

Fig. S1. RIP140 expression in PM transfected with WT or Y3F mutant RIP140 followed by control or LPS stimulation.

Protein and mRNA level were analyzed by western blotting and qPCR.

Fig. S2. Knockdown efficiency. The expression level of RIP140 protein (left) and mRNA (right) in WT and M ϕ RIP140KD mice. .

Fig. S3. Altering RIP140 levels affects IL4-induced M2 activation.

a qPCR analyses of M2 gene expression in control vs. RIP140 over-expressing (left) or RIP140-knockdown (right) mouse PM, with or without IL-4 stimulation. **b** Arginase activity assay. Data are presented as urea concentrations, and show three experimental results (means \pm s.d.). Student's t-test (n=3) was used (*P<0.05; **P<0.01; ***P<0.001).

Fig. S4. In vitro wound healing assay.

In vitro wound healing was assessed using 293TN cells supplemented with conditioned medium of macrophage with RIP140 over-expression (Flag-RIP140) or RIP140-knockdown (RIP140siRNA). IL-4 was used to trigger M2 polarization of macrophages whereas Ctrl denotes treatment with a control solution without cytokine.

Fig. S5. Bone marrow transplantation efficiency validation.

GFP intensity in bone marrow cells of the receiving mice following BMT with cells carrying either Ctrl-GFP, WT-RIP140 or Y3F-RIP140.

Fig. S6. Non-degradable mutant RIP140 (RIP140-Y3F) can also translocate to the cytosol in macrophages upon IL-4 treatment.

a Western blot analyses of FLAG-RIP140 Y3F from total cell lysate in IL-4 (10ng/ml) treated RAW264.7 cells. **b** Western blot analyses of FLAG-RIP140 Y3F from the cytosolic/nuclear fractions of IL-4 (10ng/ml) treated RAW264.7 cells. Data show three experimental results.

Fig. S7. Endogenous RIP140 translocates to the cytosol in macrophages upon IL-13 treatment.

a Western blot analyses of endogenous RIP140 from total cell lysate in IL-13 (10ng/ml) treated RAW264.7 cells. **b** Western blot analyses of RIP140 from the cytosolic/nuclear fractions of IL-13 (10ng/ml) treated RAW264.7 cells. Data show three experimental results.

Fig. S8. RIP140 knockdown still increases pSTAT6 levels in macrophages treated with kinase inhibitor, but fails to affect PTP1B gene expression.

a Western blot analyses of phospho-STAT6 and STAT6 levels in IL-4-treated (10ng/ml) control vs. RIP140 knockdown RAW264.7 cells, with or without pretreatment with kinase inhibitor Staurosporine (50ng/ml). **b** qRT-PCR analyses of RIP140 and PTP1B mRNA levels in control (flag) vs. RIP140-overexpressing RAW264.7 cells with or without IL-4 treatment (10ng/ml, 6hr). Data show three experimental results (means±s.d.). Student's t-test (n=3) was used (*P<0.05; **P<0.01; ***P<0.001).

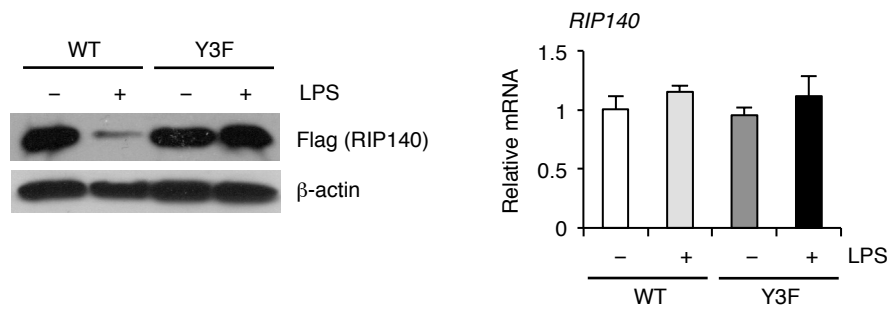


Fig. S1 Lin et al.

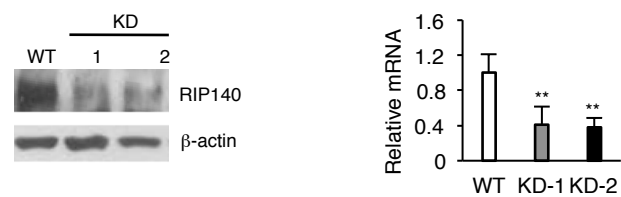


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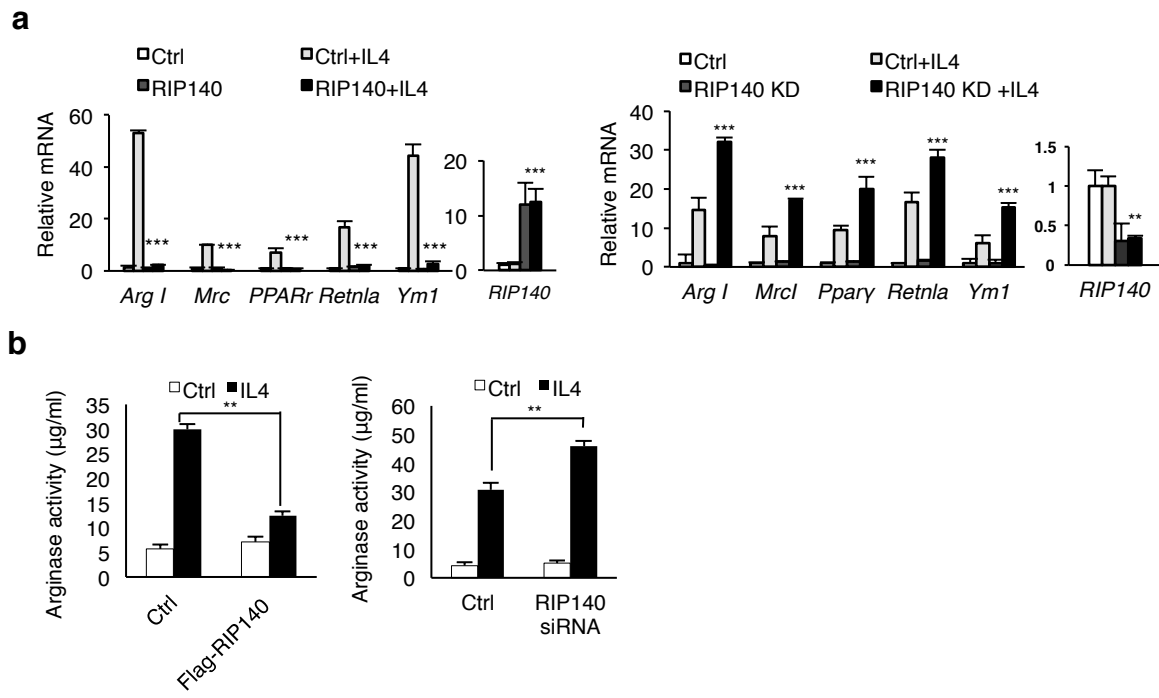


Fig. S3 Lin et al.

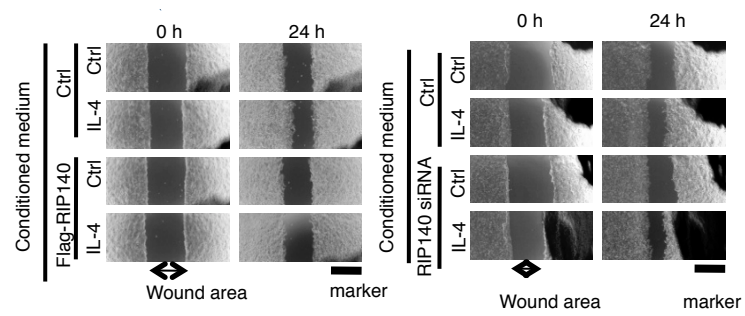


Fig. S4 Lin et al.

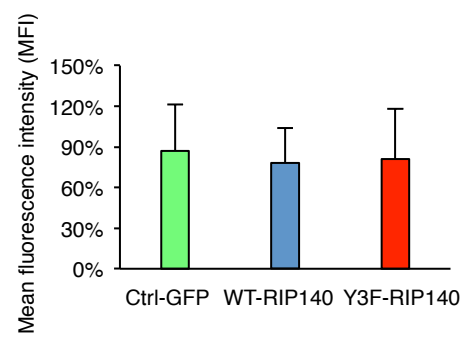


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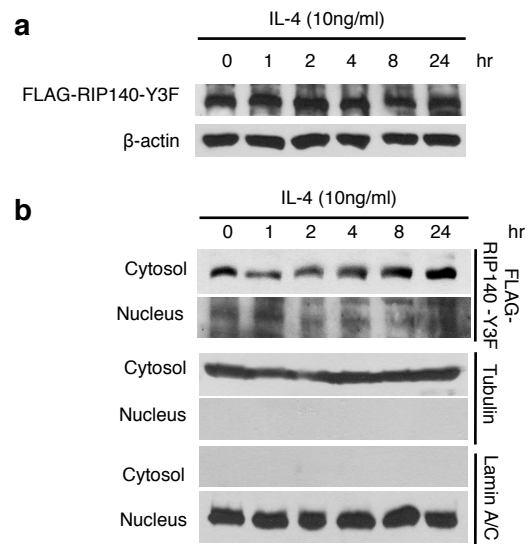


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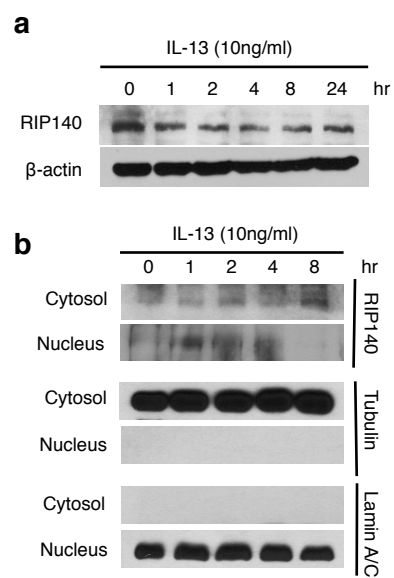
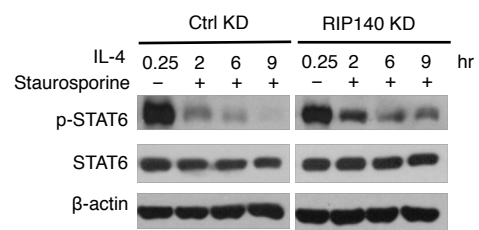
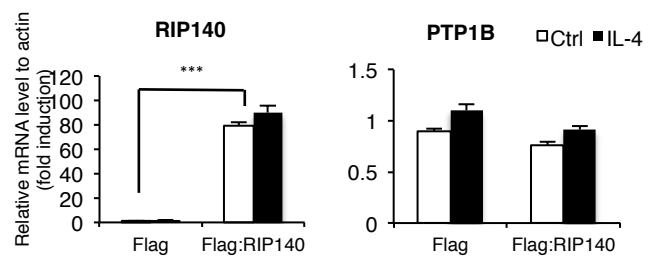


Fig. S7 Lin et al.

a**b****Fig. S8 Lin et al.**