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REVIEW

Actin acting at the Golgi

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Abstract The organization, assembly and remodeling of the actin cytoskeleton provide force and tracks for a variety of (endo)membrane-associated events such as membrane trafficking. This review illustrates in different cellular models how actin and many of its numerous binding and regulatory proteins (actin and co-workers) participate in the structural organization of the Golgi apparatus and in trafficking-associated processes such as sorting, biogenesis and motion of Golgi-derived transport carriers.

Keywords Golgi apparatus · Cytoskeleton · Actin · Spectrin · Myosin · Rho GTPases

Introduction

The function of the Golgi apparatus is the result of a complex interaction between the molecules that establish its architecture, those that determine protein transport and those that integrate signals from either outside or inside the cell. Cytoskeletal elements (microtubules, actin filaments or microfilaments and intermediate filaments) integrate these processes. Association and coordination between them as well as their respective binding and regulatory proteins are present in the majority of endomembrane systems, including the Golgi apparatus. While its basic function is highly conserved, the Golgi varies greatly in shape and number from one organism to another. Briefly, it ranges from dispersed cisternae or isolated tubular networks as occurs in algae, protozoa and the yeast *Saccharomyces cerevisiae*, to a pile of flattened cisternae aligned in parallel and known as the Golgi stack. This, depending on the organism examined, could be present in a single (fungi and the yeast *Pichia pastoris*) or multiple copies, the latter being scattered throughout the cytoplasm (plants and *Drosophila*) or organized as a ribbon around centrioles (vertebrates) (daSilva et al. 2004; Hawes and Satiat-Jeunemaitre 2005; He et al. 2004; He 2007; Henderson et al. 2007; Kondylis and Rabouille 2003; Ladinsky et al. 1999; Lowe 2011; Mogelsvang et al. 2003; Pelletier et al. 2002; Preuss et al. 1992; Rambourg and Clermont 1986; Rambourg et al. 2001; Ramírez and Lowe 2009; Rios and Bornens 2003; Rossanese et al. 1999). The cytoskeleton determines the location of the Golgi, and depending on the cellular model, either microtubules or actin filaments have the greater influence (Egea and Rios 2008), the impact of intermediate filaments being very limited (Gao and Sztul 2001; Gao et al. 2002; Styers et al. 2006; Toivola et al. 2005). Historically, microtubules were the first cytoskeleton element to be linked to the Golgi structure and function (Thyberg and Moskalewski 1999), and only later was it firmly established that actin and associated proteins (actin et al.) also played a significant role (for recent reviews see Brownhill et al. 2009; Egea et al. 2006; Harris and Tepass 2010; Hehnly and Stamnes 2007; Lanzetti 2007; Loubéry and Coudrier 2008; Myers and Casanova 2008; Ridley 2006; Smythe and Ayscough 2006; Soldati and Schliwa 2006). Here, we provide an up-to-date overview of the structural and transport consequences of the coupling

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between the actin-based cytoskeleton and the Golgi in a variety of cellular models that are commonly used to investigate membrane trafficking events.

Actin and co-workers in the structural organization of the Golgi apparatus

The first experimental evidence that actin and the Golgi interacted was that Golgi membranes and Golgi-derived vesicles contained actin and actin-binding proteins (Heimann et al. 1999) and that the Golgi invariably compacted when cells were treated with a variety of naturally occurring substances that perturbed the actin organization and its dynamics, which mainly include cytochalasins, latrunculins, jasplakinolide and botulinum toxins and are known generically as actin toxins or actin drugs (Fig. 1) (di Campli et al. 1999; Valderrama et al. 1998, 2000, 2001). At ultrastructural level, the compacted Golgi was seen to depend on whether actin drugs depolymerized or stabilized actin filaments, giving rise, respectively, to dilatation (Fig. 1) or fragmentation/perforation of cisternae. Moreover, these ultrastructural impairments occurred in a microtubule-independent manner, which ruled out synergic cooperation between microtubules and actin filaments controlling the shape and integrity of Golgi cisternae (Lazaro-Dieguez et al. 2006). Golgi compactness is consistently seen when actin partners present at the Golgi are perturbed, such as after the depletion of the Arp2/3 activator WASp homologue associated with actin, Golgi membranes and microtubules (WHAMM)

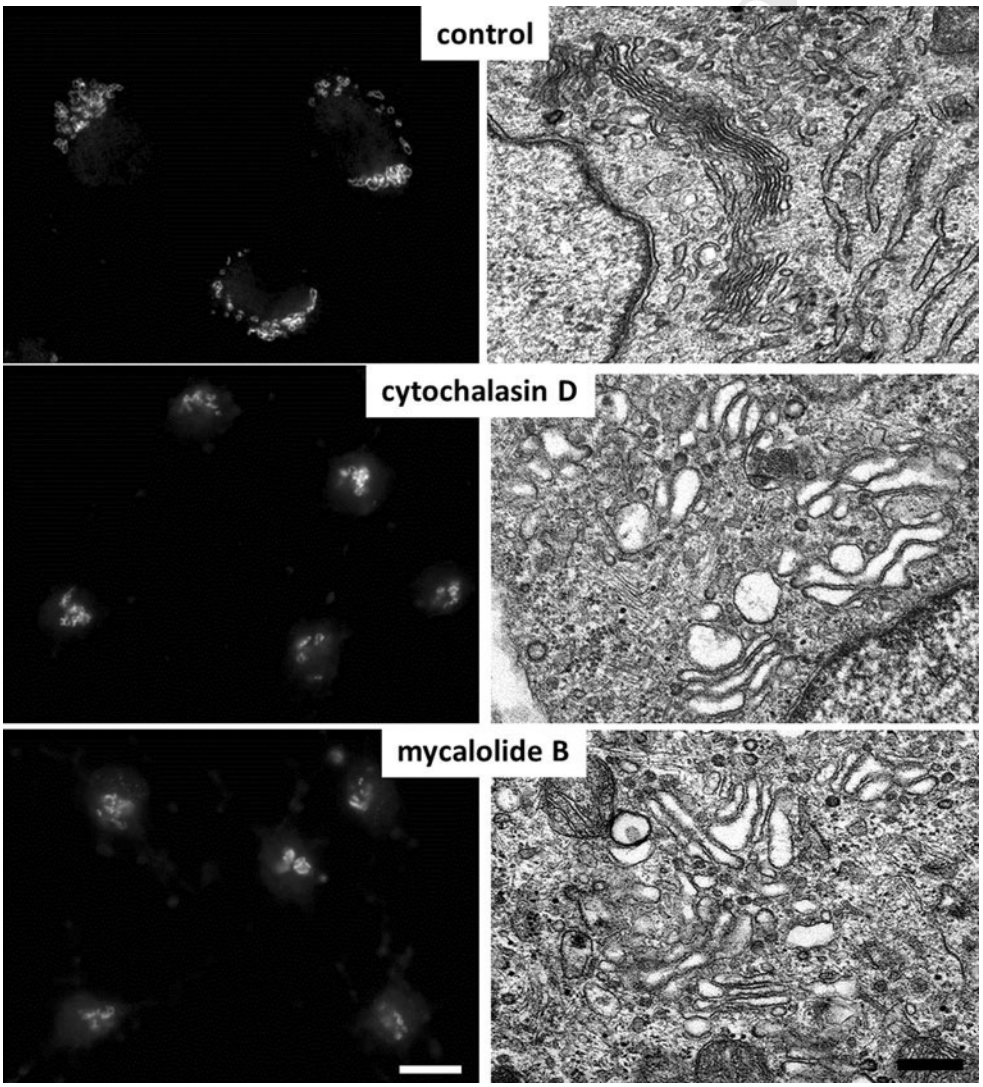

Fig. 1 Alterations in the Golgi morphology after actin cytoskeleton disruption. NRK cells treated with the filamentous-actin-depolymerizing agents cytochalasin D or mycalolide B show a compacted Golgi (stained to GM130) in contraposition with the extended one shown by untreated cells. At ultrastructural level, both actin toxins cause dilatation of cisternae and an abnormally high number of peri-Golgi vesicles. *Bar* for epifluorescence images, 10 μm; *bar* for ultrastructural images, 200 nm

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(Campellone et al. 2008), cortactin (Kirkbride et al. 2012) or myosin 18A, an unconventional myosin that connects filamentous actin to the phosphatidylinositol 4-phosphate (PI4P)-binding protein GOLPH3 (Dippold et al. 2009; Ng et al. 2013) (Fig. 2a). However, in some cases, interference with the actin machinery produces fragmentation (and dispersion) of the Golgi, which occurs after the depletion or constitutive activation of actin nucleators formin family members mDia (mammalian *Diaphanus*), the formin-like 1/FMNL1 and INF2 (Colon-Franco et al. 2011; Ramabhadran et al. 2011; Zilberman et al. 2011). Taken together, these findings reinforce the notion that the proper regulation of actin at the Golgi is necessary to maintain the structural integrity of the Golgi apparatus.

Other important cytoskeletal organization in which actin is integrated corresponds to that formed by spectrin. In red blood cells, the spectrin-based cytoskeleton determines their characteristic biconcave shape and localizes as a bidimensional network beneath the plasma membrane. Defects in major components (spectrin, ankyrin and protein 4.1) are associated with abnormal cell shape and membrane fragility (Lux 1979). By analogy with erythrocytes, the Golgi-associated spectrin skeleton could act as an extended, two-dimensional interactive platform on the cytoplasmic surface of cisternae, regulating its shape and transport functions (Beck et al. 1994; Beck and Nelson 1998; Holleran and Holzbaur 1998; Godi et al. 1998; De Matteis and Morrow 2000). While mammalian red blood cells contain only one type of spectrin tetramer (αIβI subunits), nucleated cells contain numerous isoforms of both subunits, being βIII spectrin present at the Golgi (Salcedo-Sicilia et al. 2013; Stankewich et al. 1998). Other isoforms of the spectrin-based cytoskeleton components typically present in the plasma membrane of red blood cells have also been localized in the Golgi, such as β and γ actin (Valderrama et al. 2000), ankyrins Ank_{G119} and Ank_{195} (Beck et al. 1997; Devarajan et al. 1996, 1997), protein 4.1B (Kang et al. 2009), anion exchanger AE2 (Holappa et al. 2001, 2004) and tropomyosin (Percival et al. 2004). βIII spectrin is required to maintain the characteristic Golgi architecture since its functional interference or knockdown causes fragmentation and dilation of Golgi membranes (Salcedo-Sicilia et al. 2013; Siddhanta et al. 2003). Most likely, the Golgi fragmentation is produced by the loss of the direct interaction of βIII spectrin with the dynein/dynactin motor complex subunit Arp1 (Holleran et al. 2001), and distal cisternae swelling is caused by alterations in the activity of ionic channels, or in the mechanical stability of cisternae or both. Strikingly, actin toxins did not perturb the localization of βIII spectrin at the Golgi, which indicates that actin dynamics does not participate in the association of βIII spectrin with Golgi membranes, but PI4P was crucial in such interaction (Salcedo-Sicilia et al. 2013). The ultrastructural alterations caused by actin drugs (Lazaro-Dieguez et al. 2006) and the depletion of βIII spectrin (Salcedo-Sicilia et al. 2013) indicate that they provide the necessary mechanical stability to cisternae to prevent their expected spontaneous swelling due to the hyperosmotic protein content in transit through the Golgi stack. Ion regulatory molecules such as vacuolar H^+-ATPase (Moriyama and Nelson 1989) and cation (NHEs) exchangers (Nakamura et al. 2005) either resident in the

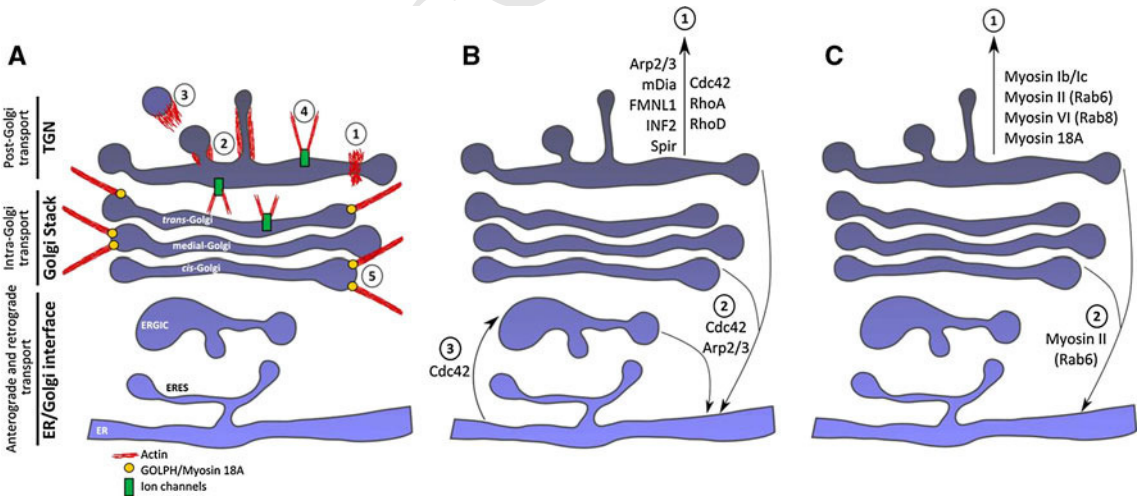

Fig. 2 Diagram of the secretory membrane trafficking pathways and events in which actin, Rho GTPases, actin nucleators and myosin motors are known to participate. **a** Actin filaments, their polymerization and dynamics could act as a force for the scission (*1*), pulling (*2*) and propelling (*3*) of the transport carrier generated in cisternae, and for maintaining the flattened shape of cisternae (*4*) and the extended Golgi ribbon (*5*). **b** Rho GTPases and actin nucleators reported to act in the post-Golgi (*1*), Golgi-to-ER (*2*) and ER-to-Golgi (*3*) protein transport. **c** Myosin motors and their known Rab protein effectors in the post-Golgi (*1*) and Golgi-to-ER (*2*) protein transport. See the text for details

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Golgi or in transit to the plasma membrane could contribute to this actin/spectrin-dependent cisternal mechanical stability, finely regulating intra-Golgi ion concentration and pH homeostasis. In accordance with this idea are the reports that the sorting of some secretory cargo at the *trans*-Golgi network (TGN) by the actin-filament-severing protein ADF/cofilin activates the calcium ATPase 1 (SPCA1) (Curwin et al. 2012; von Blume et al. 2009, 2011) and that the actin depolymerisation-induced cisternae swelling is accompanied by a rise in the intra-Golgi pH (Lazaro-Dieguez et al. 2006). Therefore, actin seems to regulate the activity of some ionic regulatory proteins present in Golgi membranes, similarly to what occurs at the plasma membrane (Mazzochi et al. 2006) (Fig. 2a).

Actin and co-workers in the sorting, biogenesis and motion of transport carriers at the Golgi

Filamentous actin

A key aspect in the structure of polarized cells is the maintenance of polarized molecular organization. This is based on highly specific sorting machinery at the exit of the TGN (Rodriguez-Boulan et al. 2005). Cytoskeleton elements form part of this machinery, and the integrity of actin filaments is necessary for efficient delivery of some proteins destined for the apical or the basolateral plasma membrane domains in both polarized and non-polarized cells, but not for the transport of lipid raft-associated proteins (Jacob et al. 2003; Lazaro-Dieguez et al. 2007; Lebreton et al. 2008) (Fig. 2a).

Rho GTPases and actin nucleators

Tight control of the coupling between Golgi-associated actin polymerization and membrane elongation and fission reactions prevents the structural and functional collapse of the Golgi. Part of this control can be achieved by regulating the activation state of Rho GTPases and downstream effectors in Golgi membranes (Fig. 2b). Briefly, classical Rho GTPases cycle between active GTP-bound and inactive GDP-bound forms. There are three types of proteins that regulate this cycle: guanine nucleotide exchange factors (GEFs) activate GTPases; GTPase-activating proteins (GAPs) inactivate them and guanine nucleotide dissociation inhibitors (GDIs) keep GTPases in a GDP-inactive form. Cdc42, which was the first RhoGTPase to be localized in the Golgi (Erickson et al. 1996; Fucini et al. 2000; Luna et al. 2002; Matas et al. 2004; Prigozhina and Waterman-Storer 2004; Wu et al. 2000), affects ER/Golgi interface and post-Golgi intracellular trafficking (Harris and Tepass 2010) (Fig. 2b). Constitutively active and inactive Cdc42 mutants block the ER-to-Golgi transport of anterograde cargo (VSV-G) (Wu et al. 2000). The overexpression and activation of Cdc42 (Luna et al. 2002) or the knockdown of Cdc42 GAP ARHGAP21 (also known as ARHGAP10) (Hehnly et al. 2009) inhibit the Golgi-to-ER transport of retrograde cargo (Shiga toxin) (Fig. 2b). Cdc42 binds γCOPI subunit recruiting N-WASP and Arp2/3 to Golgi membranes. p23 (a receptor for cargo containing the dilysine motif in the COOH-terminal) competes with Cdc42 for binding to γCOPI subunit. Cargo loading by p23 disrupts the Ccdc42–γCOP interaction and recruits dynein to promote the dynein-dependent ER-to-Golgi transport (Chen et al. 2005) (Fig. 2b). Therefore, Cdc42 coordinates actin- and microtubule-dependent motility of transport carriers at the ER/Golgi interface (Hehnly and Stamnes 2007) (Fig. 2b). In post-Golgi trafficking (Fig. 2b), the expression of constitutively active or inactive Cdc42 mutants slows the exit of basolateral protein markers and accelerates the exit of apically destined ones (Cohen et al. 2001; Kroschewski et al. 1999; Musch et al. 2001). ARHGAP21/10 and Cdc42 GEFs Fgd1 and Dbs are also present in Golgi membranes regulating post-Golgi vesicular transport (Dubois et al. 2005; Egorov et al. 2009; Estrada et al. 2001; Kostenko et al. 2005; Menetrey et al. 2007).

At first, Cdc42 was believed to be the only Rho GTPase working at the Golgi (Matas et al. 2005), but recent data also implicate other Rho GTPases and downstream effectors (Fig. 2b). This is the case for RhoA with mDia (Ziberman et al. Zilberman et al. 2011) and Citron-N (Camera et al. 2003), RhoD with WHAMM (Gad et al. 2012) and Rac1 and its exchange factor β-PIX with the clathrin heavy-chain-binding protein CYFIP/Sra/PIR121 (Anitei et al. 2010). RhoD-WHAMM and the ARF1-primed Rac1-CYFIP/Sra/PIR121 protein complexes stimulate the Arp2/3-induced actin polymerization at the Golgi and vesicle biogenesis (Anitei et al. 2010; Campellone et al. 2008). The ROCK/LIM kinase (LIMK) signaling pathway and its substrate cofilin are necessary for apical cargoes (Rosso et al. 2004; Salvarezza et al. 2009). RhoA GEF-H1 interacts with exocyst component Sec5, which in turn activates RhoA-regulating post-Golgi trafficking and assembly of other exocyst components (Pathak et al. 2012).

The presence in Golgi membranes of molecular components that trigger actin polymerization with those that control vesicular budding and fission suggests intimate molecular coupling between them, which is strongly similar to that observed during endocytosis (Mooren et al. 2012). Actin assembly provides the structural support that facilitates the formation of transport carriers in the lateral portions of Golgi membranes (Fig. 2a). This can be achieved by generating force through actin polymerization triggered by actin nucleators, which in turn can be

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accompanied by the mechanical activity of actin motors (myosins). In accordance with this idea, Arp2/3, mDia, formin-like 1/FMNL1 and INF2 and Spir1 are all present in the Golgi (Carreno et al. 2004; Chen et al. 2004; Colon-Franco et al. 2011; Kerkhoff et al. 2001; Matas et al. 2004; Ramabhadran et al. 2011; Zilberman et al. 2011) (Fig. 2b). At the TGN, there is a functional coupling between dynamin-mediated membrane fission and Arp2/3-mediated actin-based mechanisms (Cao et al. 2005; Carreno et al. 2004; Kerkhoff et al. 2001; Kessels and Qualmann 2004; Praefcke and McMahon 2004). Interference with dynamin2/cortactin or dynamin2/syndapin2/cortactin blocks post-Golgi protein transport (Cao et al. 2005; Kessels et al. 2006; Salvarezza et al. 2009). As indicated above, in early Golgi compartments, there is a functional connection between actin polymerization governed by Cdc42, coatomer (COPI)-mediated transport carrier formation and microtubule motor-mediated motion. WASH (Wiskott-Aldrich syndrome protein and SCAR homolog) is another Arp2/3 activator that regulates the cation-independent mannose phosphate receptor (CI-MPR) trafficking from endosomes to the Golgi, forming an endosomal subdomain containing Arp2/3, F-actin, tubulin and retromer components (Gomez and Billadeau 2009). The local fine regulation of actin dynamics on the transport carrier assembly could represent an early step that precedes its scission in the lateral portions of cisternae for subsequent switching to microtubule tracks for motion (Fig. 2a).

Actin nucleation/polymerization activity associated with Arp2/3 on Golgi membranes could also give rise to the formation of actin comet tails, which consist of filamentous actin and various actin-binding proteins that focally assemble and grow on a membrane surface (Campellone and Welch 2010) (Fig. 2a). After the overexpression of phosphatidylinositol 5-kinase, actin tails have been observed only in raft-enriched TGN-derived vesicles (Guerriero et al. 2006; Rozelle et al. 2000). An in vitro approach in liposomes showed actin polymerization occurring after the recruitment of the activated form of ARF1 around liposomes. This actin polymerization was dependent on Cdc42 and N-WASP present in HeLa cell extracts and resulted in the formation of actin comets, which pushed the ARF1-containing liposome forward (Heuvingh et al. 2007). However, actin comet tails do not seem to be an efficient mechanism to provide directionality for transport carriers, in which microtubule tracks, and to lesser extent actin ones, seem more suitable. However, an actin comet tail-like mechanism could easily provide brief local force to facilitate the final separation of the transport carrier at the lateral rims of Golgi cisternae (Fig. 2a) similarly to what happens during endocytosis (Merrifield 2004; Merrifield et al. 2005; Taylor et al. 2012), and/or for its translocation to closely arranged microtubule tracks (Egea et al. 2006) (see green arrows in Fig. 3).

Myosin motors

In addition to actin polymerization, myosins also generate a force, which can selectively couple protein sorting and transport carrier biogenesis and motility. Class I myosin is a monomeric, non-processive motor that binds to Golgi membranes and is present on apical Golgi-derived vesicles of polarized cells (Almeida et al. 2011; Fath and Burgess 1993; Jacob et al. 2003; Montes de Oca et al. 1997; Tyska et al. 2005). Myosin Ib together with actin polymerization have recently been shown to participate in membrane remodeling to form tubular transport carriers at the TGN directed to endosomes and the plasma membrane (Almeida et al. 2011; Coudrier and Almeida 2011) (Fig. 2c). It has been hypothesized that myosin Ib spatially controls actin assembly at the TGN, interacting with F-actin via its motor domain and at the membrane via its PH domain. Such interaction generates a force concomitantly with the polymerization of actin, leading to membrane curvature changes (Coudrier and Almeida 2011; Loubéry and Coudrier 2008). Coudrier and collaborators have suggested coordination between myosin 1b and non-muscle myosin II for the scission of tubular carriers at the TGN (Coudrier and Almeida 2011). If this is confirmed, it would represent a new level of cooperation between different actin motors for transport carrier biogenesis. It has been suggested that myosin I could also have sorting ability, which could be linked to its capacity to interact with lipid raft-associated cargo. In this respect, myosin Ic controls the delivery of GPI-linked cargo proteins to the cell surface from the endosomal recycling compartment (Brandstaetter et al. 2012), but this does not seem to be the case for myosin Ib either at the TGN or in endosomes (Almeida et al. 2011).

Non-muscle myosin II is another non-processive motor that directly interacts with Golgi membranes (Fath 2005; Heimann et al. 1999; Miserey-Lenkei et al. 2010) and mediates both Golgi-to-ER and post-Golgi protein transport (DePina et al. 2007; Duran et al. 2003; Musch et al. 1997; Stow et al. 1998) (Fig. 2c). It was postulated that this motor is tethered to the cisterna by its tail and to actin filaments by its motor head. Its subsequent motion along actin filaments could provide the force needed to extend Golgi-derived membranes away from the cisterna (Fig. 2a), which could facilitate the functional coupling of membrane scission protein(s), leading to the release of the transport carrier. In accordance with this hypothesis, myosin II forms a complex with Rab6, which facilitates its localization to Golgi membranes and controls the fission of anterograde and retrograde Rab6 transport carriers (Miserey-Lenkei et al. 2010). The Golgi-associated tropomyosin isoform

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(Percival et al. 2004) could stabilize short actin filaments formed locally in the cisternae lateral rims during vesicle biogenesis. These short actin filaments together with myosin II could equivalently act as a sarcomeric-like system to generate the force necessary to split transport carriers (Fig. 2a). Accordingly, there is an abnormal accumulation of uncoated vesicles close to cisternae after the knockdown of myosin II or the pharmacological blockade of its motor activity (Duran et al. 2003; Storrie et al. 2012) (Fig. 3). Many of them still remained attached to cisternae (see red arrows in Fig. 3), which argues in favor of the participation of myosin II in the fission process (Miserey-Lenkei et al. 2010). However, in another line of evidence, myosin II has been reported to be required only for motion but not for the biogenesis of PKD-dependent transport carriers at the TGN (Wakana et al. 2012).

In contrast to endocytic recycling and translocation of secretory vesicles/granules to cell periphery actin cytoskeleton, there is no clear experimental evidence of myosin V activity at the Golgi in mammalian cells (not the case in yeast; see below). However, a yeast two-hybrid screen of human Rab proteins for myosin Va binding has revealed that myosin Va isoform functions in post-Golgi-trafficking interacting with Rab6 together with Rab8 and/or Rab10, although myosin Va does not localize in the Golgi (B. Goud, personal communication).

Myosin VI is another myosin motor located in the Golgi (Buss et al. 2004; Warner et al. 2003) (Fig. 2c). It differs from the other processive myosins in that it only moves transport carriers toward the fast-depolymerizing minus-end pole of the microfilament. Therefore, myosin VI could provide the force and directionality for the transport carrier movement away from cisternae in accordance with the expected fast-growing plus-end polarization of the actin filaments originating in Golgi membranes. Myosin VI is involved, among many others (Buss and Kendrick-Jones 2008; Sweeney and Houdusse 2010), in the maintenance of Golgi morphology (Sahlender et al. 2005; Warner et al. 2003). The interaction

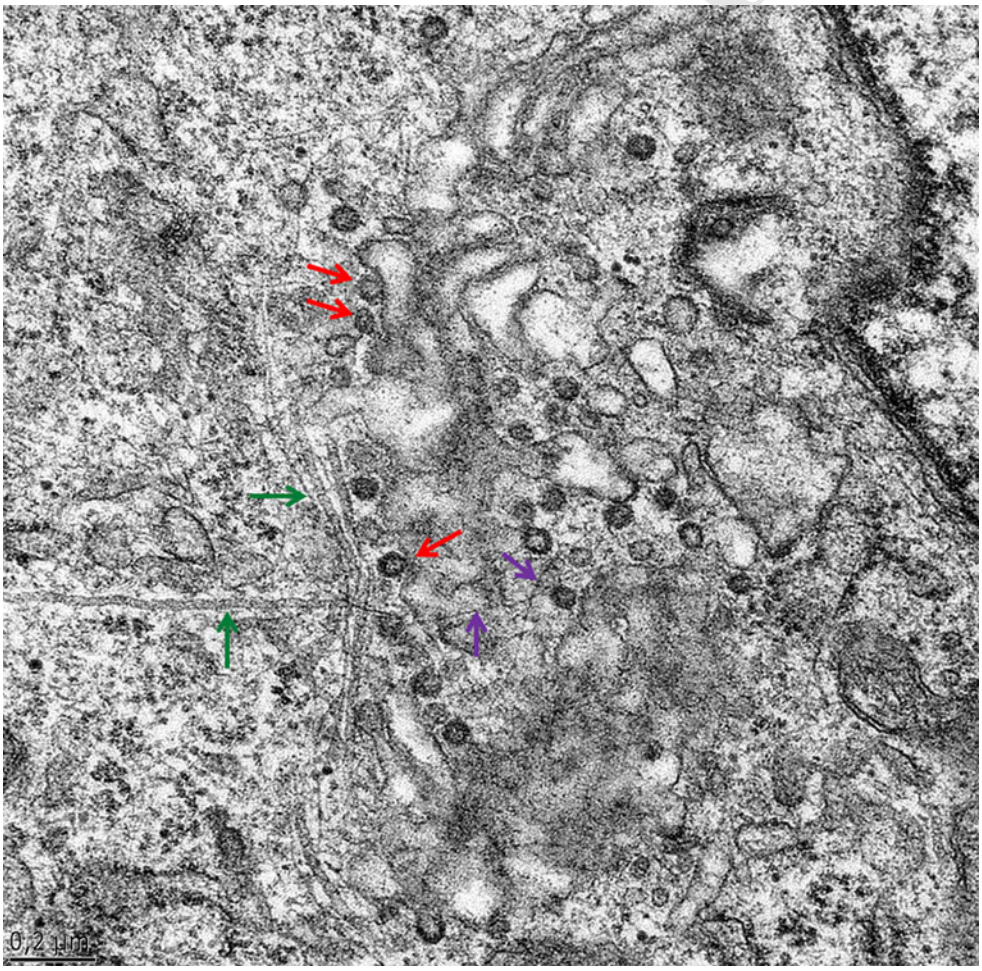

Fig. 3 The pharmacological blockade of the myosin II motor activity by blebblistatin in NRK cells produces an accumulation of peri-Golgi vesicles close to swollen cisternae. Note that some vesicles remain connected to cisternae by a narrow neck (*red arrows*). Microtubules and actin filaments are, respectively, indicated by *green and purple arrows*. *Bar* 200 nm

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between myosin VI and optineurin, a partner of Rab8 (Sahlender et al. 2005), acts at the TGN of polarized epithelial cells in the protein sorting and basolateral transport mediated by the clathrin adaptor protein complex AP-1B (Ang et al. 2003; Au et al. 2007; Jordens et al. 2005). Therefore, the known role of some Rab proteins as linkers of endocytic membranes to cytoskeletal motors is now also extended to the Golgi (Goud and Gleeson 2010).

Finally, the unconventional myosin 18A has also been located in distal Golgi membranes (Fig. 2c), where it binds to the PI4P-binding protein GOLPH3. It has been suggested that the GOLPH3–Myos18 interaction couples actin filaments to Golgi membranes and the tension generated by this interaction facilitates the maintenance of the extended Golgi ribbon organization and flattens Golgi cisternae (Fig. 2a). In addition, it also seems to support secretory function because the depletion of GOLPH3 blocks the exit of VSV-G from the TGN (Dippold et al. 2009). The contribution of this unconventional myosin- to Golgi-associated membrane trafficking requires further characterization because it exhibits low motor activity (Guzik-Lendrum et al. 2013).

The Golgi apparatus–actin interaction in other cellular models

Plant cells

Stationary actin filaments or actin bundles are the most prominent cytoskeleton element in plant cells. They are all oriented with the same polarity and aligned along the plant cell. Attached to the actin bundles are the ER, vesicles and numerous discrete or a few clustered Golgi stack-TGN units, also known as Golgi bodies. They are highly variable in number (from a few tens to hundreds) depending on the plant type, plant cell type and its developmental stage (Boutte et al. 2007; Hawes and Satiat-Jeunemaitre 2005; Kepes et al. 2005). In polarized root hairs and pollen tubes, the TGN is segregated from Golgi bodies, which localize to growing tips, where together with actin, Rho/Rac members (ROPs and Rac1, respectively), Rab (Rab4a and Rab11) and ARF (ARF1) small GTPases regulate secretory and endocytic trafficking (Samaj et al. 2006). Also in this cell type, the motility and positioning of Golgi bodies is highly dependent on the actin organization, being faster and directional in areas containing actin filament bundles and slower and non-directional in areas with fine filamentous actin (Akkerman et al. 2011).

In plants, most of the endomembrane compartments are in constant movement together with the cytoplasmic streaming whereby cellular metabolites are distributed throughout the cell (Shimmen and Yokota 2004). Golgi bodies show actin-dependent dispersal and spatial organization (Boevink et al. 1998) and contain a fine fibrillar material enriched in actin, spectrin- and myosin-like proteins (Mollenhauer and Morre 1976; Satiat-Jeunemaitre et al. 1996). The depolymerization of actin filaments with actin toxins uncouples the association between specific regions of cortical ER with individual Golgi bodies (Boevink et al. 1998; Brandizzi et al. 2003), but, and in contrast to animal cells (Valderrama et al. 2001), it does not perturb the brefeldin A (BFA)-induced Golgi disassembly (Ito et al. 2012). Thus, cytochalasin or latrunculin treatments induce the aggregation of Golgi bodies and variably alter the Golgi morphology depending on the cell type and the period of treatment (Chen et al. 2006; Satiat-Jeunemaitre et al. 1996). Actin toxins also perturb the coordinated movement of Golgi bodies and ER tubules (da Silva et al. 2004; Uemura et al. 2002; Yang et al. 2005). Actin does not participate in the ER/Golgi interface protein transport (Saint-Jore et al. 2002), but it does contribute to post-Golgi trafficking to the plasma membrane and the vacuole. In the tip of growing cells like pollen tubes, actin filaments are the tracks through which Golgi-derived secretory vesicles are transported (Picton and Steer 1981; Vidali et al. 2001). An intact actin–myosin system is required for the transport of cargo containing polysaccharides and the enzymes necessary for cell wall morphogenesis, and the local differences in the actin cytoskeleton organization determine where their secretion is required (Blancaflor 2002; Crowell et al. 2009; Hu et al. 2003; Kato et al. 2010; Miller et al. 1995; Nebenfuhr et al. 1999). Finally, Golgi bodies are propelled by plant myosin family members, especially the myosin XI class (Avisar et al. 2008, 2009; Boutte et al. 2007; Higaki et al. 2007; Peremyslov et al. 2010; Sparkes 2011).

Yeast

The use of a large number of mutants that alter intracellular traffic in the budding yeast *S. cerevisiae* has led to the identification of proteins involved in both membrane trafficking and actin organization (Kaksonen et al. 2006; Mulholland et al. 1997). Most components of the secretory pathway and many of the actin-based cytoskeleton are conserved between yeast and mammalian cells. The actin cytoskeleton in yeast consists primarily of cortical patches and cables (Moseley and Goode 2006). Actin filaments polarize growth in yeast (Novick and Botstein 1985). In this respect, many actin mutants accumulate large secretory vesicles and exhibit phenotypes consistent with defects in polarized growth (Pruyne et al. 2004). This, together with the polarized organization of actin cytoskeleton, has suggested a role for actin in the positioning and orientation of the secretory pathway and polarized transport of late

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secretory vesicles to the plasma membrane (Finger and Novick 2000; Mulholland et al. 1997; Yamaguchi and Kopecka 2010). A mutation of GRD20, a protein involved in sorting in the TGN/endosomal system, showed aberrant secretion of the vacuolar hydrolase carboxypeptidase Y (but not other TGN membrane proteins) and defects in the polarization of the actin cytoskeleton (Spelbrink and Nothwehr 1999). As in mammalian cells, the actin-severing protein cofilin concomitantly with Pmr1, the yeast orthologue of the secretory pathway calcium ATPase 1 (SPCA1), is also required for sorting at the late Golgi compartment (Curwin et al. 2012). Overexpression of Avl9p, a member of a novel protein superfamily, produces vesicle accumulation and a post-Golgi defect in secretion. Its depletion in a strain that also lacks Vps1 (dynamin) and Apl2 (adaptor protein complex 1) results in perturbed actin cytoskeleton organization and defects in polarized secretion (Harsay and Schekman 2007). Concentration of late (but not early) Golgi elements at the sites of polarized growth (the bud) depends on actin, which is transported along actin cables by yeast myosin V (Myo2) (Rossanese et al. 2001). Crucial in this process is Ypt11, a Rab GTPase that interacts with Myo2 and Ret2, a subunit of the coatomer complex. The polarization of late Golgi cisternae in the bud is not produced in Ypt11Δ mutant (Arai et al. 2008). The Rab protein Ypt31/32 present at the TGN directly interacts with Myo 2 and the secretory vesicle Rab Sec4, whose interaction is modulated by PI4P levels (Santiago-Tirado et al. 2011). Moreover, the Ypt31/32-Myo2-Sec4 complex interacts with exocyst subunit Sec15 regulating post-Golgi trafficking and cell growth (Jin et al. 2011).

With regard to the early secretory pathway, actin filament depolymerization with actin toxins does not affect anterograde ER-to-Golgi protein transport (Brazer et al. 2000). However, this is not the case in retrograde Golgi-to-ER trafficking, which is regulated by the ubiquitin ligase Rsp5, a protein that forms a complex containing COPI subunits and has as substrates the actin cytoskeleton proteins Sla1, Lsb1, Lsb2, which bind to the Arp2/3 activator Las17 (Jarmoszewicz et al. 2012; Kaminska et al. 2011).

Drosophila

The *Drosophila* cellular model is an alternative to yeast to study the Golgi because it shares many structural and functional similarities with the mammalian model, although most *Drosophila* cells and tissues lack the characteristic mammalian Golgi ribbon. Instead, they present a scattered and fairly constant number of what are known as tER-Golgi units, which are ultrastructurally constituted by a pair of Golgi stacks (Kondylis and Rabouille 2009). The integrity of the actin cytoskeleton is crucial for Golgi stack pairs since actin depolymerization causes their splitting and perturbs Golgi inheritance, which requires duplication to form the paired structure. Abi and Scar/WAVE (but not WASP) are necessary in this process (Kondylis et al. 2007). The inactivation of the golgin-like microtubule/actin-binding protein lava lamp prevented the necessary Golgi dispersal in the cellularization process (Papoulas et al. 2005; Sisson et al. 2000). The analysis of a genome-wide RNA-mediated interference screen in adherent *Drosophila* S2 cells showed that the depletion of the *tsr* gene (which codifies for destrin, also known as ADF/cofilin) induces Golgi membranes to aggregate and swell, resulting in inhibition of the HRP secretion (Bard et al. 2006). Coronin proteins dpdo1 and coro regulate the actin cytoskeleton and also govern biosynthetic and endocytic vesicular trafficking, as indicated by mutant phenotypes that show severe developmental defects, ranging from abnormal cell division to aberrant formation of morphogen gradients (Rybakin and Clemen 2005).

Dictyostelium discoideum

Cells of this social amoeba are easy to manipulate by genetic and biochemical means. They contain various types of vacuole, ER and small Golgi stacks (Becker and Melkonian 1996). Comitin (p24) is a dimeric *Dictyostelium* actin-binding protein present in the Golgi and vesicles that contains sequence motifs homologous to lectins. It seems that this protein binds Golgi-derived vesicles to the actin filaments via the cytoplasmic exposed mannosylated glycans (Jung et al. 1996; Weiner et al. 1993). Villidin is another actin-binding protein that associates with secretory vesicles and Golgi membranes (Gloss et al. 2003). The centrosomal protein LIS1 (DdLIS1) links microtubules, the nucleus and the centrosome and indirectly controls the Golgi morphology. Mutants of this protein lead to microtubule disruption, Golgi fragmentation and actin depolymerization (Rehberg et al. 2005). AmpA is a secreted protein necessary for cell migration in an environment-dependent manner that also participates in the regulation of actin polymerization. It is found in the Golgi but transported to the plasma membrane, where it regulates endocytosis (Noratel et al. 2012). In addition to Rho GTPases, *Dictyostelium* also contains other Rho-regulated signaling components such as RhoGDI, Arp2/3 complex, PAK, WASP, Scar/WAVE, formins, GEFS and GAPS (Eichinger et al. 2005). The acquisition of cell polarity during chemotaxis needs WASP, which localizes on vesicles whose formation in the Golgi requires the interaction between WASP with the pombe Cdc15 homology (PCH) family protein members Nwk/Bzz1-p-like and syndapin-like proteins (Lee et al. 2009). RacH is a closer protein to Rac and Cdc42 which localizes to compartments of the secretory

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pathway (nuclear envelope, ER and the Golgi) where it stimulates actin polymerization, and it also seems to be involved in actin-based trafficking of vesicles, but in contrast to AmpA, it is uncoupled from chemotaxis (Somesh et al. 2006).

Caenorhabditis elegans

Very little is known about the Golgi and actin cytoskeleton interaction in this organism, but consistent with a possible role of coronin 7 in Golgi trafficking (Rybakin et al. 2004; Rybakin and Clemen 2005), depletion of the coronin 7 homolog POD1 leads to aberrant accumulation of vesicles in cells of the early embryo (Rappleye et al. 1999). Moreover, CRP-1, a Cdc42-related protein, localizes at the TGN and recycling endosomes. Alteration of CRP-1 expression in epithelial-like cells perturbs apical but not basolateral trafficking (Jenna et al. 2005).

Concluding remarks and perspectives

The actin cytoskeleton usually works in tight coordination with microtubules (Disanza and Scita 2008). The functional relationship between each cytoskeleton network and Golgi dynamics is complementary. In animal cells, actin and co-workers participate in early events of transport biogenesis such as protein sorting, membrane fission and keeping cisternae flat. Microtubules and associated motors are more directly involved in the motion of Golgi-derived transport carriers to their final destinations and in the positioning and organization of the Golgi as a ribbon-like structure (at least in vertebrates) (Brownhill et al. 2009; de Forges et al. 2012). Conversely, in plant cells, endomembrane compartments and associated trafficking are almost exclusively mediated by actin filaments. In other cellular models, less is known but in general terms, actin cytoskeleton elements regularly participate in post-Golgi protein transport and Golgi inheritance. Finally, the actin cytoskeleton as a dynamic biopolymer surely affects the biophysical properties (rigidity/elasticity and tension) of Golgi membranes. Future research in this biophysical cell biology interactive area will undoubtedly provide valuable information about how actin contributes to the structural and functional organization of the Golgi. Another important question is how myosin motors participate in the genesis of transport carriers, particularly in providing the force that generates curvature and facilitates membrane fission. In vitro models such as the giant unilamellar vesicles (GUVs) (Bassereau and Goud 2011) will help to our knowledge of the precise molecular mechanism and sequence of this process. According to the evidence furnished by this particular line of research, curved membranes, but not flat ones, use phosphoinositides to stimulate Cdc42-N-WASP-Arp2/3-driven actin polymerization (Gallop et al. 2013). Finally, the results that clearly implicate actin in protein sorting and the identification of molecular targets that directly regulate the ionic environment of the TGN/Golgi provide an unexpected new perspective on the Golgi–cytoskeleton interaction.

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