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IN BRIEF

BIOFILMS

Biofilm disassembly revisited

Both D-amino acids and the polyamine norspermidine were previously reported to trigger the disassembly of *Bacillus subtilis* biofilms, but two studies now question these findings. Leiman *et al.* confirm that D-amino acids inhibit biofilm formation, but the authors show that this is not a direct effect and that it is due to the misincorporation of these amino acids into newly synthesized proteins, which also reduces growth. Furthermore, they find that the *B. subtilis* strain in which D-amino acids were previously shown to trigger biofilm disassembly has a mutation in its D-aminoacyl-tRNA deacylase, which removes D-amino acids from mischarged tRNAs and thus prevents their misincorporation into proteins. Repair of this mutation abolished the inhibitory effect of D-amino acids on biofilm formation and growth. Previous work had also reported that norspermidine is produced in aged *B. subtilis* biofilms and that it triggers biofilm disassembly. Using high-performance liquid chromatography, Hobley *et al.* now find that *B. subtilis* biofilms do not produce detectable norspermidine, whereas it is readily detectable in cultures of *Vibrio parahaemolyticus*, which is a known norspermidine producer. Furthermore, the authors show that exogenously added norspermidine promotes *B. subtilis* biofilm formation, similarly to its structural analogue spermidine, which is a known biofilm inducer. Norspermidine inhibits both growth and biofilm formation only at very high concentrations, which are unlikely to occur naturally. Moreover, a genomic analysis showed that *B. subtilis* lacks the norspermidine biosynthetic pathway. In conclusion, these two studies suggest that the roles of D-amino acids and norspermidine as specific biofilm disassembly triggers are questionable and that, under certain conditions, these substances are general growth inhibitors that can indirectly inhibit biofilm formation.

ORIGINAL RESEARCH PAPERS Leiman, S. A. *et al.* D-amino acids indirectly inhibit biofilm formation in *Bacillus subtilis* by interfering with protein synthesis. *J. Bacteriol.* **195**, 5391–5395 (2013) | Hobley, L. *et al.* Norspermidine is not a self-produced trigger for biofilm disassembly. *Cell* **156**, 844–854 (2014)

MICROBIOME

Effects of in-feed antibiotics on pig microbiota

By tracking the changes in the gut microbiome of pigs, Looft *et al.* examined whether in-feed antibiotics alter the composition of the luminal and mucosa-associated communities in the small and large intestines. In untreated pigs, there was a predominance of Firmicutes and an enrichment of phage genes in the ileum, and the mucosa-associated community was, overall, more diverse than that of the lumen. Furthermore, the composition of the mucosa-associated ileal microbiota closely resembled that of the mucosal microbiota of the large intestine, which suggests that it functions as an inoculum for the rest of the gut. Pigs that were fed antibiotics (such as chlortetracycline, sulphamethazine and penicillin) had a divergent microbiota, which included an increase in the abundance of *Escherichia coli* in the ileum and of *Lachnobacterium* spp. in all gut locations. Furthermore, the authors report an increase in antibiotic resistance genes, including resistance genes for antibiotics that were not administered. This can be attributed to co-selection with other resistance genes or to the enrichment of bacteria that already had these resistance genes.

ORIGINAL RESEARCH PAPER Looft, T. *et al.* Bacteria, phages and pigs: the effects of in-feed antibiotics on the microbiome at different gut locations. *ISME J.* http://dx.doi.org/10.1038/ismej.2014.12 (2014)