conditions that promote T3S and multiple interactions were identified within polycistronic transcripts produced from the locus of enterocyte effacement (LEE) that encodes the T3S system. The majority of Hfq binding was within the LEE5 and LEE4 operons, the latter encoding the translocon apparatus (SepL-EspADB) that is positively regulated by the RNA binding protein, CsrA. Using the identified Hfq-binding sites and a series of sRNA deletions, the sRNA Spot42 was shown to directly repress translation of LEE4 at the *sepL* 5" UTR. Strikingly, Hfq binds the *sepL* 5" UTR at the CsrA binding site indicating that translational activation by CsrA competes with the repressive Hfq-Spot42 complex. *In silico* analysis of *sepL* mRNA secondary structure and expression studies of *sepL* site-directed mutants and truncates provided evidence that the unbound *sepL* mRNA is translationally inactive. A model is proposed for transient translation of *sepL*, an OFF-ON-OFF toggle, whereby the nascent mRNA is translationally off, before being activated by CsrA, and then repressed by Hfq and Spot42.

A 07
An antisense RNA connects SigB-associated stress with SOS-response

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Advancements in RNA-sequencing have revealed that overlapping transcription is pervasive throughout bacterial genomes. Antisense transcripts present a perfect base-complementarity with mRNAs from the opposite strand and might interfere with gene expression. However, due to their slight level of expression and low degree of conservation among closely-related species, it remains unresolved if such transcripts are merely transcriptional noise or possess a regulatory function.
In a previous report, we discovered that antisense transcripts overlap the vast majority of the *Staphylococcus aureus* annotated genome. To further understand this phenomenon, we focused on deciphering the potential function of the antisense RNA (asRNA) to the *lexA* gene which encodes the master regulator of the bacterial SOS-response. In the present work, we unveil the overlapping architecture of *lexA*-asRNA, which results from the transcriptional-terminator read-through of a Sigma B controlled gene. As expected, the expression of the asRNA is activated upon cellular stress. The examination of a variety of fluorescent-reporter constructs by flow cytometry and microfluidic systems strongly indicates that the asRNA directly modulates *lexA* gene expression. Using *S. aureus* chromosomic mutants, which modified transcription of *lexA*-asRNA, we show how the asRNA affects the expression of SOS-genes through LexA.
Altogether, our data reveal a complex asRNA architecture that links the SigB-associated stress with the SOS-response in *S. aureus*. Additionally, the *lexA*-asRNA system proves to be a relevant example on how pervasive transcription might have a regulatory impact on gene expression.

A 08
RNA editing and repair by reverse polymerisation

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Embedded on the Central Dogma is the idea that all nucleotide polymerisation proceeds in 5'-3' direction. An ancient nucleotidyltransferase, capable of reverse polymerisation (3'-5'), was recently found to play an essential role in tRNA maturation. In eukaryotes, tRNAHis guanylyltransferase (Thg1) adds am essential G^{-1} residue to the 5' end of $tRNA^{His}$, as a prerequisite for aminoacylation by the cognate histidyl-tRNA synthetase. Our co-crystal structure of Thg1 with $tRNA^{His}$ revealed that Thg1 catalyzes nucleotide addition using a palm domain homologous to canonical forward polymerases, and facilitates reverse polymerisation by orienting the RNA substrate on the opposite side of the palm domain (Nakamura *et al.* 2013, PNAS).
Despite that fact that most archaea and bacteria encode the $tRNA^{His}$ G^{-1} residue in their genomes and may do not require Thg1 to establish $tRNA^{His}$ identity, many species encode a Thg1 homolog (Heinemann *et al.* 2012, NAR). These bacterial and archaeal enzymes are phylogenetically distinct from eukaryotic-type Thg1, and differ in RNA recognition motifs. We recently discovered an extended, template dependent reverse polymerisation repair activity of archaeal Thg1. *Pyrobaculum aerophilum* Thg1 was found to catalyze extended, template dependent tRNA repair, adding up to 13 nucleotides to a truncated $tRNA^{His}$ substrate, restoring a functional $tRNA^{His}$. We further show that the fingers domain of *Methanosarcina acetivorans* and *P. aerophilum* Thg1 is dispensable for enzymatic activity. In addition, we identified residues in yeast Thg1 that prevent extended polymerisation. Mutation of these residues to alanine resulted in extended reverse polymerisation (Desai *et. al.* 2017, RNA Biol.).
While reverse polymerisation is not a common RNA and DNA synthesis route, we show that templated RNA repair can occur in the reverse, 3'-5' direction. This research forms the basis for future engineering efforts towards a high fidelity, template dependent reverse polymerase.

A 09
Bacterial RNA gets an NAD-cap – But how to remove it?

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