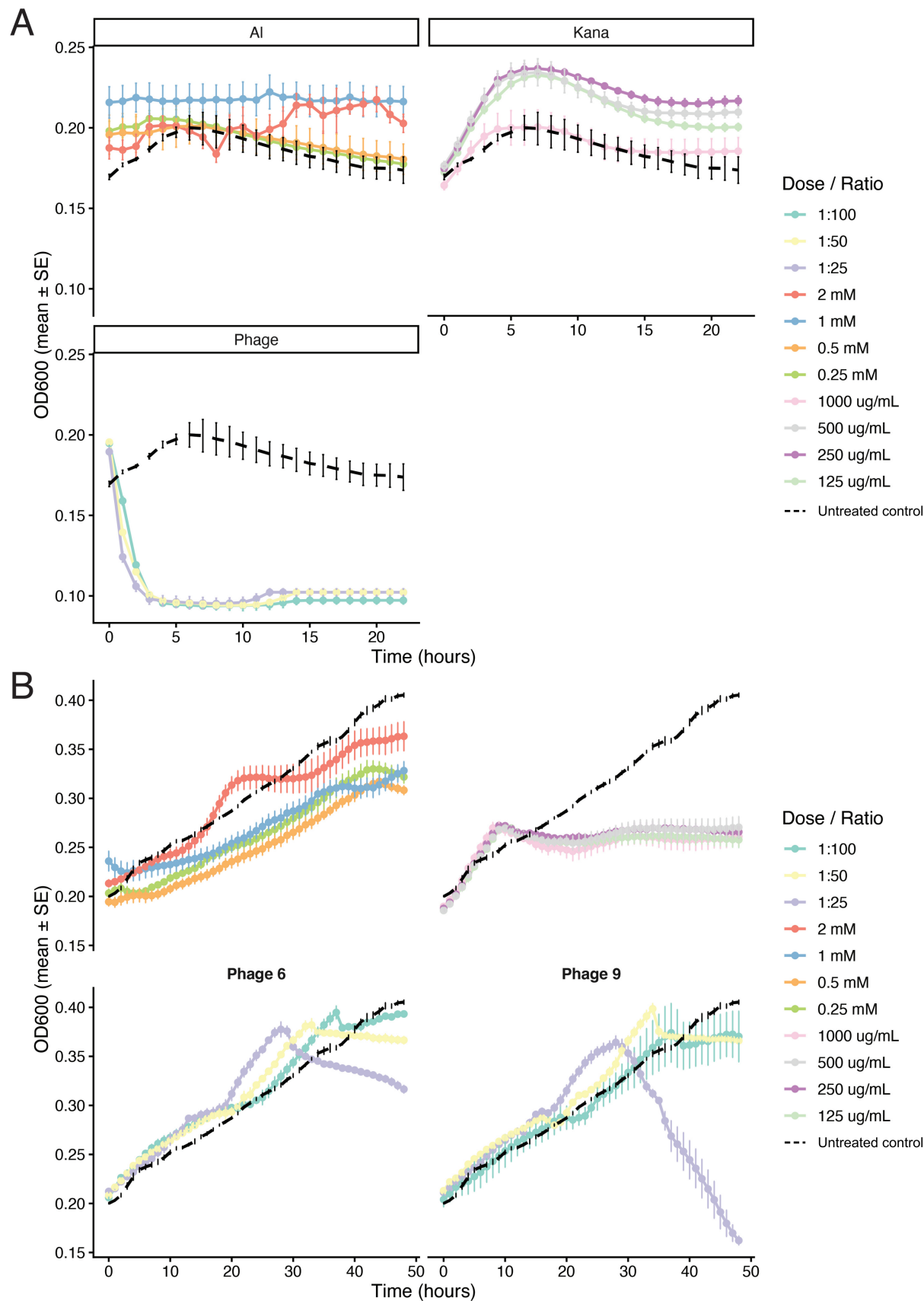
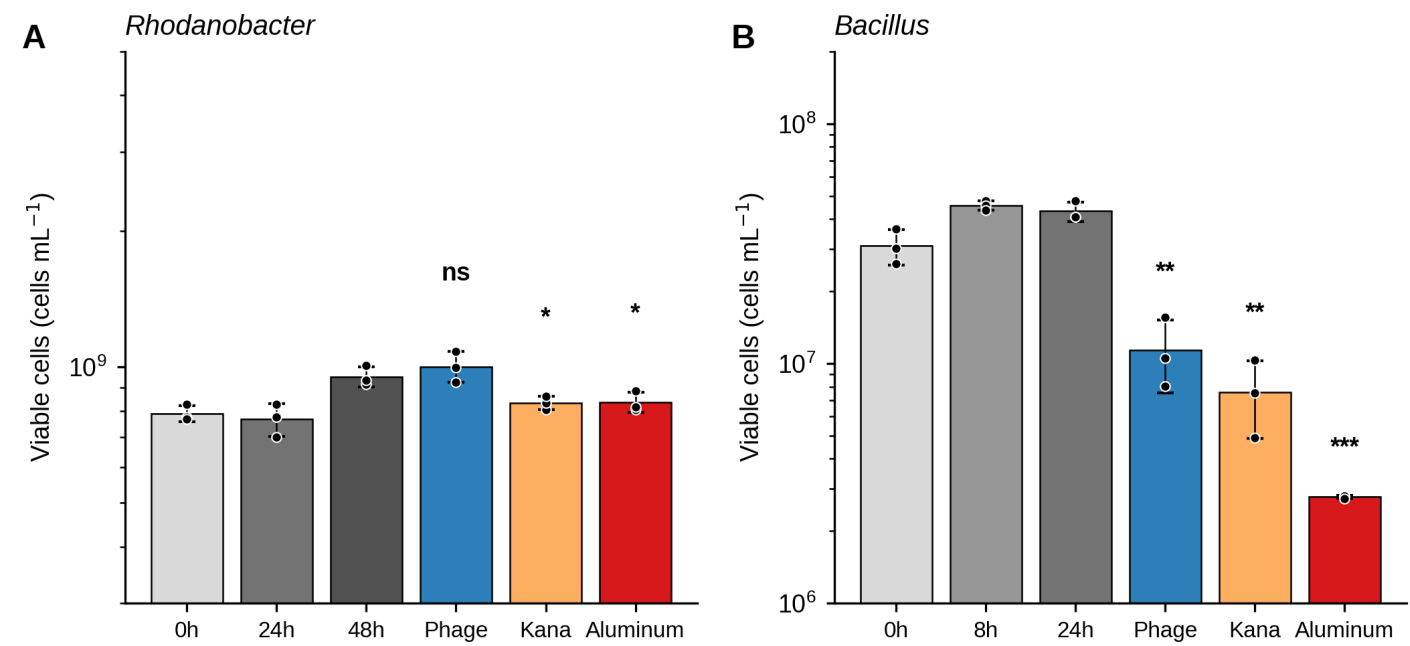


Supplementary Figure S1



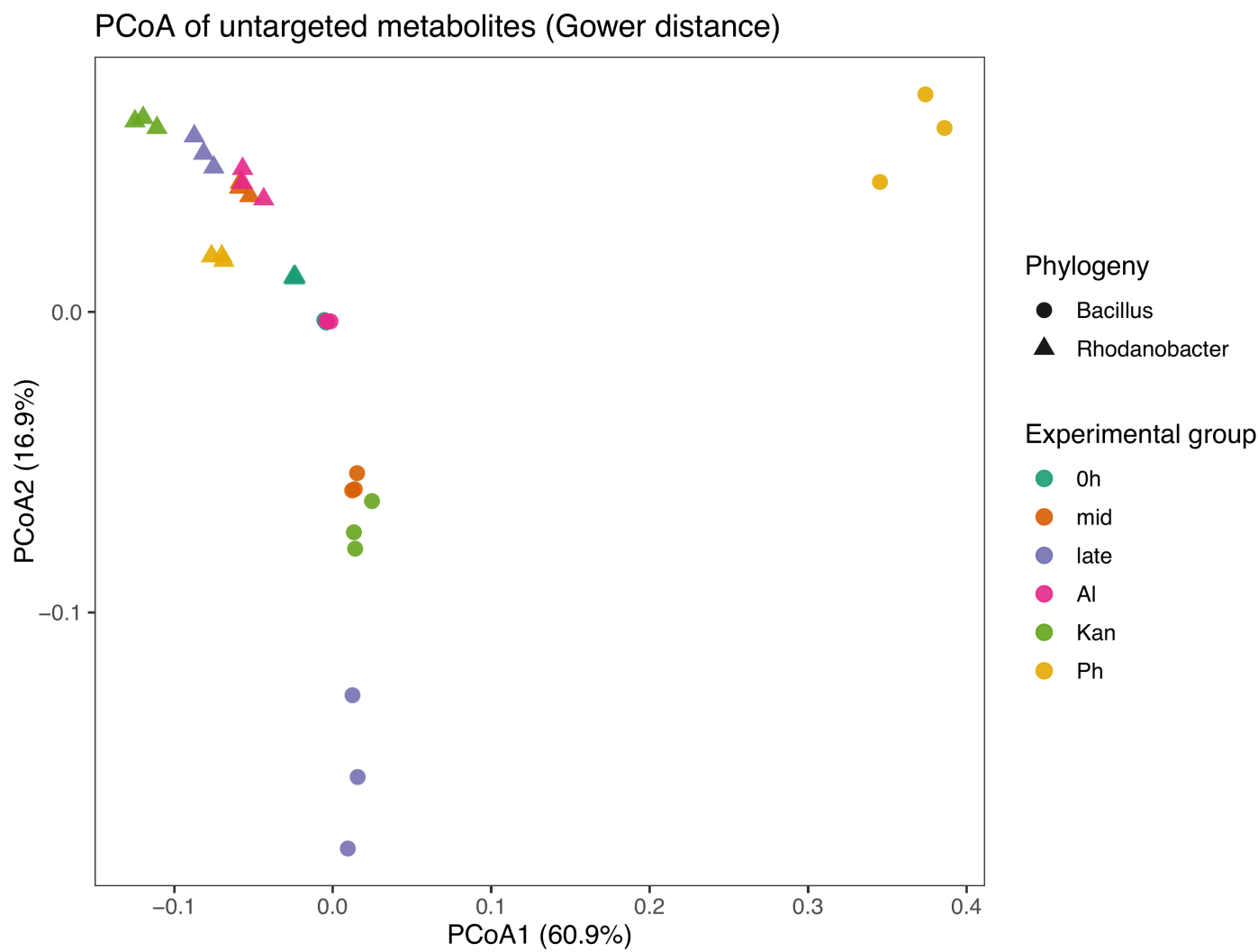
Supplementary Figure S1. Dose-response screening of aluminum, kanamycin and bacteriophage stress in *Bacillus* and *Rhodanobacter* identifies conditions producing growth inhibition or lysis. OD600 (mean $\pm$ SE) over time for *Bacillus* (A) and *Rhodanobacter* (B) across a dose range of each stressor; black dashed lines show untreated controls. Conditions selected for downstream analyses (1 mM aluminum, 500 μ g mL⁻¹ kanamycin, 1:50 (v/v) phage for *Bacillus* and 1:25 (v/v) phage for *Rhodanobacter*) balanced strong stress against sufficient biomass for multi-omic profiling. n = 4 biological replicates per condition.

Supplementary Figure S2



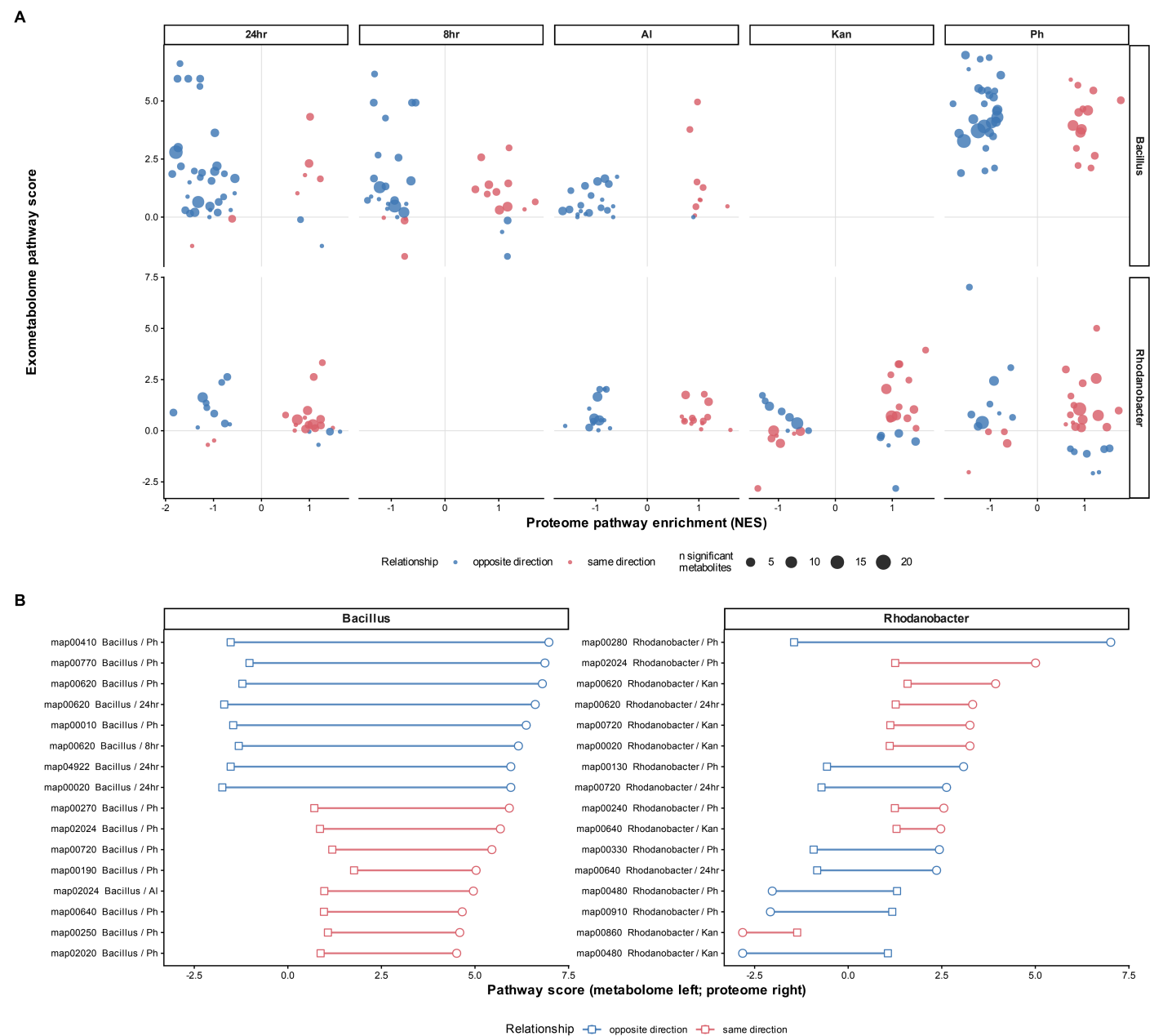
Supplementary Figure S2. Direct cell counts (cells mL⁻¹) at experimental endpoints and at time-course timepoints for *Rhodanobacter* (A) and *Bacillus* (B). Bars show mean $\pm$ SD of $n = 3$ biological replicates; black dots indicate individual replicates. Untreated time-course samples are shown in grey, phage in blue, kanamycin in orange and aluminum in red. Significance markers above stress conditions denote two-sided independent t-tests on log₁₀-transformed cell densities relative to the time-matched untreated control (48 h for *Rhodanobacter*; 24 h for *Bacillus*): *** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$, ns $p \geq 0.05$.

Supplementary Figure S3



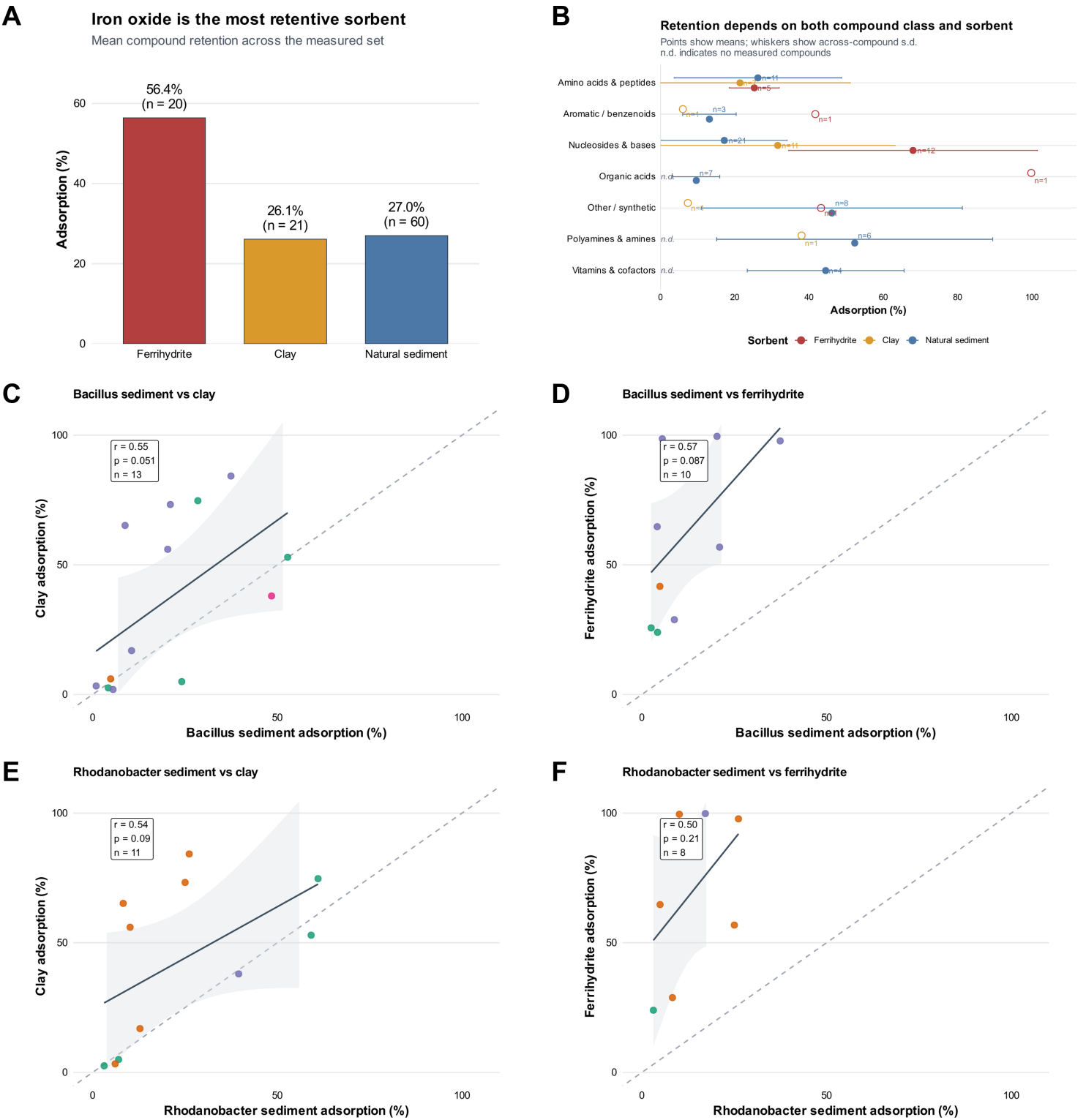
Supplementary Figure S3. Ordination of untargeted metabolite profiles. Principal coordinates analysis (PCoA) based on Gower distances among samples from *Bacillus subtilis* and *Rhodanobacter*. Each point represents a sample; symbol shape denotes organism (circles, *Bacillus*; triangles, *Rhodanobacter*), and colour denotes group: 0 h, mid, late, aluminum (Al), kanamycin (Kan) or phage (Ph). Percentages on the axes indicate the variation represented by each coordinate.

Supplementary Figure S4



Supplementary Figure S4. Proteome and exometabolome pathway scores show condition-dependent agreement. (A) Proteome pathway enrichment scores plotted against exometabolome pathway scores for available organism–condition combinations. Pathways are shown when proteome FGSEA adjusted $P \leq 0.05$ or at least one significant metabolite maps to the pathway. Point size indicates the number of significant metabolites; colour indicates whether the scores have the same, opposite or weak/zero direction. No peptide or proteomic results were obtained for *Bacillus* under kanamycin, so this condition is omitted; it does not indicate a null response. (B) Up to eight representative pathways per organism and direction, ranked by the sum of the absolute proteome and exometabolome scores. Each segment joins an open circle (exometabolome score) and an open square (proteome score); colour denotes concordant or opposing directions. Al, aluminum; Kan, kanamycin; Ph, phage.

Supplementary Figure S5



Supplementary Figure S5. Aqueous-phase metabolite removal across sorbents and organisms. (A) Mean aqueous-phase removal by ferrihydrite, clay and natural sediment; labels give the number of compounds measured. (B) Mean removal by compound class and sorbent. Points show means, whiskers show across-compound s.d.; n labels give the number of compounds, and n.d. indicates no measurements. (C,D) Matched-compound aqueous-phase removal by clay and ferrihydrite, respectively, compared with *Bacillus*-derived natural-sediment samples. (E,F) Corresponding comparisons for *Rhodanobacter*-derived samples. Points represent individual compounds and are coloured by compound class. Dashed lines show the identity relationship; solid lines and shaded bands show linear fits and 95% confidence intervals. Insets report Pearson's r , p and n . The plotted term "adsorption" denotes operationally measured aqueous-phase removal; loss from the supernatant does not distinguish surface adsorption from other removal processes.