SL301

Cell Signaling Through Redox Control of Gene Regulation in *Rhodobacter sphaeroide*

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We reported earlier that inactivation of the cbb_3 oxidase leads to derepression of PS genes (PS^D) under aerobic conditions, which is accompanied by the oxygen-insensitive formation of the photosynthetic spectral complexes of *Rhodobacter sphaeroides*. Inactivation of the PrrBA two-component activation system in a cbb_3 background overrides the cbb_3 minus phenotype. The spectrum of PS genes which are derepressed under aerobic conditions by the inactivation of the cbb_3 oxidase (*puc, puf, hemA, bchE, hemN,* and *hemZ*) is coincident with those genes which are shown to be regulated by the PrrBA two-component system Based on these and other findings, we proposed that the cbb_3 cytochrome *c* oxidase functions as an O_2/redox sensor which transduces an inhibitory signal to the membrane-bound sensor histidine kinase, PrrB, under aerobic conditions to prevent PS gene expression. The unique role of the cbb_3 oxidase in this sensory transduction pathway comes from studies of a cbb_3 lacking the CcoQ subunit. This mutant contains a fully functional cbb_3 oxidase but it produces spectral complexes aerobically like other Cco mutant strains, further suggesting that the cbb_3 oxidase itself is an O_2/redox sensor.

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Localization of F Plasmid SopB Protein and Gene Silencing via Protein-mediated Subcellular Localization of DNA

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The subcellular localization of the SopB protein, which is encoded by the *Escherichia coli* F plasmid and is involved in the partition of the single-copy plasmid, was directly visualized through the expression of the protein fused to the jellyfish green fluorescent protein (GFP). The fusion protein was found to localize to positions close but not at the poles of exponentially growing cells. Examination of derivatives of the fusion protein lacking various regions of SopB suggests that the signal for the cellular localization of SopB resides in a region close to its N terminus. Overexpression of SopB led to silencing of genes linked to, but well-separated from, a cluster of SopB-binding sites termed *sopC*. In this SopB-mediated repression of *sopC*-linked genes, all but the N-terminal 82 amino acids of SopB can be replaced by the DNA-binding domain of a sequence-specific DNA-binding protein, provided that the *sopC* locus is also replaced by the recognition sequence of the DNA-binding domain. These results suggest a mechanism of gene silencing: patches of closely packed DNA-binding protein is localized to specific cellular sites; such a patch can capture a DNA carrying the recognition site of the DNA-binding domain and sequestrate genes adjacent to the recognition site through nonspecific binding of DNA.

SL303

Regulation of SoxR, the Superoxide-sensory Regulator in *Escherichia coli*

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We isolated Tn10-insertion mutants affecting the activity of SoxR, screening for constitutive expression of SoxS using *soxS-lacZ* fusion. One of the mutations was mapped in *rseB*, a gene in *rseABC* (Regulation of SigmaE) operon. The constitutive *soxS*-expressing phenotype was due to the polar mutation of downstream gene, *rseC*. RseC is likely to function as a component of SoxR reduction system because SoxR was kept in oxidized form to activate *soxS* expression in *rseC* mutant. RseC is a membrane protein with a cysteine-rich N-terminal domain facing the cytoplasm and a transmembrane domain in the C-terminal region. The functionally critical cysteines were determined by cysteine to serine substitution mutagenesis. The transmembrane domain of RseC was also required for RseC function in reducing SoxR. The truncated N-terminal domain of RseC reduced the *soxS* transcription by 50% as judged by in vitro transcription assay. RseC was subject to conformational change according to the redox condition and had an antioxidant activity in vitro. RseC-overproducing cell became more resistant to H_2O_2 and cumene hydroperoxide in vivo but, not to the superoxide generating agents, such as menadione and plumbagin. The phenotype of *rseC* mutant in the stationary phase revealed some interesting properties. Expression of hydroperoxidase I (KatG) did not increase and the expression level of SoxS remained high, although SoxRS system is silent to superoxide generating agents in the stationary phase.

SL304

Regulation of Pseudohyphal Growth in *Candida albicans*

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The pathogenic fungus, *Candida albicans*, undergoes reversible morphogenetic transition ranging among budding yeast, true hypha and pseudohypha. Although pseudohyphae, which were considered as the third form, vary in shape from attached strings of yeast-like cells to long filaments with constriction at the septa, the developmental process has not yet established. A *C. albicans PRF1* (pseudohypha- regulating factor) gene that encoded a protein highly homologous to *Saccharomyces cerevisiae* Ssn6p and *Dictyostelium discoideum* TRFA was isolated. Mutants lacking Prf1p did not develop into true hypha, but rather grew exclusively as pseudohyphae on a variety of aerobic conditions tested at hyphal inducing temperature (37 °C). Under anaerobic or embedded condition, the *prf1/prf1* cells showed a markedly suppressed filamentous growth. Furthermore, the *prf1/prf1* strains exhibited a severe growth defect in serum at 37°C and were unable to established systemic infection in mice. Thus Prf1p is an important regulator determining the morphological transition and virulence in *C. albicans*, and may be a putative target for the exploration of candidacidal drug.

SL305

Identification of a Domain in Yeast Chitin Synthase 3 Required for Biogenesis of Chitin Ring, But Not Cellular Chitin Synthesis

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Chitin, the most abundant polymer