IN BRIEF

BACTERIAL TRANSCRIPTION

Turning the switch on gene expression

Metabolite-binding riboswitches undergo structural changes to regulate transcription or translation of downstream genes. Two papers now identify a riboswitch in a non-coding small RNA (sRNA) that coordinates the expression of the ethanolamine utilization (*eut*) locus with the availability of essential cofactors of this pathway. DebRoy *et al.* showed that, in *Enterococcus faecalis*, the sRNA *eutX*, which is encoded by an intergenic region of the *eut* operon, inhibits *eut* gene expression by sequestering a positive regulator of actively transcribed *eut* mRNAs, termed EutV. Binding of the cofactor adenosyl cobalamine to the *eutX* riboswitch led to premature termination of *eutX* and truncated *eutX* transcripts that cannot bind EutV. Similarly, Mellin *et al.* report that full-length *rli55* sRNA transcripts encoded by the *eut* locus sequester EutV in *Listeria monocytogenes*. Following binding of the cofactor vitamin B12 to the *rli55* riboswitch, truncated *rli55* transcripts are generated, which enables EutV to drive gene expression. Together, these studies provide evidence for a new class of riboswitch that ensures the appropriate activation of a metabolic pathway only in the presence of essential cofactors.

ORIGINAL RESEARCH PAPERS DebRoy, S. *et al.* A riboswitch-containing sRNA controls gene expression by sequestration of a response regulator. *Science* **345**, 937–940 (2014) | Mellin, J. R. *et al.* Sequestration of a two-component response regulator by a riboswitch-regulated noncoding RNA. *Science* **345**, 940–943 (2014)

MICROBIOME

Clostridia spp. combat food allergy in mice

Environmentally induced changes in the composition of the intestinal microbiota have been implicated in allergic sensitization. In this report, Nagler and colleagues show that mice that have been treated with antibiotics have an altered gut microbial diversity and increased sensitization to food allergens. The selective colonization of mice devoid of commensal microorganisms with *Clostridia* spp. provided protection against dietary allergens. Moreover, the authors show that the induction of IL-22 by *Clostridia* spp. in the intestine decreased the access of a peanut allergen to the bloodstream and led to reduced serum allergen concentrations. Thus, *Clostridia* spp. could potentially have a beneficial effect on the treatment of food allergies.

ORIGINAL RESEARCH PAPER Stefka, A. T. *et al.* Commensal bacteria protect against food allergen sensitization. *Proc. Natl Acad. Sci.* USA http://dx.doi.org/10.1073/pnas.1412008111 (2014)

BACTERIAL PATHOGENESIS

Entry through a lipid zipper

It has been suggested that entry of bacterial pathogens into host cells depends on actin polymerization-driven deformation of the plasma membrane as well as additional host and bacterial factors. A new study now shows that binding of the host cell membrane component glycosphingolipid Gb3 to the bacterial surface lectin LecA is sufficient for the engulfment of the opportunistic pathogen *Pseudomonas aeruginosa*. LecA–Gb3 interactions induced cholesterol-dependent formation of a 'lipid zipper' at the membrane, which led to membrane invagination and bacterial invasion. Importantly, actin polymerization was not required for the formation of plasma membrane invaginations. In summary, this study shows that LecA and Gb3 are key factors that promote invasion of *P. aeruginosa* and are potential drug targets.

ORIGINAL RESEARCH PAPER Eierhoff T. *et al.* A lipid zipper triggers bacterial invasion. *Proc. Natl Acad. Sci. USA* **111**, 12895–12900 (2014)