Rvs161p and Rvs167p, the Two Yeast Amphiphysin Homologs, Function Together in Vivo

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Mutations in RVS161 and RVS167, the two yeast amphiphysin homologs, cause very similar growth phenotypes, a depolarized actin cytoskeleton, and a defect in the internalization step of endocytosis. Rvs161p and Rvs167p have been shown to interact in the two-hybrid system, but their localization in the cell may be different thus raising the question whether the interaction is physiologically relevant. Here we demonstrate that the two proteins function together in vivo. We find that the steady state level of Rvs167p is strongly reduced in an rvs161Δ strain. Similarly, the level of Rvs161p is strongly reduced in an rvs167Δ strain. We demonstrate that these reduced protein levels at steady state are due to a decreased stability of either Rvs protein in the absence of the other protein. Furthermore, we find that the amount and ratio of Rvs161p and Rvs167p are critical parameters for receptor-mediated endocytosis. In addition, by using the two-hybrid system we show that the interaction of Rvs167p with actin is not abolished in an abp1Δ strain suggesting that Abp1p is not essential for this interaction.

The yeast Rvs161p and Rvs167p, together with mammalian amphiphysin I and II, nematode, and fission yeast isoforms, constitute a family of conserved proteins (1). The N termini of the different proteins share the highest homology, and this common domain was called the BAR domain (BIN/Amphiphysin/RVS domain (2)). Rvs161p consists only of the BAR domain (Fig. 1), whereas the other members of the family have an SH3 domain at their C termini and a central domain varying among the different proteins. In the case of Rvs167p, the central domain is rich in glycine, proline, and alanine and therefore is called the GPA domain (Fig. 1).

The mammalian homolog, amphiphysin I, was first identified as a brain protein enriched at presynaptic regions (3). The identification of dynamin, synaptotagmin, the α-s-subunit of AP2-adaptin, and clathrin as amphiphysin I-interacting proteins further implicated amphiphysin I in endocytosis (1). A more broadly expressed isoform, amphiphysin II, has been identified by several groups and was found to interact with the same proteins that interact with amphiphysin I (1). Interestingly, the two isoforms can be coimmunoprecipitated from brain extracts suggesting that they act in concert (4). Indeed, two studies (4, 31) found that the two amphiphysin isoforms colocalize in brain. However, a third study identified a different subcellular localization of the two isoforms (5). Amphiphysin I was shown to be concentrated in the cortical cytoplasm of nerve terminals, whereas amphiphysin II was concentrated in axon initial segments and nodes of Ranvier (5).

The two yeast members of the amphiphysin family, encoded by RVS161 and RVS167, were first identified in a screen for mutations causing reduced viability upon nutrient starvation (6, 7). RVS161 was also identified in a screen for endocytosis mutants (8). Mutations in rvs161 or rvs167 exhibit the same phenotypes except for a defect in cell fusion only found in the rvs161 mutant (9). The mutant phenotypes include defects in endocytosis, cell polarization, bud site selection in diploid cells, and a depolarized actin cytoskeleton (7, 8, 10). Rvs161p and Rvs167p have been shown to interact through the BAR domain in the two-hybrid system (11). However, their localization in the cell seems to be different, raising the question whether the Rvs161p-Rvs167p interaction is relevant in vivo. Rvs161p was shown to be mainly cytosolic in unbudded cells, and upon bud emergence it localizes mainly to the mother-bud neck region (9). In contrast, in unbudded cells Rvs167p is localized mainly in small cortical patches throughout the cell which polarize at the bud emergence site and in small buds (12). By using the two-hybrid system the BAR domain of Rvs167 has also been shown to mediate homodimerization in one study (13), and another study failed to detect any Rvs167p-Rvs167p interaction (11).

Mutations in rvs161 and rvs167 affect the actin cytoskeleton (7, 10). Interestingly, the SH3 domain of Rvs167p has been shown to interact with actin in the two-hybrid system (14). The finding that Rvs167p interacts with the actin-binding protein Abp1p through its GPA/SH3 domains (15) led to the hypothesis that Abp1p could mediate the interaction between Rvs167p and actin.

In this study we investigated whether Rvs161p and Rvs167p indeed function together in vivo. We find that the steady state level of Rvs161p is strongly reduced in an rvs167Δ strain. Similarly, the level of Rvs167p is strongly reduced in an rvs161Δ strain. We demonstrate that these reduced protein levels at steady state are caused by a decreased stability of either Rvs protein in a strain mutated in the other rvs gene. Furthermore, we provide evidence that the amount and ratio of Rvs161p and Rvs167p are critical parameters for receptor-mediated endocytosis. In addition, by using the two-hybrid system we find that Abp1p is not required to mediate the interaction of Rvs167p with actin.

MATERIALS AND METHODS

Yeast Strains, Media, and General Techniques—Yeast strains used in this study were EGY48 (Mat a his3 trp1 ura3-52 leu2-3,112 lys2-801 ura3-52 leu2::lexAop6-LEU2), RH448 (Mat a his4 leu2 lys2 ura3 bar1), RH376 (Mat a his3 leu2 trp1 ura3 bar1), RH2600 (Mat a his4 ura3 rvs161Δ bar1), RH2950 (Mat a his4 leu2 trp1::URA3 ura3 rvs167Δ TRP1 bar1), RH5238 (Mat a his3 trp1 ura3 leu2::lexAop6-LEU2 abp1::KanMX6), and RH5239 (Mat a his3 trp1 ura3 leu2::lexAop6-LEU2 rvs161Δ::KanMX6). Strains that did
Plasmid Constructions—pEG202 and pG4-5 were described elsewhere (18); both plasmids contain a 2-μm origin of replication. The reporter gene plasmid pSH18-34 was described elsewhere (19), and it contains the LexA operators upstream of the reporter gene GALI-IacZ. To construct pEGRVS161 the RVS161-ORF was amplified by the polymerase chain reaction with 5′-primer poly(pur) and 3′-primer pEGRVS161, introducing an EcoRI site upstream and a BamHI site downstream of the RVS161-ORF. The fragment was then inserted as an EcoRI-Xhol fragment into pEG202. To construct pMgACTI the ACTI-ORF was amplified by the polymerase chain reaction using the 5′-primer GATACCGGATCC and 3′-primer ACTGAGATTCCGCTAGCTCTTTAAG) and EcoRI domain. The fragment was then inserted as an EcoRI-Xhol fragment into pG4-5 which was cut with Xhol, the 5′-overhang filled in with Klenow, and cut with EcoRI. To construct pEGRVS167 the RVS167-ORF was amplified by the polymerase chain reaction using the 5′-primer RVS167-1, GATACTCGGATCC and 3′-primer RVS167-2, GATACCGGATCC and introducing an EcoRI site upstream and a Xhol site downstream of the RVS167-ORF. The fragment was then inserted as an Xhol-RVS167 fragment into pEG202. pGRVS167 was described elsewhere (20). To construct pMGBKAR, the BAR domain of Rvs167 was amplified by the polymerase chain reaction using the 5′-primer RVS167-1, GATACTCGGATCC and 3′-primer RVS167-2, GATACCGGATCC and introducing an EcoRI site upstream and a Xhol site downstream of the RVS167-ORF. The fragment was then inserted as an Xhol-BAR fragment into pG4-5. To construct pMGBKAR/GPA the BAR and GPA domains of RVS167 were amplified by the polymerase chain reaction using the 5′-primer RVS167-1, GATACTCGGATCC and 3′-primer RVS167-2, GATACCGGATCC and EcoRI domain. The fragment was then inserted as an EcoRI-Xhol fragment into pG4-5. To construct pMGBKAR/GPA the BAR and GPA domains of RVS167 were amplified by the polymerase chain reaction using the 5′-primer RVS167-1, GATACTCGGATCC and 3′-primer RVS167-2, GATACCGGATCC and introducing an EcoRI site upstream and an Xhol site downstream of the BAR/GPA domains. The fragment was then inserted as an EcoRI-Xhol fragment into the SphI site downstream of the BAR/GPA domains. The reporter gene plasmid pSH18-34 with the reporter gene GALI-IacZ was amplified by the polymerase chain reaction using the 5′-primer TACGC-9 and 3′-primer GATACGGAATTC. The fragment was then inserted as an Xhol-BAR fragment into pJG4-5. To construct pJGBAR/GPA the BAR and GPA domains of RVS167 were amplified by the polymerase chain reaction using the 5′-primer RVS167-1, GATACTCGGATCC and 3′-primer RVS167-2, GATACCGGATCC and introducing an EcoRI site upstream and a Xhol site downstream of the RVS167-ORF. The fragment was then inserted as an EcoRI-Xhol fragment into pJG4-5. To construct pJGBAR the BAR domain of Rvs167 was amplified by the polymerase chain reaction using the 5′-primer RVS167-1, GATACTCGGATCC and 3′-primer RVS167-2, GATACCGGATCC and introducing an EcoRI site upstream and an Xhol site downstream of the RVS167-ORF. The fragment was then inserted as an Xhol-BAR fragment into pJG4-5. To construct pJGBKAR/GPA the BAR and GPA domains of RVS167 were amplified by the polymerase chain reaction using the 5′-primer RVS167-1, GATACTCGGATCC and 3′-primer RVS167-2, GATACCGGATCC and introducing an EcoRI site upstream and an Xhol site downstream of the RVS167-ORF. The fragment was then inserted as an EcoRI-Xhol fragment into pJG4-5. To construct pJGBKAR the BAR domain of Rvs167 was amplified by the polymerase chain reaction using the 5′-primer RVS167-1, GATACTCGGATCC and 3′-primer RVS167-2, GATACCGGATCC and introducing an EcoRI site upstream and an Xhol site downstream of the RVS167-ORF. The fragment was then inserted as an EcoRI-Xhol fragment into pJG4-5. To construct pJGBKAR/GPA the BAR and GPA domains of RVS167 were amplified by the polymerase chain reaction using the 5′-primer RVS167-1, GATACTCGGATCC and 3′-primer RVS167-2, GATACCGGATCC and introducing an EcoRI site upstream and an Xhol site downstream of the RVS167-ORF. The fragment was then inserted as an EcoRI-Xhol fragment into pJG4-5.
itself is indirect and mediated via Rvs161p. To test this we performed a two-hybrid analysis in an rvs161Δ strain, and we still detected a strong Rvs167p-Rvs167p interaction (Fig. 2C), suggesting that the interaction is direct or mediated by a protein other than Rvs161p. To confirm the report that Rvs161p and Rvs167p form a complex in vivo (11), we showed that the two proteins could be coimmunoprecipitated under native conditions with antibodies against Rvs161p and Rvs167p (Fig. 2D).

Reduced Stability of Rvs Proteins in the Absence of Its Partner—Interestingly, the localization of Rvs161p (mostly cytoplasmic, at mother-bud neck region in small budded cells) and Rvs167p (small cortical patches that polarize upon bud emergence) seems to be different (9, 12). These findings raise the question whether the interaction of Rvs161p with Rvs167p is required in vivo. We found that when compared with a WT strain at steady state, the level of Rvs167p is strongly reduced in an rvs161Δ strain (Fig. 3A). Similarly, the level of Rvs161p is strongly reduced in an rvs167Δ strain (Fig. 3A). As control we detected Sla2p, a protein that is also required for endocytosis and actin organization (24). Its levels were constant in the three strains (Fig. 3A). A decreased synthesis or an increased instability of the proteins in the mutant strains could cause these reduced protein levels at steady state. To address this question we performed pulse-chase experiments in the different strains. As shown in Fig. 3B, both Rvs161p in an rvs167Δ strain and Rvs167p in an rvs161Δ strain are unstable when compared with a WT strain. Taken together these data suggest an in vivo function for the Rvs161p-Rvs167p interaction in stabilizing both proteins.

Amount and Ratio of Rvs161p and Rvs167p Are Critical Parameters for Endocytosis—Mutations in rvs161 or rvs167 have both been shown to affect the internalization step of receptor-mediated endocytosis (8). To learn more about the involvement of the proteins in endocytosis, we decided to overexpress them and measure how they affect α-factor internalization (Fig. 4A). Interestingly, overexpression of Rvs167p in an rvs161Δ strain exacerbated the endocytic defect of this strain (Fig. 4B). Similarly, overexpression of Rvs161p in an rvs167Δ strain exacerbated the endocytic defect of this strain (Fig. 4C). The protein levels of Rvs161p and Rvs167p upon overexpression in a WT or a mutated strain are similar (data not shown). Interestingly, co-overexpression of both Rvs161p and Rvs167p in a WT strain reduces α-factor internalization to about two-thirds of WT rate (Fig. 4D). Taken together these data suggest that not only the amounts of Rvs161p and Rvs167p but also their ratio are critical parameters for endocytosis. We have tested the same strains overexpressing the Rvs proteins for an actin and a growth phenotype. As shown in Fig. 5, A–H, overexpression of the Rvs proteins either individually or together in a WT strain produces a clear synergistic effect on the actin and cell growth phenotype.
does not affect the actin cytoskeleton profoundly. The cells have a normal polarized actin cytoskeleton with cables running along the mother-bud axis. Also the growth rates of the different strains are not greatly affected (Fig. 5I). As shown in Fig. 6, A–H, overexpression of Rvs167p in an rvs161Δ strain or overexpression of Rvs161p in an rvs167Δ strain does not further deteriorate or ameliorate the actin defect exhibited by the mutants. We detect a mild growth phenotype upon overexpression of Rvs167p in the rvs161Δ strain when compared with the strain with the empty vector (Fig. 6I). Overexpression of Rvs161p in an rvs167Δ strain has no obvious effect on the growth rate (Fig. 6I).

Interaction of Rvs167p with Actin Does Not Require Abp1p—Mutations in rvs167 affect the actin cytoskeleton (7), and Rvs167p has been shown to interact with actin via its SH3 domain (14). The actin-binding protein Abp1p has been proposed to mediate this interaction of Rvs167p and actin since it interacts with the GPA/SH3 domains of Rvs167p (15). As shown in Fig. 7, the interaction of Rvs167p with actin in the two-hybrid system is not abolished in an abp1Δ strain showing that Abp1p is not required to mediate the interaction of Rvs167p with actin. Interestingly, we also detect an interaction of Rvs161p with actin in the two-hybrid system. However, this interaction is abolished in an rvs167Δ strain suggesting that the interaction of Rvs161p with actin is mediated via Rvs167p (data not shown).

DISCUSSION

In this study we provide direct evidence that Rvs161p and Rvs167p function together in vivo. We find that the steady state levels of the two proteins are interdependent. This effect is caused by a dramatically decreased stability of either Rvs protein in the absence of its partner. These data provide evidence that the interaction of Rvs161p and Rvs167p is physiologically relevant and required for the stability of both proteins. Furthermore, these findings might explain the almost identical phenotypes that are seen upon mutation of the two genes individually. The mutant phenotypes detected in either mutant strain might be a combination of loss of both Rvs proteins. Nevertheless, the function of Rvs161p in cell fusion (9) does not seem to require Rvs protein-protein interaction. Apparently, the highly reduced levels of Rvs161p in the rvs167Δ mutant are
sufficient for this function. As seen in Fig. 3B, Rvs161p is more sensitive to Rvs167p levels than vice versa. Since Rvs167p has two additional domains when compared with Rvs161p, this difference might reflect a partial stabilization of Rvs167p in the absence of Rvs161p via interactions mediated by these domains with other proteins (e.g. interaction with actin).

As mentioned in the Introduction, two previous studies have investigated the cellular localization of Rvs161p (mostly cytoplasmic, at mother-bud neck region in small budded cells (9)) and Rvs167p (small cortical patches that polarize upon bud emergence (12)). The two proteins apparently do not colocalize in the cell; however, in this study we have provided evidence that these two proteins do function together in vivo. This discrepancy might be explained if one considers that Rvs161p localization is mainly cytosolic. Therefore, it might also be localized to the Rvs167p-containing patches but is not concentrated there. In the case of Rvs167p, the bright fluorescence caused by its high concentration in cortical patches might make it difficult to detect an additional weak diffuse cytosolic staining. Therefore, it is possible that a certain amount of Rvs167p is localized to the cytosol and interacts with Rvs161p.

Interestingly, overexpression of either Rvs161p or Rvs167p alone in a WT strain has no major effect on the internalization step of endocytosis. However, co-overexpression of both proteins reduced the α-factor internalization rate to two-thirds of WT levels. Also, overexpression of Rvs161p in an rvs167Δ
strain, as well as overexpression of Rvs167p in an rvs161Δ strain, exacerbated the endocytic defect found in the mutated strains. Since the amount of protein upon overexpression is similar in all the strains (WT, rvs161Δ, and rvs167Δ, data not shown), we conclude that both the amount and the ratio of Rvs161p and Rvs167p are critical parameters for the internalization step of endocytosis. We have tested the strains used for the endocytosis assays for an actin and a growth phenotype. None of the strains overexpressing the Rvs proteins exhibited an obvious change in their actin cytoskeleton when compared with the strains with the empty vectors. Also the growth rates were almost identical with the exception of a mild defect detected upon overexpression of Rvs167p in the rvs161Δ strain.

The endocytic defect we detect in some of the strains overexpressing the Rvs proteins might be explained in two ways. One possibility would be that overexpression of the Rvs proteins causes a very subtle actin defect not detectable by immunofluorescence but affecting endocytosis. Another possibility would

FIG. 6. Actin staining and growth curve. A–D, rvs161Δ strain (RH2600) transformed with either p195 (A and B) or p195RVS167 (C and D) were fixed, and filamentous actin was visualized using TRITC-phalloidin (A and C) or Nomarski optics (B and D). E–H, rvs167Δ strain (RH2950) transformed with either p181 (E and F) or p181RVS161 (G and H) were fixed, and filamentous actin was visualized using TRITC-phalloidin (E and G) or Nomarski optics (F and H). The same strains were grown overnight. The cultures were diluted to about 10⁶ cells/ml and incubated at 24 °C, and the cell densities were determined every 3 h.

FIG. 7. Two-hybrid analysis. The interaction of Rvs167p (pEGRVS167) with actin (pJGACT1) was tested in a WT (EGY48) and in an abp1Δ (RH5238) strain. The strains contain the reporter gene plasmid pSH18-34 with the lacZ gene under the control of eight LexA operators, a bait and a prey. They were streaked out on plates containing X-gal. Positive interactors turn dark whereas negative colonies remain white.
be that by overexpressing the Rvs proteins another protein required for endocytosis is titrated out and therefore endocytosis is affected.

By using the two-hybrid system, we detected an interaction of Rvs161p with itself mediated via the BAR domain as previously described (13). Since the same domain also mediates the interaction with Rvs161p, we wanted to determine whether the Rvs167p-Rvs167p interaction is direct or mediated via Rvs161p. Two-hybrid analysis in an rvs161Δ strain demonstrated that Rvs161p is not required for this interaction and, therefore, Rvs167p either interacts directly with itself or the interaction is mediated via a protein other than Rvs161p. Interestingly, amphiphysin I has also been shown to interact with itself in mammalian cells (27).

Mutations in rvs161 and rvs167 have been shown to affect the actin cytoskeleton (7, 10). A previous study has shown that Rvs167p interacts with actin via its SH3 domain (14), and it was suggested that the actin-binding protein Abp1p could mediate this Rvs167p-actin interaction (15). Here we show that in the two-hybrid system Rvs167p interacts with actin in an abp1Δ strain showing that Abp1p is not required for this interaction. There are several possible explanations for this finding. First, it could be that in the absence of Abp1p another protein takes over its function. Second, there could be more than one protein involved in mediating this interaction. Third, Rvs167p could interact directly with actin. Another potential candidate to mediate the Rvs167p-actin interaction could be Las17p, since it was shown to interact with the GPA/SH3-domains of Rvs167p in the two-hybrid system (32).

rus mutant cells show pleiotropic phenotypes including sensitivity to nonoptimal growth conditions, defects in the organization of the actin cytoskeleton, defects in bud-site selection, and endocytosis (7, 8, 10). Interestingly, Rvs167p was identified as a target of the Pho85 cyclin-dependent kinase and thus might be involved in the regulation of the actin cytoskeleton in early in cell cycle (28). Also its mammalian homolog, amphiphysin, has been shown to be regulated by phosphorylation (27, 29). In addition, amphiphysin I has been implicated in the autoimmune Stiff-Man syndrome disorder associated with breast cancer, and amphiphysin II has been shown to interact with the MYC oncoprotein implicating it in cell cycle control (2, 14). Taken together these data suggest that some general functions and regulation of these proteins have been conserved throughout evolution. In addition, these findings implicate the members of this protein family not only as important players in intracellular membrane trafficking but also in mediating signals during the cell cycle.

In summary, we demonstrate for the first time that the yeast amphiphysin homologs Rvs161p and Rvs167p function together in vivo. The interaction between the proteins seems to be crucial for their stability. Furthermore, we show that the amount and ratio of Rvs161p and Rvs167p are critical parameters for the internalization step of endocytosis.

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