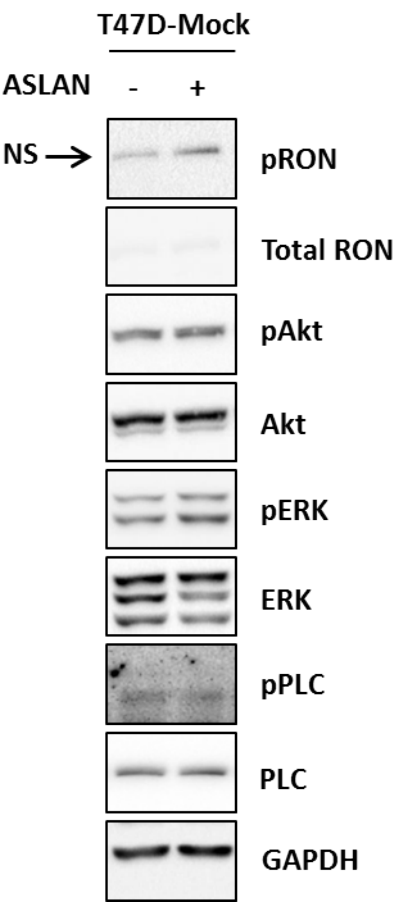


Supplementary Figure S1



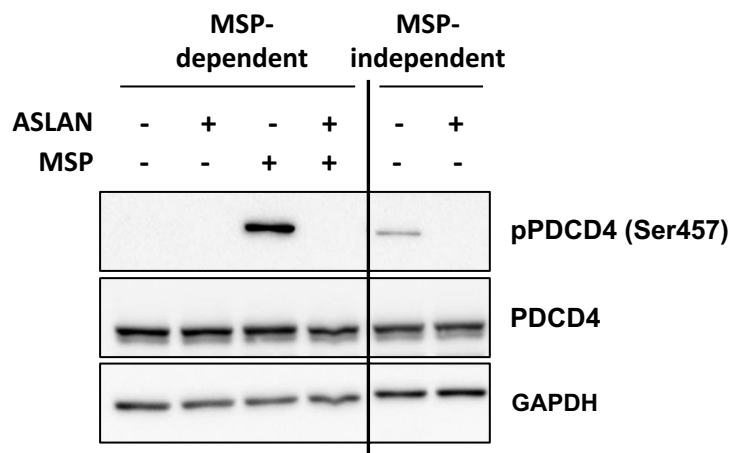
Supplementary Figure S1. kinetic analysis of RON activation following MSP stimulation and the effect on downstream signaling. A) Immunoprecipitation (IP)–Western blot analysis showing kinetic activation of RON by MSP. T47D-RON cells were treated with 100 ng/ml of doxycycline for 48 h, and were serum starved for 24 h followed by MSP stimulation (100 ng/ml) for different time points. Cell lysates from each time point were immunoprecipitated with 4G10 antibody and analyzed by Western blot. B) Whole cell lysates of the same samples were analyzed for activation of different signaling pathways downstream of RON. β-actin was used as loading control.

Supplementary Figure S2



Supplementary Figure S2. Effect of small molecule RON inhibitor, ASLAN002, on T47D-Mock and downstream signaling. Western blot showing status of different signaling pathways in T47D-Mock cells in the absence and presence of ASLAN002 for 4 h. As is indicated, 1 uM of ASLAN002 used to inhibit RON activity in T47D-RON cells shows no off-target effect on activity status of different signaling pathways when RON is not present. GAPDH was used as loading control. NS, non-specific band.

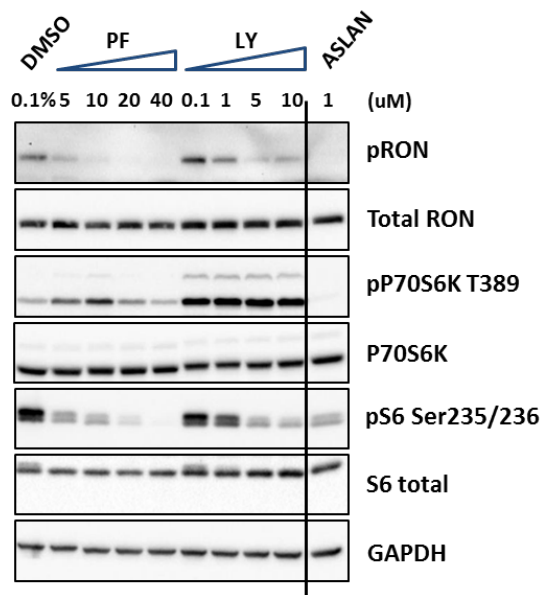
Supplementary Figure S3



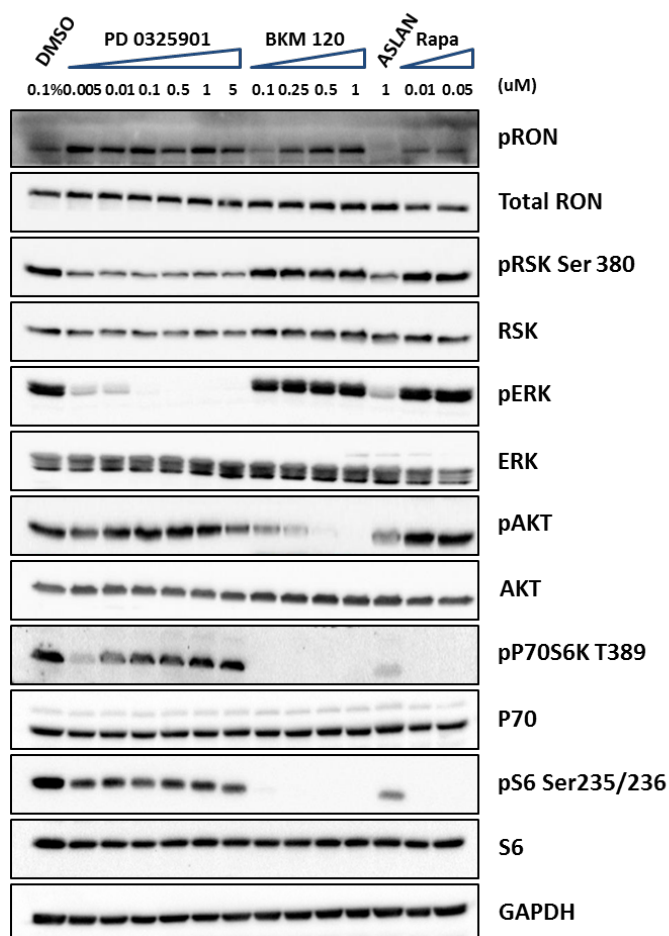
Supplementary Figure S3. Validation of RPPA analysis in T47D-RON cells in MSP-dependent and – independent conditions. Western blot shows phosphorylation of PDCD4 tumor suppressor in response to MSP stimulation or RON overexpression. GAPDH was used as loading control. Line indicates separation between lanes on the same Western blots.

Supplementary Figure S4

A

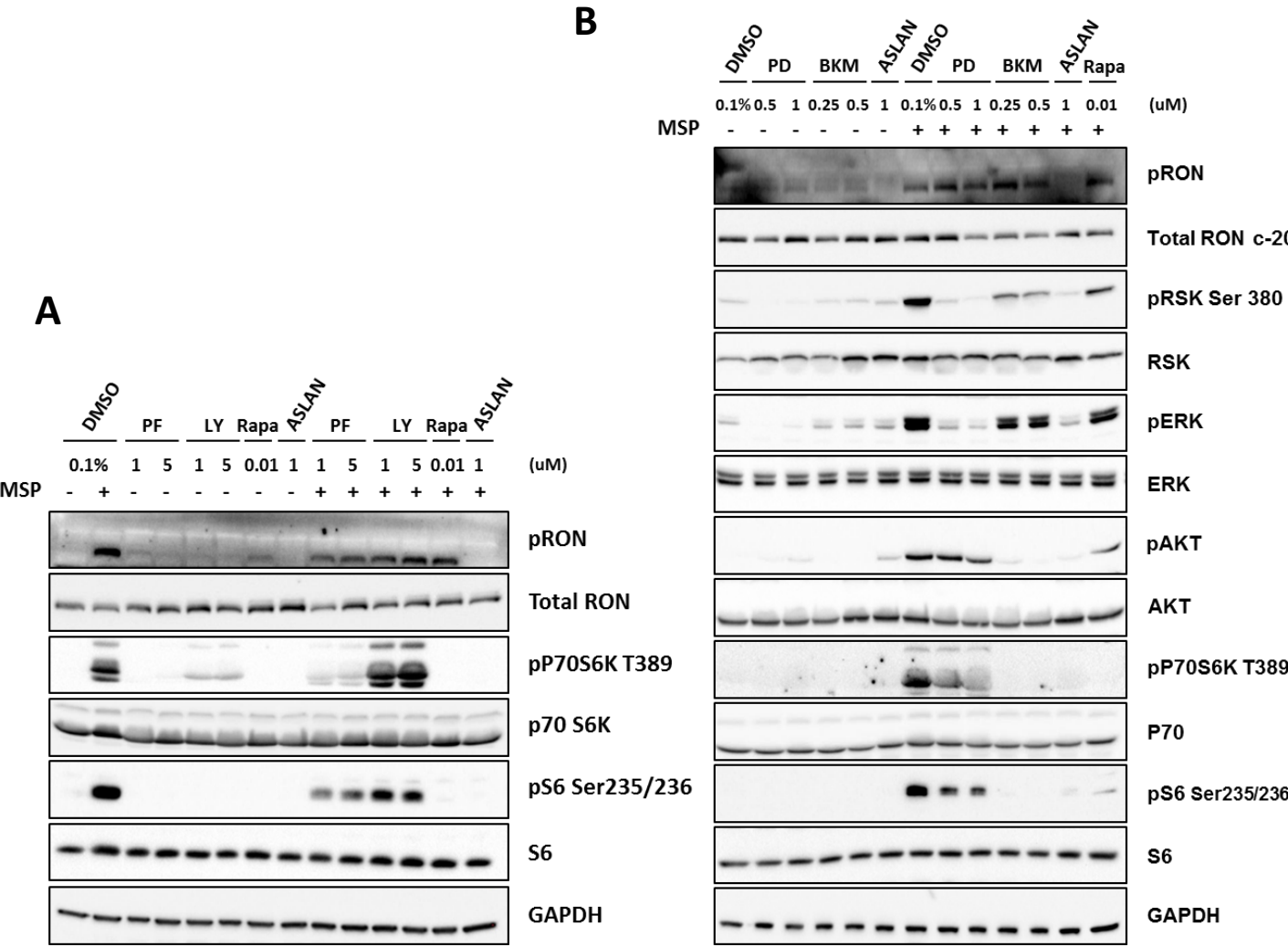


B



Supplementary Figure S4. Analysis of MSP-independent RON signaling using inhibitors of p70S6K, PI3K, and MAPK pathways. A) Representative Western blots for analysis of p70S6K as a potential kinase upstream of rpS6 using two inhibitors: pan-p70S6K (LY2584702) and p70S6K1 (PF-4708671). B) Western blot analysis showing PI3K, rather than MAPK, is the dominant kinase downstream of RON and upstream of mTORC1 responsible for phosphorylation of rpS6 in T47D-RON cells. Inhibition of PI3K or MAPK signaling for 4 h using different doses of NVP-BKM120 and PD0325901 respectively, shows that PI3K inhibition causes complete abrogation of rpS6 phosphorylation downstream of RON. See also Supplementary Figure S5 for signaling analysis using these inhibitors in the context of MSP-induced RON activation in T47D-RON cells. Line indicates separation between lanes on the same Western blots.

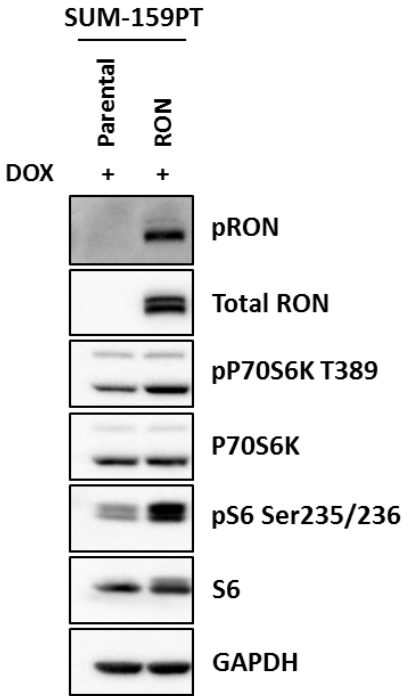
Supplementary Figure S5



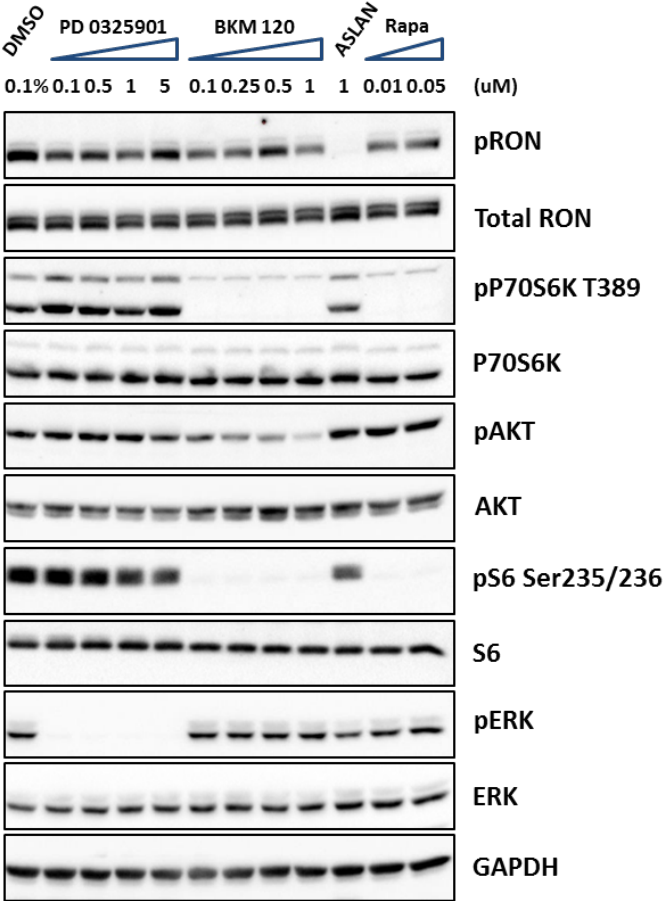
Supplementary Figure S5. Analysis of MSP-induced RON signaling using inhibitors of p70S6K, RSK, mTORC1, PI3K, and MAPK pathways. A) Representative Western blot showing effect of different kinase inhibitors on phosphorylation of rpS6 in the context of MSP-induced RON activation. T47D-RON cells were cultured in the presence of doxycycline (50 ng/ml) for 48 h and in serum starved media for 24 h, followed by 4 h treatment with different inhibitors. Cells were then stimulated with MSP (100 ng/ml) for 30 min. Inhibition of mTORC1 by rapamycin caused complete inhibition of phospho-rpS6, which is mediated through inhibition of p70S6K. PF-4708671, as a specific inhibitor of p70S6K1, shows partial efficacy on inhibiting rpS6 phosphorylation downstream of RON. B) Western blot analysis showing PI3K as the dominant kinase downstream of RON and upstream of mTORC1 responsible for phosphorylation of rpS6. Doxycycline-treated T47D-RON cells (50 ng/ml, for 48 h) were serum starved for 24 h and treated with NVP-BKM120 (PI3K inhibitor) and PD0325901 (MAPK inhibitor) for 4 h, followed by MSP stimulation (100 ng/ml) for 30 min. GAPDH was used as loading control.

Supplementary Figure S6

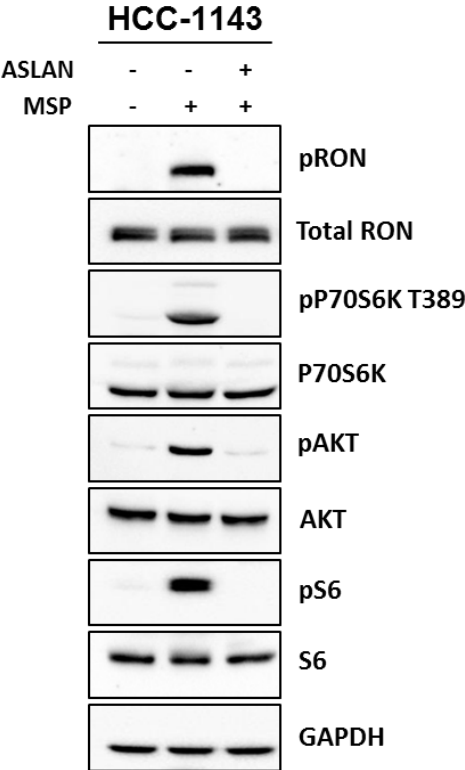
A



B

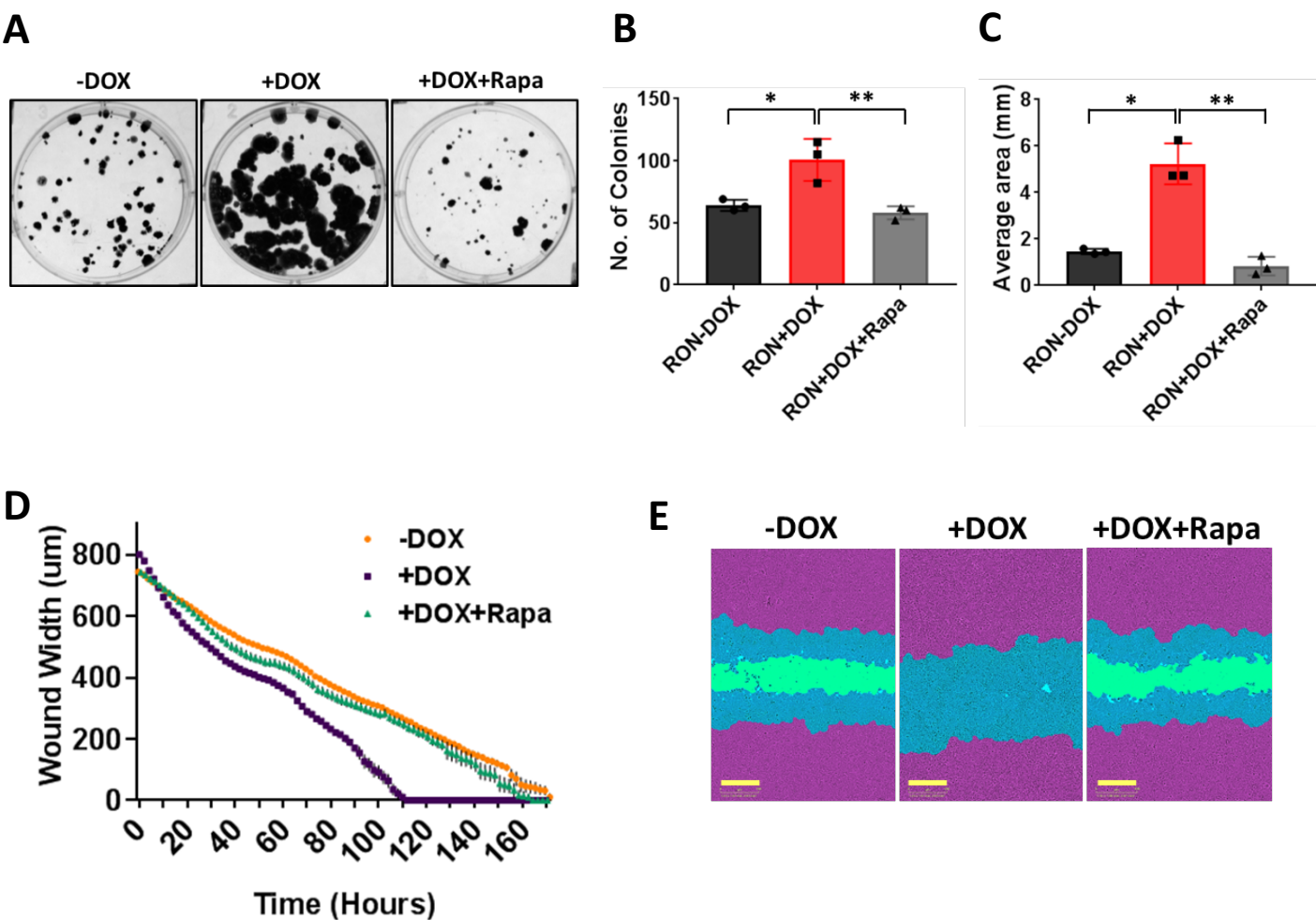


C



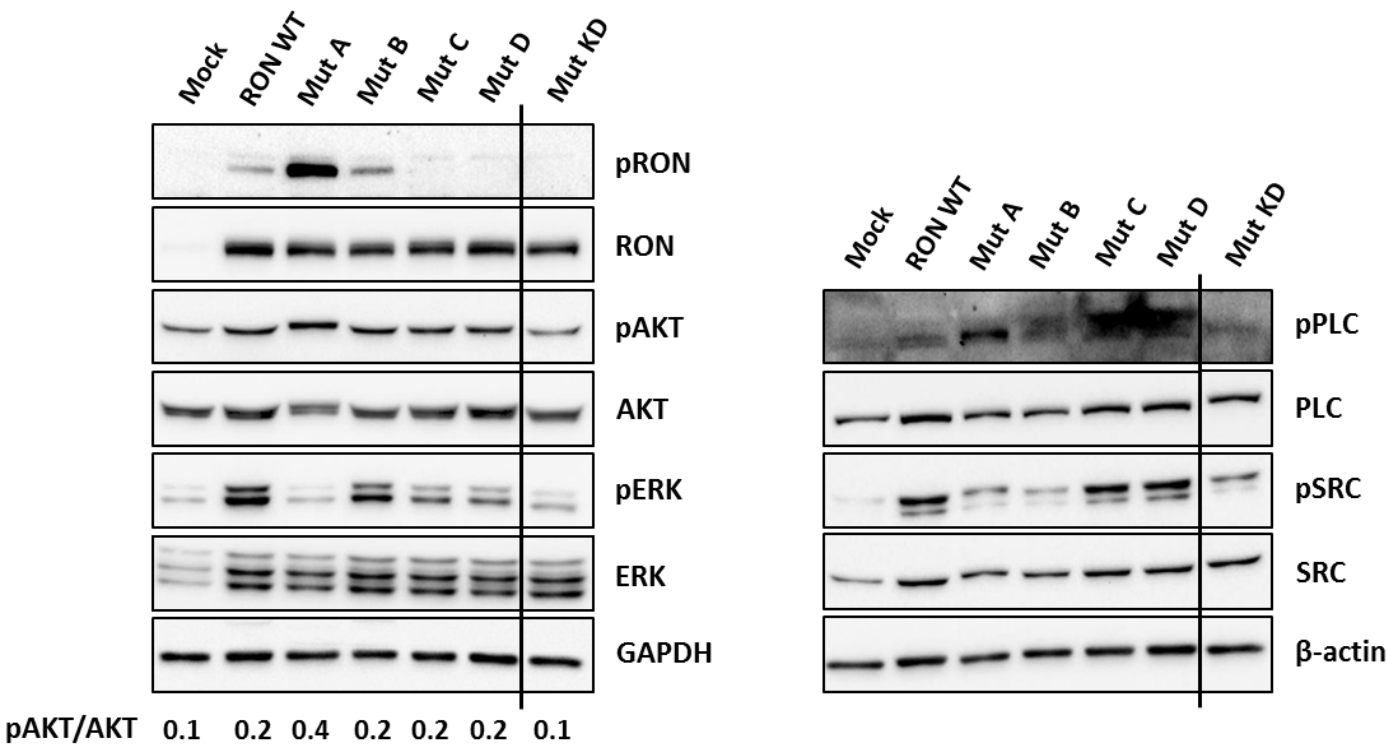
Supplementary Figure S6. RON signals through the PI3K/mTORC1 pathway in TNBC cell lines regardless of type of activation, similar to T47D ER+ cell line. A) Representative Western blots showing status of mTORC1 pathway components in SUM-159PT cells engineered for RON overexpression. SUM-159PT cells were infected with RON in the Tet-inducible lentiviral backbone, and treated with 500 ng/ml of doxycycline for 48 h. Western blot shows increased phosphorylation of p70S6K and rpS6 upon overexpression of RON. B) Signaling analysis of SUM-159PT-RON cells using inhibitors of PI3K, MAPK, and mTORC1 pathway. SUM-159PT-RON cells were cultured in the presence of doxycycline (500 ng/ml) for 48 h, followed by 4 h treatment with NVP-BKM120, PD0325901, and rapamycin, as inhibitors of PI3K, MAPK, and mTORC1 pathways respectively. Results show PI3K is the main kinase upstream of mTORC1, responsible for phosphorylation of rpS6 in a MSP-independent condition, similar to T47D-RON cells. C) Signaling analysis of HCC-1143 TNBC cells with high endogenous level of RON in the presence or absence of MSP. HCC-1143 cells were serum starved for 24 h, and treated with RON inhibitor ASLAN002 for 4 h followed by MSP stimulation (100 ng/ml) for 30 min. Western blot shows robust activation of AKT, p70S6K, and rpS6 as a result of RON activation, which is reversed by ASLAN002. GAPDH was used as loading control.

Supplementary Figure S7



Supplementary Figure S7. mTORC1 is required for RON-mediated colony formation and migration in T47D-RON cells. A-C) Effect of mTORC1 inhibition on colony formation of T47D-RON cells. Doxycycline-treated (500 ng/ml) T47D-RON cells were seeded at very low density, treated $\pm$ Rapamycin (50 nM) every other day and were allowed to form colonies, followed by Crystal Violet staining. Representative images are shown in panel A. Number and average area per colony, quantified by ImageJ are shown in panel B & C. D and E) Effect of mTORC1 inhibition on migration of T47D-RON cells. Doxycycline-treated (500 ng/ml) T47D-RON cells were seeded at high density the day before wounding, and were treated with Rapamycin (50 nM) every other day. The rate of wound closure in each group over the course of treatment is shown in panel D. Representative images at day 5 is shown in panel E. The initial wound in each group is shown in blue/green; migrating cells from the initial wound are shown in blue. Scale bars represent 300 μ m. Data are shown as mean $\pm$ SEM, $n=7$.

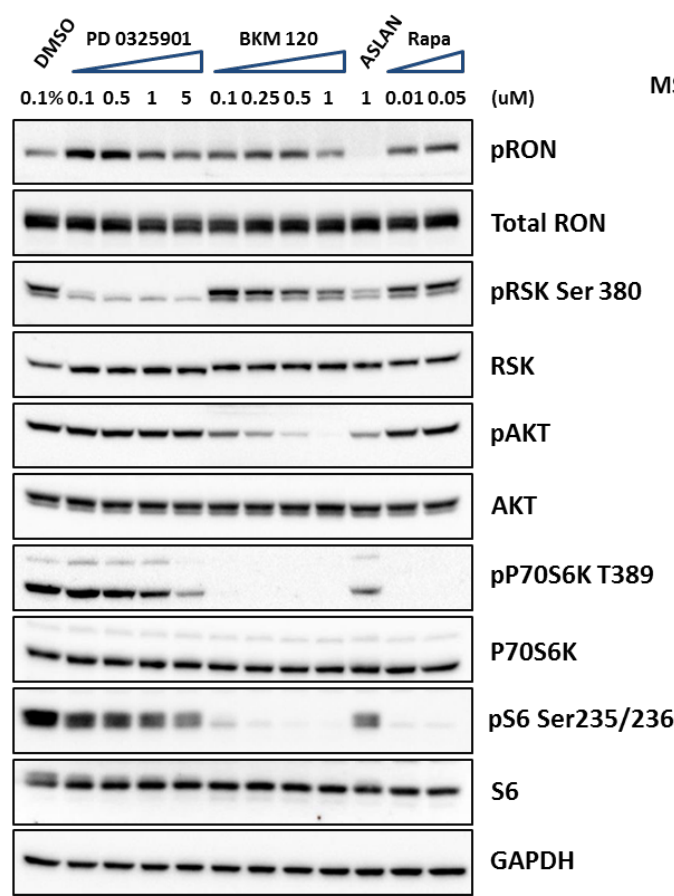
Supplementary Figure S8



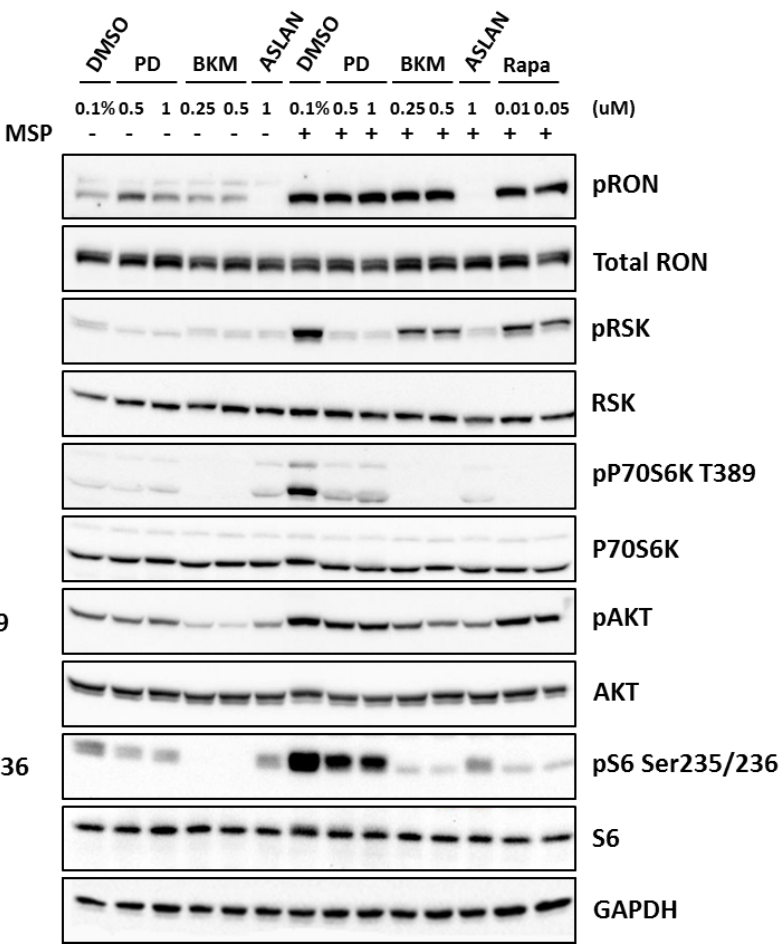
Supplementary Figure S8. Signaling analysis of RON wild type and mutants in T47D cells. T47D cells infected with RON WT and different mutants were treated with 100 ng/ml of doxycycline for 48 h and cultured in serum starved media for 24 h. Differential activation of downstream signaling pathways is shown among RON mutants. The ratio of pAKT to AKT is shown for different mutants, which indicates more AKT activation in Mut A compared to other RON mutants. GAPDH and β -actin were used as internal controls.

Supplementary Figure S9

A

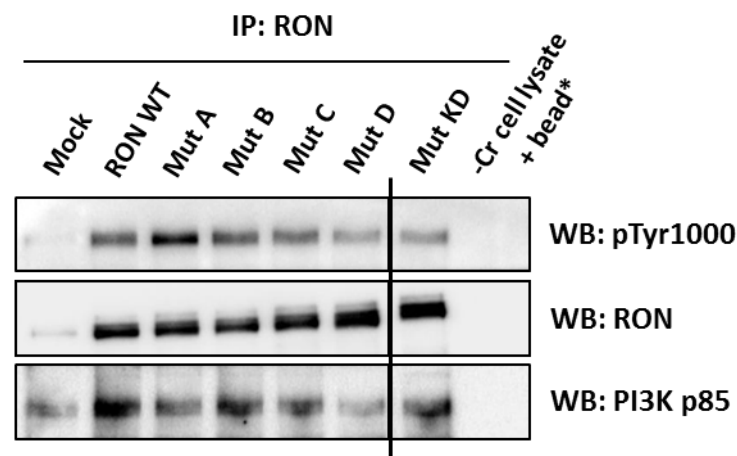


B



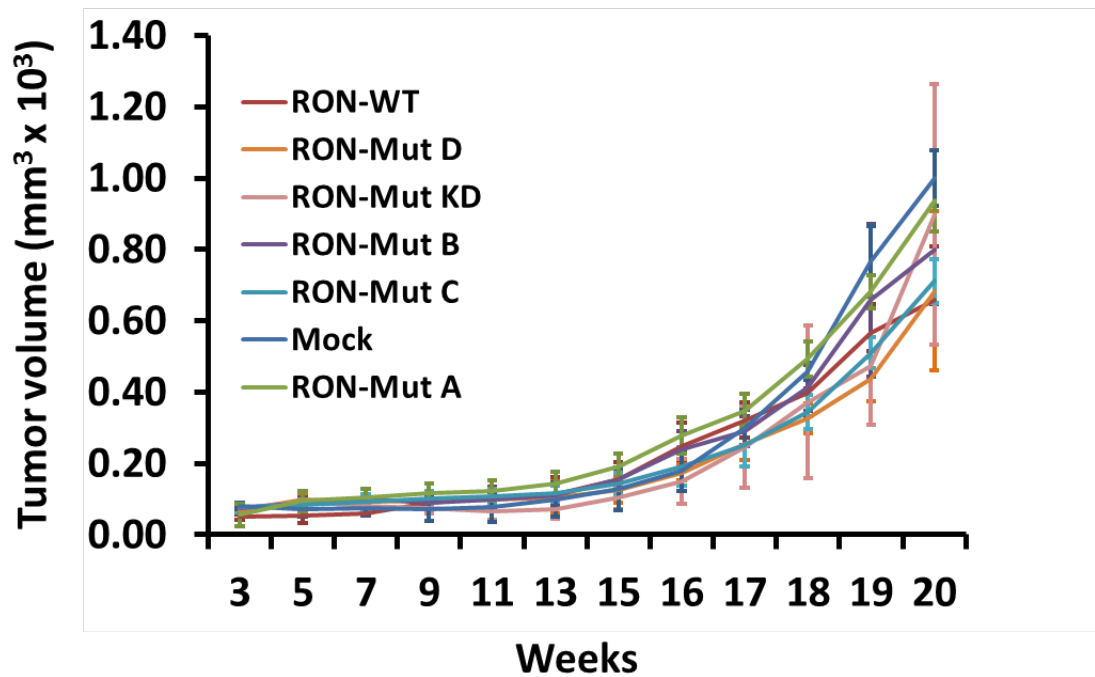
Supplementary Figure S9. RON-Mut A Signals strongly through mTORC1 downstream of PI3K in both MSP-dependent and –independent types of RON activation. A) T47D-RON-Mut A cells were cultured in the presence of doxycycline (500 ng/ml) for 48 h, followed by 4 h treatment with NVP-BKM120 and PD0325901, as inhibitors of PI3K and MAPK pathways respectively. As is shown, RON-Mut A signals strongly through p70S6K/rpS6 which is fed by mTORC1 downstream of PI3K. B) Representative Western blots showing PI3K inhibition in T47D-RON Mut A cells causes dramatic decrease of rpS6 phosphorylation downstream of mTORC1. T47D-RON Mut A cells were cultured in the presence of doxycycline (50 ng/ml) for 48 h, and in serum-starved media for 24 h. Cells were then treated with various doses of inhibitors for 4 h, followed by MSP stimulation (100 ng/ml) for 30 min. GAPDH was used as loading control.

Supplementary Figure S10



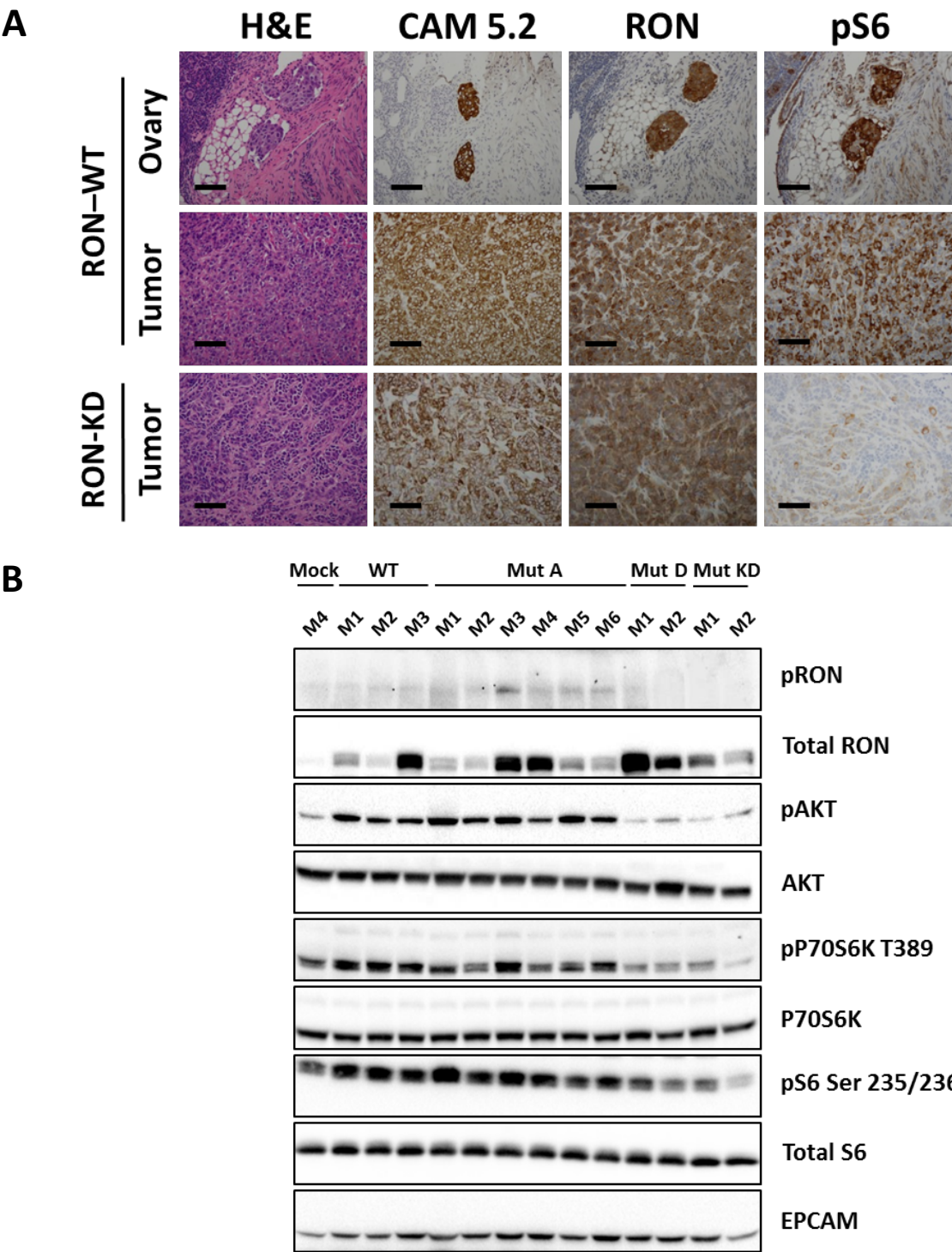
Supplementary Figure S10. Co-immunoprecipitation analysis of RON wild type and mutants in T47D cells. T47D cells infected with RON WT or different mutants were treated with 100 ng/ml of doxycycline for 48 h and cultured in serum starved media for 24 h. Lysates were immunoprecipitated with RON antibody and probed for PI3K p85, the regulatory subunit of PI3K. Phosphorylation and expression status of RON among the mutants are shown by pTyr1000 and RON antibodies.

Supplementary Figure S11



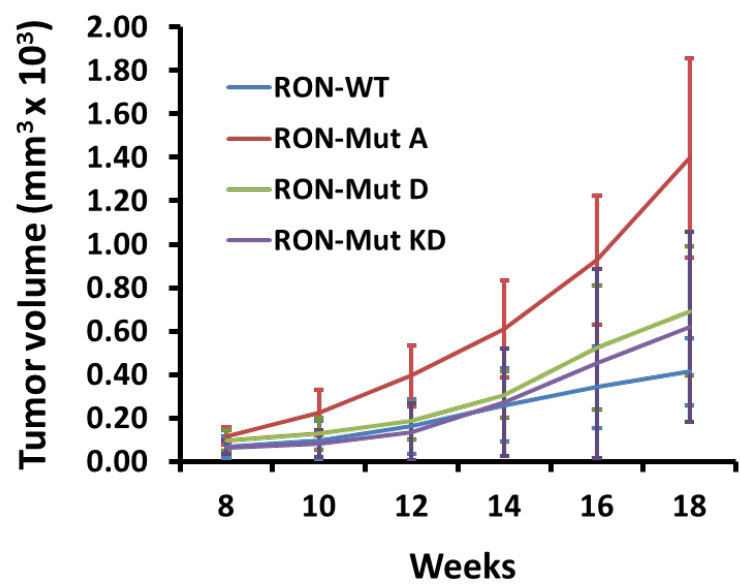
Supplementary Figure S11. Growth rate of T47D tumors expressing various RON mutants in NOD-SCID mice. Graph represents growth rate of tumors from T47D-RON WT and different mutants following orthotopic transplantation into NOD/SCID mice.

Supplementary Figure S12



Supplementary Figure S12. Phosphorylation status of rpS6 and components of the PI3K/mTORC1 pathway in T47D tumors expressing RON WT or mutants. A) Representative images showing H&E, CAM5.2, RON and pS6 (Ser 235/236) staining for metastatic RON-WT (ovary metastasis and primary tumor) vs non-metastatic RON-Mut KD (primary tumor) following orthotopic transplantation into NOD/SCID mice. Scale bars represent 100 μ m. B) Western blot shows status of pAKT, pP70 and pS6 in lysates from primary tumors of T47D-RON WT, RON- Mut A, Mut D and Mut KD. EPCAM was used as loading control, as an epithelial marker for human tumors.

Supplementary Figure S13



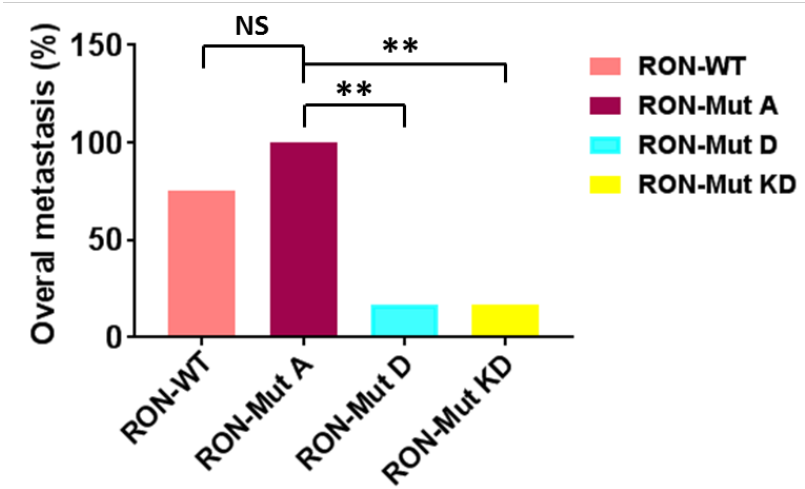
Supplementary Figure S13. Tumor growth rate of RON WT and mutants in hMSP-NOD-SCID mice. Graph represents growth rate of tumors from T47D-RON WT, RON Mut A, Mut D, and Mut KD following orthotopic transplantation into hMSP-NOD/SCID mice (n= 6-8 per group). The difference in tumor growth rate between different groups was not statistically significant (One-Way ANOVA, Multiple comparisons).

Supplementary Figure S14

A

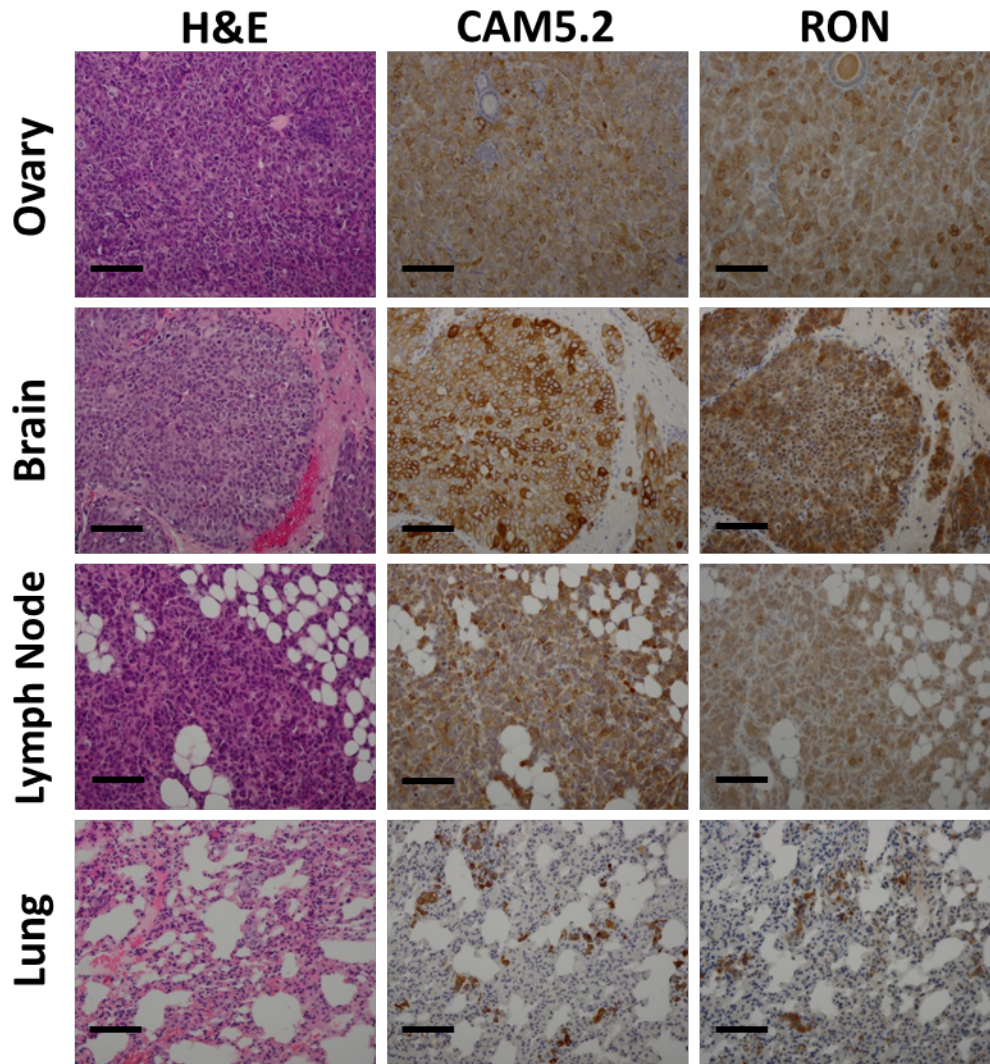
	WT	Mut A	Mut D	Mut KD
Tumor volume average (mm ³)	1408.0	1487.0	1385.0	1493.0
Overall Metastasis	6/8 (75%)	8/8 (100%)	1/6 (16.7%)	1/6 (16.7%)

B



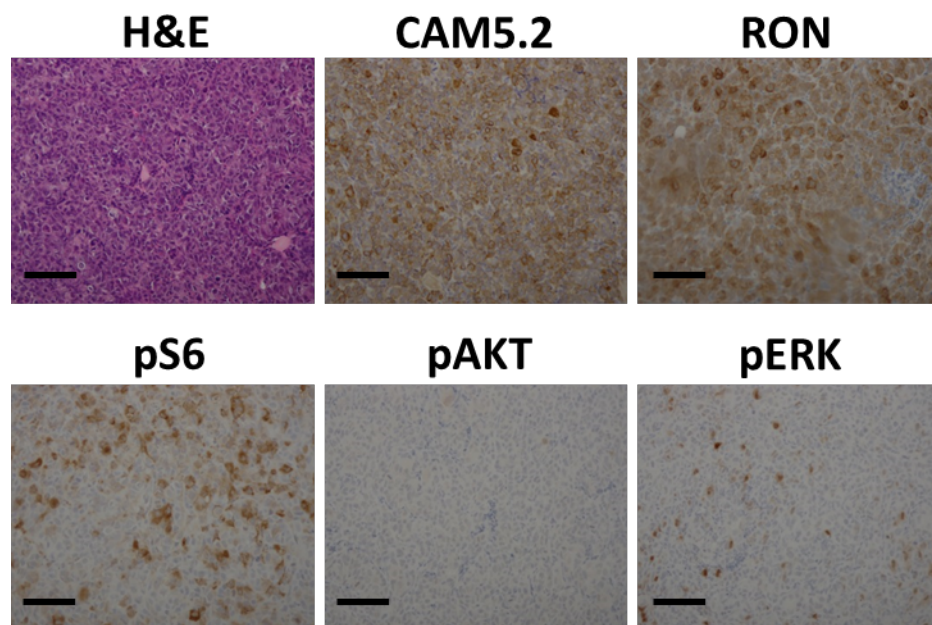
Supplementary Figure S14. Effect of hMSP on metastatic burden from tumors expressing RON WT or mutants. A) Average tumor volume at the endpoint, number of mice in each group, and overall metastasis frequency from tumors expressing different RON mutants in hMSP mice are provided. B) Quantification of overall spontaneous metastasis in hMSP-NOD/SCID mice following transplantation of RON WT, RON Mut A, Mut D, and Mut KD is shown. ***P*<0.005, NS, non-significant (Chi-square analysis, Fisher's exact test).

Supplementary Figure S15



Supplementary Figure S15. Tissue tropism of metastasis in NSG mice following tail vein injection of T47D-RON cells as an experimental metastasis model. Representative images showing H&E, RON, and CAM5.2 staining on metastatic lesions in different organs of NSG mice following tail vein injection of T47D-RON cells. Mice were put on doxy diet (50 ppm) upon injection of cells to induce RON expression. Scale bars represent 100 μ m.

Supplementary Figure S16



Supplementary Figure S16. Phosphorylation status of AKT and ERK in metastatic lesions induced by T47D-RON cells in vehicle-treated mice. Representative images indicate basal phosphorylation level of rpS6, AKT, and ERK in metastatic ovaries of mice treated with vehicle. Treatment with vehicle started at day 30 following tail vein injection of T47D-RON cells and continued until the end of the experiment. Representative images are from mice euthanized at week 4 of treatment. Scale bars represent 100 μm .

Supplementary Table S1.

Protein name	-MSP	+MSP	+MSP +ASLAN	<i>p</i> -value
rpS6-pS235/S236	0.52	1.58	0.42	0.00035
rpS6-pS240/S244	0.59	1.43	0.38	0.000447
MAPK-pT202/Y204	0.95	2.20	0.92	0.000447
PDCD4	0.95	1.67	0.60	0.010733
Akt-pS473	1.10	1.69	0.94	0.016835
p90RSK-pT573	0.95	1.46	0.98	0.000447
p70S6K-pT389	0.89	1.15	0.86	0.006453
Collagen-VI	1.00	1.47	1.44	0.086766

Supplementary Table S1. List of proteins whose phosphorylation or expression significantly changed upon MSP stimulation. Absolute values of significantly changed proteins or phosphoproteins with >1.5 fold change in response to MSP treatment in T47D-RON cells are shown. Data represents mean ± SEM (P values by unpaired, two-sided t-test).

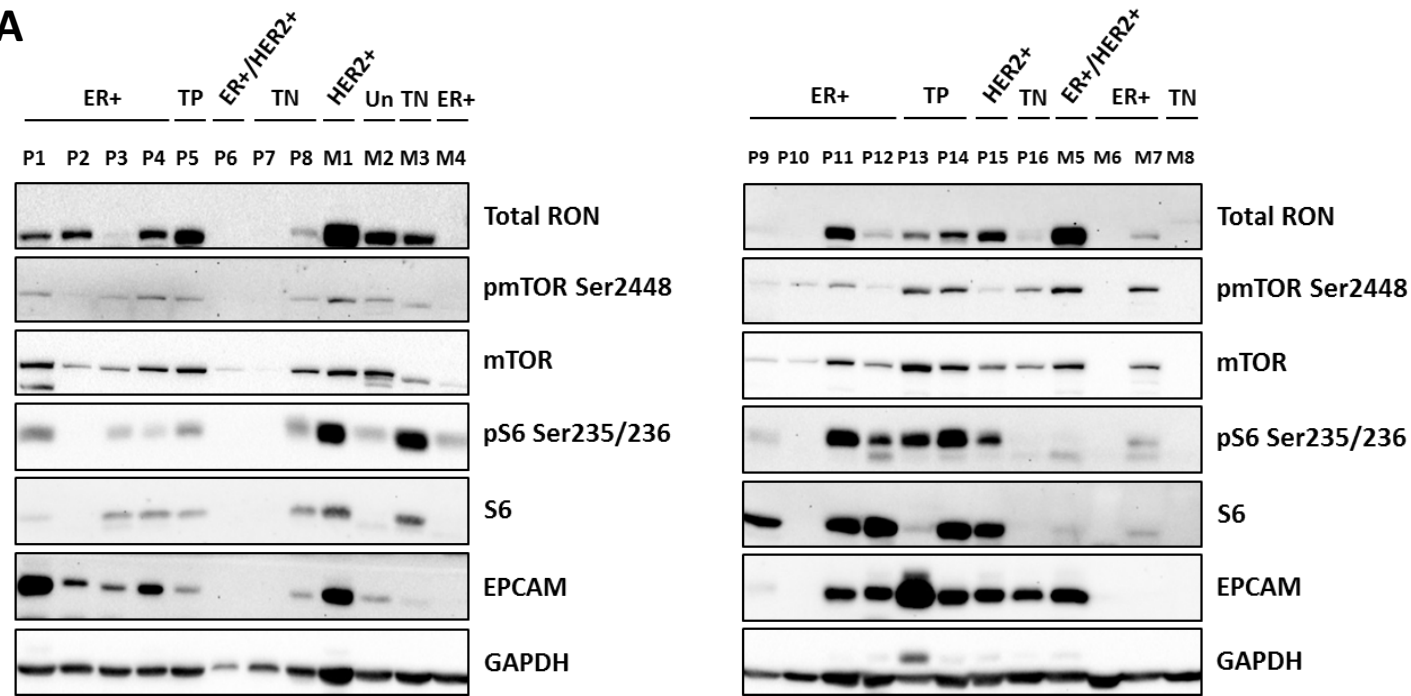
Supplementary Table S2.

	Mock	WT	Mut A	Mut B	Mut C	Mut D	Mut KD
Tumor volume average (mm³)	1394.00	1421.0	1582.0	1464.0	1285.0	1350.0	1323.0
Overall Metastasis	1/8 (12%)	6/14 (43%)	8/8 (100%)	4/11 (36%)	6/13 (46%)	1/19 (5.3%)	1/17 (5.9%)
Liver	1/1	2/6	6/8	2/4	3/6	1/1	1/1
Lung	0/1	2/6	6/8	3/4	4/6	1/1	0/1
Brain	0/1	0/6	1/8	1/4	0/6	0/1	0/1
Spleen	1/1	1/6	5/8	1/4	3/6	1/1	1/1
Ovary	1/1	4/6	6/8	3/4	5/6	1/1	1/1
Kidney	0/1	4/6	5/8	3/4	5/6	1/1	0/1
Lymph nodes	0/1	1/6	2/8	0/4	1/6	0/1	0/1

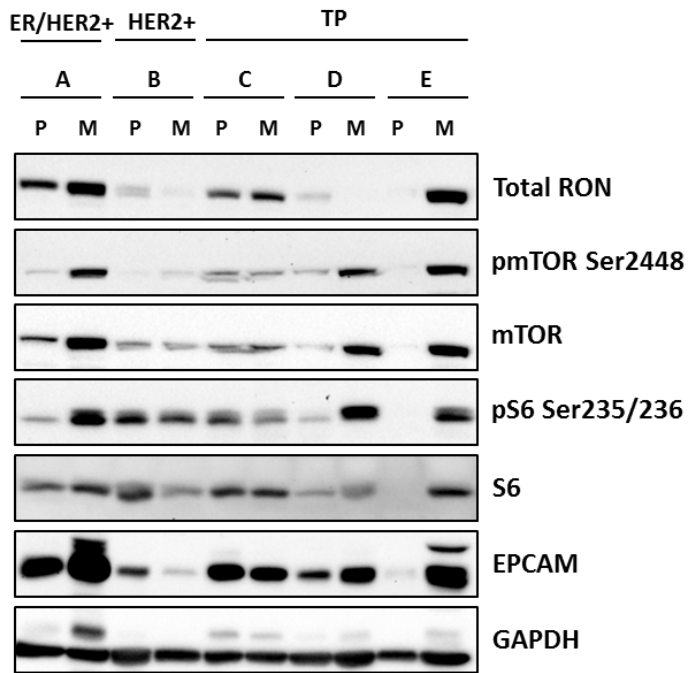
Supplementary Table S2. Gross analysis of metastasis in mice bearing RON mutant tumors. Average tumor volume at the endpoint, number of mice in each group, and tissue tropism of metastasis for different RON mutants are provided.

Supplementary Figure S17

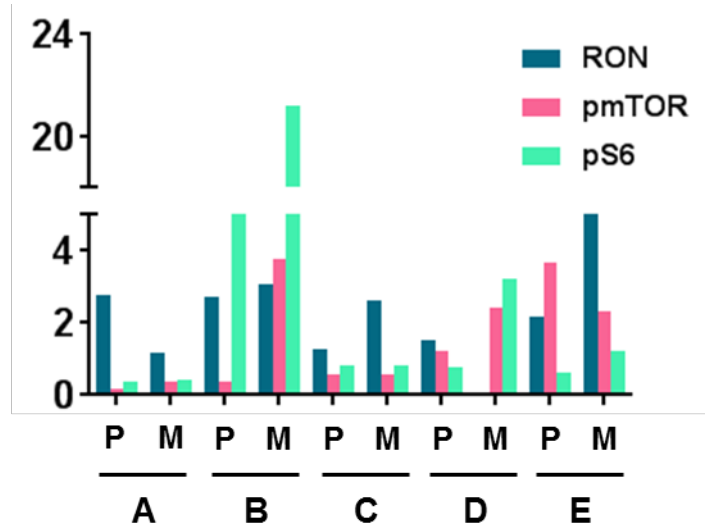
A



B

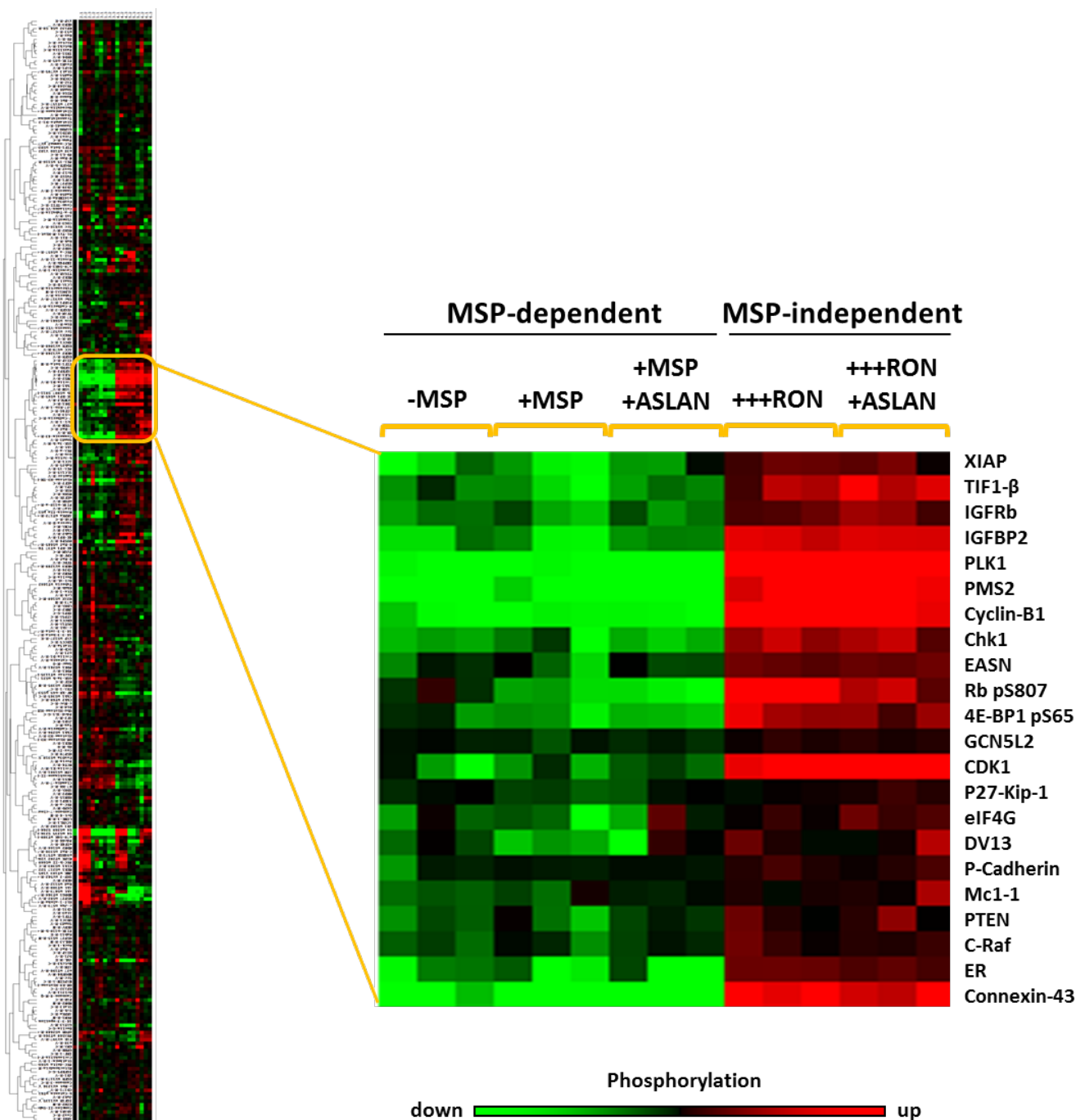


C



Supplementary Figure S17. Status of RON expression and mTORC1 signaling in human primary and metastatic breast tumors. A) Representative Western blots showing presence of RON and phosphorylated mTORC1 and mTORC1 substrate, rpS6, across different subtypes of unmatched primary and metastatic breast tumors. B) Status of RON and mTORC1 signaling in matched primary and metastatic tumors. EPCAM was used as an epithelial marker to indicate presence of cancer cells, and GAPDH was used as loading control. C) Quantification of RON, pmTOR, and pS6 levels normalized to EPCAM from samples in B. ER, Estrogen receptor, TP: Triple positive (ER/PR/HER2), TN: Triple negative, Un: Unknown.

Supplementary Figure S18



Supplementary Figure S18. Clustered heatmap of 305 proteins is shown for T47D-RON cells in MSP-dependent and -independent conditions and with or without the RON inhibitor ASLAN002. An enlarged cluster of proteins that are particularly changed due to presence or absence of serum are shown on the right.

raw RPPA data[illegible]

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Complex I	ND3	U00001	ND3	ND3	1014	11.5	5.5	+	1.0	Complex I	ND3	U00001	ND3	ND3	1014	11.5	5.5	+	1.0
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Complex I	ND8	U00001	ND8	ND8	1014	11.5	5.5	+	1.0	Complex I	ND8	U00001	ND8	ND8	1014	11.5	5.5	+	1.0
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Complex I	ND11	U00001	ND11	ND11	1014	11.5	5.5	+	1.0	Complex I	ND11	U00001	ND11	ND11	1014	11.5	5.5	+	1.0
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Complex I	ND34	U00001	ND34	ND34	1014	11.5	5.5	+	1.0	Complex I	ND34	U00001	ND34	ND34	1014	11.5	5.5	+	1.0
Complex I	ND35	U00001	ND35	ND35	1014	11.5	5.5	+	1.0	Complex I	ND35	U00001	ND35	ND35	1014	11.5	5.5	+	1.0
Complex I	ND36	U00001	ND36	ND36	1014	11.5	5.5	+	1.0	Complex I	ND36	U00001	ND36	ND36	1014	11.5	5.5	+	1.0
Complex I	ND37	U00001	ND37	ND37	1014	11.5	5.5	+	1.0	Complex I	ND37	U00001	ND37	ND37	1014	11.5	5.5	+	1.0
Complex I	ND38	U00001	ND38	ND38	1014	11.5	5.5	+	1.0	Complex I	ND38	U00001	ND38	ND38	1014	11.5	5.5	+	1.0
Complex I	ND39	U00001	ND39	ND39	1014	11.5	5.5	+	1.0	Complex I	ND39	U00001	ND39	ND39	1014	11.5	5.5	+	1.0
Complex I	ND40	U00001	ND40	ND40	1014	11.5	5.5	+	1.0	Complex I	ND40	U00001	ND40	ND40	1014	11.5	5.5	+	1.0
Complex I	ND41	U00001	ND41	ND41	1014	11.5	5.5	+	1.0	Complex I	ND41	U00001	ND41	ND41	1014	11.5	5.5	+	1.0
Complex I	ND42	U00001	ND42	ND42	1014	11.5	5.5	+	1.0	Complex I	ND42	U00001	ND42	ND42	1014	11.5	5.5	+	1.0
Complex I	ND43	U00001	ND43	ND43	1014	11.5	5.5	+	1.0	Complex I	ND43	U00001	ND43	ND43	1014	11.5	5.5	+	1.0
Complex I	ND44	U00001	ND44	ND44	1014	11.5	5.5	+	1.0	Complex I	ND44	U00001	ND44	ND44	1014	11.5	5.5	+	1.0
Complex I	ND45	U00001	ND45	ND45	1014	11.5	5.5	+	1.0	Complex I	ND45	U00001	ND45	ND45	1014	11.5	5.5	+	1.0
Complex I	ND46	U00001	ND46	ND46	1014	11.5	5.5	+	1.0	Complex I	ND46	U00001	ND46	ND46	1014	11.5	5.5	+	1.0
Complex I	ND47	U00001	ND47	ND47	1014	11.5	5.5	+	1.0	Complex I	ND47	U00001	ND47	ND47	1014	11.5	5.5	+	1.0
Complex I	ND48	U00001	ND48	ND48	1014	11.5	5.5	+	1.0	Complex I	ND48	U00001	ND48	ND48	1014	11.5	5.5	+	1.0
Complex I	ND49	U00001	ND49	ND49	1014	11.5	5.5	+	1.0	Complex I	ND49	U00001	ND49	ND49	1014	11.5	5.5	+	1.0
Complex I	ND50	U00001	ND50	ND50	1014	11.5	5.5	+	1.0	Complex I	ND50	U00001	ND50	ND50	1014	11.5	5.5	+	1.0
Complex I	ND51	U00001	ND51	ND51	1014	11.5	5.5	+	1.0	Complex I	ND51	U00001	ND51	ND51	1014	11.5	5.5	+	1.0
Complex I	ND52	U00001	ND52	ND52	1014	11.5	5.5	+	1.0	Complex I	ND52	U00001	ND52	ND52	1014	11.5	5.5	+	1.0
Complex I	ND53	U00001	ND53	ND53	1014	11.5	5.5	+	1.0	Complex I	ND53	U00001	ND53	ND53	1014	11.5	5.5	+	1.0
Complex I	ND54	U00001	ND54	ND54	1014	11.5	5.5	+	1.0	Complex I	ND54	U00001	ND54	ND54	1014	11.5	5.5	+	1.0
Complex I	ND55	U00001	ND55	ND55	1014	11.5	5.5	+	1.0	Complex I	ND55	U00001	ND55	ND55	1014	11.5	5.5	+	1.0
Complex I	ND56	U00001	ND56	ND56	1014	11.5	5.5	+	1.0	Complex I	ND56	U00001	ND56	ND56	1014	11.5	5.5	+	1.0
Complex I	ND57	U00001	ND57	ND57	1014	11.5	5.5	+	1.0	Complex I	ND57	U00001	ND57	ND57	1014	11.5	5.5	+	1.0
Complex I	ND58	U00001	ND58	ND58	1014	11.5	5.5	+	1.0	Complex I	ND58	U00001	ND58	ND58	1014	11.5	5.5	+	1.0
Complex I	ND59	U00001	ND59	ND59	1014	11.5	5.5	+	1.0	Complex I	ND59	U00001	ND59	ND59	1014	11.5	5.5	+	1.0
Complex I	ND60	U00001	ND60	ND60	1014	11.5	5.5	+	1.0	Complex I	ND60	U00001	ND60	ND60	1014	11.5	5.5	+	1.0
Complex I	ND61	U00001	ND61	ND61	1014	11.5	5.5	+	1.0	Complex I	ND61	U00001	ND61	ND61	1014	11.5	5.5	+	1.0
Complex I	ND62	U00001	ND62	ND62	1014	11.5	5.5	+	1.0	Complex I	ND62	U00001	ND62	ND62	1014	11.5	5.5	+	1.0
Complex I	ND63	U00001	ND63	ND63	1014	11.5	5.5	+	1.0	Complex I	ND63	U00001	ND63	ND63	1014	11.5	5.5	+	1.0
Complex I	ND64	U00001	ND64	ND64	1014	11.5	5.5	+	1.0	Complex I	ND64	U00001	ND64	ND64	1014	11.5	5.5	+	1.0
Complex I	ND65	U00001	ND65	ND65	1014	11.5	5.5	+	1.0	Complex I	ND65	U00001	ND65	ND65	1014	11.5	5.5	+	1.0
Complex I	ND66	U00001	ND66	ND66	1014	11.5	5.5	+	1.0	Complex I	ND66	U00001	ND66	ND66	1014	11.5	5.5	+	1.0
Complex I	ND67	U00001	ND67	ND67	1014	11.5	5.5	+	1.0	Complex I	ND67	U00001	ND67	ND67	1014	11.5	5.5	+	1.0
Complex I	ND68	U00001	ND68	ND68	1014	11.5	5.5	+	1.0	Complex I	ND68	U00001	ND68	ND68	1014	11.5	5.5	+	1.0
Complex I	ND69	U00001	ND69	ND69	1014	11.5	5.5	+	1.0	Complex I	ND69	U00001	ND69	ND69	1014	11.5	5.5	+	1.0
Complex I	ND70	U00001	ND70	ND70	1014	11.5	5.5	+	1.0	Complex I	ND70	U00001	ND70	ND70	1014	11.5	5.5	+	1.0
Complex I	ND71	U00001	ND71	ND71	1014	11.5	5.5	+	1.0	Complex I	ND71	U00001	ND71	ND71	1014	11.5	5.5	+	1.0
Complex I	ND72	U00001	ND72	ND72	1014	11.5	5.5	+	1.0	Complex I	ND72	U00001	ND72	ND72	1014	11.5	5.5	+	1.0
Complex I	ND73	U00001	ND73	ND73	1014	11.5	5.5	+	1.0	Complex I	ND73	U00001	ND73	ND73	1014	11.5	5.5	+	1.0
Complex I	ND74	U00001	ND74	ND74	1014	11.5	5.5	+	1.0	Complex I	ND74	U00001	ND74	ND74	1014	11.5	5.5	+	1.0
Complex I	ND75	U00001	ND75	ND75	1014	11.5	5.5	+	1.0	Complex I	ND75	U00001	ND75	ND75	1014	11.5	5.5		

V306
CF 0.93539503 0.99765639 0.99908042 0.85816182 1.01932608 1.07415407 0.9643216 0.98119395 1.00575692 0.90889623 1.03754238 1.05063742 0.91998689 1.07558912 1.0947771