FUNGAL GENETIC ANALYSIS

Flexible switching

New research published in *Cell* shows that switching on and off members of a family of adhesion genes in *Saccharomyces cerevisiae* is linked to morphological development.

The FLO gene family of S. cerevisiae encodes cell-wall glycoproteins. FLO11 is usually the only FLO gene expressed in the strain used by Halme et al. Four silent FLO genes, FLO1, 5, 9 and 10, are located in subtelomeric regions. FLO11 expression is required for surface adherence, filament formation and sliding motility. Expression of different individual FLO genes radically alters cell-adhesion properties. Variation in surface-gene expression is a common tactic used by pathogens to evade the host immune response. Now, Halme et al. show that genetic and epigenetic mechanisms vary FLO gene expression.

Using immunofluorescence and a transcriptional reporter, Halme et al. showed that FLO11 expression was variegated — some cells express it, others don't. Pedigree analysis showed that switching was reversible. Moving FLO11 to a new location prevented switching — showing that FLO11 switching was position-dependent, but switching was also shown to be FLO-promoter specific. The authors suggest that Sf11p recruits Hda1p to silence FLO11. So, FLO11 regulation is both promoter-dependent and position-specific — this was a surprise because most previously characterized position-silencing-dependent switches are promoter-independent. Plus, because FLO11 is ~46 kb from the telomere, this is the third example in S. cerevisiae of a telomere-independent silencing mechanism — it might be more widespread in yeast than previously thought.

What about the reservoir of silent FLO genes? Mutations in the IRA1 and IRA2 genes — the yeast Ras GTPase-activating proteins — which are inherently unstable, unlike the rest of the yeast genome, switch on and off silent FLO loci. Once silent FLO genes are switched on, epigenetic regulation imposes flexibility onto the phenotype-switching mechanism.

In diploid cells, switching *FLO11* expression on and off correlated with the ability to form filaments. Epigenetic and genetic control of *FLO* expression and morphology could be important in pathogenesis — in *Candida albicans* white-opaque phenotype switching is necessary for full virulence. Switching might allow members of the colony to sample new niches, without committing each cell to a new, and potentially less advantageous, phenotype.

Susan Jones

References and links

ORIGINAL RESEARCH PAPER Halme, A. et al. Genetic and epigenetic regulation of the FLO gene family generates cell-surface variation in yeast. Cell 116, 405–415 (2004)



IN BRIEF

INDUSTRIAL MICROBIOLOGY

Engineering Escherichia coli for efficient conversion of glucose to pyruvate

Causey, T. B. et al. Proc. Natl Acad. Sci. USA 101, 2235-2240 (2004)

For industrial purposes, the main microorganisms involved in the microbial production of pyruvate — mutant strains of the yeast *T. glabrata* and *E. coli* — both require precise regulation of the culture conditions, and the associated production costs are high. This paper reports on the development of an *E. coli* strain, strain TC44, which, by the appropriate combination of mutations, gives a high yield of pyruvate from glucose and only requires simple mineral salts as nutrients.

BACTERIAL VIRULENCE

Clustering of Nck by a 12-residue Tir phosphopeptide is sufficient to trigger localized actin assembly

Campellone, K. G. et al. J. Cell Biol. 164, 407-416 (2004)

Enteropathogenic *E. coli* attach to intestinal epithelial cells and induce attaching and effacing (A/E) lesions. One of the main features of A/E lesions is the pronounced alteration to the cytoskeleton directly beneath the attached bacterium that results in the formation of an actin pedestal. Now, Campellone *et al.* report that Tir is the only translocated bacterial effector protein that is required for pedestal formation. Additionally, they have pinpointed a key domain of just 12 amino acids in the carboxyl terminus that is sufficient to induce actin tail formation.

INNATE IMMUNITY

Inactivation of a *Pseudomonas aeruginosa* quorumsensing signal by human airway epithelia

Chun, C. K. et al. Proc. Natl Acad. Sci. USA (17 Feb 2004) doi: 10.1073/pnas.0308750101

P. aeruginosa is the main cause of respiratory tract infections in cystic fibrosis patients. Here, Chun *et al.* demonstrate that an activity associated with differentiated mammalian airway epithelial cells can inactivate the *P. aeruginosa* quorum-sensing signal molecule 3OC12-HSL. The inactivation of this host defence mechanism could be an important factor in chronic bacterial infections.

VIROLOGY

The AU-rich RNA recombination hot spot sequence of brome mosaic virus is functional in tombusviruses: implications for the mechanism of RNA recombination

Shapka, N. & Nagy, P. D. J. Virol. 78, 2288-2300 (2004)

This study shows that RNA from a tombusvirus, an RNA virus that is pathogenic to plants, carrying a well-defined recombination signal from a different plant virus, brome mosaic virus, undergoes frequent recombination in both plant and protoplast cells. The recognition of the same recombination signal by different viruses could promote interviral recombination.