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### **REGULATION OF GLUCAN SYNTHESIS BY CLATHRIN AND THE EXOMER IN *Schizosaccharomyces pombe*.**

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The cell wall is a protective barrier and an important morphogenetic element. In most fungi, the main components of the cell wall are chitin,  $\beta$ -glucans and mannoproteins. A great effort has been directed to study the regulation of chitin synthesis, which has led to the characterization of a series of specific regulators, termed Chs proteins. The situation is different in the case of  $\beta$ -glucan, where practically the only regulators that are under study are the Rho GTPases and their modulators. Our objective is to study the regulation of the synthesis of  $\beta$ -glucan by the mechanisms of vesicle transport. We have cloned and deleted the *S. pombe clc1*<sup>+</sup> gene, coding for the clathrin light chain; we have found that a *clc1 $\Delta$*  deletion is lethal and that this lethality is remedied by the osmotic protector sorbitol. *clc1 $\Delta$*  cells have an aberrant morphology and an altered composition of the cell wall. A conditional *clc1* mutant with a reduced level of Clc1p has been constructed; this mutant exhibits reduced  $\beta$ -glucan synthase activity and an altered distribution of the  $\beta(1,3)$ glucan synthases Bgs1p, Bgs3p and Bgs4p. These results show that clathrin is important for the correct function of the three  $\beta$ -glucan synthases.

The term exomer refers to a complex of proteins, described in *Saccharomyces cerevisiae*, which has been proposed to form a vesicle coat for the transport of proteins from the trans-Golgi Network to the plasma membrane. The complex contains Chs5p (a scaffold) and four ChAPs (Chs5-Associated Proteins; responsible for cargo recognition). The exomer is required for the regulation of chitin synthesis, cell fusion during mating and polarity. *S. pombe* Cfr1p is similar to Chs5p and Csr1p is the only protein in fission yeast with similarity to the ChAPs. We have found that a *cfr1 $\Delta$*  mutant is sensitive to compounds that interfere with glucan synthesis, exhibits genetic interaction with *cps1/bgs1* and *cwg1/bgs4* mutants, and have a reduced level of  $\beta$ -glucan in their cell wall. Csr1p co-localizes with Cfr1p at the Golgi and both proteins co-immunoprecipitate, showing that they interact physically. Surprisingly, we have found that the localization of Bgs1p, Bgs3p and Bgs4p is different in *cfr1 $\Delta$*  and *csr1 $\Delta$*  mutants. These results show that the  $\beta$ -glucan synthases are differentially regulated by the exomer and questions the idea of Cfr1p and Csr1p working as a complex.