'Friend or foe' bacteria kill their algal hosts when coexisting is no longer beneficial

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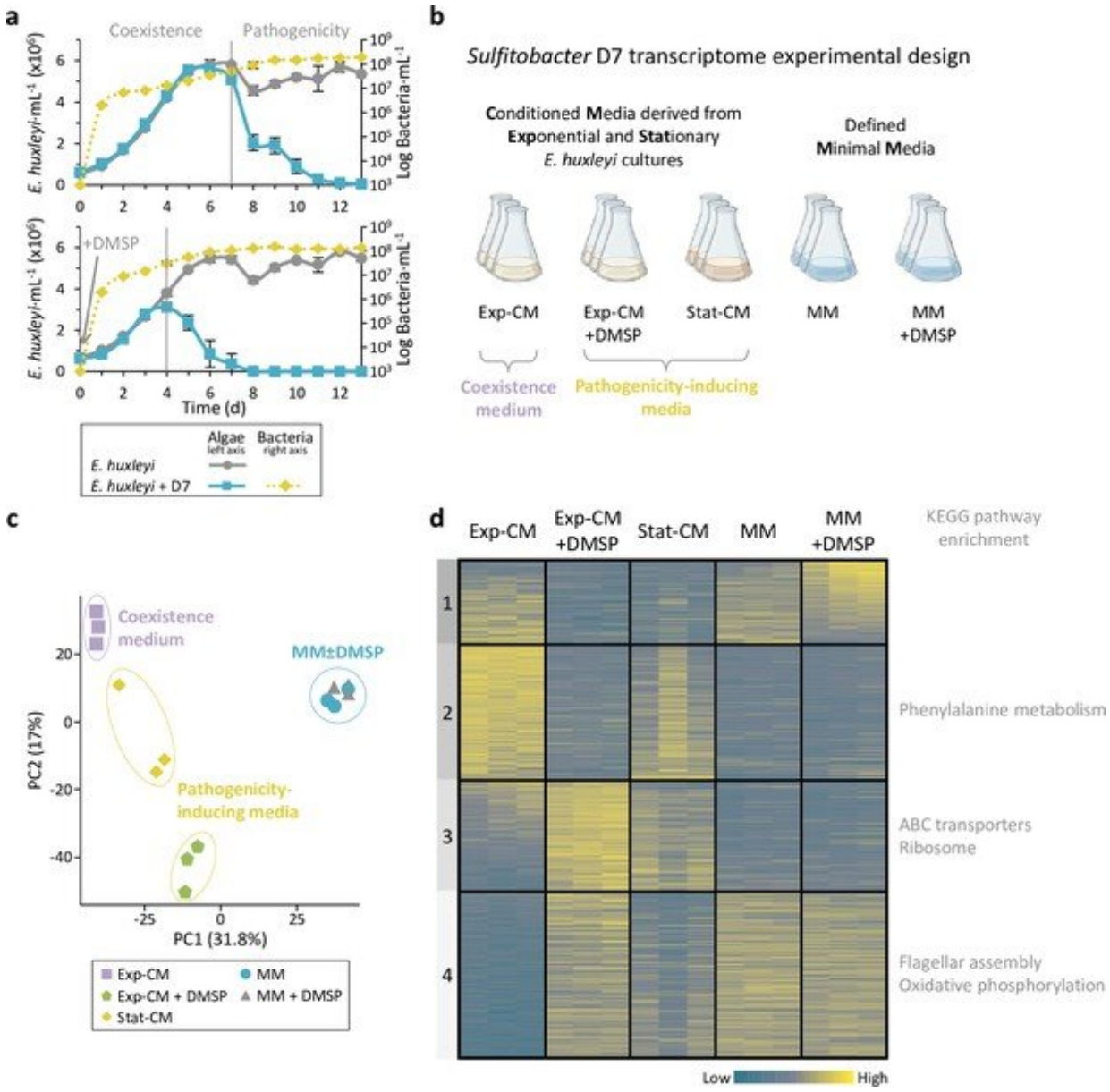

Transcriptional profiling of Sulfitobacter D7 in response to dimethylsulfoniopropionate (DMSP) and additional Emiliania huxleyi infochemicals reveals the signaling role of DMSP. (a) Time course of E. huxleyi CCMP379 and bacterial abundance (full and dashed lines, left and right axes, respectively) in algal mono-cultures or during co-culturing with Sulfitobacter D7. Top panel: Co-cultures display two phases with distinct bacterial lifestyles: coexistence and pathogenicity. Bottom panel: DMSP was added at day 0 to a final concentration of 100 μM. Results represent average ± SD (n=3). Figure 1a has been adapted from Figure 5 of Barak-Gavish et al., 2018. (b) Design of Sulfitobacter D7 transcriptome experiment aiming to explore gene expression profiles in response to E. huxleyi-derived media and in response to DMSP. Growth media consisted of conditioned media (CM) derived from E. huxleyi at exponential growth or stationary phase (Exp-CM and Stat-CM, respectively), and an additional treatment in which 100 μM of DMSP was added (Exp-CM + DMSP). These media differentially induce the coexistence and pathogenicity lifestyles of Sulfitobacter D7. In order to identify DMSP-responsive genes we inoculated Sulfitobacter D7 in defined minimal media (MM), lacking E. huxleyi-derived exudates, without and with 100 μM DMSP (MM and MM + DMSP, respectively). Sulfitobacter D7 was inoculated into each media and harvested for RNA profiling after 24 hr of growth. Initial conditions of the media and bacterial growth are elaborated in Table 1. (c) Principle component analysis of Sulfitobacter D7 detected genes in all treatments (2588 genes). Triplicates of each treatment are shown. (d) Heatmap of gene expression of all differentially expressed genes in the comparisons Exp-CM + DMSP vs. Exp-CM, Stat-CM vs. Exp-CM, and MM + DMSP vs. MM (1179 genes). Clusters were determined based on k-means analysis. Significant functional enrichment in each cluster, based on Kyoto encyclopedia of genes and genomes (KEGG) pathways, is denoted. Each row represents one gene, and the color intensity corresponds to the standardized expression across all samples (triplicates of each treatment are shown). Expression values are scaled by row. Genes in cluster 1 are ordered based on the mean expression values in the MM + DMSP treatment. Genes in cluster 2–4 are ordered based on the mean expression values in the Exp-CM treatment. Credit: *eLife* (2023). DOI: 10.7554/eLife.84400

Scientists have detailed a lifestyle switch that occurs in marine bacteria, in which they change from coexisting with algae hosts in a mutually beneficial interaction to suddenly killing them. The results are published today in *eLife*.

Details of this lifestyle switch could provide new insights into the regulation of algal bloom dynamics and its impact on large-scale biogeochemical processes in marine environments.

Single-celled algae, known as phytoplankton, form oceanic blooms that are responsible for around half of the photosynthesis that occurs on Earth, and form the basis of marine food webs. Therefore, understanding the factors controlling phytoplankton growth and death is crucial to maintaining a healthy marine ecosystem. Marine bacteria from the Roseobacter group are known to pair up and coexist with phytoplankton in a mutually beneficial interaction. The phytoplankton provide the Roseobacter with organic matter useful for bacterial growth, such as sugar and amino acids, and the Roseobacter in return provides B-vitamins and growth-promoting factors.

However, recent studies have revealed that Roseobacters undergo a lifestyle switch from coexistence to pathogenicity, a state in which they kill their phytoplankton hosts. A chemical compound called DMSP is produced by the algae and is hypothesized to play a role in this switch.

"We have previously identified that the Roseobacter Sulfitobacter D7 displays a lifestyle switch when interacting with the phytoplankter Emiliania huxleyi," states first author Noa Barak-Gavish, a Ph.D. graduate in the Department of Plant and Environmental Sciences, Weizmann Institute of Science, Israel. "However, our knowledge about the factors that determine this switch was still limited."

To characterize this lifestyle switch, Barak-Gavish and colleagues

performed a transcriptomics experiment, allowing them to compare the genes that are differentially expressed by Sulfitobacter D7 in coexistence or pathogenicity stages.

Their experimental setup demonstrated that Sulfitobacter D7 grown in a pathogenicity-inducing medium have a higher expression of transporters for metabolites such as amino acids and carbohydrates than those grown in a coexistence medium. These transporters serve to maximize the uptake of metabolites released from dying Emiliania huxleyi (E. huxleyi) . Furthermore, in pathogenic Sulfitobacter D7, the team observed an increased activation of flagellar genes that are responsible for the movement of the bacteria. These two factors allow Sulfitobacter D7 to utilize an "eat-and-run" strategy, where they beat competitors to the material released upon E. huxleyi cell death and swim away in search of another suitable host.

The team confirmed the role of DMSP in bringing about the switch to this killer behavior by mapping the genes activated in Sulfitobacter D7 in response to the presence of DMSP and other algae-derived compounds. However, when only DMSP was present, the lifestyle switch did not occur. This implies that although DMSP mediates the lifestyle switch, it is also dependent on the presence of other E. huxleyi-derived infochemicals—compounds that are produced and used by organisms to communicate.

DMSP is an infochemical produced by many phytoplankton, so it is likely that the other required infochemicals allow the bacteria to recognize a specific phytoplankton host. In natural environments, where many different microbial species exist together, this specificity would ensure that bacteria only invests in altering gene expression and its metabolism when the correct algal partner is present.

The study also uncovers the role of algae-derived benzoate in

Sulfitobacter D7 and E. huxleyi interactions. Even in high concentrations of DMSP, benzoate functions to maintain the coexistence lifestyle. Benzoate is an efficient growth factor and is provided by E. huxleyi to Sulfitobacter D7 during coexistence. The authors propose that as long as Sulfitobacter D7 benefits from coexistence by receiving materials for growth, it will maintain the mutualistic interaction. When less benzoate and other growth substrates are provided, the bacteria undergoes the lifestyle switch and kills its phytoplankton host, swallowing up any remaining useful materials.

The exact mechanism of Sulfitobacter D7 pathogenicity against E. huxleyi remains to be discovered, and the authors call for further work in this area. The cellular machinery Type 2 secretion system—a complex that many bacteria use to move materials across their cell membrane—is more prevalent in Sulfitobacter D7 compared to other Roseobacters, hinting at a unique method of pathogenicity that requires further investigation.

"Our work provides a contextual framework for the switch from coexistence to pathogenicity in Roseobacter-phytoplankton interactions," concludes senior author Assaf Vardi, a professor in the Department of Plant and Environmental Sciences, Weizmann Institute of Science. "These interactions are an underappreciated component in the regulation of algal bloom dynamics and further study in this area could provide insights into their impact on the fate of carbon and sulfur in the marine environment."

More information: Noa Barak-Gavish et al, Bacterial lifestyle switch in response to algal metabolites, *eLife* (2023). DOI: 10.7554/eLife.84400

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