Figure legend for the supplementary figures

Figure S1. Dissection of the Rabaptin5–GAT interface (**A**) Stereo view of GAT-binding site of the Rabaptin5 homodimer. Backbones of the two helices are shown in ribbon representation, and colored in cyan and green, respectively. Residues directly involved in Rabaptin5 binding (with a 3.6-Å cutoff) are shown in stick models and colored in yellow (carbon atoms), blue (nitrogen), red (oxygen), and magenta (sulfur). Carbon atoms in other residues are colored as the same as the backbone (**B**) Stereo view of the three-helix bundle of GGA1 GAT domain. The three helices, $\alpha_2-\alpha_4$, are labeled as A2, A3, and A4 and colored in white. Representation and color schemes are the same as in S1A. The mobile loop connecting α_2 and α_3 is represented putatively by a thin curve. The orientation is similar to that of Fig. 2B.

Figure S2. Schematic representation of the GAT-Rabaptin5 interface. Residue pairs of hydrogen bonds (<3.2 Å) are connected with solid lines. Water molecules are depicted as orange dots. Otherwise, pairs of van de Waals interactions (<3.6 Å) are connected with dash-lines. Residues from different structural motifs are clustered in boxes of different colors. Names of the residues with mutations shown to be deleterious for the binding are colored in red.

Figure S3. Schematic diagram of putative interactions between GGA and some partner proteins. A Rabaptin5 dimer is shown in green. The N-terminal VHS domain of GGA binds to the cytosolic sorting signal peptide of receptors or an auto-inhibition peptide in the hinge region. The GAT domain interacts with membrane-bound ARF as well as Rabaptin5 coiled coil homodimer region (residues 551-575). The GAE domain interacts with the Fxx Φ motif in Rabaptin5. Some of the illustrated interactions may not co-exist; they can be either competitive or sequential.