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Review Article

The role of VPS4 in ESCRT-III polymer remodeling

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The endosomal sorting complex required for transport-III (ESCRT-III) and VPS4 catalyze a variety of membrane-remodeling processes in eukaryotes and archaea. Common to these processes is the dynamic recruitment of ESCRT-III proteins from the cytosol to the inner face of a membrane neck structure, their activation and filament formation inside or at the membrane neck and the subsequent or concomitant recruitment of the AAA-type ATPase VPS4. The dynamic assembly of ESCRT-III filaments and VPS4 on cellular membranes induces constriction of membrane necks with large diameters such as the cytokinetic midbody and necks with small diameters such as those of intraluminal vesicles or enveloped viruses. The two processes seem to use different sets of ESCRT-III filaments. Constriction is then thought to set the stage for membrane fission. Here, we review recent progress in understanding the structural transitions of ESCRT-III proteins required for filament formation, the functional role of VPS4 in dynamic ESCRT-III assembly and its active role in filament constriction. The recent data will be discussed in the context of different mechanistic models for inside-out membrane fission.

Introduction

Endosomal sorting complex required for transport-III (ESCRT-III) is an evolutionarily conserved protein machinery that mediates membrane-remodeling including scission in a variety of cellular contexts in eukaryotes and archaea [1,2]. ESCRT complexes have been first discovered in yeast to catalyze receptor trafficking via endosomes and multi-vesicular bodies (MVB) [3,4]. Since then, the ESCRT machinery has been implicated in many topologically equivalent membrane-remodeling processes. These include budding of enveloped viruses [5,6], cytokinesis [7,8], biogenesis of microvesicles and exosomes, plasma membrane and endolysosomal repair [9–11], neuron pruning [12], dendritic spine maintenance [13] and nuclear envelope reformation [14–16].

Yeast ESCRT-III comprises four subunits, Vps20, Snf7, Vps2 and Vps24, which assemble sequentially at the membrane [4]. Higher eukaryotes express the corresponding homologs in different isoforms, CHMP6, CHMP4A, B, C, CHMP2A, B and CHMP3 and in addition CHMP1A, B, CHMP5, CHMP7 and IST1 [17]. Comparable to the yeast system, CHMP4 recruitment is followed by CHMP3 and CHMP2 [18], two ESCRT-III members that block Snf7 (CHMP4) polymerization and cap ESCRT-III assembly prior to recycling [19,20]. Although some functions of individual ESCRT-III proteins have been linked to a specific cellular membrane-remodeling process, such as CHMP2B to dendritic spine regulation [21], CHMP4C to abscission checkpoint regulation [22] and CHMP7 to nuclear envelope sealing [14,23], the precise functions of most CHMP isoforms remain to be determined. While some remodeling processes such as cytokinesis require almost all ESCRT-III members and VPS4 [24], others such as HIV-1 budding can be achieved with a minimal set of one CHMP4 and one CHMP2 isoform plus VPS4 [18] or with CHMP3 acting synergistically with CHMP2A but not with CHMP2B [25]. This suggests that core ESCRT-III members CHMP4, CHMP2 and CHMP3 constitute a minimal membrane fission machinery together with VPS4, while the other ESCRT-III subunit exert functions specific for a given membrane-remodeling process. Common to all processes

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is that VPS4 is an indispensable factor of the fission machinery [17,26–28]. Consistent with a minimal membrane fission machinery CHMP4, CHMP3, CHMP2A and VPS4 likely catalyze membrane fission as proposed by the release of membrane tubules in an *in vitro* reconstitution experiment [29].

ESCRT-III polymerization

ESCRT-III proteins reside in the cytosol in an inactive closed helical conformation [30–32], which opens upon activation [33–35]. Although activation is likely first regulated by membrane recruitment of CHMP6 or CHMP4 via ESCRT-II VPS25 [36], Alix [37] or ESCRT-I Vps28 [38], fine tuning mechanisms such as phosphorylation of IST1 [39] and ubiquitination of CHMP1B [40] have been implicated in controlling their polymerization. Furthermore, CC2D1A and B act as negative regulators of CHMP4 polymerization [41,42] thereby controlling ESCRT-III recruitment during nuclear envelope reformation [43].

ESCRT-III polymerization is achieved by extending the helical hairpin and aligning the hairpins into a polymer that is further stabilized by domain exchange [44,45]. This generates extensive basic surfaces that stabilize positively curved membranes in case of CHMP1B [44] and negative membrane curvature in case of yeast Snf7 (CHMP4) [45]. The same structural principles are most likely employed to assemble CHMP4 spirals *in vitro* [46–48], helical CHMP2A–CHMP3 tubular structures [31,49,50], CHMP2A filaments [25], Vps24 (CHMP3) filaments [51] as well as archaeal CdvB filaments [52,53]. *In vivo*, CHMP4 (Snf7) spirals [54,55], as well as a CHMP2B helical scaffold, deform the plasma membrane into tubular structures [56]. Thicker spirals of ESCRT-III containing CHMP2A and IST1 have been directly imaged at the cytokinetic midbody [57–59], which presumably comprise bundles of ESCRT-III filaments [26]. In summary, although we know some of the structural principles of ESCRT-III polymerization, we still lack the high-resolution architecture and composition of a native ESCRT-III complex assembled at budding sites or during other ESCRT-regulated processes.

Membrane curvature is likely to play an important role in initiating ESCRT-III membrane assembly consistent with preferred Snf7 assembly on negatively curved membranes [60]. This is further in line with Vps20 (CHMP6) generating a curvature sensitive complex that nucleates Vps32 (Snf7/CHMP4) polymerization when bound to ESCRT-II [61]. ESCRT-III membrane interaction requires negatively charged membranes [62–64], which is consistent with mutation of basic CHMP3 residues abrogating plasma membrane interaction [30] and the extensive basic membrane binding surfaces generated by CHMP1B and Snf7 polymer structures [44,45]. Most ESCRT-III proteins also have an N-terminal conserved membrane insertion motif [50,56] that is important for CHMP2B membrane tube formation [56] and ESCRT-III anchoring at endosomes [65].

Structural basis of ESCRT-III disassembly by VPS4

Human cells express two paralogs of *vps4* (VPS4A and VPS4B) [66], while archaea and yeast express one [3]. Little is yet known about the differential functions of VPS4A versus VPS4B in higher eukaryotes. VPS4 is a member of the type-I or meiotic clade of AAA family of proteins (ATPase associated with various activities) [67]. It disassembles ESCRT-III polymers thereby recycling them [68]. Several studies show that *in vitro*-assembled ESCRT-III polymers are efficiently disassembled by VPS4 indirectly corroborating the physiological relevance of the assemblies [49,51,69].

VPS4 recruits ESCRT-III members via its N-terminal MIT (Microtubule Interacting and Trafficking) domain interacting with the ESCRT-III MIM (MIT domain interacting motif) [70–72]. A short linker connects the MIT domain to the ATPase domain, which has been implicated in the regulation of VPS4 assembly and activity [69,73]. The ATPase activity is formed by the large and small ATPase domains that harbor the nucleotide binding site at their interface [74–76]. Nucleotide binding is required for hexamer assembly and induces conformational changes associated with the ATPase reaction cycle [77–80]. Assembly and ATPase activity are further regulated by Vta1p/LIP5 [76,80–83]. In the hexamer structure, a Vta1 dimer bridges two adjacent Vps4 subunits [84,85]. ESCRT-III protein substrate passes via the central pore [86] by global unfolding ESCRT-III subunits upon ATP hydrolysis [87]. However, it remains to be determined whether the entire ESCRT-III protein is being unfolded by passing through the central pore or only part of each protomer passes thereby inducing polymer disassembly. Structures of active VPS4 hexamers revealed a pseudohexameric helical assembly that binds ESCRT-III substrates through a repeating array of dipeptide-binding pockets formed by pore loop 1 residues, which led to the suggestion of a ‘conveyor belt’ model of translocation. In this model, ATP binding induces assembly at the growing end of the helix and substrate engagement while hydrolysis promotes helix disassembly and substrate release at the lagging end [84,88,89].

ESCRT-III remodeling by VPS4

The residence time of ESCRT-III during HIV-1 budding is rather short and complemented by a short transient recruitment of several VPS4 complexes [90–93], which most likely induce constriction. Furthermore, live cell imaging suggested that ESCRT-III and VPS4 must dissociate from the budding site to proceed to fission [28]. If fission fails upon ESCRT release, ESCRT-III and VPS4 are recruited for another trial [28]. Short dynamic, almost simultaneous recruitment of ESCRT-III and VPS4 including their continuous stochastic exchange has been also observed during intraluminal vesicle (ILV) formation at endosomes. However, Vps4 was not required to maintain dynamic assembly and disassembly of ESCRT-III [27] but is essential for ILV release [94]. The number of ESCRT-III involved in endosomal budding seems to be in the range of 75–200 Snf7 (CHMP4), 15–50 Vps24 (CHMP3) and several hexamers of Vps4 [27]. Similarly, ESCRT-III assembly at the midbody follows a dynamic turnover that depends on VPS4 ATPase activity [26]. *In vitro*, VPS4 can promote the growth and turnover of ESCRT-III Vps2/VPS24-Snf7 (CHMP2A/CHMP3–CHMP4) filaments assembled on flat membranes [26] further corroborating the dynamic features of ESCRT-III assemblies.

A central question is how ESCRT-III filaments constrict small 10–50 nm in diameter bud necks such as those of ILVs or enveloped viruses and large (μm) diameters as the cytokinetic midbody. Because the multi-stranded bundles of ESCRT-III forming dynamic spirals at the midbody [26,57–59] have never been observed at smaller bud sites, constriction of larger and smaller diameters seems to require different sets of ESCRT-III filaments. This does not exclude that ESCRT-III assemblies employed to constrict small bud necks are employed as well once larger diameters have been constricted to a compatible diameter.

ESCRT-III CHMP3 and CHMP2A follow CHMP4 polymerization and recruit VPS4. CHMP2A and CHMP3 assemble *in vitro* into helical tubular structures with diameters ranging from 47 to 51 nm [25]. Such tubular structures bind membrane on the outside, VPS4 hexamers inside, which disassemble the polymer in the presence of ATP *in vitro* [95]. Using atomic force microscopy (AFM) [96–98], the structure and dynamics of

Figure 1. Model of VPS4-catalyzed constriction of CHMP2A-3 tubular filaments leading to membrane constriction.

(A) CHMP2A-3 helical filaments assemble within a membrane neck structure such as a vesicle or virus bud; note that these filaments may be constantly remodeled by growing at one end and shrinking at the other. The number of turns present at budding sites is yet unknown. The filaments formed by CHMP2A-3 have diameters of ~50 nm allowing VPS4 to diffuse inside. (B) VPS4 assembles within the polymer into a hexameric/pseudohexameric structure in the presence of ATP and Mg^{2+} starting disassembly in an asymmetric fashion to a point of complete constriction (C) that leads to filament breakage. One or several VPS4 complexes may be implicated in this process. Constriction induces the formation of a dome-like end cap that might constrict the membrane thereby setting the stage for membrane fission (Figure adapted from [99]).

the ESCRT-III system have been studied at the nm-scale before [48,61]. Studying CHMP2A–CHMP3 tube remodeling by high-speed AFM revealed further that the tubular structures are progressively constricted and cleaved at limited concentrations of VPS4B. This was confirmed by electron microscopy imaging which corroborated that CHMP2A–CHMP3 tube cleavage produced dome-like end caps [99]. Based on the spontaneous formation of ‘domes’ observed upon CHMP2A–CHMP3 tube assembly [49], it has been suggested that such a structure could constrict a membrane tube from 50 nm to a few nanometers thereby setting the stage for membrane fission [100]. Thus, the finding that one or several VPS4 complexes actively remodel ESCRT-III generating dome-like structures *in vitro* supports a dome-like model for the structural basis of ESCRT-III-catalyzed membrane constriction (Figure 1) that sets the stage for membrane fission. Furthermore, CHMP2A–CHMP3 filament constriction and cleavage by VPS4 leads eventually to complete disassembly, indicating that the dome is a highly dynamic structure, which is consistent with the proposal that ESCRT-III and VPS4 must have left the constriction site prior to completion of fission [28].

Models for membrane fission

Besides the dome model [101], which is supported by the fact that VPS4 generates dome-like structures from CHMP2A–CHMP3 tubular filaments that might be able to constrict membranes, other models for ESCRT-III/VPS4-catalyzed membrane constriction and fission have been put forward. Given the dynamics of ESCRT-III, it has been suggested that VPS4 complexes might exchange ESCRT-III subunits that favor higher curvature, which could constrict a helical tube or create a cone [17] (Figure 2A). Notably, cones of CHMP2A–CHMP3 and Vps2–Vps24 as well as of positive curvature stabilizing IST1–CHMP1B have been observed *in vitro* [31,44,50]. The role of a cone structure was further developed in the flattening cone model that predicts that flattening of the cone leads to neck closure. Calculations indicate that the minimal adhesive strength of the membrane-protein interactions required for scission is lower for cones than for domes. Furthermore, the adhesion-induced constriction forces exerted by the ESCRT assemblies onto the membrane necks seem to be slightly higher for cones than for domes [102]. Since straight tubes of CHMP2A–CHMP3 are constricted by VPS4, cone-shaped tubes might as well be constricted actively by VPS4.

Figure 2. ESCRT-III filament remodeling inducing membrane-remodeling.

(A) The dynamic exchange of subunits with high intrinsic curvature into filaments representing a lower curvature might lead to constriction. (B) Out-of-plane buckling by a spiraling filament growing beyond its preferred radius of curvature due to the accumulation of elastic stress can as well induce remodeling (figure adapted from [17,104]).

Another model suggests that membrane extrusion and fission are driven by filament buckling [48,103]. In this model, ESCRT-III assembles on a flat membrane into filaments with a preferred radius. Filament polymerization with smaller radii (than the preferred one) will store over bending energy and polymerization producing larger radii will exert an axial force. The forces generated by these two types of filaments can catapult the filaments out of plane, thereby producing vesiculation [104] (Figure 2B). Subsequent membrane fission might be induced by VPS4-catalyzed reversal of the tubular filaments to planar filaments [105].

Conclusions and perspectives

ESCRT complexes catalyze universal membrane-remodeling processes with a preferred inside-out topology [17]. It is thus crucial to understand the mechanics of the machinery in order to fully understand how they catalyze membrane constriction and control membrane fission. The emerging picture of their function is that they are assembled in a dynamic fashion with or without VPS4 action, which needs to be consolidated. Secondly, prior to disassembly or during disassembly, at least one class of ESCRT-III filaments composed of CHMP2A and CHMP3 are constricted. It remains to be determined whether such ESCRT-III filament constriction can constrict membrane tubes as well and cleave them by inducing membrane fission. Furthermore, future experiments are required to determine how VPS4 remodels different ESCRT-III filaments or tubular structures. Finally, the function of larger diameter multi-stranded ESCRT-III spirals imaged at the midbody need to be reconciled or combined with the function of smaller diameter filaments such as CHMP2A–CHMP3 likely acting at narrow bud neck structures.

Abbreviations

AFM, atomic force microscopy; ESCRT-III, endosomal sorting complex required for transport-III; ILV, intraluminal vesicle; MIT, microtubule interacting and trafficking.

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Competing Interests

The Authors declare that there are no competing interests associated with the manuscript.

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