

Interplay of Gic1 or Gic2 proteins with translation termination factors Sup35 and Sup45 in the yeast

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In the *Saccharomyces cerevisiae* yeast, the SUP35 and SUP45 genes encode eRF3 and eRF1 proteins, respectively. Mutations in these genes lead not only to omnipotent nonsense suppression, but also to a number of other pleiotropic effects. This may indicate that the translation termination factors are involved in multiple cellular processes besides of their main function -- recognition of stop codons and termination of protein synthesis. Previously, we performed a screening for the genes that influence viability of the sup35 and sup45 nonsense-mutants; among such genes we identified the GIC1 and GIC2 paralogues. The corresponding proteins are involved in control of bud formation, cell size, and bud localization. Gic1 and Gic2 take part in regulation of actin cytoskeleton formation and control of cell cycle switch to G1. These proteins affect GTPase Cdc42, which has conservative CRIB (Cdc42/Rac-Interactive Binding) sequence for this interaction. In our previous work, interaction between Gic2 and Sup35 was shown using the yeast two-hybrid system, and a CRIB-like sequence was identified in the C-terminal part of Sup35. Additional copies of GIC1 and GIC2 affected the termination efficiency in the sup35 and sup45 mutants. In some strains where either GIC1 or GIC2 was overexpressed, we observed alterations in the level of one or both translation termination factors. We constructed the yeast strains with the disruptions of the chromosomal copies of either SUP35 or SUP45 harboring single and double deletions of GIC1 and GIC2. This allowed us to appreevaluate how the sup35 and sup45 mutations influence formation of the actin cytoskeleton and budding process in cells lacking the GIC1 and GIC2 genes.

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