Vacuolar H\textsuperscript{+}-ATPases (V-ATPases) are highly conserved ATP-driven proton pumps responsible for acidification of intracellular compartments. V-ATPase proton transport energizes secondary transport systems and is essential for lysosomal/vacuolar and endosomal functions. These dynamic molecular motors are composed of multiple subunits regulated in part by reversible disassembly, which reversibly inactivates them. Reversible disassembly is intertwined with glycolysis, the RAS/cyclic AMP (cAMP)/protein kinase A (PKA) pathway, and phosphoinositides, but the mechanisms involved are elusive. The atomic- and pseudo-atomic-resolution structures of the V-ATPases are shedding light on the molecular dynamics that regulate V-ATPase assembly. Although all eukaryotic V-ATPases may be built with an inherent capacity to reversibly disassemble, not all do so. V-ATPase subunit isoforms and their interactions with membrane lipids and a V-ATPase-exclusive chaperone influence V-ATPase assembly. This minireview reports on the mechanisms governing reversible disassembly in the yeast Saccharomyces cerevisiae, keeping in perspective our present understanding of the V-ATPase architecture and its alignment with the cellular processes and signals involved.
have been shown to modulate V-ATPase assembly by a process that requires PIP 3-kinase and mTOR activation (24–27).

This review reports on the mechanisms of reversible disassembly in yeast, particularly in regard to our present understanding of the V-ATPase architecture. Next, we summarize recent structural discoveries on the yeast V-ATPase, their interrelation with V-ATPase regulation by reversible disassembly, and our current understanding of the mechanisms and signals involved.

ARCHITECTURE OF EUKARYOTIC V-ATPase

ATPase rotary motors include F-ATP synthase, archaeal A-type ATP synthase, bacterial A/V-like ATPase, and eukaryotic V-ATPase (28). V-ATPase and other members in this family share common structural features essential for the mechanical rotation of protein subunits during ATP catalysis. They all have (i) a protuberant globular domain peripherally attached to the membrane that houses three catalytic sites, (ii) a membrane domain that forms the path for ion transport, (iii) a centrally located rotor that couples ATP hydrolysis and ion transport across membranes, and (iv) one or more peripheral stalks that connect the peripheral and membrane domains.

Rotation of rotor-forming subunits relative to the steady catalytic sites is driven by hydrolysis of ATP inside the globular structure of V1 (A3B3) (Fig. 1). ATP hydrolysis promotes rotation of the rotor’s shaft (subunits D, F, and d) at the center of the A3B3 hexamer. The shaft is connected to a hydrophobic proteolipid ring inside the membrane (c-ring), which consists of subunits c, c’, and c’’ and transfers the protons. Active transport requires entrance of cytosolic protons to the Vo subunit a in order to reach the c-ring. The protons bind to an acidic residue in the c-ring, and after a 360° rotation, protons exit the other side of the membrane, traveling through the Vo subunit a. This general mechanism of rotational catalysis is shared with all rotary ATPases (28).
Eukaryotic V-ATPases distinguish themselves from other rotary ATPases in three ways. First, V-ATPases are dedicated proton pumps. Second, V-ATPases are regulated by reversible disassembly. Third, V-ATPases contain three peripheral stalks. In contrast, the A and bacterial A/V-ATPases have two peripheral stalks and F-ATPases have one (28). The V-ATPase peripheral stalks are made of a heterodimer of E and G subunits; reversible disassembly requires the third peripheral stalk (EG3) (Fig. 3) (6, 29). It also requires a soluble subunit that is absent in other rotary ATPases (subunit C). The yeast subunit C contains two globular domains, the head (Chead) and foot (Cfoot) (30). The Chead domain interacts with EG3 with high affinity (6, 31). Through its Cfoot domain, subunit C interacts with the second peripheral stalk (EG2) and the N terminus of the Vo subunit a (a-NT). These subunit interactions are broken and reformed when V-ATPases disassemble and reassemble.

Subunit C is released to the cytosol during disassembly (8). Reassembly requires the subunit C to be rapidly reincorporated into the complex and its interactions with EG3, EG2, and a-NT to be restored. Reintroduction of subunit C into V₁Vo requires significant bending of the third peripheral stalk (6, 29). This compression imposes physical stress in its coiled-coil structure, like “spring-loading.” The EG3 tension, which persists within assembled V₁Vo complexes, is released when V₁Vo disassembles. Thus, it is proposed that spring-loading requires energy for reassembly and primes V-ATPases to easily disassemble after glucose depletion, when ATP must be preserved.

These new structural discoveries hopefully will lead to a better understanding of V-ATPase regulation and function.

**FIG 2** Reversible disassembly of V-ATPase: extracellular stimuli and intracellular signals. V-ATPase disassembly breaks the complex apart, as V₁, V₀, and the V₁ subunit C separate. Disassembly is reversible, and reassembly of the three components restores ATP hydrolysis and proton transport. Yeast cells adjust the number of assembled V-ATPases in response to environmental stressors, including changes in glucose, pH, and salts. These extracellular cues are communicated to V-ATPases by several signals and unknown mechanisms that require an assembly factor (RAVE) and are intertwined with glycolysis and glycolytic enzymes, RAS/cAMP/PKA components, cytosolic pH (pHcyt) homeostasis, and PI(3,5)P₂.
understanding of how glucose and other cellular signals regulate V-ATPase function and assembly. The spring-loading mechanism of reversible disassembly is compatible with our current understanding of the structural architecture of the eukaryotic V-ATPase complex. It is also consistent with our knowledge of the major cellular processes associated with V1, V0 disassembly and reassembly. Below, we discuss our view of the alignment of the V-ATPase architecture with these cellular processes and signals.

CONNECTING GLUCOSE METABOLISM TO V-ATPase ASSEMBLY

The concept of spring-loading requires energy to bend EG3 and reassociate subunit C with the peripheral stalks EG3 and EG2 and the N terminus domain of the V0 subunit a (a-NT). Restoration of the native complex probably requires bending of EG3, like spring-loading, which is accomplished with the assistance of the chaperone complex RAVE. The tension contained in EG3 within the assembled V1V0 complex, is then released when V1V0 disassembles. The tension contained in EG3 within the assembled V1V0 complex, is then released when V1V0 disassembles. The spring-loading mechanism of reversible disassembly is compatible with our current understanding of the structural architecture of the eukaryotic V-ATPase complex. It is also consistent with our knowledge of the major cellular processes associated with V1, V0 disassembly and reassembly. Below, we discuss our view of the alignment of the V-ATPase architecture with these cellular processes and signals.

FIG 3 Spring-loading: a model for disassembly and reassembly. The V1 domain and subunit C detach from V0 at the membrane and are released into the cytosol during disassembly. Reassembly requires reassociation of subunit C with the peripheral stalks EG3 and EG2 and the N terminus domain of the V0 subunit a (a-NT). Restoration of the native complex probably requires bending of EG3, like spring-loading, which is accomplished with the assistance of the chaperone complex RAVE. The tension contained in EG3 within the assembled V1V0 complex, is then released when V1V0 disassembles.

The regulator of ATPase of vacuoles and endosomes (RAVE) complex is a V-ATPase-exclusive assembly factor. It is required for V1V0 assembly at steady state (biosynthetic assembly) and reassembly in response to glucose readdition to glucose-deprived cells. The RAVE complex chaperones loading of subunit C into V1V0, a job that requires aligning Chead with the EG3 and EG2 peripheral stalks in addition to introducing structural stress in EG3. In the absence of RAVE, V-ATPases at the vacuolar membrane are unstable and inactive, with V1 and subunit C loosely associated. Importantly, although several assembly factors are required for V-ATPase assembly, only RAVE appears to be involved in V-ATPase reversible disassembly. The RAVE complex has three components, the adaptor protein.
Skp1p and its two subunits, Rav1p and Rav2p (46). Skp1p associates with other cellular complexes. Rav1p and Rav2p are solely found in the RAVE complex. Of the two subunits, Rav1p constitutes the central component; it binds Rav2p and Skp1p (47). Rav1p also forms the interface between RAVE and V-ATPase subunits. In the cytosol, Rav1p binds V-ATPase subunit C and the V1 peripheral stalk-forming subunits EG (48). At the membrane, Rav1p interacts with the N-terminal domain of Vph1, subunit a (17). Genetic and biochemical data have shown that binding of Rav1p to subunit C can occur independently of its binding to V1. Preloading RAVE with subunit C and V1 simultaneously in the cytosol may expedite reassembly, which is known to be a fast response completed within 3 to 5 min of glucose readdition (19, 55). Importantly, formation of RAVE-C and RAVE-V1 subcomplexes in the cytosol is not glucose dependent, indicating that RAVE binding is not the signal for V1Vo reassembly.

Deletion of the genes RAV1 and RAV2 leads to growth defects characteristic of V-ATPase mutants (46, 47); the vacuolar membrane ATPase (vma) growth phenotype displays growth sensitivity at pH 7.5 and in the presence of calcium (1). The raviΔ and rav2Δ mutant cells also exhibit temperature sensitivity, but the vma traits are detected at 37°C. This phenotype is more substantial in raviΔ than rav2Δ cells (46, 47), likely because Rav1p constitutes the functional subunit of the RAVE complex. The raviΔ mutant has major V-ATPase assembly and functional defects in vivo, although its vma growth phenotype is fairly mild and considered “partial.”

The raviΔ mutant resembles the yeast mutant strain vph1Δ, which lacks the isoform Vph1p of the V0 subunit a (56). The Vo subunit a is the only yeast V-ATPase subunit encoded by two functional homologs, VP1 and STV1 (56, 57); VP1 encodes the vacuolar isoform and STV1 has sorting information for the Golgi/endosomal compartments (58). Genome-level synthetic genetic analyses (17) showed that a synthetic vma growth phenotype can be generated after combining the raviΔ mutation with class E mutants of endosomal and vacuolar transport (59), suggesting that the physiological basis for the raviΔ partial vma phenotype is that RAVE is a Vph1p-specific chaperone. The discovery that RAVE assists in the assembly of Vph1p-containing V-ATPases but that Stv1p-containing complexes do not need RAVE to properly assemble is in agreement with prior studies showing that Vph1p-containing V-ATPases are more responsive to glucose than are Stv1p-containing pumps (60). Since Vph1p targets V-ATPase to the vacuole and Stv1p to the Golgi and endosomal compartments, these results also suggest that only vacuole-associated pumps reversibly disassemble.

The functions of the RAVE complex are likely conserved in other eukaryotes. The Rav1p sequence homologs, rabcoctins, are necessary for acidification of endosomes and synaptic vesicles (61–63). The human V0 subunit a exists in four different isoforms; mutations in particular isoforms cause osteopetrosis and renal tubular acidosis (64). Identifying the V0 subunit a isoform(s) recognized by rabcoctins could help in understanding these and other pathologies associated with V-ATPases. The RAVE subunit Rav2p is found only in fungi and does not have human homologs. Therefore, Rav2p offers unique opportunities to selectively disrupt RAVE complex functions in fungal human pathogens for which V-ATPase pumps are desirable targets (65–72).

IS ATP HYDROLYSIS NECESSARY FOR V-ATPase DISASSEMBLY AND REASSEMBLY?

How energy can be used to reassemble V1Vo is virtually unknown. ATP facilitates in vitro reconstitution of V1 and V0, (73–75), suggesting that ATP could promote reassembly in vivo. Genetic screens aimed at identifying V-ATPase mutants defective in V1Vo reassembly are challenging because V1Vo disassembly is not absolute. There is a cellular fraction of V-ATPase complexes that does not disassemble in response to glucose depletion; it probably yields basal V-ATPase activity necessary to support critical cellular functions (1, 10). Those pumps constitute about 30% of the total V1Vo and likely include V-ATPases at nonvacuolar membranes (Stv1p-containing V-ATPase pumps) (19, 60). Coincidentally, 25 to 30% of V-ATPase activity is sufficient to support wild-type growth, which makes the growth phenotype of these types of mutants very subtle. A few mutants, primarily disassembly mutants, have been identified by site-directed mutagenesis experiments. Intriguingly, many of those mutations also alter V-ATPase catalysis. Those studies suggest that peripheral stalks may regulate rotational catalysis by influencing ATP binding, chemical reaction, or release of ADP/Pi (76–78).

As expected, site-directed mutations at conserved amino acids of the peripheral stalk subunits E and G can suppress glucose-dependent disassembly (Fig. 1) (76, 79). The mutations vma4-D44A and vma10-R25A/L in subunits E and G, respectively, suppress disassembly; they also stimulate the enzyme activity (76, 79). The vma4-T202A mutation near the C terminus enhances Vmax, by about 2-fold without significantly affecting the Kms of the enzyme (77), resembling the mutant vma4-D44A (76). Although the ability of vma4-T202A to reversibly disassemble has not been determined, these studies indicate that the peripheral stalks can communicate with the catalytic sites inside the A2B3 hexamer and they can affect disassembly and catalysis.

Paradoxically, the rate of ATP hydrolysis by V-ATPase can alter V1Vo disassembly (Fig. 1). Pharmacologic inhibition of V-ATPase pumps with the V-ATPase inhibitor concanamycin A reduces V1Vo disassembly, without affecting reassembly (19). Inactive V-ATPases carrying mutations at a V0 proton-binding subunit (vma11-E145L) of the c-ring (19) or the V1 catalytic subunit (vma1–R219K) (78) are defective for disassembly. Likewise, vma1-P177V and vma1–R219A, which are partially inactive (by 30% to 50%), are also defective in disassembly. Thus, it appears that wild-type levels of activity are necessary to disassemble V1Vo; hyperactive, hypoactive, and inactive pumps cannot sufficiently disassemble (19, 45, 76). Obviously, not all disassembly mutants are catalytically impaired. The mutations vma1-G150 and vma1-D157E inhibit disassembly without affecting V-ATPase activity (78), perhaps by stabilizing subunit interactions.

From these studies, it becomes clear how little we still know about the intramolecular mechanisms that drive disassembly and reassembly. Although additional studies will be necessary to determine how intrinsic subunit interactions and differential conformations impact disassembly and reassembly, it appears that V-ATPases adopt conformations prone for disassembly during a catalytic cycle of rotation driven by ATP hydrolysis (19). Still, we cannot exclude the possibility that catalysis may also drive reassembly. In this context, ATP-driven subunit rotation in V1 may stimulate its own reassembly in the presence of RAVE. This process will require the inhibitory subunit of V1 (subunit H) to be activated.
released from its rotor-locking inhibitory position during reassembly (Fig. 3) (7, 11, 12, 80).

YEAST V-ATPase: AT THE CROSSROADS OF MULTIPLE INTRACELLULAR SIGNALS

In support of the spring-loading hypothesis, there are no known chaperones that aid in V-ATPase disassembly; V-ATPase may be primed to disassemble (29). In addition to glucose depletion, exposure to less preferred carbon sources (galactose, glycerol/ethanol, and raffinose) causes disassembly (8, 19). These data further argue that little or no glucose is the driving signal of disassembly.

There is evidence indicating that the Ras/cAMP/PKA pathway probably mediates reversible disassembly (18, 20). Ras/cAMP/PKA signaling controls metabolism in response to sudden availability of rapidly fermentable sugars (81), compatible with a role for Ras/cAMP/PKA during V₁Vₒ reassembly (Fig. 2). Constitutively active Ras and PKA suppress disassembly by glucose depletion (18). These studies suggest that the Ras/cAMP/PKA pathway acts upstream of V-ATPase. In an independent study linking V-ATPase reassembly to cAMP and PKA, reassembly appeared to be an upstream activator of PKA (20). That study suggested that alkalization of the cytosol after glucose readdition is the signal for reassembly. Although these results seem contradictory, the possibility that a positive feedback mechanism may regulate V-ATPase assembly cannot be excluded (Fig. 4). The reassembled V-ATPase may activate PKA signaling, which in turn enhances the V-ATPase assembly.

Cytosolic pH is emerging as a key regulator for various cellular functions (82), and V-ATPase affects cytosolic pH homeostasis (83). In addition to activating V-ATPase catalysis and proton transport, readdition of glucose activates the plasma membrane ATPase, Pma1p, which is the main efflux pump responsible for yeast cytosolic pH regulation (83, 84). V-ATPases are necessary for cytosolic pH homeostasis because (i) active V-ATPases are necessary for normal Pma1p levels to be present at the plasma membrane, and (ii) cytosolic pH homeostasis is maintained by the coordinated action of V-ATPase and Pma1p (83). In the evaluation of the signals for reassembly, the contribution of cytosolic pH merits additional investigation. Addressing whether glucose-dependent Pma1p activation precedes glucose-dependent V-ATPase activation may help clarify the role of cytosolic pH for reassembly.

Fungi grow more rapidly at acidic than neutral pH (85). It should come as no surprise that V₁Vₒ disassembly in response to glucose depletion is affected by environmental stress signals, such as elevated pH (86). At pH 7.0, V-ATPase disassembly is significantly suppressed compared to disassembly at pH 5.0, the optimal pH for yeast growth. Although the mechanisms involved in the prevention of disassembly by pH remain elusive, adaptation to high pH appears to have both PI(3,5)P₂-dependent and -independent components (16). Knowing whether glucose and pH use common mechanisms to regulate V₁Vₒ disassembly requires additional studies. Notably, it may help in the understanding of fungal pathogenicity; Candida albicans adaptation to neutral-to-alkaline pH environments in the host stimulates cellular signals that trigger its morphological change from the yeast form (nonpathogenic) to the hyphal form (pathogenic) (65, 68, 72, 87).

V-ATPase function is necessary for adaptation to stress condi-
tions. Vacuoles are yeast storage compartments and an important mechanism of protection against toxic metals and drugs (88). By modulating V-ATPase disassembly, yeast protects the vacuolar luminal pH and maintains secondary transport systems across the membrane. Exposure of yeast to osmotic shock increases the total pool of vacuolar V$_i$, V$_o$ assembled (89). This involves a mechanism that requires the signaling lipid PI(3,5)P$_2$, interacting with the V$_o$ subunit a isoform Vph1p (16). Interestingly, PI(3,5)P$_2$ has little or no effect on glucose-dependent reversible disassembly of the V-ATPase, indicating that the cellular signals behind hyperosmotic stress- and glucose-induced V-ATPase reassembly are independent. High salts and high pH can increase V$_i$, V$_o$ assembly levels at steady state in the presence of glucose, when cellular energy is abundant and most V-ATPase complexes are assembled. How this may work is not clear. It suggests that vacuolar membranes may contain subpopulations of V-ATPases specialized to respond to different signals, adding a layer of complexity to this intricate regulatory event.

CONCLUDING REMARKS

Structural data are beginning to support a collection of studies investigating how glucose signals are communicated to V-ATPases. The new concept is that V$_i$, V$_o$ may be structurally built with an inherent facility to disassemble but that its reassembly imposes energetic constrains. This concept has reinforced our view of disassembly and reassembly as two independently controlled events. A variety of extracellular cues that control V-ATPase assembly and/or disassembly are emerging, although glucose is the main and strongest external stimulus. We do not know what is the glucose sensor or the mechanism involved in this communication. Our understanding of V-ATPase regulation by reversible disassembly is incomplete. The spring-loading hypothesis has not been experimentally tested. If all V-ATPases are structurally suited to reversibly disassemble, why do not all of them do so? Vph1p-containing V-ATPases disassemble and reassemble, but not Stv1p-containing V-ATPases. There are many questions that remain unanswered regarding the roles of glycolysis, RAS/cAMP/ PKA, and V$_i$, catalysis. Some of these questions include the following: (i) do glycolytic enzymes and/or glycolysis control V-ATPase at steady state and during glucose depletion/readition; (ii) are glycolysis and RAS/cAMP/PKA elements of a common pathway or different pathways that work in parallel to control V-ATPase assembly and function; (iii) is V-ATPase upstream of PKA or downstream; (iv) what is the yeast V-ATPase subunit that is phosphorylated, if any; and (v) what phosphatase enzyme is involved.

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