The discovery of molecules required for membrane fusion has revealed a remarkably conserved mechanism that centers upon the formation of a complex of SNARE proteins. However, whether the SNARE proteins or other components catalyze the final steps of membrane fusion in vivo remains unclear. Understanding this last step depends on the identification of molecules that act late in the fusion process. Here we demonstrate that in Saccharomyces cerevisiae, Vac8p, a myristoylated and palmitoylated armadillo repeat protein, is required for homotypic vacuole fusion. Vac8p is palmitoylated during the fusion reaction, and the ability of Vac8p to be palmitoylated appears to be necessary for its function in fusion. Both in vivo and in vitro analyses show that Vac8p functions after both Rab-dependent vacuole docking and the formation of trans-SNARE pairs. We propose that Vac8p may bind the fusion machinery through its armadillo repeats and that palmitoylation brings this machinery to a specialized lipid domain that facilitates bilayer mixing.

Homotypic organelle fusion is essential for the maintenance of organelle structure, copy number, and function. In Saccharomyces cerevisiae, lysosome (vacuole) homotypic fusion is involved in three distinct aspects of vacuole biogenesis. First, during cell division the mother cell vacuoles form vesicular/tubular segregation structures that migrate into the daughter cells and fuse to establish a new vacuole (1–5). Second, during zygote formation the segregation structures from each mother cell fuse, generating the bud vacuole and exchanging vacuole material (2). Third, yeast vacuoles undergo regulated rapid fission and fusion events. This allows yeast cells to rapidly change the volume of the vacuole in response to changes in osmolarity of their environment.

Development of an in vitro vacuole fusion assay (6–8) has revealed that most of the molecules known to function in homotypic vacuole fusion are the same or direct homologues of those required for heterotypic fusion (9). Vacuole fusion requires NSF (Sec18p), α-SNAP (α-soluble NSF attachment protein) (Sec17p) (7), the trafficking factor LMA1 (10, 11), a Rab GTPase (Ypt7p) (12), a large protein complex containing the Class B vps proteins Yam2/Vps41p and Vam6/Vps39p (13, 14) and Class C vps proteins (15), t-SNAREs (Vam3p and Vam7p), v-SNAREs (Nyw1p) (16, 17) (Vti1p and Ykt6p) (18) (see Fukuda et al. for a discussion of the assignment of the SNARE types (19)), phosphatidylinositol 4,5-bisphosphate (20), calmodulin (21), and protein phosphatase I (22). Extensive kinetic analysis performed by Wickner and co-workers (9) reveal three distinct events in the vacuole fusion pathway: priming, docking, and fusion. Several molecules required for priming and docking have been identified. However, details of the molecular basis of fusion are controversial. Several lines of evidence suggest that formation of the v-SNARE-t-SNARE complex catalyzes membrane fusion (23–25). However, equally compelling functional studies suggest that the formation of trans-SNARE pairs is required for docking of membranes but does not catalyze fusion (26–28). Moreover, some molecules required for fusion have been shown to function after SNARE pair formation (21, 22). Thus, to distinguish between these models, we need to know more about the identity and function of molecules that act after membrane docking.

Vac8p is an armadillo (ARM)-repeat-containing protein (29–31). ARM repeat proteins contain tandem arrays of multiple imperfect repeats. These repeats often have multiple binding partners, and a single ARM protein often has multiple functions (32). Yeast Vac8p contains 11 ARM repeats and, although not a functional homologue, it is closely related by sequence to plakoglobin and β-catenin. Like plakoglobin and β-catenin, Vac8p has multiple binding partners and interacts with its known partners through its ARM repeats (33, 34). Vac8p is localized on the vacuole membrane and is enriched at vacuole-vacuole contact sites. Vac8p is both myristoylated and palmitoylated.

It had been shown that palmitoyl-CoA, by transfer to a target protein(s), stimulates vacuole fusion (7). Palmitoylation may be a universal requirement for fusion since palmitoyl-CoA also stimulates Golgi vesicle fusion with the Golgi (35). However, the role of palmitoyl-CoA had not been well defined. Here, we demonstrate that Vac8p plays a direct role in vacuole fusion both in vivo and in vitro. Vac8p is not necessary for priming and docking but acts after most of the previously identified fusion components and is likely required for fusion per se. Moreover, we show that Vac8p is the target for palmitoyl-CoA and is palmitoylated during the fusion reaction.

**EXPERIMENTAL PROCEDURES**

**Yeast Strains and Reagents**—The yeast strains utilized were BJ3505 (MAT a Δ pep4::HIS3 pdr1Δ16R HIS3 lys2–208 trplΔ101 ura3–52 gln2-3,112 aux2–801 his3–Δ200 trp1–Δ901 lys2–801 suc2–29), BY4741 (MATa Δ phosph::TRP1 leu2–3,-112 ura3–52 his3Δ101 trplΔ901 lys2–801 suc2–29), and K91-1A (MATα ura3 pho8::pAL134 pho13::pH13 lys1–8) (8); LWY4789 (BJ3505 Δ vac8::::TRP1)

triethylenemiammoniumpropyl)-4-(4-diethylamino)phenyl) hexatrienylypyridinium dibromide; NSF, N-ethylmaleimide sensitive factor; SNARE, soluble N-ethylmaleimide factor attachment protein receptor; t-, target membrane; v-vesicle; PIPES, 1,4-piperazinediethanesulfonic acid.
and LWTY4502 (DK6281 Δvac8::HIS3) (this study); LWTY7213 (VACS) and LWTY2887 (Δvac8::HIS3) (29); LWTY5929 (BJS505 Δpsa1::TRP1), LWTY5930 (DK6281 Δvac3::LEU2), LWTY6196 (BJS505 Δvac1::TRP1 Δvac8::HIS3), and LWTY6197 (DK6281 Δvac3::LEU2 Δvac8::HIS3) (this study). DNA sequencing was performed as described. Cells were grown in synthetic complete medium supplemented with 0.2% caffeine. The mixture was incubated with aeration at 24°C for 1 h. The cells were harvested in a microcentrifuge at 10,000 rpm for 1 min. The genomic DNA was isolated and purified as described (38), but the calcium chelator, BAPTA inhibitor (10 mM), was substituted for Microcystin LR. The use of BAPTA provided the ability to wash out the inhibitor and measure subsequent fusion. For visualization of docking, aliquots of the reaction were labeled with 80 μM FM4-64 in fusion buffer containing 1% (w/v) Triton X-100, and Vac8p was immunoprecipitated as described (29).

RESULTS

Vac8p Is Required for Vacuole Fusion—Preliminary evidence that Vac8p is required for homotypic vacuole fusion came from observations that the vacuole is extremely fragmented in vac8 mutants (29–31) (for examples, see Figs. 2 and 6). This fragmentation does not result from defects in vacuole inheritance per se because other vac mutants display normal vacuole morphology (36). A requirement for Vac8p in homotypic vacuole fusion was confirmed by the demonstration that Δvac8 vacuoles cannot fuse in vitro or in vivo. The in vitro assay measures the extent of content mixing of two sets of vacuoles. Vacuoles with proalkaline phosphatase that lack the activating proteinases A and B are mixed with vacuoles that contain these proteinases but that lack proalkaline phosphatase (8, 37). The amount of the resulting activated alkaline phosphatase reflects the extent of vacuole fusion.

Wild-type levels of vacuole fusion require Vac8p on both partner vacuoles (Fig. 1a). This requirement for fusion-related molecules on both vacuole types has been observed in several instances (12, 38). The lowered levels of alkaline phosphatase activation with vacuoles from the Δvac8 mutant result from defective vacuole fusion, as levels of both the activating proteases and proalkaline phosphatase are normal (Fig. 1b).

The Δvac8 fusion defect is not due to the absence of a previously described molecule(s) that is directly required for fusion. As shown in Fig. 1c, Sec18p, Sec17p (7), Ypt1p (12), Vam3p, Nvy1p (16), and calmodulin (21) are all present on Δvac8 vacuoles at the same levels found on the wild-type organelle.

Vac8p Functions Directly in Fusion—To further test for a direct role for Vac8p in fusion, we identified a temperature-sensitive vac8 mutant, vac8–15ts, that is defective in vacuole fusion at elevated temperatures. This vac8 mutant has normal vacuole morphology at 24°C but has fragmented vacuoles after a short incubation period at 37°C. Isolated vacuoles from the vac8–15ts mutant fuse with near wild-type efficiency at 24°C, but at 33°C, fusion occurs at about 50% that is seen with wild-type (Fig. 1d) (at temperatures above 33°C, vacuole fusion in vitro is inhibited in both the wild-type and mutant vacuoles).

To test the immediate outcome of loss of Vac8p function, we developed an in vivo assay to look at the fusion of vacuoles over time. It had been observed that transferring Schizosaccharomyces pombe to hypotonic media induces vacuolar fusion (39). Thus we tested the effect of hypotonic media on S. cerevisiae. We found that a majority of the vacuoles from both the wild-type and the vac8–15ts strain fused within 45 s at 24°C (84 and 79%, respectively), whereas few of the vacuoles in the vac8Δ strain fused (10%). After a 5-min shift to 37°C, a majority of the wild-type vacuoles still fused (80%), but few Δvac8 and vac8–15ts vacuoles fused (11 and 14%, respectively (Fig. 2 and Table 1). We attribute the greater block in fusion seen in vac8–15ts in the in vivo assay to be due to the ability to perform this assay at 37°C rather than 33°C. That a defect in fusion of vac8–15ts vacuoles can be detected after just 5 min at the non-permissive temperature strongly suggests that the role of Vac8p in fusion is direct.
Several lines of evidence suggest that Vac8p is a target for the palmitoyl-CoA stimulation of vacuole fusion. First, in contrast to wild-type, the addition of palmitoyl-CoA to Δvac8 vacuoles does not stimulate fusion (Fig. 3a). Second, the mutant vac8Δ3 (C4G, C5T, C7S), which cannot be palmitoylated (29), is defective in fusion in vitro (Fig. 3b), and most importantly, there is no stimulation of fusion by palmitoyl-CoA (Fig. 3b). Moreover, in parallel with these in vitro defects, vac8Δ3 is defective in vacuole fusion in vivo (Fig. 2 and Table 1). Although it is possible that the specific residue changes in vac8Δ3 abolish Vac8p function independently of its ability to be palmitoylated, this is unlikely. vac8Δ3 functions normally in the cytoplasm to vacuole targeting pathway, demonstrating that this mutant fully retains some of the functions of Vac8p (29). Third, when [3H]palmitoyl-CoA is added during fusion of wild-type vacuoles, significant levels of [3H]palmitate are incorporated into Vac8p (Fig. 3c). Vac8p is ~0.1–0.5% of the total vacuolar protein (not shown). In the reaction utilized, there were 0.12 pmol of [3H]palmitate incorporated (as measured by counts associated with immunoprecipitated Vac8p) into 0.48–2.4 total pmol of Vac8p (based on total vacuole protein per reaction). Therefore, 1 in every 4–20 Vac8p molecules is modified by exogenous palmitate during fusion. Furthermore, modification of Vac8p might be concentrated to regions of vacuole-vacuole contact, where a much higher fraction of Vac8p molecules may be palmitoylated. Additional vacuolar proteins also are palmitoylated (Fig. 3c). Vac8p is the band at 64 kDa (absent in Δvac8 vacuoles). The four other palmitoylated polypeptides migrate with apparent molecular masses of ~46, 40, and 30 kDa; their identities remain to be determined.

Vac8p Functions after Vacuole Docking and Trans-SNARE Pair Formation—To determine the step where Vac8p functions in vacuole fusion, we examined each partial reaction. First, we examined priming. During priming, ATP hydrolysis by Sec18p releases Sec17p from the vacuolar membrane and disassembles cis-SNARE pairs (41, 42). Priming was first measured by examining the ability of exogenous Sec18p to disrupt cis-SNARE pairs. The extent of SNARE pair disruption was the same for wild-type and Δvac8 vacuoles (Fig. 4a). We also examined priming in the presence of endogenous Sec18p. In this case a time course of Sec17p release from the SNARE complex was assayed. Vacuoles were incubated under fusion conditions and transferred to ice at the times indicated. The amount of Sec17p associated with the SNARE complex was measured by Western blot analysis after immunoprecipitation with anti-Ypt7p antibody. Both wild-type and Δvac8 vacuoles show the same kinetics of Sec17p release (Fig. 4b), again indicating that Vac8p is not required for vacuole priming.

We next examined docking. First, we used a staged assay where vacuoles are arrested at docking with BAPTA, a calcium chelator. This assay is based on studies showing that the addition of BAPTA prevents vacuole fusion and that BAPTA inhibits a post-docking step (21, 43). Importantly, the BAPTA inhibition is reversible. To visualize docking, we used a previously developed morphological assay of isolated vacuoles (38). Both wild-type and Δvac8 vacuoles form extensive clusters in vivo (Fig. 5, b and c). To show that the Δvac8 vacuoles are specifically docked and not simply aggregated, ATP was omitted from the incubation; this prevents priming and subsequent docking (Fig. 5, a and d). Moreover, vacuole docking does not occur when Δypt7 vacuoles are utilized (Fig. 5h). The lack of docking of Δypt7 vacuoles is consistent with previous studies showing that anti-Ypt7p antibody blocks vacuole docking (38). The BAPTA was subsequently removed, and vacuoles were incubated under fusion conditions for 90 min. Fusion occurred for docked, wild-type vacuoles but not for

The rapidity of vacuole fusion in response to osmotic stress, shown by the in vitro assay, suggests that specific regulatory mechanisms control fusion. This helps explain recent observations that, like regulated secretory events at the synapse, homotypic vacuole fusion is regulated by Ca2+ (21). Thus, this assay should be useful both for testing molecules involved in fusion per se and also in testing those molecules that play a regulatory role.

Palmitoylation of Vac8p Is Required for Fusion Activity—Fusion of Golgi transport vesicles with their acceptor compartment is stimulated by palmitoyl-CoA (35), as is vacuole-vacuole fusion (7) (Fig. 3a). This stimulation was proposed to be due to palmitoylation of a target protein(s). Palmitoylation is generally reversible and can serve a regulatory role (40).
docked Δvac8 vacuoles (Fig. 5, c and f). Thus, Δvac8 vacuoles dock as well as wild-type vacuoles but are defective at a subsequent step in fusion.

A further test of the ability of Δvac8 vacuoles to dock can be seen in Fig. 4a (0 time), where it can be seen that the t-SNARE, Vam3p, complexes normally with the v-SNARE, Nyv1p. SNARE pairing was measured by assaying the degree of Nyv1p co-immunoprecipitation with Vam3p. The majority of SNARE pairs assayed are cis pairs, meaning that the v- and t-SNAREs are on the same vacuole (27). The extent of pairing was the same for wild-type and Δvac8 vacuoles.

The above experiments demonstrate that SNARE pairs both form and can be disrupted in Δvac8. However, because wild-type vacuoles contain the same v- and t-SNAREs, it remained to be shown whether the v-SNAREs from a single vacuole will complex with t-SNAREs on another vacuole, forming a trans-SNARE pair. As described below, we find that trans-SNARE pairing is normal as well. Nichols et al. (16) previously demonstrated that one could assay solely for trans-SNARE formation by measuring the Nyv1p-Vam3p complex formed when vacuoles dock.

**Table I**

| Strain        | % Vacuoles fused | % Vacuoles not fused |
|---------------|------------------|----------------------|
| 24 °C         |                  |                      |
| Wild-type     | 93               | 16                   |
| Δvac8 n = 100 | 10               | 90                   |
| vac8–15° n = 116 | 79        | 21                   |
| vac8–3 n = 93 | 16               | 84                   |
| 37 °C         |                  |                      |
| Wild-type     | 117              | 20                   |
| Δvac8 n = 100 | 11               | 89                   |
| vac8–15° n = 100 | 14      | 86                   |
| vac8–3 n = 100 | 7                | 93                   |
Fusion of Docked Membranes Requires Vac8p

Differences in the in vivo phenotypes of ypt7 and Δvam3 compared with Δvac8 further support a model whereby Ypt7p
Our discovery that Vac8p is required for homotypic vacuole fusion addresses three questions. 1) Is the act of SNARE pair formation sufficient for membrane fusion? 2) Is membrane fusion coordinated with other membrane-related processes? 3) Is a specialized lipid domain required to promote bilayer mixing, and is this domain brought together with proteins that comprise the fusion machinery?

Two major lines of evidence support the prevailing view that formation of the v-SNARE/t-SNARE complex catalyzes membrane fusion. Liposomes containing a purified v-SNARE fuse with liposomes containing purified t-SNAREs (23, 25). Moreover, structural analysis of the SNARE pair complex suggests that SNARE pairing may be the fundamental event in membrane fusion. The complex forms four parallel helices with structural similarities between the SNARE complex and viral fusion proteins (for discussion see, Skehel and Wiley (24)).

Although SNARE pairing as a catalyst of fusion is an attractive model, several studies indicate that SNARE pairing is not sufficient for fusion. First, functional studies with both yeast vacuoles and sea urchin cortical vesicles demonstrate that when the SNARE complex is disrupted, fusion still proceeds (26, 27). Furthermore, in synaptosomes, N-ethylmaleimide treatment causes an increase in SNARE pair formation, yet it does not result in a concomitant increase in fusion (28). In addition, staged assays have revealed molecules that function after SNARE pairing, protein phosphatase I (22), and Ca\(^{2+}\)/calmodulin (21).

To build an accurate model of how fusion occurs, the key players in this event need to be identified and characterized. Our studies present a new type of molecule required for fusion. Results from both in vivo and in vitro studies demonstrate that Vac8p is required for fusion and strongly suggest that Vac8p functions downstream of SNARE pairing. Most compelling is our finding that trans-SNARE pair formation appears to be normal in \(\Delta vac8\) vacuoles. The SNARE pairs from \(\Delta vac8\) vacuoles formed within 30 min and to the same extent as that seen with wild-type vacuoles. Although \(\Delta vac8\) vacuoles are not competent for fusion, they are competent for the earlier steps of priming and docking.

Analysis of both the localization and the sequence of Vac8p suggests a model of how Vac8p may function in fusion. Vac8p accumulates at vacuole-vacuole junctions in vivo (30, 31), a site consistent with a direct role in fusion. Vac8p has two types of binding motifs. One motif is composed of 11 ARM repeats of Vac8p. These repeats are found in a diverse set of proteins, have no known catalytic function, and are generally sites of protein-protein interaction. The other motif is composed of acyl chains at the Vac8p amino terminus, and as discussed below, this acylation may bring Vac8p to a specific lipid domain. Thus, an attractive model for Vac8p function is that through its ARM repeats, Vac8p binds the protein portion of the fusion machinery, and when Vac8p is acylated, it brings the protein complex to a specialized lipid domain.

In addition to its role in homotypic vacuole fusion presented here, Vac8p is required in at least two other distinct membrane processes, where it interacts with a unique protein partner specific for that process. Vac8p complexes with Apg13p to facilitate the closure of vesicles in the cytoplasm-to-vacuole-targeting pathway (34). Vac8p complexes with Nvj1p to form specific junctions between the nucleus and vacuole (33). The interactions of Vac8p with these proteins are mediated through the Vac8p ARM repeats. Interestingly, the functional role of each of the Vac8p partners above is restricted to a specific process, and none are required for homotypic vacuole fusion. Thus, it is likely that there is a Vac8p binding partner dedicated to homotypic vacuole fusion. Discovery of this partner...
should provide further insight into the role that Vac8p plays in fusion.

That Vac8p is required for multiple vacuole membrane-related events yet acts through several partners suggests that there is a mechanism to prevent these diverse processes from occurring simultaneously. Perhaps each occurs at a similar region of the vacuolar membrane, creating the need to coordinate these events with each other.

Are There Functional Homologues of Vac8p?

Most of the proteins that have previously been described to play a role in homotypic vacuole fusion have been shown to have direct homologues in other organisms and, moreover, to have direct homologues that function in fusion of other membranes. Although Vac8p homologues are likely to be present in metazoans, none has yet been identified. The difficulty in searching for a Vac8p homologue by sequence alone is that there are at least 25 ARM repeat proteins in *Caenorhabditis elegans*, 30 in *Drosophila melanogaster*, and 93 in *Arabidopsis thaliana*. Moreover, there is not just one consensus sequence for palmitoylation. Thus, it is likely that a metazoan Vac8p homologue will need to be identified by functional analysis.

Although Vac8p homologues may exist in other organisms, it appears that Vac8p functions in some but not all yeast membrane fusion pathways. The role of Vac8p in nuclear vacuole junction formation and in the cytoplasm-to-vacuole targeting pathway may be a specific function in fusion; however, other yeast membrane fusion events do not appear to require Vac8p. Some membrane fusion events are essential for yeast viability, yet VAC8 is not an essential gene. However, the fact that palmitoyl-CoA stimulates fusion of mammalian Golgi vesicles with the Golgi and also stimulates homotypic vacuole fusion strongly suggests that palmitoyl-CoA provides a general, fundamental role in membrane fusion.

What Is the Role of Palmitoyl-CoA in Fusion?

The lipid binding motif of Vac8p is composed of Gly2, which is myristoylated, and Cys4, Cys5, and Cys7, which are palmitoylated. Interestingly, the sites of myristoylation and palmitoylation on Vac8p are very similar to those found in the plasma membrane Src-family kinases (29). Moreover, these proteins share a conserved Ser at position 6 and a conserved Lys at position 9. These motifs are not common, and it is tempting to speculate that they serve a similar function in these diverse proteins. In the case of the Src-family kinases, palmitoylation drives them into cholesterol-rich lipid rafts (50). By analogy, palmitoylation of Vac8p may bring it (and the fusion machinery that is attached to the ARM repeats of Vac8p) to an ergosterol-rich membrane domain (ergosterol is the sterol in yeast that functions similarly to cholesterol).

Properties of cholesterol are consistent with it playing a major role in facilitating fusion. Cholesterol is intercalated in the cytoplasmic side of the phospholipid bilayer and stabilizes the region near the polar hydroxyl group. However, the region of the acyl chains that are most distal from the phospholipid head group are destabilized by the presence of cholesterol, precisely the situation that is expected to accompany bilayer mixing. In addition, cholesterol has been shown to have an important role in viral fusion (for examples, see Kielian and Helenius (51) and Chatterjee et al. (52)). Moreover, SNARE proteins concentrate at sites of exocytosis in cholesterol-rich domains (53). In addition, it has been proposed that the Vo subunit of the vacuolar ATPase plays a key role late in fusion (49). Interestingly, the Vo subunit of the v-ATPase in synaptic-
like vesicles has been shown to be a major protein that cross-links to cholesterol (54).

The observation that palmitoylation of Vac8p is required for fusion strongly suggests that this region of Vac8p attaches to a defined membrane patch that promotes bilayer mixing. Determination of the composition of this patch is likely to yield further insight into the final steps in fusion.

Acknowledgments—Special thanks to Drs. William Wickner, Albert Haas, Andreas Mayer, Christian Ungermann, Zuoyu Xu, and Rob Piper for providing numerous strains, plasmids, and antibodies and for many helpful discussions concerning the vacuole-vacuole docking and fusion assays. We thank Dr. Scott Emr for providing TDI2 (SEY6210 Δavl3::LEU2). We thank Drs. Rob Piper, Peter Rubenstein, Mark Stammes, and Robert Cohen for helpful discussions of the manuscript.

REFERENCES

1. Bachmair, A., Finley, D., and Varshavsky, A. (1986) Science 234, 179–186
2. Weisman, L. S., and Wickner, W. (1988) Science 241, 589–591
3. Weisman, L. S., Emr, S. D., and Wickner, W. T. (1990) Proc. Natl. Acad. Sci. U. S. A. 87, 1076–1080
4. Gomes de Mesquita, D. S., ten Hoopen, R., and Woldringh, C. L. (1991) J. Gen. Microbiol. 137, 2447–2454
5. Raymond, C. K., Roberts, C. J., Moore, K. E., Howald, I., and Stevens, T. H. (1997) Biochim. Biophys. Acta 1330, 221–228
6. Nichols, B. J., Ungermann, C., Pelham, H. R., Wickner, W. T., and Haas, A. (1998) J. Cell Biol. 141, 1499–1509
7. Pan, X., and Goldfarb, D. S. (1998) J. Cell Sci. 111, 2137–2147
8. Haas, A., Conradt, B., and Wickner, W. (1994) EMBO J. 13, 3296–3305
9. Haas, A., Conradt, B., and Wickner, W. (1994) J. Cell Biol. 126, 87–97
10. Wickner, W., and Haas, A. (2000) Annu. Rev. Biochem. 69, 247–275
11. Xu, Z., and Wickner, W. (1996) J. Cell Biol. 132, 787–794
12. Xu, Z., Mayer, A., Muller, E., and Wickner, W. (1997) J. Cell Biol. 136, 299–306
13. Haas, A., Schegelmuller, D., Lazar, T., Gallwitz, D., and Wickner, W. (1995) EMBO J. 14, 5258–5270
14. Price, A., Wickner, W., and Ungermann, C. (2000) J. Cell Biol. 148, 1223–1230
15. Sato, T. K., Rehling, P., Peterson, M. R., and Emr, S. D. (2000) Mol. Cell 6, 661–671
16. Nichols, B. J., Ungermann, C., Pelham, H. R., Wickner, W. T., and Haas, A. (1997) Nature 387, 199–202
17. Ungermann, C., and Wickner, W. (1998) EMBO J. 17, 3269–3276
18. Ungermann, C., van Mollard, G. F., Jensen, O. N., Margolis, N., Stevens, T. H., and Wickner, W. (1999) J. Cell Biol. 145, 1435–1442
19. Fukuda, R., McNew, J. A., Weber, T., Parlati, F., Engel, T., Nickel, W., Rothman, J. E., and Sollner, T. H. (2000) Nature 407, 198–202
20. Mayer, A., Scheglmann, D., Dove, S., Glatz, A., Wickner, W., and Haas, A. (2000) Mol. Biol. Cell 11, 807–817
21. Peters, C., and Mayer, A. (1998) Nature 396, 575–580
22. Peters, C., Andrews, P. D., Stark, M. J., Cesario-Tadic, S., Glatz, A., Podtelejnikov, A., Mann, M., and Mayer, A. (1999) Science 285, 1084–1087
23. Weber, T., Zemelman, B. V., McNew, J. A., Westermarck, B., Gmachl, M., Parlati, F., Sollner, T. H., and Rothman, J. E. (1998) Cell 92, 759–772
Fusion of Docked Membranes Requires the Armadillo Repeat Protein Vac8p
Yong-Xu Wang, Emily J. Kauffman, Jason E. Duex and Lois S. Weisman

J. Biol. Chem. 2001, 276:35133-35140.
doi: 10.1074/jbc.M103937200 originally published online July 5, 2001

Access the most updated version of this article at doi: 10.1074/jbc.M103937200

Alerts:
• When this article is cited
• When a correction for this article is posted

Click here to choose from all of JBC's e-mail alerts

This article cites 57 references, 35 of which can be accessed free at
http://www.jbc.org/content/276/37/35133.full.html#ref-list-1