Supporting Information

Gallop et al. 10.1073/pnas.1305286110

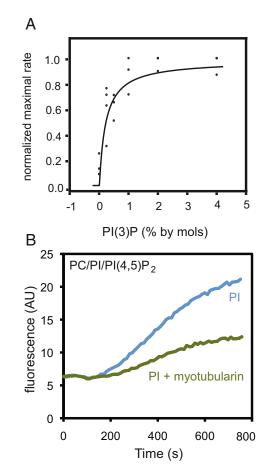


Fig. S1. (*A*) Dose–response of actin polymerization by pyrene actin assay from liposomes with phosphatidylinositol 3-phosphate [PI(3)P] and PI(4,5)P₂. (*B*) Pyrene actin assay on addition of 48% pheochromocytoma (PC), 47% PI, and 5% $PI(4,5)P_2$ liposomes in extracts that were incubated with 3-phosphatase myotubularin showing a role for 3-kinase activity.

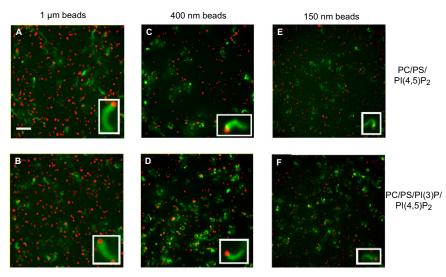


Fig. 52. (A-F) Representative images of actin structures (green) growing at the surface of glass beads coated with membranes containing PI(4,5)P₂ only or PI(4,5)P₂ and PI(3)P (red). (*Insets*) Enlargements of actin structures. (Scale bar, 10 μ m.)

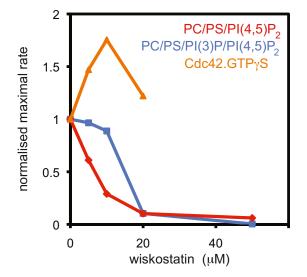


Fig. S3. Titration of neural Wiskott–Aldrich syndrome protein (N-WASP) inhibitor wiskostatin showing that it can completely inhibit actin polymerization from $PI(4,5)P_2$ and also $PI(3)P/PI(4,5)P_2$ liposomes. Wiskostatin stabilizes the autoinhibited confirmation of N-WASP, which is relieved by binding to cell cycle division 42 (Cdc42).GTP (1). Actin polymerization stimulated by Cdc42.GTP is refractory to wiskostatin. $PI(3)P/PI(4,5)P_2$ -stimulated actin polymerization is still sensitive to wiskostatin, again suggesting that PI(3)P is not acting via increasing Cdc42 GEF activity.

1. Peterson JR, et al. (2004) Chemical inhibition of N-WASP by stabilization of a native autoinhibited conformation. Nat Struct Mol Biol 11(8):747–755.

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