

P004 Influence of an additional genomic mutation on localisation and signalling capacity of several Gap1 alleles in *Saccharomyces cerevisiae*.

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Yeast cells starved for nitrogen on a glucose-containing medium enter G0 and acquire several features characteristic for stationary phase, such as accumulation of reserve carbohydrates, induction of STRE-controlled genes, repression of ribosomal protein genes and enhanced stress resistance. Addition of amino acids to such cells causes growth resumption and rapid reversal of these phenotypes. We previously showed that the General Amino acid Permease Gap1 acts as an amino acid sensor for the pathway responsible for this rapid cellular adaptation upon re-addition of a nitrogen source. C-terminal truncations of Gap1 were constructed that caused a constitutive activation of the pathway. Here we report that the latter phenotype is dependent on an additional mutation in the genome of the *gap1Δ* strain used. We found that this mutation has a large influence on localisation of different Gap1 alleles. Gap1 alleles that are thought to be blocked in the ER show a wild type phenotype in a strain containing this mutation where they have a *gap1Δ* phenotype in the wild type background. Therefore we called this mutation *seg1-1* (for Suppressor of ER exit deficient Gap1 alleles). Also the alleles showing a constitutive activation are much more accumulated in the plasma membrane in this background. The role of this *seg1-1* mutation will be further analysed and the gene containing the *seg1-1* mutation will be identified by screening with a genomic library.