Activated ADP-ribosylation Factor Assembles Distinct Pools of Actin on Golgi Membranes*

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The small GTP-binding protein ADP-ribosylation factor (ARF) has been shown to regulate the interaction of actin and actin-binding proteins with the Golgi apparatus. Here we report that ARF activation stimulates the assembly of distinct pools of actin on Golgi membranes. One pool of actin cofractionates with coatomer (COPI)-coated vesicles and is sensitive to salt extraction and the plus end actin-binding toxin cytochalasin D. A second ARF-dependent actin pool remains on the Golgi membranes following vesicle extraction and is insensitive to cytochalasin D. Isolation of the salt-extractable ARF-dependent actin from the Golgi reveals that it is bound to a distinct repertoire of actin-binding proteins. The two abundant actin-binding proteins of the ARF-dependent actin complex are identified as spectrin and drebrin. We show that drebrin is a specific component of the cytochalasin D-sensitive, ARF-dependent actin pool on the Golgi. Finally, we show that depolymerization of this actin pool with cytochalasin D increases the extent of the salt-dependent release of COPI-coated vesicles from the Golgi following cell-free budding reactions. Together these data suggest that regulation of the actin-based cytoskeleton may play an important role during ARF-mediated transport vesicle assembly or release on the Golgi.

Extensive biochemical and genetic studies have allowed a detailed description of the molecular events surrounding the assembly of coated transport vesicles (1, 2). In particular, GTPases of the ARF family act as molecular switches to trigger the assembly of the coat proteins. The ARF-dependent oligomerization of coat proteins has been shown to be sufficient to deform the membrane into a vesicle bud (3, 4). Despite this progress, many details have yet to emerge regarding the mechanisms of cargo packaging, the regulation of vesicle formation, and the nature of interactions with molecular motors and other cytoskeletal proteins that allow directed movement.

Although ARF is best characterized for its role in vesicle formation, recent studies suggest it may serve additional functions in the cell. One role for ARF may be to regulate the activity of the phospholipid-modifying enzymes phospholipase D, phosphatidylinositol 4-kinase, and phosphatidylinositol 5-kinase (5–7). This regulation may play a role in vesicle release and/or be part of signaling pathways not directly related to transport. A second role for ARF may be in directing the assembly of the actin-based cytoskeleton. ARF has been shown to regulate the binding of spectrin, ankyrin, and actin to the Golgi membranes (8, 9). ARF6 has been implicated in rearranging the actin cytoskeleton at the plasma membrane (7, 10, 11). ARF isoforms may also play a similar role in yeast (12).

There is considerable evidence that the actin cytoskeleton and myosin motors are important for Golgi function in the cell (13, 14). Recent studies show that actin is likely to be a component of Golgi-derived transport vesicles (15, 16). In this study, centrinactin, tropomyosin isoforms, and myosin isoforms were found to be selectively associated with distinct classes of Golgi-derived vesicles. Some isoforms of spectrin and ankyrin localize to the Golgi apparatus, suggesting that this is a site of their action (9, 17, 18). The actin cytoskeleton may also play a role in Golgi morphology and positioning (19).

Here we present evidence that at least two distinct pools of actin are assembled on the Golgi membrane upon ARF activation. This actin affects the release of COPI-coated vesicles from the Golgi and regulates the association of spectrin and the actin-binding protein drebrin with the membrane. The implications of these findings for the ability of COPI-coated vesicles to mediate multiple trafficking steps and for the role of the cytoskeleton in Golgi function will be discussed.

**EXPERIMENTAL PROCEDURES**

**Materials—** Rat liver Golgi membranes and bovine brain cytosol were isolated as described previously (20). The following antibodies were used in this study: M3A5 (21), anti-actin (Sigma), anti-drebrin (Medical & Biological Laboratories Co.), anti- e-COP (22), anti-KDEL receptor (23).

**Golgi Binding Assays—** The final reaction conditions included 25 mM HEPES (pH 7.2), 2.5 mM magnesium acetate, 15 mM potassium chloride, 0.2 mM succrose, Golgi membranes (0.2 mg/ml), bovine brain cytosol (1.0 mg/ml), and an ATP-regenerating system. The incubations were carried out for 20 min at 37 °C, and the final reaction volume was 1 ml. When specified, brefeldin A, cytochalasin D, or latrunculin A was added at the indicated concentration. For reactions containing brefeldin A, the membranes and cytosol were preincubated for 10 min at 37 °C with the toxin or with a solvent control. For "float-up" binding assays, the membranes were isolated following the incubation by centrifugation at 15,000 × g for 30 min at 4 °C in a refrigerated microcentrifuge. The membranes were then resuspended in 50 μl of 45% (w/w) sucrose in 25 mM HEPES (pH 7.2), 25 mM KCl and placed into a 7 × 20-mm ultracentrifugation tube. The sample was overlaid with 125 μl of 35% (w/w) sucrose in 25 mM HEPES (pH 7.2), 25 mM KCl and then 25 μl of 15% (w/w) sucrose in 25 mM HEPES (pH 7.2), 25 mM KCl. The sample was spun at 100,000 rpm for 30 min in a TLA-100 rotor (Beckman Instruments). A 100-μl sample containing the Golgi membranes was removed from the top of the step gradient, and the membrane proteins were

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‡ The abbreviations used are: ARF, ADP-ribosylation factor; SDS-PAGE, SDS-polyacrylamide gel electrophoresis; GTPγS, guanosine 5′-3-O-(thio)triphosphate.

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precipitated by the addition of trichloroacetic acid to 10%. The pellet from the step gradient and the TCA precipitate were analyzed by SDS-PAGE and Western blotting. For two-stage incubations the conditions were just as described above except that following the first incubation, the membranes were resolated by microcentrifugation and incubated with a second reaction mixture prior to the float-up centrifugation.

**COPI-coated Vesicle Budding Reactions**—Unless otherwise indicated, the final budding reaction conditions were identical to those used for binding reactions except that the reaction volume was 2.0 ml. Following the incubation, the Golgi membranes were isolated by centrifugation at 15,000 × g for 15 min and washed one time with LSSB (25 mM HEPES (pH 7.2), 2.5 mM magnesium acetate, 50 mM potassium chloride, 0.2 mM sucrose). The vesicles were stripped from the membrane by incubating in HSSB (25 mM HEPES (pH 7.2), 2.5 mM magnesium acetate, 250 mM potassium chloride, 0.2 mM sucrose) for 10 min on ice. Following the incubation, the membranes were centrifuged at 15,000 × g for 15 min. Where indicated, the pellet containing the Golgi remnants was resuspended in Laemmli sample buffer for the Western blot analysis. The supernatant was loaded onto a 35% (w/v) sucrose cushion (25 mM HEPES (pH 7.2), 2.5 mM magnesium acetate, 250 mM potassium chloride, 35% (w/v) sucrose) and centrifuged at 350,000 × g for 30 min. The 35% sucrose pellets were resuspended in Laemmli sample buffer and analyzed by Western blotting.

**Western Blotting**—Proteins were fractionated using SDS-PAGE and blotted onto polyvinylidene difluoride membranes using standard protocols for the Bio-Rad minigel and blotting apparatuses. Following the transfer, the membranes were dried and incubated with appropriate dilutions of the antibodies. The signal was visualized using horseradish peroxidase-conjugated secondary antibodies (Bio-Rad) and ECL (Amershall Pharmacia Biotech). Where indicated, the signals were quantitated using densitometry.

**Polypeptide Sequence Determination and Mass Spectrometry**—Identification by mass spectrometry was done by cutting protein bands from SDS-PAGE gels and digesting the material with 0.2 μg of trypsin (24). The resulting peptide mixture was then loaded onto a 2-μl bed volume of POROS 50 R2 (PerSeptive Biotechnologies, Foster City, CA) reversed-phase beads (packed into an Eppendorf gel-loading tip) and eluted stepwise with 4 μl of 16% (and then with 4 μl of 30%) acetonitrile, 0.1% formic acid (25). The “16%” and “30%” peptide pools were each analyzed twice by matrix-assisted laser desorption/ionization time of flight mass spectrometry in the presence and absence of peptide calibrants (25) using a REFLEX III (Bruker-Franzen, Bremen, Germany) instrument equipped with a gridless pulsed extraction ion source and a 2-GHz digitizer and operated in reflectron mode. Spectra were obtained by averaging multiple signals. After recalibration with internal standards, monoisotopic masses were assigned for the most prominent peaks, and a peptide mass list was generated to search a protein non-redundant database (National Center for Biotechnology Information, Bethesda, MD) using the PeptideSearch (26) algorithm with an accuracy requirement of 40 ppm. For the identification of the 280-kDa band, 55 of 63 experimental masses matched human α-spectrin, and 15 of the remaining 28 masses matched human β-spectrin (random matches, 12 of 63). For the identification of the 120-kDa band, 8 of 13 experimental masses matched human drebrin (random matches, 4 of 13).

**RESULTS**

While characterizing the protein components of Golgi-derived transport vesicles, we observed that non-muscle actin is a major GTPγS-dependent protein constituent of both Golgi membranes and the Golgi-derived vesicles following incubations with cytosol (data not shown). This finding, together with recent reports indicating that ARF regulates the levels of actin, spectrin, and ankyrin on Golgi membranes (9) and that actin-binding proteins associate with vesicles (15, 16), led us to begin dissecting the nature and role of actin on the Golgi apparatus. As a first step, we developed a cell-free Golgi binding assay that allowed actin polymerization to be assayed independently of Golgi binding (Fig. 1, see “Experimental Procedures”). In short, Golgi membranes were incubated with cytosol under conditions known to promote coated vesicle formation. Following the incubation, the membranes were resuspended in 45% sucrose and then overlaid with 35% sucrose in a centrifuge tube. Because Golgi membranes have a buoyant density equivalent to about 30% sucrose, they float to the top of the gradient during ultracentrifugation (Fig. 1A, G fraction). By contrast, large cytoskeletal polymers pellet at the bottom of the centrifuge tube (Fig. 1A, P fraction). The presence of the Golgi membranes in the G fraction is confirmed by blotting for the cis-Golgi marker, KDEL receptor (Fig. 1A).

In the absence of GTPγS, the Golgi membranes bound neither to G-actin, present in the cytosol (Fig. 1A, lanes 1 and 2), nor to F-actin, assembled by the addition of the actin-polymerizing toxin phalloidin (Fig. 1A, lanes 5 and 6). In the presence of phalloidin, the F-actin was completely resolved from the Golgi membranes on the step gradient. By contrast, when GTPγS was included in the incubation (Fig. 1A, lanes 3 and 4), 50–80% of the actin was present in the G fraction. The GTPγS-dependent binding of actin to the Golgi was completely inhibited by the addition of brefeldin A, indicating that the GTPγS effect is mediated by ARF (Fig. 1B). Brefeldin A inhibits nucleotide exchange for ARF1 but not for ARF6 or the rho family of GTPases (27, 28), indicating that the brefeldin A-sensitive actin binding we observe is distinct from the modifications of the actin cytoskeleton involving these GTPases. If either cytosol or the membranes are omitted from the reaction, no actin appears in the G fraction (Fig. 1C). These findings suggest that actin is assembled directly by Golgi membranes upon activation of ARF with GTPγS.

**Fig. 1. Actin binding to Golgi membranes**. A, Golgi membranes were incubated with cytosol, and GTPγS and phalloidin were added to the reaction where indicated. Following the incubation, the reaction was loaded onto a float-up gradient. Shown is a Western blot of the top Golgi fraction (G) and the bottom pellet fraction (P) from the gradients. The blot was probed for actin and the Golgi marker, KDEL receptor. B, a Western blot probed with the anti-actin antibody of the top Golgi fractions from float-up Golgi binding. Shown are the results from duplicate incubations containing brefeldin A (BFA, 200 μM) or the methanol solvent alone as indicated. C, a Western blot of the top Golgi fraction probed with antibodies against actin and the cotransfected subunit ε-COP. The incubations were devoid of cytosol, Golgi membranes, or GTPγS as indicated.
them to assemble actin, we used two-stage binding reactions (Fig. 2). In the first stage, cytosol, GTPγS, and membranes were incubated either with or without the actin monomer-binding toxin latrunculin A to block actin assembly. Fig. 2 shows that upon reisolation of the membranes following the first stage, actin bound in a GTPγS-dependent manner and that this binding was completely blocked by the presence of latrunculin A. If the reisolated Golgi membranes were then incubated with cytosol in a second stage without GTPγS and latrunculin A, actin was found to be present on the membranes provided that GTPγS had been included in stage 1. Actin assembled on the Golgi without GTPγS in stage 2 even though actin assembly had been blocked by latrunculin A in stage 1. This result supports the model that actin assembles directly on the Golgi in the presence of membrane-bound activated ARF, as opposed to a model whereby the Golgi membranes associate with actin that had been previously assembled through a membrane-independent but GTPγS-dependent mechanism. Because the 21-kDa ARF proteins are the major small GTPases to associate with the Golgi membranes when incubated with GTPγS (29) and no additional G proteins are activated in stage 2, this result also provides strong additional evidence that the observed GTPγS effects on actin assembly are ARF-mediated.

Because ARF triggers coated vesicle assembly and the conditions used above for ARF-dependent actin binding are known to bud COPI-coated vesicles from the Golgi, we wished to examine whether the ARF-dependent actin pool was involved in vesicle assembly or release. Following cell-free budding reactions, COPI-coated vesicles are typically stripped from the membrane with 250 mM KCl and then purified using an isopycnic sucrose gradient (20) or by pelleting through a sucrose cushion (30). We find that much of the ARF-dependent actin is extracted from the Golgi with 250 mM KCl (as are COPI-coated vesicles) and pellets through a 35% cushion during ultracentrifugation (Fig. 3). This finding could indicate that the ARF-dependent actin is directly associated with the vesicles.

In addition to the actin that cofractionated with the vesicles, we observed that some actin is resistant to the salt extraction and remained on the Golgi membranes left behind after removing the vesicles (Fig. 3, Golgi remnants). Surprisingly, although the appearance of actin in the vesicle-enriched 35% sucrose pellet was completely blocked by the addition of the plus-end-binding toxin cytochalasin D, the levels of actin on the Golgi remnants were largely unaffected by the presence of this toxin (Fig. 3). Blotting for the vesicle marker ε-COP revealed that the levels of Golgi membrane and vesicles were unaffected by the toxin treatment. Tubulin levels, both on the Golgi remnants and in the 35% sucrose pellet, were also unaffected by GTPγS or cytochalasin D, indicating that these reagents did not have effects on microtubule assembly or disassembly. The actin

![Diagram showing ARF-bound Golgi assemble actin independently of additional G protein activation.](http://www.jbc.org/)
FIG. 4. Actin-binding proteins associated with GTPγS versus phalloidin-polymerized actin. Shown is a Coomassie blue-stained gel of the isolated actin pools. Lanes 1 and 2 show the 35% sucrose pellet from budding reactions containing Golgi membranes, cytosol, and GTPγS. 20 μm cytochalasin D (CytD) was present in the incubation shown in lane 1 and absent in the incubation shown in lane 2. Lanes 3 and 4 show actin isolated from incubations carried out under standard budding reaction conditions except that Golgi membranes and GTPγS were omitted from the reaction. Phalloidin was included in the reaction shown in lane 3. The actin pellet was extracted with 250 mM KCl and precipitated with 10% trichloroacetic acid. The reaction size was 5 ml.

**FIG. 5.** Drebrin is selectively associated with the cytochalasin D-sensitive actin. Budding reactions were carried out in the presence or absence of 50 μM GTPγS, 20 μM cytochalasin D (CytD), or 200 μM brefeldin A (BfA) as indicated. Following the incubation the Golgi membranes were stripped with 250 mM KCl, and the Golgi remnants and salt extract were fractionated by SDS-PAGE. Shown are Western blots probed with antibodies against drebrin or actin.

**DISCUSSION**

We describe an ARF-dependent salt extractable actin pool assembled from whole cytosol with a remarkably simple protein composition. One of the two major actin-binding proteins of this complex was identified as a mixture of α- and β-spectrin, confirming previous studies showing an ARF-mediated interaction between spectrin and the Golgi (8). The second protein was identified as drebrin. Previously, drebrin had been characterized as a neuron-specific protein that may play a role in neuronal development. In particular, it has been localized to dendrites and may play a role in dendrite outgrowth. If drebrin were restricted to neuronal dendrites, it would be unlikely that it plays a general role in Golgi transport or other vesicular trafficking steps. Several findings indicate that this is not the case. First, we find by Western blot analysis that a protein identical in size to brain cytosol drebrin is recognized by the drebrin antibody in Chinese hamster ovary cells (data not shown), indicating that drebrin or a closely related isoform is
present in non-neuronal cells. Second, other recent studies have found drebrin to be present in several non-neuronal tissues (34, 35). Finally, a homolog of drebrin, ABP1, is present in the yeast Saccharomyces cerevisiae. ABP1 interacts genetically with genes encoding proteins required for endocytosis (33). This finding, together with the results we report here, raises the possibility that drebrin is involved in vesicular trafficking. Additional studies will be required to determine the precise role of drebrin or drebrin-related proteins in these processes.

The effects of cytochalasin D on the salt-dependent extraction of COPI-coated vesicles could indicate a role for actin in the fission reaction or vesicle release. It is, however, equally likely that actin plays a role in a vesicle tethering or docking event that is not directly related to the fission reaction. In this respect, it is interesting that similar results have been obtained when the function of the GM130/giantin complex is compromised. This complex is thought to tether vesicles to the Golgi after they are formed (36) or to dock vesicles to a target organelle prior to SNARE-mediated fusion (37, 38). Although additional studies will be required to establish the precise role of actin in these trafficking steps, the cytochalasin D effects indicate that the ARF-dependent actin plays a role in the interaction of COPI vesicles with the Golgi and therefore is likely to function during COPI-mediated trafficking to, from, or within the Golgi.

It is of interest to speculate why pools of actin with distinct properties are assembled on the Golgi. One possibility is that the different pools of actin reflect different classes of assembling vesicles. ARF triggers the assembly of both COPI-coated and AP1/clathrin-coated vesicles on the Golgi apparatus (39, 40). It is possible that ARF directs the assembly of distinct actin pools for different classes of vesicles. Additional evidence supporting this model comes from the recent finding that different classes of Golgi-derived vesicles associate with distinct actin-binding proteins (15). A second possibility is that ARF directs the assembly of different actin pools on different types of membranes. For example, actin assembled on cis-Golgi membranes may have different properties from actin assembled on trans-Golgi membranes. Assembling actin with specific properties on each type of membrane or at each class of vesicle assembly site could direct the overall organization, positioning, or morphology of these organelles. Alternatively or in addition, it could be involved in conferring cytoskeleton-dependent directionality and targeting to the transport process.

Targeting proteins in the cell almost certainly requires specific interactions with molecular motor proteins and cytoskeletal elements to move them in the proper direction to the correct location in the cell. In some instances, the same class of vesicle coat can be used for multiple transport steps; for example, COPI-coated vesicles appear to be used for anterograde and retrograde trafficking from Golgi cisternae as well as in the endocytic pathway (41–45). Therefore, it is highly probable that there is cellular machinery in addition to the coat proteins that specify interactions with the cytoskeleton. The ARF-mediated changes in the actin cytoskeleton may be part of this machinery. Thus ARF-regulated vesicle assembly may not only involve the directed oligomerization of coat proteins but also additional complex regulation of cytoskeletal components to ensure that the assembled vesicle moves along a specific cytoskeletal filament with the correct molecular motor and thus arrives at the proper destination in the cell.

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