BioArchitecture 1:5, 230-235; September/October 2011; © 2011 Landes Bioscience

Myosin 1 controls membrane shape by coupling F-Actin to membrane

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Keywords: Myosins, actin, membrane remodeling, membrane traffic, trans-Golgi network

Abbreviations: F-Actin, actin filaments; G-actin, globular actin; KD, knockdown; KO, knockout; MPR, CI-mannose-6-phophate receptor; NPF, nucleation-promoting factors; TGN, trans-Golgi network

Submitted: 10/06/11

Revised: 10/10/11

Accepted: 10/12/11

http://dx.doi.org/10.4161/bioa.1.5.18406

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Cellular functions are intimately associated with rapid changes in membrane shape. Different mechanisms interfering with the lipid bilayer, such as the insertion of proteins with amphipatic helices or the association of a protein scaffold, trigger membrane bending. By exerting force on membranes, molecular motors can also contribute to membrane remodeling. Previous studies have shown that actin and myosin 1 participate in the invagination of the plasma membrane during endocytosis while kinesins and dynein with microtubules provide the force to elongate membrane buds at recycling endosomes and at the trans-Golgi network (TGN). Using live cell imaging we have recently shown that a myosin 1 (myosin 1b) regulates the actin dependent post-Golgi traffic of cargo and generates force that controls the assembly of F-actin foci and promotes with the actin cytoskeleton the formation of tubules at the TGN. Our data provide evidence that actin and myosin 1 can regulate membrane remodeling of organelles as well as having an unexpected role in the spatial organization of the actin cytoskeleton. Here, we discuss our results together with the role of actin and other myosins that have been implicated in the traffic of cargo.

Introduction

During embryonic development numerous biological processes, including cell migration, membrane traffic and tissue reorganization, require rapid changes in membrane shape. For example during intracellular transport of cargo, membranes of organelles acquire curved shapes leading to the formation of tubular and vesicular carrier precursors. The current model regarding membrane remodeling is largely based on studies of plasma membrane deformations that lead to membrane invagination during endocytosis. This model involves two major steps for membrane deformation and a third step that implies change in membrane continuity and triggers the scission of vesicles from the plasma membrane.

In the first step, membrane bending can be achieved by different mechanisms. Change in lipid composition or integral membrane proteins can influence membrane bending.[1] Proteins that insert amphipatic helices into the proximal lipid monolayer, such as proteins with ENTH (Epsin N-Terminal Homology) or ANTH (AP180N-Terminal Homology) domains, also induce membrane curvature.[2] These proteins have been involved at the plasma membrane for endocytosis and at the trans-Golgi network (TGN) for the exit of CI-mannose-6-phosphate receptor (MPR). Scaffolding proteins, including proteins with BAR domains, make membrane curvature by imposing their crescent shape on the membrane with which they interact.[3,4] F-BAR proteins with a positive curvature have been implicated at the plasma membrane for endocytosis.[5,6] Clathrin, or other coats that are recruited to membrane domains by adaptor complexes like AP1, AP2, AP3, AP4, are believed to also contribute to membrane curvature.[7] Interestingly, proteins with ENTH, ANTH or BAR domains interact with the adaptor complexes, suggesting that the adaptor/coat machinery coordinates the formation of membrane curvature with the sorting of cargo addressed to different cellular destinations.[2,8]

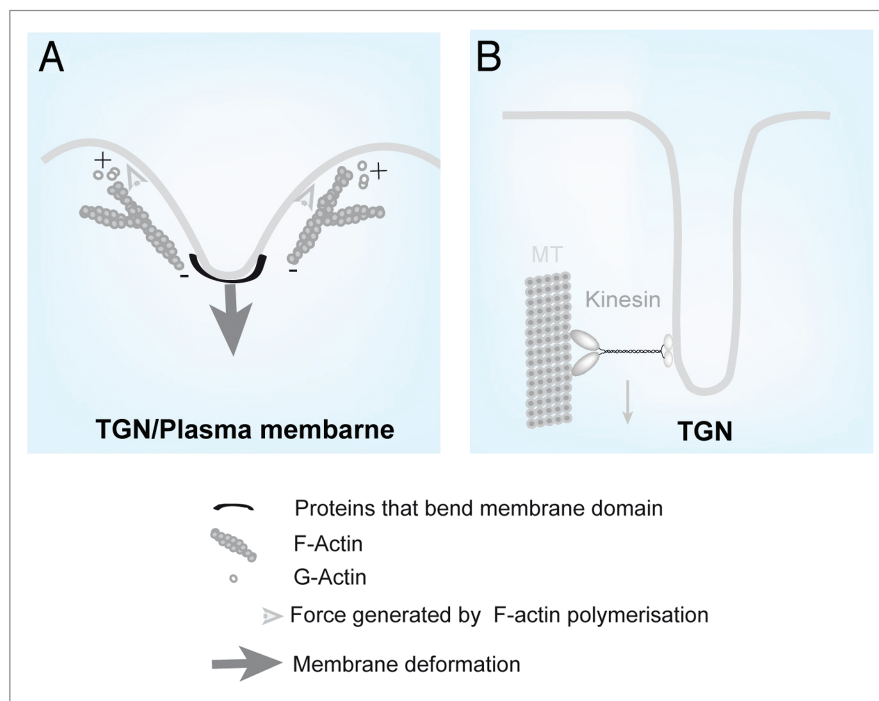

Figure 1. Formation of carrier vesicles. Model for the sequential events that induce membrane deformation at the plasma membrane or at the TGN. (A) By interacting with NPF, F-BAR protein or ETNH proteins control the side of actin polymerization for membrane invagination at the plasma membrane and at the TGN. (B) Kinesin and microtubules then elongate the membrane of the TGN.

In the second step, in the case of morphogenesis of carrier vesicles, a generation of forces allows for the elongation of the membrane leading to membrane tubules prior to scission. Motor proteins and cytoskeleton are commonly employed to generate forces. Actin polymerization itself can push membranes. At the plasma membrane, proteins with F-BAR domains interact with members of the Wiskott Aldrich Syndrome protein (WASP) family, also named nucleation-promoting factors (NPF), that activate the Arp2/3 complex and hence induce actin polymerization.[5] If the BAR domain protein is located between NPF molecules, the force generated by actin polymerization leads to membrane invagination (**Fig. 1**). In addition to actin polymerization, a yeast myosin from class I has been proposed to contribute to membrane invagination during endocytosis (**Fig. 1**).[9,10] In contrast, kinesins and dynein with microtubules provide the force to elongate membrane buds at recycling endosomes and at the TGN, two of the main protein sorting stations of the cell at the cross roads of the exocytic and endocytic pathways (**Fig. 1**).[11-13] Recent experimental evidence indicates that several NPFs, including WASP, WASH and WHAMM, and several myosins from the classes II, V, VI and XVIII, contribute to cargo trafficking at the TGN and endosomes.[14-19] Do these myosins and actin polymerization participate in membrane remodeling leading to the formation of carrier vesicles? We recently raised this question and showed that the actin cytoskeleton and a myosin I (myosin 1b) promote the formation of tubular-carrier precursors at the TGN.

Myo1b Regulates TGN Exit and Formation of Tubular-Precursor Carriers

We have shown previously that myosin1b (Myo1b) localizes at the plasma membrane in regions enriched with actin filaments, and at early endosomes, multivesicular endosomes and lysosomes.[20,21] Furthermore, Myo1b was involved in the traffic of cargo along the endocytic pathway and together with F-actin it modulated the shape of sorting endosomes.[20,22] In our recent study we found that a pool of Myo1b localizes at the TGN in HeLa cells.[23] We thus investigated whether Myo1b participates in the trafficking of different cargo that transit through the TGN. We showed that the cellular distribution of MPR that cycles between the TGN and sorting endosomes was impaired by down expression or overexpression of Myo1b in HeLa cells. Using live cell imaging we showed that the exit of GFP-MPR from the TGN was inhibited in cells depleted for Myo1b by siRNA, while the traffic of MPR from endosomes to the TGN was not altered. The delivery of newly synthesized lysosomal hydrolases to endosomes, such as cathepsin D and β-hexosaminidase, was also affected. However, instead of being secreted, as previously reported in KO cells for MPR,[24] β-hexosaminidase activity was decreased in cell-culture media in the absence of Myo1b. These findings suggest that Myo1b inhibits not only post-Golgi transport to endosomes but also post-Golgi transport to the plasma membrane. Indeed, the kinetics of TGN exit of the neurotrophin receptor p75 (p75), that localizes at the plasma membrane at steady-state,[12] was slowed down in the absence of Myo1b. Interestingly, the exit of MPR and p75 depends on an intact actin cytoskeleton.[25-27] In contrast, the exit of a lipid-raft anchored protein (GPI-GFP), that leaves the TGN independently of the actin cytoskeleton, was not regulated by Myo1b.[26,27] We concluded that Myo1b controls the exit of cargos requiring actin dynamics and that are delivered either to endosomes or via the exocytic pathway to the plasma membrane. Previous reports have suggested that inhibition of cargo exit from TGN impairs the homeostasis of the Golgi complex and consequently disturbs its morphology.[28-30] We observed by conventional electron microscopy that depletion of Myo1b produced shorter Golgi stacks, dilated mostly at the rims, and an abnormal accumulation of peri-Golgi vesicular profiles with various shapes and sizes. Exit of MPR from the TGN depends on the formation of tubular-carrier precursors that elongate and break into post-Golgi carrier vesicles.[14,31] Using fast time-lapse imaging we observed a dramatic

reduction of tubular-carrier precursors and cytoplasmic carrier vesicles in absence of Myo1b. Conversely, overexpression of Cherry-Myo1b induced the formation of long GFP-MPR tubular-precursors. To determine whether the force generated by Myo1b triggers cargo exit from the TGN and could be at the origin of membrane deformation, we analyzed whether Myo1b rigor mutant (N160A) with a single mutation in the motor domain and a Myo1b mutant (E476K) with a single mutation in the "switch 2" of the motor domain, both being unable to generate force to move F-actin in vitro, could rescue the normal distribution of MPR upon endogenous Myo1b KD. We found that expression of recombinant wild-type Myo1b, but not Myo1b-rigor or Myo1b-E476K, rescued the normal steady-state distribution of MPR. We concluded that Myo1b regulates the exit of cargo from the TGN by generating a force that could contribute to the formation of tubular-precursor carriers.

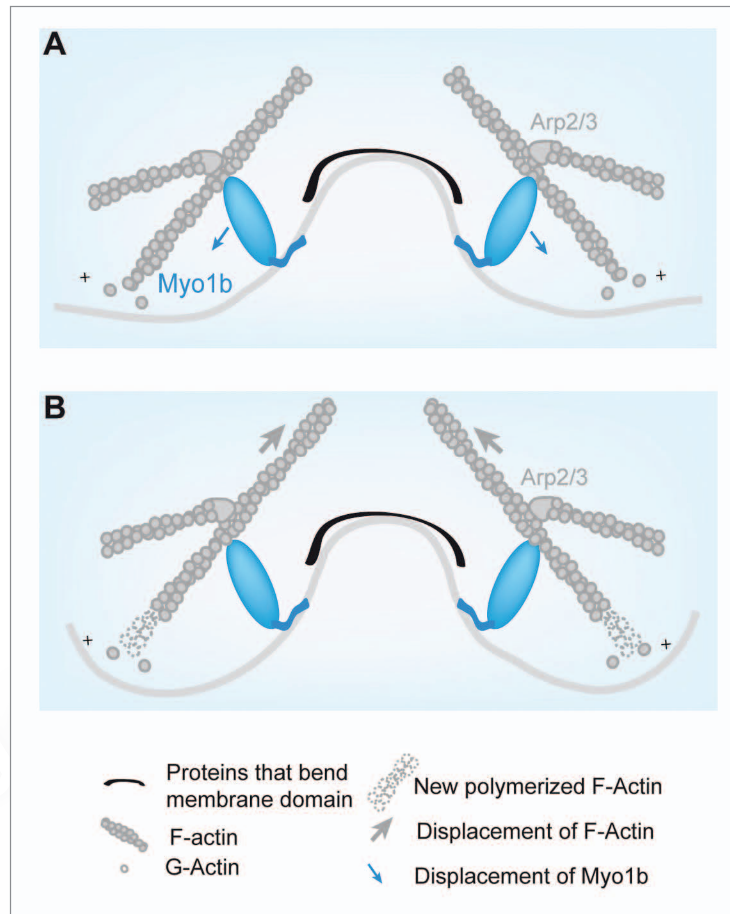

Figure 2. Model for the role of Myo1b in membrane remodeling. Myo1b moves toward the plus end of F-actin. If Myo1b is immobilized because of its interaction with the membrane then the Myo1b motor activity will move F-actin backward thereby facilitating addition of new G actin next to the plasma membrane. (A) Shows the orientation of Myo1b movement (see blue arrows) and (B) the consequence of this movement on actin dynamics (see gray arrows and new addition of G-actin).

Myo1b Controls the Assembly of F-Actin Foci at the TGN

Growing evidence indicates that the actin cytoskeleton plays a role in membrane trafficking at the TGN.[14,25,28,29] and that actin foci localizes in the TGN region.[28,32] However, the role of actin dynamics at an early stage of post-Golgi carrier biogenesis, such as formation of tubular-carrier precursors, was unknown. By monitoring the behavior of F-actin foci at the TGN in cells expressing Cherry-LifeAct and GFP-MPR, we observed that F-actin foci remained relatively non motile and stable at the opposite of the behavior of F-actin at the plasma membrane where a burst of actin occurs during the formation of coated pits to enable vesicle formation and ligand uptake.[33] Thirty percent of F-actin foci codistributed with MPR associated to the TGN and F-actin foci were in close proximity to the TGN membrane when MPR tubules formed, suggesting that F-actin foci interact with the TGN membrane. We showed that F-actin foci are required for the formation of tubular-carrier precursors emanating from the TGN and give rise to the formation of post-Golgi carriers. The Arp2/3 complex has been proposed to nucleate the actin filaments that compose F-actin foci at the TGN membrane.[34] The reduction of F-actin foci in the TGN region, upon Arp2/3 complex KD by siRNA, phenocopied the accumulation of MPR in the TGN, as seen in the Myo1b KD. Furthermore, KD of the Arp2/3 complex reduced the formation of tubular-carrier precursors and consequently the number of cytoplasmic MPR carrier vesicles. Myosins generate force by interacting in an ATP dependant manner with F-actin filaments. We found that Myo1b was in association with F-actin foci in the TGN region and that its depletion reduced the number of F-actin foci at the TGN by 60%. Myo1b depletion also decreased the number of Arp2/3 structures in the volume occupied by MPR in the TGN area, but not at the ventral plasma membrane. While Myo1b overexpression increased the number of Arp2/3 structures in the Golgi region with no significant increase at the ventral plasma membrane.

Together, these findings allowed us to propose that Myo1b tethers F-actin foci to the TGN membrane. Recently, Kobama and colleagues reported the ability of Myo1b, to bind to phosphoinositides.[35] As Myo1b contains, in its tail domain, a cluster of basic amino acids as well as a PH-like motif, Myo1b could bind the TGN membrane via its PH motif. Similar to the other members of the subgroup of short tail myosins 1, Myo1b does not contain a second actin binding domain or a protein-protein interacting motif, such as a single Src homology domain 3 (SH3), that can bind proteins involved in actin

polymerization.[36] However, another member of the short tail myosin 1 subgroup, myosin 1c has been proposed to spatially control actin assembly to the plasma membrane in Xenopus Oocytes via its motor domain.[37] Myo1b, similarly to Myo1c, could spatially control actin assembly at the TGN membrane by interacting with F-actin via its motor domain and at the membrane via its PH motif. In agreement with this hypothesis, we found that the force generated by Myo1b was required to tether F-actin foci to the TGN membrane. Using an in vitro motility assay, we observed that the rigor mutant remained bound to F-actin while the E476K mutant displayed a weak affinity for F-actin. Myo1b, but not Myo1b-rigor or Myo1b-E476K, rescued the normal steady-state distribution of F-actin foci at the TGN.

Our findings can be summarized in a model where the force generated by Myo1b controls F-actin foci orientation and binding to the TGN membrane. The force generated by Myo1b could thereby facilitate actin polymerization and contribute to membrane curvature of the TGN (**Fig. 2**). According to this model, Myo1b-Tail interactions with the TGN membrane should be strong enough to avoid Myo1b-Tail slipping and Myo1b dissociation in the TGN membrane during the force produced by Myo1b-motor on F-actin. Further work needs to be done to test this hypothesis.

Is Myo1b Function Coordinated with Actin Nucleation?

The Arp2/3 complex can be activated and recruited to the TGN through the interaction of clathrin coat with Cyfip1/2, triggering WAVE/N-WASP actin nucleation.[14] However, this mechanism cannot account for the actin dependent exit of p75 since this receptor exits the TGN independently of clathrin.[38] ARF1 (ADP-ribosylation factor 1) recruits COPI and clathrin coat complexes on Golgi subdomains and also activates the Arp2/3 complex via cdc42 and N-WASP.[39-41] Furthermore, expression of an active cdc42 mutant activates the TGN exit of p75 (ref. 42). In addition, several NPFs, including WASP and WHAMM, contribute to the trafficking of cargo at the Golgi and TGN.[14,17] These data, together, highlights the existence of multiple TGN exit pathways with distinct mechanisms to recruit and activate the Arp2/3 complex at the TGN membrane. It is likely that Myo1b functions in these different pathways by tethering the newly polymerized F-Actin to the TGN membrane. Furthermore, we have previously reported that Myo1b participates in membrane trafficking along the endocytic pathway and in particular to the transfer of cargo from the membrane of the sorting endosomes to the internal vesicles.[22] We observed that overexpression of Myo1b increases membrane extensions at the surface of these endosomes. Conversely, we recently observed that Myo1b KD decreases the formation of such extensions (Almeida CG, unpublished data). Thus, Myo1b may function to tether newly polymerized F-actin to the membranes of different types of organelles and thereby trigger membrane remodeling of organelles.

Do Other Myosins Implicated in Membrane Traffic Contribute to Membrane Remodeling of Organelles?

Myosins from classes I and II could contribute to organelle and plasma membrane remodeling. Yeast myosins 1, with a long tail that interacts with NPF via an acidic motif at its C-terminus, were the first myosins reported to be involved in membrane remodeling.[9,10] More recently, another long tail myosin 1, myosin 1e, that could interact with NSF via its SH3 domain, has been suggested to play a similar role in mammalian cells.[43,44] It has been proposed that these myosins elongate the plasma membrane and/or cleave the newly-formed membrane invagination leading to the endosomes.[10] A recent report showed that depletion of myosin II inhibits scission of tubular-carrier precursors positive for Rab6.[16] We observed that inhibition of the ATPase activity of this myosin inhibits the scission of MPR tubules. This suggests that myosin II contributes to membrane remodeling leading to scission of tubular carrier precursors downstream of Myo1b function at the TGN (Almeida CG, unpublished data). Whether this myosin contributes directly to the scission or whether it acts in coordination with dynamin remains to be clarified.

Recent experimental evidence indicates that members of other classes of myosins contribute to membrane trafficking along both the endocytic and exocytic pathways and suggests that these myosins function by using distinct molecular mechanisms in distinct pathways. For example, myosin VI has been found to be involved in the first step of endocytosis as well as in the endocytic recycling pathway and more recently in the fusion of secretory vesicles to the plasma membrane.[19,45-47] Although depletion of myosin VI reduces tubule formation in the endocytic recycling pathway, its direct role in membrane remodeling for these different steps of membrane trafficking needs to be further examined.[19] Myosins V contribute to endocytic recycling of a variety of receptors and cargos. Yet, although the isoform Vb is associated with tubular networks, there is no evidence indicating that myosins V can remodel membrane of the recycling endosomes.[48-50] It is likely that myosin 18a participates in retrograde trafficking since its membrane receptor, GOLPH3, has been shown to contribute to retrograde Golgi trafficking in yeast and to be associated with the retromer complex in mammalian cells.[51] Myosin 18a depletion inhibits the frequency of formation of Golgi carriers. However, due to the question raised by the sequence of its motor domain, it is not clear whether this myosin can generate a force to remodel membrane. Myosin 18a presents a non-conserved residue in the "switch 2" region of the motor domain that abolishes cellular function of myosin II in *dictyostellium* and instead, myosin 18a presents an actin-binding site in an unusual PDZ domain at its N-terminus in order to have an ATP dependent actin-binding site.[52] Further work needs to be done to determine whether these myosins contribute to membrane remodeling of organelles by generating, with F-actin, a force on organelle membrane.

In conclusion, our recent work with that of Miserey-Lenkei and colleagues, provides two examples of myosins involved in membrane trafficking that control membrane remodeling of organelles.[16,23] These new results provide an emerging concept

that the force generated by myosins is part of the machinery implicated in membrane elongation and/or membrane scission, two steps of membrane remodeling that lead to the formation of carrier vesicles. Furthermore, they suggest that the function of some of these myosins needs to be coordinated to achieve the formation of carrier vesicles.

Acknowledgments

This work has been supported by the Institut Curie, the CNRS and the Agence Nationale pour la Recherche (grant ANR 09-BLAN-0027). C.G.A. has been the recipient of an EMBO long-term fellowship (ALTF 607-2006) and a Marie Curie action intra-European fellowship for career development (FP7-PEOPLE-2007–2-1-IEF N. 2200088).

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