Golgi Biogenesis

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The Golgi is an essential membrane-bound organelle in the secretory pathway of eukaryotic cells. In mammalian cells, the Golgi stacks are integrated into a continuous perinuclear ribbon, which poses a challenge for the daughter cells to inherit this membrane organelle during cell division. To facilitate proper partitioning, the mammalian Golgi ribbon is disassembled into vesicles in early mitosis. Following segregation into the daughter cells, a functional Golgi is reformed. Here we summarize our current understanding of the molecular mechanisms that control the mitotic Golgi disassembly and postmitotic reassembly cycle in mammalian cells.

THE FUNCTION OF THE GOLGI

The Golgi is a vital membrane-bound organelle found in all eukaryotic cells, including plants, animals, and fungi (Klute et al. 2011). Its basic structure is comprised of a stack of closely apposed and flattened cisternae with fenestrated rims. The primary function of the Golgi is to modify and package proteins and lipids into transport carriers and send them to the proper locations. Secretory or transmembrane proteins are delivered from the endoplasmic reticulum (ER) to the *cis*-Golgi network. Subsequently, the cargo molecules travel through the different cisternae of the Golgi where they are posttranslationally modified by resident enzymes. Modifications include glycosylation, phosphorylation, sulfation, and proteolysis (Goldfischer 1982). Cargo molecules have been described to move through the Golgi stack

in several different ways (Emr et al. 2009). One possible mechanism is through cisternal maturation, in which the cargo remains in the cisternae and new cisternae form at the *cis* side. The newly formed cisternae then mature by receiving Golgi enzymes via retrograde transport of COPI vesicles (Bonfanti et al. 1998; Losev et al. 2006; Matsuura-Tokita et al. 2006). Alternatively, proteins are shuttled by COPI vesicles between cisternae, whereas Golgi resident enzymes reside in the relatively stable cisternal compartments (Orci et al. 1997; Elsner et al. 2003). In addition, cargo molecules may flow through transient tubular connections between adjacent cisternae (Trucco et al. 2004), or by rapid partitioning between different lipid domains (Patterson et al. 2008). At the *trans*-Golgi network (TGN), cargo molecules are sorted and transported to their proper destinations such as the endosomal-lysosomal system,

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the plasma membrane, or outside of the cell. In addition to its function in protein trafficking, the Golgi in mammalian cells plays an important role in cell polarity (Jaffe and Hall 2005) and cell cycle control (Sutterlin et al. 2002), suggesting a direct link between membrane trafficking, cell growth, and cell polarity.

The Structure of the Golgi in Different Organisms

Although the most characteristic structure of the Golgi is the stack of closely aligned cisternal membranes, some species and specialized cell types show variations in the number and morphological organization of the stacks. In some unicellular eukaryotes, including *Toxoplasma gondii* (Hager et al. 1999), *Trypanosoma brucei* (He et al. 2004), and the green algae *Ostreococcus tauri* (one of the smallest free-living unicellular eukaryotes) (Henderson et al. 2007), each cell contains only one single Golgi stack. In the vast majority of organisms, including most fungi, plants, and invertebrates, each cell hosts multiple Golgi stacks that are scattered throughout the cytoplasm. However, several eukaryotic species do not seem to have a stacked Golgi (Dacks et al. 2003; Klute et al. 2011). One prominent example is the budding yeast *Saccharomyces cerevisiae*, in which Golgi membranes do not form stacks under normal growth conditions. Instead, individual cisternae are distributed throughout the cytoplasm (Preuss et al. 1992). Notably, Golgi cisternae appear to be stacked in certain mutant strains of *S. cerevisiae* (Novick et al. 1981; Franzusoff et al. 1991; Weinberger et al. 2005), but it is unclear whether the stacks show structural and functional *cis*-to-*trans* polarity as in most other species.

The fact that Golgi cisternae in *S. cerevisiae* are not aligned into stacks indicates that stacking is not essential for secretion. However, Golgi stacking is a pronounced feature of the majority of eukaryotic organisms (Mowbrey and Dacks 2009; Klute et al. 2011), suggesting that stacking has important functional consequences. Indeed, several lines of evidence indicate that stacking increases the complexity of Golgi function. Stacking of Golgi membranes

impacts the rate of protein trafficking and the accuracy of its processing. The close spatial arrangement of cisternae in stacks minimizes the distance the molecules have to travel, which may improve the efficiency of cargo transport. By reducing the space between the cisternae, budding transport vesicles are already in close proximity to the target membrane with which they fuse (Linstedt 1999). Furthermore, during budding, local tethering complexes link the vesicles to the target cisternae, which may prevent the vesicles from diffusing away (Lupashin and Sztul 2005). Perhaps more importantly, cisternal stacking directly controls the flux rate of protein trafficking through the Golgi stack. The progression of molecules through the stack depends on the budding and fusion rate of COPI vesicles. With fully stacked cisternae, vesicles can only form and fuse at the rims. Once the cisternae unstack, however, more membrane area becomes accessible, thereby increasing the rate of vesicle budding and cargo transport through the Golgi (Wang et al. 2008). Moreover, stacking ensures efficient and accurate posttranslational modifications of cargo proteins by the resident glycosylation enzymes (Varki 1998). Each cisterna corresponds to a different reaction compartment, in which a specific subset of enzymes is concentrated. These enzymes are distributed across the stack in the order by which they operate (Kornfeld and Kornfeld 1985; Roth 2002). This ensures that the cargo proteins are modified sequentially and accurately as they travel through the stack.

Unlike the discrete individual stacks in lower organisms, the Golgi in vertebrate cells shows a more complex organization (Wei and Seemann 2010). Each mammalian cell typically contains about 40–100 Golgi stacks (Shima et al. 1998) that usually interconnect with each other into a continuous ribbon-like structure that is localized adjacent to the nucleus (Fig. 1A) (Rambourg et al. 1987). The formation and maintenance of the perinuclear localization of the Golgi ribbon relies on the well-organized microtubule cytoskeleton emanating from the perinuclear centrosomes (Glick and Nakano 2009). The minus end-directed motor cytoplasmic dynein associates with the

Figure 1. Morphology of the Golgi during the cell cycle in mammalian cells. Untreated NRK cells (A–E) or cells treated with the kinesin 5 inhibitor s-trityl-L-cystein, that inhibits centrosome separation and thereby induces the formation of monoasters (F), were fixed and triple-stained for the Golgi resident protein GM130 (green), α -tubulin (red), and DNA (Hoechst, blue). Images are z-projections of confocal image stacks. The continuous Golgi ribbon (A) is disassembled in early mitosis (B). The Golgi membranes concentrate at the spindle poles in metaphase cells (C) as well as in cells with monoasters (F). During anaphase (D), the Golgi membranes are partitioned and reform two ribbons on the opposite sides of the nucleus (E). Scale bar 10 μ m (A,B,E); 5 μ m (C,D,F).

Golgi and moves the membranes along the microtubules toward the centrosomes, leading to the concentration of the Golgi stacks in the pericentriolar region and formation of the Golgi ribbon (Rios and Bornens 2003; Miller et al. 2009). The variations in Golgi architecture among species suggest that the molecular mechanisms that control the Golgi structure and its inheritance may have evolved differently. Here we focus on the mechanisms that facilitate the inheritance of the mammalian Golgi during mitosis.

MOLECULAR MECHANISMS OF GOLGI BIOGENESIS IN MAMMALIAN CELLS

In mammalian cells, the continuous Golgi ribbon needs to be segregated into the two daughter cells when the cell divides. This is achieved through a three-step process (Fig. 2) that involves disassembly (consisting of ribbon unlinking, cisternal unstacking, and vesiculation), partitioning, and reassembly of Golgi membranes (Wei and Seemann 2009b). In late G2 phase of the cell cycle, the Golgi ribbon is

unlinked upon separation of the lateral connections between the stacks. In early mitosis, the cisternae then unstack and further disassemble into vesicular and tubular membranes (Lucocq et al. 1989). These mitotic Golgi membranes are then divided into the two daughter cells where they are reassembled into a functional Golgi (Shorter and Warren 2002; Lowe and Barr 2007).

The Biological Significance of Mitotic Golgi Disassembly in Mammalian Cells

Several lines of evidence indicate that the disassembly of the Golgi ribbon is important for its division process (Shorter and Warren 2002) as well as for mitotic progression (Colanzi and Corda 2007). The simplest explanation for mitotic Golgi disassembly is the necessity to divide the continuous ribbon between the two daughters. In support of this notion, the Golgi in some primitive organisms undergoes binary fission to split the organelle into two entities (Pelletier et al. 2002). The comparable process in mammalian cells is the unlinking of the Golgi

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Figure 2. Schematic illustration of the mitotic division of the mammalian Golgi. During interphase, the Golgi stacks (green) are interconnected into a ribbon-like structure near the centrosomes (red) and next to the nucleus (blue). In late G2, before the cells enter mitosis, the connections between the stacks are severed. In early mitosis, the Golgi cisternae unstack and vesiculate, leading to the disassembly of the Golgi. By metaphase, mitotic Golgi membranes cluster at the spindle poles, associate with astral microtubules, and are dispersed throughout the cytoplasm. The membranes are then partitioned as the spindle elongates and segregates the chromosomes during anaphase. During telophase and cytokinesis, the Golgi membranes reassemble in each daughter cell into a larger ribbon next to the centrosome and a smaller ribbon adjacent to the cleavage furrow. In the final stage, the smaller ribbon moves to the opposite side of the nucleus to merge with the larger ribbon.

ribbon in late G2 phase. This first step in the disassembly process has been described to contribute to the transition between G2 and mitosis (Colanzi and Corda 2007). Inhibition of this step by interfering with a particular set of Golgi structural proteins (GRASP65 and GRASP55), kinases (MEK1, ERK, and Plk3), or membrane fission molecules (CtBP/BARS) delays G2/M transition. These observations led to the postulation of a Golgi-specific checkpoint (Sutterlin et al. 2002; Hidalgo Carcedo et al. 2004; Yoshimura et al. 2005; Colanzi et al. 2007; Feinstein and Linstedt 2007) that monitors the structural integrity of the Golgi ribbon and delays entry into mitosis, but differs from the conventional

G2/M DNA damage checkpoint. Inhibition of Golgi ribbon unlinking prevents a key mitotic regulator, the Aurora A kinase, from being recruited to the centrosomes and thus impairs its activation (Persico et al. 2010).

After the ribbon is unlinked and the cell enters mitosis, the Golgi cisternae are unstacked and converted into vesicular membranes. Complete vesiculation does not appear to be an absolute requirement for Golgi partitioning or mitotic progression. Expression of nonregulatable mutants of the Golgi stacking protein GRASP65 (Tang et al. 2010b) or microinjection of a nonphosphorylatable p47 (Uchiyama et al. 2003), an adaptor protein of the AAA ATPase

p97 that is involved in postmitotic Golgi membrane fusion, leads to larger, less disassembled clusters of Golgi membranes during mitosis, but does not seem to block the segregation of Golgi membranes into the daughter cells. Instead, a more possible role for Golgi vesiculation may be to release components from the Golgi that have an important role in late mitosis or cytokinesis (Wei and Seemann 2010). For example, Nir2, a Golgi-associated protein in interphase, is released from the Golgi membrane in mitosis and moves to the cleavage furrow where it is required for cytokinesis (Litvak et al. 2004). In addition, the Golgi also provides membranes for the abscission step (Gromley et al. 2005; Goss and Toomre 2008).

Mechanisms of Mitotic Golgi Disassembly

Unlinking the Golgi Ribbon

In late G2 phase, the Golgi ribbon is unlinked by severing the tubular connections between the stacks (Colanzi and Corda 2007). The conversion of the ribbon into stacks depends on the MAP kinase, kinase MEK1 (Acharya et al. 1998; Colanzi et al. 2000; Kano et al. 2000; Sutterlin et al. 2001), but not the classical MEK1 substrates ERK1 or ERK2 (Acharya et al. 1998; Colanzi et al. 2000). Instead, ERK1c and Plk3/VRK1 were shown to contribute to Golgi ribbon unlinking in late G2 phase (Xie et al. 2004; Shaul and Seger 2006; Lopez-Sanchez et al. 2009), but their substrates on the Golgi membranes have not yet been identified. Another study showed that MEK1 causes Golgi ribbon unlinking through mitotic phosphorylation of GRASP55 by ERK2 (Jesch et al. 2001a). Expression of a nonphosphorylatable GRASP55 mutant interferes with ribbon unlinking in late G2 (Feinstein and Linstedt 2008), although this result was not confirmed by others (Duran et al. 2008; Xiang and Wang 2010). Precisely how GRASP55 is involved in Golgi ribbon unlinking remains unclear. Similarly, microinjection of antibodies against GRASP65 also indicates a role for GRASP65 in severing of the ribbon (Puthenveedu et al. 2006) and the progression from G2 into M-phase (Sutterlin et al. 2002). Besides these kinases, the membrane fission protein

CtBP1/BARS is important for Golgi ribbon unlinking (Hidalgo Carcedo et al. 2004). Inhibition of BARS activity prevents severing of the ribbon as well as G2/M transition (Colanzi et al. 2007), but the underlying molecular mechanisms as well as the signaling events that activate BARS await further investigation.

Vesiculation and Unstacking of the Cisternae

Following the conversion of the Golgi ribbon into stacks, the cisternae are unstacked and vesiculated through COPI-dependent vesicle formation (Misteli and Warren 1994; Xiang et al. 2007; Tang et al. 2008). Vesiculation and cisternal unstacking occurs simultaneously in early mitosis, leading to rapid conversion of the Golgi into vesicular and tubular membranes. Vesiculation of the Golgi cisternae is triggered by an imbalance of membrane budding and fusion during mitosis (Warren 1993; Lowe et al. 1998b). In interphase, COPI vesicles are captured and tethered to the *cis*-cisternae by the ternary giantin-p115-GM130 tethering complex prior to membrane fusion (Fig. 3A) (Nakamura et al. 1997; Sonnichsen et al. 1998). Giantin on the vesicle is linked by p115 to GM130 on the Golgi cisternae (Nakamura 2010), which is regulated by the small GTPase Rab1 (Moyer et al. 2001; Weide et al. 2001; Beard et al. 2005). Once the complex is formed, p115 directly catalyzes the formation of SNARE complexes, leading to fusion of the two membranes (Shorter et al. 2002; Diao et al. 2008). During mitosis, Arf1 remains active and COPI vesicles continue to form (Xiang et al. 2007), but the vesicles cannot fuse with their target membranes because of the disruption of vesicle tethering complex (Misteli and Warren 1994; Nakamura et al. 1997; Lowe et al. 1998b). Phosphorylation of GM130 by Cdk1 in early mitosis prevents p115 from binding, thereby blocking vesicle tethering and subsequent fusion (Fig. 3B) (Levine et al. 1996; Lowe et al. 1998b). Rab1 is also phosphorylated by Cdk1, which may contribute to the inhibition of vesicle tethering and fusion (Bailly et al. 1991; Preisinger et al. 2005). The persistent budding of COPI vesicles without fusion

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Figure 3. Schematic illustration of putative Golgi disassembly and reassembly mechanisms during the cell cycle. (A) During interphase, Golgi cisternae are held together by *trans*-oligomers of GRASP65 and GRASP55 (not shown). COPI transport vesicles are tethered to the Golgi by the giantin-p115-GM130 complex, which facilitates fusion with the targeting membrane. (B) Early in mitosis, phosphorylation of GM130 by Cdk1 inhibits binding of p115, which helps block vesicle tethering and subsequent fusion. GRASP65 is phosphorylated by Cdk1 and Plk, which disrupts GRASP65 oligomerization and results in cisternal unstacking. Unstacking increases the membrane surface for the formation of COPI vesicles that fail to fuse because of the disruption of the tethering complex, leading to disassembly of the Golgi (C). (D) During late mitosis, the Golgi reforms by two fusion processes mediated by NSF and p97 ATPases together with their adaptor proteins. Following dephosphorylation of GM130, tethering and NSF-mediated fusion are restored, generating short cisternae, whereas p97-mediated fusion reassembles membranes into longer, but single cisternae. Reformed cisternae are first linked by p115 and then stacked once GRASP65 is dephosphorylated by PP2A.

gradually consumes the cisternae (Lowe et al. 1998a). In addition to COPI-dependent vesiculation, the Golgi cisternae are also disassembled by a COPI-independent pathway (Misteli and Warren 1995), but the exact role and mechanism of this pathway remains elusive.

Efficient disassembly of the Golgi in early mitosis is facilitated by unstacking of the cisternae, which significantly enhances COPI vesicle formation. Compared to stacked cisternae, unstacked membranes provide a larger surface for COPI vesicle budding (Fig. 3B) (Wang et al. 2008). Mitotic unstacking of Golgi cisternae is achieved through phosphorylation of GRASP65 and its homolog GRASP55. The GRASP proteins are Golgi peripheral membrane proteins that form dimers. These dimers from adjacent membranes interact with each

other to form *trans*-oligomers that hold the Golgi cisternae together in a stack (Wang et al. 2003). Mitotic phosphorylation leads to GRASP deoligomerization and thus Golgi unstacking (Wang 2008). GRASP65 is regulated by the Polo-like kinase Plk1 and Cdk1 (Fig. 3B) (Lin et al. 2000; Wang et al. 2003), whereas GRASP55 is mitotically phosphorylated by ERK2 and Cdk1 (Jesch et al. 2001a; Xiang and Wang 2010).

Partitioning the Golgi into the Daughter Cells

Once disassembled, the mitotic Golgi membranes are present in at least two pools. In addition to vesicles that are evenly dispersed throughout the cytoplasm (Jesch et al. 2001b; Axelsson and Warren 2004), a significant portion of the mitotic Golgi membranes

concentrate at the spindle poles and associate with astral microtubules (Fig. 1C, metaphase; Fig. 2) (Shima et al. 1998; Jokitalo et al. 2001; Seemann et al. 2002). These spindle-associated membranes are organized in clusters of tubulovesicular structures with a relatively constant number within each cell. The mitotic clusters are polarized, with *cis*-Golgi proteins spatially separated from those of the *trans*-Golgi (Shima et al. 1997), a similar pattern to that of the Golgi stacks during interphase. The spindle association of the mitotic Golgi clusters, which is also prominent in cells with monoasters (Fig. 1F), has prompted the idea that Golgi partitioning is regulated by the spindle (Shorter and Warren 2002). This has been tested by inducing asymmetrical cell division to segregate the entire spindle into only one of the daughter cells (Wei and Seemann 2009a). Upon division, both daughter cells assemble functional Golgi stacks, but the ribbon of interconnected stacks are only observed in the daughter cell that receives mitotic Golgi clusters partitioned together with the spindle. This indicates that the spindle plays a vital role in partitioning an intact Golgi ribbon (Wei and Seemann 2009c).

Postmitotic Golgi Reassembly

Upon segregation into the nascent daughter cells, the mitotic Golgi membranes reassemble during telophase and cytokinesis into two distinct ribbons on opposite sides of the nucleus (Fig. 1E, cytokinesis; Fig. 2). A smaller Golgi ribbon reforms adjacent to the midbody, whereas a larger ribbon is found in the pericentriolar region on the side away from the cleavage furrow (Shima et al. 1998; Bartz et al. 2008). The function of the smaller ribbon is not clear, but its positioning next to the midbody may facilitate an efficient and polarized delivery of membranes into the cleavage furrow to seal the plasma membrane during abscission. Consistent with this idea, it has been observed that Golgi-derived vesicles are directly delivered from both daughter cells into the cleavage furrow where these membranes fuse (Goss and Toomre 2008). Following abscission, the smaller ribbon moves to the opposite side of

the nucleus and merges with the larger Golgi into a continuous ribbon (Gaietta et al. 2006).

Mechanistically, the Golgi is reformed at the end of mitosis by two interrelated processes, formation of flattened cisternae by membrane fusion and stacking of cisternae. Postmitotic Golgi membrane fusion is mediated by two AAA-ATPases, the N-ethylmaleimide-sensitive factor (NSF) and the valosin-containing protein (VCP)/p97 (Cdc48 in yeast) (Fig. 3D) (Acharya et al. 1995; Rabouille et al. 1995; Kondo et al. 1997). Stacking of Golgi cisternae requires the tethering factor p115 that initially links adjacent membranes (Shorter and Warren 1999) and the GRASP proteins that hold the cisternae into stacks (Fig. 3E) (Barr et al. 1998; Shorter et al. 1999).

Two Membrane Fusion Pathways and Cisternal Formation

Like most intracellular membrane fusion processes, postmitotic Golgi membrane fusion requires SNARE proteins. The assembly of SNARE proteins on opposite membranes into a SNAREpin complex leads to membrane fusion (Rothman and Warren 1994; Weber et al. 1998). Once the membranes are fused, the SNAREpin complex is then disassembled by NSF, which allows the next round of SNAREpin assembly and membrane fusion. Purified NSF, together with its cofactors α - and γ -SNAP (soluble NSF attachment protein), and the tethering factor p115 are sufficient for in vitro reassembly of mitotic Golgi membranes into cisternae (Rabouille et al. 1995; Tang et al. 2008, 2010a). However, although the ATPase activity of NSF is required for SNARE disassembly and sustained membrane fusion in most membrane fusion events, this activity is not required for postmitotic Golgi membrane fusion, suggesting a different role for NSF in Golgi reassembly (Muller et al. 1999). In telophase, dephosphorylation of GM130 (Lowe et al. 2000) allows the reformation of the giantin-p115-GM130 tethers (Fig. 3D). This reassociation may contribute to membrane fusion and cisternal formation (Shorter and Warren 1999) and is regulated by a Rab-GTPase (Shorter et al. 2002).

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Compared to NSF-dependent membrane fusion, p97-mediated cisternal regrowth is less well understood (Fig. 3D). Treatment of mitotic Golgi membranes with purified p97 and its cofactor p47 generates long, single cisternae (Rabouille et al. 1995; Kondo et al. 1997), indicating that p97/p47 drives homotypic membrane fusion. Furthermore, p97 can bind another adaptor protein, p37, and the p97/p37 complex also promotes Golgi membrane fusion in vitro. Unlike the p97/p47 complex, however, the p97/p37 membrane fusion activity depends on p115 (Uchiyama et al. 2006). Although NSF- and p97-mediated Golgi reassembly share common components of SNARE proteins, these two pathways contribute non-additively to cisternal regrowth (Rabouille et al. 1998). This suggests that they may have distinct roles in postmitotic Golgi membrane fusion, but the coordination between two pathways has not been fully elucidated.

The activity of the p97/p47 complex in postmitotic Golgi reassembly depends on ubiquitin (Meyer 2005). Although p97 and ubiquitination are often associated with protein degradation, the membrane fusion activity of p97 does not involve the proteasome (Meyer et al. 2002; Wang et al. 2004). p47 binds mono-ubiquitin through its ubiquitin-associated (UBA) domain (Meyer et al. 2002). Deletion of the UBA domain of p47 or adding a mutant ubiquitin that does not bind to p47 suppresses p97/p47-mediated postmitotic fusion of Golgi membranes in vitro (Meyer et al. 2002; Wang et al. 2004). Furthermore, the p97/p47 membrane fusion process requires the activity of the deubiquitinating enzyme VCI135 to remove ubiquitin moieties that were added during mitotic disassembly (Uchiyama et al. 2002; Wang et al. 2004). Full understanding of the underlying mechanism, however, requires the identification of the ubiquitin ligase and the substrate(s) on the Golgi membranes.

Cisternal Stacking

In telophase, cisternae start to reform by NSF- and p97-mediated membrane fusion and begin to align with each other into stacks. The

tethering factor p115 is required not only for NSF- and p97/p37-dependent cisternal formation, but also for initial stack formation (Shorter and Warren 1999). However, p115 is only temporarily required in the early steps of stacking. Following formation of the initial connections, stacking of the newly formed cisternae relies on the stacking proteins GRASP65 and GRASP55 (Fig. 3E). Both GRASPs are dephosphorylated at the end of mitosis, which restores their capability of oligomerization and thus allows cisternal restacking (Wang et al. 2003, 2005; Tang et al. 2010b). PP2A has been identified as the phosphatase that reverses phosphorylation of GRASP65 (Tang et al. 2008), but whether the same enzyme is responsible for GRASP55 dephosphorylation remains to be tested. Because GRASP65 and GRASP55 are localized on different parts of the Golgi (Shorter et al. 1999), these proteins may play essential roles in generation of the polarized, stacked structure (Xiang and Wang 2010).

CONCLUDING REMARKS

Despite recent advances, the mechanisms that regulate Golgi biogenesis remain largely mysterious. In particular, the interplay between the Golgi membranes and the mitotic machinery in the partitioning process must be further investigated at the mechanistic level. There are also only hints as to why the Golgi undergoes such extensive disassembly and reassembly during mammalian cell division and how this process regulates cell cycle progression. In addition, identification of the ubiquitin ligase and the substrates on the Golgi membranes will advance our understanding of p97/p47-mediated postmitotic Golgi membrane fusion.

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