

Among the genes which have been known to play a role in defence mechanism against oxidative stress, the genes for catalase and glutathione peroxidase showed decreased level of expression in *lkh* null mutant. Our results indicate that LAMMER kinase may play a role not only in morphogenetic control but also in defense mechanism against oxidative stress in the fission yeast.

**D 302****LAMMER Kinase Plays a Role in Morphogenesis of the Yeast**

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We have previously cloned the *Schizosaccharomyces pombe* *lkh1*<sup>+</sup> gene encoding a novel kinase belonging to the LAMMER kinase family. For functional analysis, we have constructed the *lkh1 D* null mutant. The cell lengths of *lkh1 D* changed throughout the growth stages different from the wild type cells. Interestingly enough, the *lkh1 D* cells in liquid culture showed increased flocculation, in which, a Ca<sup>2+</sup>-dependent galactose specific lectin-like proteins were involved. In addition, the prolonged cultivation of the *lkh1 D* cells on solid medium showed an abnormal colony margin and an invasion-like growth as the well-known invasive growth of haploid *Saccharomyces cerevisiae*.

*S. cerevisiae* gene, *KNS1*, which also known as the LAMMER kinase family was disrupted in this study. *D kns1* did not show any differences from the wild type cells during normal growth conditions. Notably we have found when S 1278 background cells and the *KNS1* disruptant were grown on minimal media containing 1% butanol, *D kns1* could not induce pseudohyphal growth, even in liquid medium. Furthermore, the *Sch. pombe* *lkh1 D* transformed with *lkh1* and *KNS1*

showed the morphological change including a functional complementation of flocculation. Together, these results suggest that the LAMMER kinase homologue in yeast may not function in exactly the same pathway, but it does have a role in regulation of morphology and the cell cycle. This is the first report on the function of LAMMER kinase homologue in the yeast.

**D 303****Expression of *nsdD* that Controls Sexual Development of *Aspergillus nidulans***

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The *nsdD* encodes a GATA type transcription factor, carrying a type IVb zinc finger DNA binding domain, which functions in activating sexual development of *A. nidulans*. The *nsdD* over-expression by placing the gene downstream the *niiA* promoter resulted in the cleistothecial development even in the presence of 0.6 M KCl that inhibited sexual development specifically. The *nsdD* expression was repressed by 0.6M KCl. A lot of suppressors for *DnsdD* showed the common phenotype similar to that of *nsdD* over-expressed mutant, the salt independent sexual development. These results strongly suggest that the inhibition of sexual development by salts was carried out via *nsdD* involved regulatory network. In several allelic mutants of *nsdD* that resulted in non sense mutations and lacked C terminal zinc finger, the

transcription level was greatly increased. Also in over-expressed mutants, the transcription under its own promotor was reduced. These results suggest that *nsdD* expression is autoregulated. The *nsdD* expression was greatly reduced in *DflbA* mutant, indicating that FlbA is required for *nsdD* expression. However, GA, GB or FluG apparently did not have significant relationship with *nsdD* expression. This result suggest that FlbA is not specifically required for signalling of asexual sporulation but also required for that of sexual development, but FluG is specific to asexual signalling.

**D 801**

**The Possible Role of Akt in the Cell  
Cycle Progression of G2/M Phase in  
PC12 Cells**

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The involvement of Akt in multiple signaling pathway during cell survival and differentiation is well understood, but it is less clear how Akt interact with the cell cycle machinery to regulate cell proliferation. To investigate the role of Akt in cell growth regulation, PC12 cells ectopically expressing wild-type or dominant-inhibitory Akt were analyzed. Overexpression of wild-type Akt markedly accelerated the proliferation of PC12 cells, whereas the growth of cells expressing mutant Akt was arrested at G2/M phase of the cell cycle up to 45%. Consistent with this, the expressions of proteins and mRNA of CDC2 and cyclin B1 which regulate cell cycle transition at G2/M were up-regulated in cells overexpressing wild-type Akt compared to those of parental PC12 cells but down-regulated in cells expressing mutant Akt. Moreover, according

to EMZA analysis, activity of NF-Y transcription factor, regulating the transcription level of CDC2 and cyclin B1 was also dramatically increased in cells overexpressing wild-type Akt. Taken together, our results indicate that Akt may play a role in the cell cycle progression of G2/M phase through activation of transcription of the *cdc2* and cyclin B1 gene.

**D 802**

**Molecular Cloning of a Polycomb  
Group Gene, Xmel18, in Xenopus  
Embryo**

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Genes of the Polycomb and trithroax group (PcG and trxG) are part of a cellular memory system that maintains inactive and active states of homeotic gene expression. Mouse *mel18* is similar to the *Drosophila* PcG members Posterior sex comb (Psc) and Suppressor two of zeste (Su[2]z). One of the conserved protein motifs, the Ring finger, is required for the binding of Mel18 to a GACTNGACT target site and provides us with a basic information in designing degenerate primer corresponding to the motif. Here we report the cloning of a Polycomb gene, *mel18*, from *Xenopus* embryo. Using the combination of PCR techniques with degenerate primers, 5'RACE (rapid amplification of cDNA ends) and 3'RACE, the full length cDNA of *mel18* in the *Xenopus* embryo is successfully cloned without constructing and screening a library. *Xenopus mel18* exhibits a high homology to the mouse *mel18* and this newly cloned *Xenopus* gene is therefore designated Xmel18. The expression of Xmel18 is observed in *Xenopus* embryo. The presence of mRNA of these genes in the embryo