OP5.1 - THE UNIQUE ABILITY OF *STAPHYLOCOCCUS EPIDERMIDIS* PHAGE SEP1 ACTIVATING DORMANT CELLS

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ABSTRACT

Most bacteriophages fail to replicate in dormant hosts. The lower metabolic activity of these cells, together with their thicker cell wall and the fewer adsorption sites available impair the action of phages against them. The SEP1 phage, isolated from *Staphylococcus epidermidis*, has the rare ability to reduce the number of cells in a stationary (dormant) state.
To uncover how cells respond to SEP1 infection, both exponential and stationary cultures were challenged with phage. RNA was extracted from samples collected before and after infection (5, 15 and 30 min) and the transcriptomes analyzed by total RNA sequencing.
SEP1 transcripts gradually increased over time, corresponding to 88-95% and 59-76% of the total transcriptome in exponential and stationary cultures, respectively, at 30 min. In exponential cells, there was a logical temporal progression of expressed phage genes, from host adaptation to DNA replication genes and, finally, structural and lysis genes. In stationary cells, SEP1 transcription was delayed, with a significant expression of genes putatively involved in host takeover (gp142 – gp152) observed, mainly at 5 min. Exponential cells responded to SEP1 infection only by upregulating 3 genes involved a DNA restriction and modification system at 5 min post-infection. Two of these genes were substantially overexpressed 15 min after SEP1 infection in stationary cells, with the 3 being upregulated at 30 min post-infection. While on exponential cells, 70 and 78 genes were differentially more expressed at 15- and 30-min post-infection, in stationary cells, 894 and 1309 genes were shown to be upregulated at the same time points.

Functional enrichment analysis grouped these genes in structural constituents of the ribosome, involvement in ribosome and purine nucleoside biosynthetic processes, translation, RNA metabolic processes, etc, demonstrating for the first time that a phage can activate the metabolic machinery of stationary cells.

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