In mating mixtures of Saccharomyces cerevisiae, cells polarize their growth toward their conjugation partners along a pheromone gradient. This chemotrophic phenomenon is mediated by structural proteins such as Far1 and Bem1 and by signaling proteins such as Cdc24, Cdc42, and Gβγ. The Gβγ subunit is thought to provide a positional cue that recruits the polarity establishment proteins, and thereby induces polarization of the actin cytoskeleton. We identified RHO1 in a screen for allele-specific high-copy suppressors of Gβγ overexpression, suggesting that Rho1 binds Gβγ in vivo. Inactivation of Rho1 GTPase activity augmented the rescue phenotype, suggesting that it is the activated form of Rho1 that binds Gβγ. We also found, in a pull-down assay, that Rho1 associates with GST-Ste4 and that Rho1 is localized to the neck and tip of mating projections. Moreover, a mutation in STE4 that disrupts Gβγ/Rho1 interaction reduces the projection tip localization of Rho1 and compromises the integrity of pheromone-treated cells deficient in Rho1 activity. In addition to its roles as a positive regulator of 1,3-β-glucan synthase and of the cell integrity MAP kinase cascade, it was recently shown that Rho1 is necessary for the formation of mating projections. Together, these results suggest that Gβγ recruits Rho1 to the site of polarized growth during mating.

Signal-induced polarized growth is a fundamental mechanism of cellular differentiation and environmental response. The function of many mammalian cell types depends on their ability to sense relevant stimuli and grow in a directed fashion. An excellent model system with which to study such chemotropic phenomena is the mating response of the budding yeast, Saccharomyces cerevisiae. In preparation to mate, haploid yeast strain used in the cell lysis experiment was isolated by Saka et al. (43). All other yeast strains used in this study were derived from strain 15Dau (MATa bar1 ade1 his2 leu2-3,-112 trpl1 ura3-3), which is isogenic with strain BF264–15D (17). Both strains A35 and ELY115 contain the STE4A405V allele at the STE4 locus. Strain A35 is the original mutant STE4 isolate (18). It was back-crossed three times prior to use in this study. Strain ELY115 was created by replacing STE4 with STE4A405V in the wild type 15Dau background (18). Yeast transformants were performed by the lithium acetate method (19). Escherichia coli transformations were performed by electroporation (16). The plasmids used in this study are listed in Table I. Plasmids YCplac33/GAL1-STE4 and YCplac33/GAL1-STE4A405V were constructed as follows: STE4 was PCR-amplified from YCplac33/STE4 and YCplac33/STE4A405V (18) with added BamHI-EcoRI ends. The priming oligonucleotides were: 5'-CGGGATCCCTGTAGCTAATAGCAGCTAATGA-3' and 5'-CG-GAATTCTGAGGAGCAGCACGATAGTGC3' (boldface letters indicate the bases that comprise the added restriction sites). The products were subcloned into the pCRII vector (Invitrogen), and subsequently subcloned as BamHI fragments into YCplac33/GAL. PYES2.0/GAL1-RHO1 was created as follows: RHO1 was PCR-amplified using strain 15Dau genomic DNA as template, and the product was cloned into the pYES2.0 vector (Invitrogen) as a BamHI-EcoRI fragment. The priming oligonucleotides were: 5'-CGGGTACCCTGTAGCAGCAAAATCATAAGACG3' and 5'-GGGATCCAAAGCGTACCACTACATTAGA3'.

**Experimental Procedures**

Molecular and Microbiological Techniques—Standard methods were used for microfial and molecular manipulation (15, 16). The rho1ts yeast strain used in this study was isolated by Saka et al. (43). All other yeast strains used in this study were derived from strain 15Dau (MATa bar1 ade1 his2 leu2-3,-112 trpl1 ura3-3), which is isogenic with strain BF264–15D (17). Both strains A35 and ELY115 contain the STE4A405V allele at the STE4 locus. Strain A35 is the original mutant STE4 isolate (18). It was back-crossed three times prior to use in this study. Strain ELY115 was created by replacing STE4 with STE4A405V in the wild type 15Dau background (18). Yeast transformants were performed by the lithium acetate method (19). Escherichia coli transformations were performed by electroporation (16). The plasmids used in this study are listed in Table I. Plasmids YCplac33/GAL1-STE4 and YCplac33/GAL1-STE4A405V were constructed as follows:

- STE4 was PCR-amplified from YCplac33/STE4 and YCplac33/STE4A405V (18) with added BamHI-EcoRI ends.
- The priming oligonucleotides were: 5'-CGGGATCCCTGTAGCTAATAGCAGCTAATGA-3' and 5'-CG-GAATTCTGAGGAGCAGCACGATAGTGC3' (boldface letters indicate the bases that comprise the added restriction sites).
- The products were subcloned into the pCRII vector (Invitrogen), and subsequently subcloned as BamHI fragments into YCplac33/GAL.
- PYES2.0/GAL1-RHO1 was created as follows: RHO1 was PCR-amplified using strain 15Dau genomic DNA as template, and the product was cloned into the pYES2.0 vector (Invitrogen) as a BamHI-EcoRI fragment.
- The priming oligonucleotides were: 5'-CGGGTACCCTGTAGCAGCAAAATCATAAGACG3' and 5'-GGGATCCAAAGCGTACCACTACATTAGA3'.
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Fig. 1. The effect of Rho1 activation and overexpression on mating pathway signaling. A, colony formation assays. Strain 15Dau was cotransformed with either the GAL1-STE4 (closed bars) or GAL1-STE4+Q68H (open bar) plasmids, along with low-copy (CEV) or high-copy (2μ) RHO1 plasmid. Rho1° (Q68H) forms of RHO1. Transformants were grown to saturation in galactose medium, and the relative ability of each strain to form colonies was determined as described under “Experimental Procedures.” B, pheromone-induced transcription assay. Strain 15Dau was cotransformed with either the pYES2.0/Gal1-Rho1 or pYES2.0 plasmids along with the FUS1-lacZ reporter plasmid, pSB231. Transformants were grown to mid-log phase in galactose medium and treated with 15 nM α-factor. Aliquots were taken at the indicated time points, and β-galactosidase activity was determined as described under “Experimental Procedures.” The closed bars correspond to the pYES2.0 cells and the open bars to the pYES2.0/Gal1-Rho1 cells. C, epistasis analysis. 15Dau cells transformed with pSB231 and the following plasmids were grown to saturation on solid medium containing galactose: left, GAL1-STE4; middle, STE11-4 and GAL1-RHO1; right, GAL1-STE4 and GAL1-RHO1. β-Galactosidase activity was then assayed as described under “Experimental Procedures.”

pEB15.0 was created as follows: RHO1 was PCR-amplified from strain 15Dau genomic DNA, and the product was cloned into pESC-URA (Stratagene, La Jolla, CA) as a Km1-Xhol fragment, thereby placing RHO1 under GAL1 promoter control. The priming oligonucleotides were: 5′-GGCTGAAGATCTACATCACAACTTAAAGTGGTTA-3′ and 5′-GGGTTACCTATAAAGACAGACATTCT-3′. FAR1 was PCR-amplified from strain 15Dau genomic DNA, and the product was cloned into pESC-URA as a PvuII fragment, thereby placing FAR1 under GAL10 promoter control. The priming oligonucleotides were: 5′-CTTATTAAAGTGAGTAAGACAGTGAGG-3′ and 5′-GAAGATCT-TGAAGACACAAACAGTGGTTG-3′. pRS183.0 was created as follows: PKC1378 was PCR-amplified from strain 15Dau genomic DNA, and the product was cloned into pESC-URA as a KpnI fragment. The priming oligonucleotides were: 5′-GGGTTACCTATAAAGACAGACATTCT-3′ and 5′-GAAGATCT-TGAAGACACAAACAGTGGTTG-3′. The PCR product was ligated into pCRII (Invitrogen), and subsequently subcloned into YCplac22/GAL1 as a BamHI-HindIII fragment. The GAL1-RHO1 transcriptional fusion identified in the screen was isolated from a cDNA library kindly provided by Stephen J. Elledge. Plasmids pRS151/RHO1, pRS425/RHO1, pRS151/RHO1Q68H, and pRS425/RHO1Q68H were kindly provided by Alan M. Myers (20).

By Allele-specific High-copy Suppressor Screen—Strain ELY105 (a derivative of strain 15Dau containing an integrated copy of GAL1-STE4) was transformed first with YCP33/GAL1-STE4 and subsequently with the yeast pTRP1 cDNA library (kindly provided by Stephen J. Elledge). Transformants were spread on selective sucrose medium lacking uracil and tryptophan. Approximately 100,000 colonies were then replica-plated to selective dextrose or to selective galactose medium lacking uracil and tryptophan. Rescue of growth arrest was verified, and sterile mutants were discarded. The YCPlac33/GAL1-STE4 plasmid was cured using 5-fluoroorotic acid, and the strains were transformed with YCP333/GAL1-STE4+Q68H. The ability of the library clone to rescue overexpression of STE4+Q68H was then determined.Library clones that showed allele specificity were characterized further.

Colony Formation Assays—Colony formation assays were performed by spreading 750 cells on the appropriate selective medium containing a range of α-factor concentrations. Plates were incubated at 30 °C for 3–5 days. Resistance to α-factor was quantified by plating on selective medium containing 3, 6, 15, 30, and 60 nM α-factor as compared with the number of colonies that formed on medium lacking pheromone.

FUS1 Expression Assays—Expression of the pheromone-inducible FUS1 transcript was assayed by measuring β-galactosidase levels in cells containing a FUS1-lacZ reporter gene. Strain 15Dau was transformed with the FUS1-LacZ reporter vector (pSB251) (21) and either pYES2 or pYES2/GAL1-RHO1. Cultures were grown at 30 °C to an A600 of 0.5 in selective galactose medium. α-Factor was added to a final concentration of 15 nM, and the cultures were shaken at 30 °C. Cells were harvested, and β-galactosidase activity was determined as described previously (22). To assay β-galactosidase in cells grown on solid medium, nitrocellulose lift assays were performed as described (23).

Immunoblot—Yeast whole cell extracts were prepared by bead beating and clarified by centrifugation. 15 μg of total protein/well was electrophoresed on discontinuous SDS-polyacrylamide gels and electroblotted to polyvinylidene difluoride membranes (PVDF-PLUS, MSI Inc., Westborough, MA) according to the manufacturer’s protocol. Blots...
were then blocked with 5% nonfat dry-milk in TBS (20 mM Tris-HCl, pH 7.5, 500 mM NaCl) for 1 h and incubated with a 1:2000 dilution of a rabbit anti-GST antibody (Santa Cruz Biotechnology, Inc., Santa Cruz, CA) or a 1:500 dilution of the 9E10 mouse anti c-Myc antibody (Santa Cruz). Membranes were incubated at 4 °C overnight and then washed three times with TBS plus 0.2% Tween for 10 min. The washed membranes were probed with either horseradish peroxidase-conjugated goat anti-rabbit antibody (Promega, Madison, WI) or horseradish peroxidase-conjugated sheep anti-mouse antibody (Amersham Biosciences). Membranes were washed three times with TBS plus 2% Tween, and peroxidase activity was visualized using ECL (Amersham Biosciences) and Fuji RX film (Fuji Medical Systems, Stanford, CT).

**GST Pull-down**—Yeast strains EBY185, EBY186, and EBY187 were derived from strain 15Dau by transformation. They each carry the YCplac22/GAL1-His6-STE18 and pEB15.0 plasmids. In addition, EBY185 carries the pEB13.0 plasmid, EBY186 carries the pEB13.1 plasmid, and EBY187 carries the pEB13.2 plasmid. All three strains were grown in selective medium at 30 °C to an A600 of 0.4, at which point galactose was added to a final concentration of 3%. Cultures were then split and incubated for 2 h at 30 °C. α-Factor was added to a final concentration of 15 nM, and the cultures were incubated for an additional 4 h. Cells were harvested by centrifugation, washed once with cold water, and frozen in liquid nitrogen. Upon thawing, cell pellets were washed once with TBS and lysed with glass beads in radioimmune precipitation buffer (50 mM Tris, pH 7.5, 1% sodium deoxycholate, 1% Triton X-100, 1 mM Na3VO4, 150 mM NaCl, 1 mM Na2VO4, 50 mM NaF). Protease inhibitors were added just before lysis: 1 mM phenylmethylsulfonyl fluoride (Roche Applied Science), 1 g/ml pepstatin A, 1 g/ml leupeptin, and 5 g/ml aprotinin (Sigma). The crude lysates were cleared by centrifugation at 12,000 rpm for 15 min. Protein concentration was determined using the Bio-Rad protein assay kit. For each sample, 1 mg of total protein was transferred to a chilled microcentrifuge tube containing 20 μl of glutathione-Sepharose 4B (Amersham Biosciences); the tubes were incubated for 30 min at room temperature and then for an additional 30 min at 4 °C. The beads were washed several times with phosphate-buffered saline supplemented with the protease inhibitor mixture described.
RESULTS

Rho1 Associates with Ste4—On the basis of genetic and structural evidence, we have inferred that an unknown protein binds and down-regulates the \( \beta \gamma \) subunit of the pheromone responsive G protein. \( \beta \) and \( \gamma \) are encoded by the \( \text{STE4} \) and \( \text{STE18} \) loci, respectively. A tight cluster of mutations in \( \text{STE4} \) (A405V, G409D, S410L, and W411L/S) disrupts interaction with the putative regulator (18). To identify the unknown element, we took advantage of the observation that \( \beta \gamma \) overexpression strongly induces the mating signal and thereby confers permanent cell cycle arrest (25–27). Plasmids were constructed that allow for the galactose-inducible expression of \( \beta \) (\( \text{GALI-STE4} \)) and \( \gamma \) (\( \text{GALI-STE18} \)). A high-copy \( \text{GAL1} \)-cDNA yeast library was then screened for plasmids that could rescue the overexpression of wild type \( \beta \gamma \). Plasmids recovered in this step were re-screened for the inability to rescue the overexpression of a mutant form of \( \beta \gamma \), encoded by \( \text{STE4A405V} \) and \( \text{STE18} \). We reasoned that genes identified in this allele-specific screen might encode proteins that interact with Ste4. Of the 250,000 transformants screened, the most frequently isolated plasmid contained \( \text{RHO1} \). Rho1 is a highly conserved and well studied monomeric G protein. The mammalian homologue of Rho1, RhoA, is involved in polarization of the actin cytoskeleton. It has also been implicated in transcription, adhesion, and transformation (28–30). Like mammalian Rho proteins, Rho1 is thought to play a role in polarization of the actin cytoskeleton (10, 31). However, it also stimulates 1,3-beta-glucan synthase (10, 32) and the cell integrity MAP kinase cascade (33, 34), both of which are necessary for growth of the yeast cell wall.

To confirm and quantitate the effect of Rho1 overexpression on \( \beta \gamma \)-induced lethality, we performed single-colony formation assays. Strain 15Dau (17) was co-transformed with either the wild type or mutant \( \beta \gamma \)-overexpressing plasmids and either \( \text{RHO1} \) low-copy, \( \text{RHO1} \) high-copy, or control plasmids. Transformants were plated on glucose and on galactose medium, respectively, to repress and induce expression of \( \beta \gamma \). Only about 0.5% of the cells overexpressing wild type \( \beta \gamma \) in the absence of excess Rho1 were able to form colonies (Fig. 1A). In contrast, about 5% of the cells transformed with the low-copy number \( \text{RHO1} \) plasmid and about 13% of the cells transformed with the high-copy number \( \text{RHO1} \) plasmid could overcome the excess \( \beta \gamma \) and form colonies. Interestingly, overexpression of a mutationally activated form of Rho1, \( \text{RHO1}^{Q68H} \), enhanced the rescue phenotype by about 3-fold. However, neither Rho1 nor Rho1\(^{Q68H} \) could significantly increase the plating efficiency of cells forced to overexpress the mutant form of \( \beta \gamma \). The simplest interpretation of these data is that excess Rho1 antagonizes \( \beta \gamma \)-induced cell cycle arrest by directly interacting with Ste4. A less likely possibility is that Rho1 rescues cells overexpressing \( \beta \gamma \) by promoting cell cycle progression rather than by relieving the inhibitory effects of the mating signal. To distinguish these hypotheses, we assessed the effect of Rho1 overexpression on mating specific transcription using a \( \text{FUS1-lacZ} \) reporter. The reporter was stimulated either by treating cells with pheromone or by expression of Ste11-4, a dominant mutant form of the Ste11 MEK kinase that constitutively activates the mating pathway (35). Excess wild type Rho1 reduced pheromone-induction of \( \text{FUS1-lacZ} \) by about 50% (Fig. 1B) but had no effect on the activity of Ste11-4 (Fig. 1C). Thus, Rho1 overexpression inhibits the effects of free \( \beta \gamma \) on both transcription and cell cycle progression, and it does so at or above the level of the MEK kinase.
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To further evaluate the possibility that Gβγ physically interacts with Rho1, we constructed tagged versions of Ste4, Ste18, and Rho1, and performed pull-down assays. The tagged forms of all three proteins proved to be functional (data not shown). As shown in Fig. 2, myc-Rho1 specifically associated with GST-Ste4 but failed to associate with GST-Ste4<sup>ΔA405V</sup> or GST alone. Interestingly, the Far1 protein, which links Gβγ with Bem1 and Cdc42, also failed to associate with GST-Ste4<sup>ΔA405V</sup> (data not shown).

### The Localization of Rho1 to the Tips of Mating Projections Depends on Ste4—
In dividing cells, Rho1 associates with cortical actin patches, concentrating at the site of bud emergence, the tip of growing buds, and the mother-bud neck region prior to cytokinesis (31, 36). This subcellular localization of Rho1 is the tip of growing buds, and the mother-bud neck region prior to cytokinesis (31, 36). This subcellular localization of Rho1 is revealed by the localization of Pkc1, which plays a role in recruiting Rho1 to the site of polarized growth in mating cells.

Because inactivating the GTPase function of Rho1 augments its ability to rescue Gβγ overexpression (Fig. 1A), we wondered whether Gβγ preferentially associates with the activated form of Rho1. To answer this question, we took advantage of the finding that an internal domain of Pkc1, residues 378–640, specifically binds to activated Rho1 in the two-hybrid assay (40). We fused this Rho1 interaction domain (RID) in-frame with the gene encoding green fluorescence protein (GFP). The resulting reporter, Pkc1<sup>RID-GFP</sup>, was then used to probe the localization of Rho1 during vegetative growth (Fig. 3A). From left to right, the strains are as follows: 1) wild type (YOC1943); 2) ELY112, an STE4<sup>ΔA405V</sup> derivative of strain 15dau (18); 3) EBY246, a derivative of strain YOC774 (43) of relevant genotype <i>rho1–4 ste4Δ</i> YCplac33/STE4; 4) EBY247, a derivative of strain YOC774 of relevant genotype <i>rho1–4 ste4–4</i> YCplac33/STE4<sup>ΔA405V</sup>. The results of this experiment were identical in three independent trials.

### Table I

**Plasmids used in this study**

| Plasmid | Markers/constructs | Source |
|---------|--------------------|--------|
| YCplac33/GAL1-STE4 | URA3 CEN GAL1-STE4 | This study |
| YCplac33/GAL1-STE4<sup>ΔA405V</sup> | URA3 CEN GAL1-STE4 | This study |
| YCplac33/GAL1-STE18 | URA3 CEN STE18 | (18) |
| YCplac33/GAL1-STE18<sup>ΔA405V</sup> | URA3 CEN STE18<sup>ΔA405V</sup> | (18) |
| pEB13.0 | URA3 GAL1-cMyc-RHO1; GAL10-FLAG-FAR1 | This study |
| pEB13.5 (PKC1<sup>ΔA405V</sup>-GFP) | URA3 CEN CUP1-GFP-PKC1<sup>ΔA405V</sup> | This study |
| pEB13.9 (PKC1<sup>ΔA405V</sup>-GFP) | URA3 CEN CUP1-GFP-PKC1<sup>ΔA405V</sup> | This study |
| pEB13.1 | URA3 CEN GAL1-RHO1 | This study |
| pPSIS5/RHO1 | URA3 CEN RHO1 | (8) |
| pPSIS5/RHO1<sup>Q68H</sup> | URA3 CEN RHO1<sup>Q68H</sup> | (8) |
| pPSIS42/RHO1 | URA3 CEN RHO1 | (8) |
| pPSIS42/RHO1<sup>Q68H</sup> | URA3 CEN RHO1<sup>Q68H</sup> | (8) |
| pSB231 | URA3 CEN CUP1-GFP | (21) |
| pSL1509 | URA3 CEN STE11-4 | (35) |
| pRS516-2XHA-RHO1 | URA3 CEN HA-RHO1 | (39) |
| pGAD424/PKC1 | URA3 CEN GAL1-RHO1 | (40) |
| pSIS16CG | URA3 CEN CUP1-GFP | (50) |

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**Fig. 5.** Effect of <i>rho1–4</i> and STE4<sup>ΔA405V</sup> on cell integrity. Cells were grown and assayed for lysis as described under “Experimental Procedures.” Vegetative cells are shown in the upper row. Pheromone-treated cells are shown in the lower row. From left to right, the strains are as follows: 1) wild type (YOC1943); 2) ELY112, an STE4<sup>ΔA405V</sup> derivative of strain 15dau (18); 3) EBY246, a derivative of strain YOC774 (43) of relevant genotype <i>rho1–4 ste4Δ</i> YCplac33/STE4; 4) EBY247, a derivative of strain YOC774 of relevant genotype <i>rho1–4 ste4–4</i> YCplac33/STE4<sup>ΔA405V</sup>. The results of this experiment were identical in three independent trials.
localization of Pkc1\textsuperscript{RHD}-GFP in wild type and \textit{STE4}^{A4OSV} cells responding to pheromone. As we found when assaying the localization of HA-Rho1, the localization of Pkc1\textsuperscript{RHD}-GFP to the tips of mating projections was significantly reduced in the mutant cells (Fig. 4B). This result suggests that it is the activated form of Rho1 that associates with G\textbeta\gamma at the shmoo tip. \textit{STE4}^{A4OSV} and \textit{rho1–4 Confer a Synthetic Defect in the Integrity of Pheromone-treated Cells—Rho1 is essential for projection formation (31), presumably because it is essential for cell wall synthesis at the shmoo tip. If recruitment of Rho1 to the growth site is necessary for this process, then how are \textit{STE4}^{A4OSV} cells able to shmoo? First, it is clear that \textit{STE4}^{A4OSV} cells are only partially defective in localizing Rho1 to the shmoo tip. Perhaps the mutant form of G\textbeta\gamma is not completely deficient in Rho1 recruitment, or perhaps Rho1 is attracted by additional factors at the growth site. Second, Sekiya-Kawasaki et al. (42) have recently found that only about 20% of the normalational factors at the growth site. Second, Sekiya-Kawasaki in Rho1 recruitment, or perhaps Rho1 is attracted by addi-
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Gβγ Recruits Rho1 to the Site of Polarized Growth during Mating in Budding Yeast
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