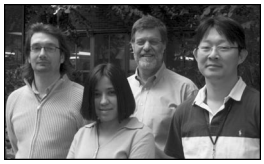
Journal Highlights

Possible Explanation for Unpaired Quorum Sensing Receptor



(l-r) Lequette, Chugani, Greenberg, and Lee

Quorum sensing systems control hundreds of genes, including genes that code for exoenzymes and extracellular virulence factor synthesis. *Pseudomonas aeruginosa* has two complete quorum sensing signaling systems. As is typical, the signal-encoding gene and the signal receptor gene lie adjacent on the chromosome. Several years ago, E. Peter Greenberg, now at the University of Washington, and collaborators showed in *P. aeruginosa* that QscR, an "orphan" signal receptor—a receptor which lacks an adjacent signal generating gene—served to repress virulence. Now Greenberg et al. have shown that QscR responds to a signal generated by one of the paired systems in *P. aeruginosa*, and they have identified a QscR-dependent regulon. They show that in addition to repressing some genes, QscR also activates others. "Repressed genes may be important in causing acute infections, while activated genes may dampen infection," says Greenberg. These findings also raise the question, why the unpaired receptor? Greenberg and coworkers show that unlike the paired receptor for the signal to which QscR binds, QscR interacts reversibly with the signal. "This provides flexibility in response to the signal," says Greenberg. "Genes activated by QscR can be turned off quickly, but activation of genes by the paired receptor will be more committed."

(Y. Lequette, J.-H. Lee, F. Ledgham, A. Lazdunski, and E. P. Greenberg. 2006. A distinct QscR regulon in the *Pseudomonas aeruginosa* quorum-sensing circuit. J. Bacteriol. 188:3365–3370.)

Abundant Phosphoprotein Drives Ribosome Formation

Weber

Nucleophosmin (NPM) is an abundant phosphoprotein residing within the nucleolus that is strongly expressed by proliferating cells, and which has been associated with ribosomal biogenesis, protein chaperoning, and centrosome duplication. Cells lacking nucleophosmin do not grow, for reasons that were unknown. Now, Jason D. Weber of Washington University School of Medicine, St. Louis, and colleagues, as well as collaborators from Oregon Health Sciences University, Portland, have identified a unique binding partner for nucleophosmin: the ribosomal protein L5 (rpL5). rpL5 functions as a chaperone to 5S rRNA. "We showed that the nuclear export of rpL5 and also 5S rRNA was contingent on functional nucleophosmin," says Weber. "In this sense, nucleophosmin guides the nuclear export of the rpL5–5S rRNA complex. Its interaction with rpL5 and 5S rRNA places it in a unique position for driving cell growth. Nucleophosmin could be thought of as a driver of ribosome biogenesis and ultimately protein synthesis. This is important, because in cancer, dysregulation of cell growth pathways is thought to be essential for tumor formation. The next step is to determine whether nucleophosmin is rate-limiting in ribosome nuclear export."

(Y. Yu, L. B. Maggi, Jr., S. N. Brady, A. J. Apicelli, M.-S. Dai, H. Lu, and J. D. Weber. 2006. Nucleophosmin is essential for ribosomal protein L5 nuclear export. Mol. Cell. Biol. 26: 3798–3809.)

Studies of Cellular Response to Pressure May Help Food Processors Develop Effective Preservation Strategies


Yousef

Ultrahigh pressure is the basis for new food preservation technology designed to inactivate pathogens such as *Escherichia coli* O157:H7 and *L. monocytogenes*. However, the method does not uniformly eliminate various strains of the same pathogen, and some strains can adapt, creating considerable risk to consumers. Now Ahmed E. Yousef and colleagues at The Ohio State University have discovered several pathways through which high pressure acts against *E. coli* O157:H7. Using DNA microarray technology, and confirming the resulting transcriptional profile by testing large numbers of knockout mutants, the researchers have found some genes that decrease resistance to pressure, and others that promote pressure resistance. "The study also reveals some of the physiological strategies that marine life may use to cope with extreme pressures in the deep ocean," says Yousef. "We recently completed a follow-up study which sheds still more light on cellular mechanisms for coping with pressure."

(A. S. Malone, Y.-K. Chung, and A. E. Yousef. 2006. Genes of *Escherichia coli* O157:H7 that are involved in high pressure resistance. Appl. Environ. Microbiol. 72:2661–2671.)

N. gonorrhoeae Shows Novel Two-Pronged Strategy for Adapting to the Host


Simms (l), and Jerse

Neisseria gonorrhoeae is a classic example of a pathogen that uses phase variable expression of genes to maintain subpopulations of antigenically and functionally different bacteria. In vitro studies have shown that gonococci expressing and not expressing opacity (Opa) proteins–antigenically distinct outer membrane proteins— differ in their capacity to interact with host cells or evade innate defenses. Amy N. Simms and Ann E. Jerse of the Uniformed Services University of the Health Sciences, Bethesda, Md., show that Opa-expressing gonococci have an unidentified advantage early during experimental genital tract infection of female mice. "We also observed that the recovery of Opa-positive variants is cyclical—a first—which suggests that by always having both bacteria expressing and not expressing Opa available, the gonococcus can evade or capitalize on fluctuating host factors," says Jerse. This is also the first in vivo model for gonococcal infection in women.

(A. N. Simms and A. E. Jerse. 2006. In vivo selection for *Neisseria gonorrhoeae* opacity protein expression in the absence of human carcinoembryonic antigen cell adhesion molecules. Infect. Immun. 74:2965–2974.)

Powerful β-Lactam Resistance Strategy Revealed in *P. aeruginosa*

Hyperproduction of the chromosomal cephalosporinase AmpC in *Pseudomonas aeruginosa* is a major cause of resistance to β-lactam antibiotics. Antonio Oliver and colleagues of the Hospital Son Dureta, Palma de Mallorca, Spain, show that *P. aeruginosa* has three AmpD proteins that act as β-lactamase repressors, and that sequential inactivation of these proteins boosts expression of chromosomal β-lactamase up to 1,000-fold, which, not surprisingly, boosts β-lactam resistance. "We show for the first time that expression of a resistance mechanism can be dramatically and efficiently amplified through multiple steps of derepression," says Oliver. "These findings are a major step forward in understanding resistance to β-lactams, but we still have a lot of details to fill in. Determining the dynamics of in vivo selection of this resistance mechanism is one of our priorities."

(C. Juan, B. Moyá, J. L. Pérez, and A. Oliver. 2006. Stepwise upregulation of the *Pseudomonas aeruginosa* chromosomal cephalosporinase conferring high-level β-lactam resistance involves three AmpD homologues. Antimicrob. Agents Chemother. 50:1780–1787.)