Supplemental material

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Christis and Munro, http://www.jcb.org/cgi/content/full/jcb.201107115/DC1

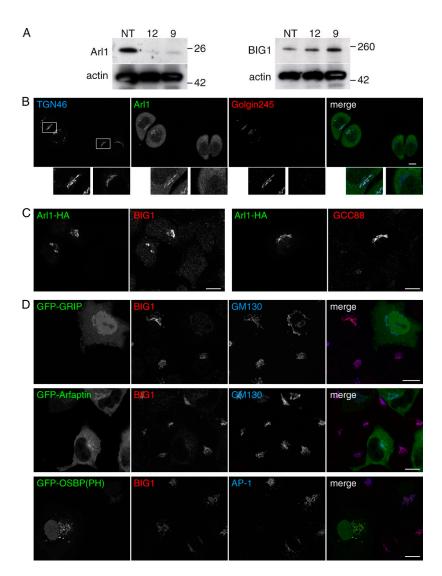


Figure S1. Arl1 recruits BIG1 to the Golgi in mammalian cells. (A) Immunoblots of total cell proteins prepared from HeLaM cells treated with control siRNAs (NT) or two different siRNAs against Arl1 (12 and 9). Blots were probed for Arl1, BIG1, or actin as a loading control. Molecular mass is indicated in kilodaltons. (B) Confocal micrographs of HeLaM cells treated with siRNA against Arl1 and mixed 50:50 with untreated cells before plating on slides for staining with antibodies to the indicated endogenous proteins. Representative Golgi regions in the boxed areas are shown magnified in the insets. (C) Confocal micrographs of HeLaM cells treated with siRNA against Arl1 and transfected with an HA-tagged siRNA-resistant Arl1 and then stained with antibodies against the HA tag and either BIG1 or the GRIP domain protein GCC88. The red channel is overexposed to show the untransfected cells where the proteins remain displaced from the Golgi. (D) Confocal micrographs of COS cells expressing the indicated GFP fusion proteins and stained with antibodies against BIG1 and the indicated Golgi markers. GFP-GRIP contains the C-terminal 82 residues of human golgin-245, GFP-arfaptin contains the entire protein, and GFP-OSBP is the PH domain of human OSBP. The Arl1 effectors GRIP and arfaptin displace BIG1 from the Golgi, but the PtdIns(4)P-binding PH domain does not. Bars, 15 μm.

Table S1 is provided as an Excel file and lists the mass spectrometric peptides from proteins bound to liposomes coated with GDP- and GTP-bound forms of *Drosophila* Arf1, Arf4, Arl1, and ARFRP1.