The S. cerevisiae genome encodes two genes that are members of the formin family, BNI1 and BNR1, which assemble linear actin cables in the bud and bud neck, respectively. Formins are a conserved family of proteins that promote the assembly of actin filaments, a function that is necessary for remodeling of the actin cytoskeleton during such processes as budding, mating, cytokinesis, endocytosis, and in higher cells, cell adhesion and migration. The hallmark of formin proteins is the presence of two highly conserved FHdomains: the FH1 domain, containing polyproline motifs that mediate binding to profilin, which in turn binds actin monomers; and the FH2 domain, which nucleates actin assembly. Bni1p and Bnr1p are classified in the diaphanous-related formingroup of metazoan formins, named for the founding Drosophila gene diaphanous. The FH2 domains of Bni1p and Bnr1p are distinct from those of the metazoan groups, containing a yeast-specific insert that is not found in other organisms. In addition to FH1 and FH2 domains, DRFs also contain a regulatory Rho-binding domainand a Dia-autoregulatory-domain. Null mutations in either BNI1 or BNR1 do not impair cell viability, but the double bni1 bnr1 mutant is inviable, indicating that formins play an essential role in S. cerevisiae. A model for formin-mediated actin assembly proposes the following sequence of events. Activated Rho protein binds to the formin RBD and releases the formin from a conformation in which it is autoinhibited, due to interaction between its amino and carboxy termini, to a conformation that exposes the FH1 and FH2 domains. The FH1 domain interacts with profilin-bound actin monomers, \"delivering\" them to the FH2 domain, which is dimeric in structure and thus may interact with two actin monomers to stabilize a dimeric actin form, prior to polymerization to form actin cables. The FH2 domain remains associated with the growing end of the filament to protect it from interaction with capping proteins.Consistent with the model, Bnr1p interacts with GTP-bound Rho4p and is thus a potential target. Bud14p has also been identified as a regulator of BNR1; Bud14p inhibits the Bnr1p FH2 domain, and displaces Bnr1p from growing ends. Bnr1p localizes exclusively to the bud neck, and localization depends on the bud neck-localized septins Cdc3p, Cdc10p, Cdc11p, Cdc12p, and Shs1p. At this locale, Bnr1p assembles actin cables that extend from the bud neck into the mother cell and therefore help stabilize the mother-bud axis. In vitro experiments have demonstrated that Bnr1p nucleating activity is 10-fold stronger than Bni1p activity, likely due to the fact that Bnr1p has a higher affinity for filament ends than Bni1p; in addition, Bnr1p binds to the sides of actin filaments and bundles them, whereas Bni1p does not. Haploid bnr1 null mutant cells exhibit a random budding pattern but grow well at 23, 30, and 37 degrees Celsius.