**Figure and Table Legends**

**Figure 1. Growth and metabolism of *N. aromaticivorans* DSM12444, ΔsacB strain during growth on G-diketone and glucose.** (Panel A) Increases in *N. aromaticivorans* cell density as monitored by Klett colorimeter units. (Panel B) Extracellular concentration of G-diketone. (Panel C) Extracellular abundance of an unknown UV-absorbing compound (measured at a wavelength of XX nm). Based on MS analysis of this compound, it is most likely a derivative of the G-diketone with either the Cα or Cβ ketone reduced (see text).

**Figure 2. Changes in transcript abundance when cells were grown on glucose plus G-diketone, GP-1 (**a derivative of G-diketone where the β-ketone is reduced)**, or PCA, vanillic acid, vanillin, and ferulic acid (referred to as base G) compared to glucose-grown cells.** The colors in each bar indicate the condition where a significant change in transcript abundance compared to glucose-grown cells was first observed. The base G (light blue) category counts the number of genes with a significant change in transcript abundance with increased (Panel A) or decreased (Panel B) abundance compared to cells grown in the presence of glucose on one or more of the tested aromatic substrates. The GP-1 (green) category indicates genes with a significant change in transcript abundance compared to cells grown in the presence of glucose but not on any of the base G compounds. The G-diketone category (dark blue) indicates genes with a significant change in transcript abundance compared to cells grown in the presence of glucose, but no significant change on GP-1 or any of the base G compounds.

**Figure 3. Changes in transcript abundance for indicated genes associated when cells are grown in the presence of G-type aromatics.** Each plot displays the log2-fold change in reads per kilobase million (RPKM) compared to the glucose control for *N. aromaticivorans* genes previously identified as encoding enzymes involved in aromatic metabolism. Black stars above a gene indicate levels of significantly different expression (\*p < 0.05, \*\*p<0.01, \*\*\*p<0.001) compared to expression of cells grown in the presence of glucose alone. Bars in each panel are colored to denote steps in aromatic metabolism that gene products are known to function (dimer degradation, aromatic ring processing, side chain processing/demethylation).

**Figure 4. Kinetic parameters of LigL, LigN and LigD dehydrogenases with indicated aromatic substrates.** Shown are the measured Kcat and apparent Km using recombinant LigL, LigN, and LigD enzymes with the indicated aromatic substrates and either NADH (G-diketone) or NAD+ (GGE, GD) as a cofactor.

**Figure 5. Time-dependent loss of G-diketone *in vitro* when incubated with recombinant LigL, LigN, and LigD,** **with and without NADH.**

**Figure 6. GC-MS analysis of aromatic substrates and *in vitro* reaction products of individual LigLND dehydrogenases with G-diketone and GP-1.** GC-MS analysis of aromatic substrates and products after indicated Lig dehydrogenases were incubated for 24 hours with the G-diketone and NADH (Panel A, C) or GP-1 and NADH (Panel B, D). Mass spectra of each peak are presented in Panel E.

**Figure 7. Proposed mechanism of G-diketone degradation by *N. aromaticivorans.*** We hypothesize that degradation of G-diketone is initiated by reduction of the Cα ketone to GP-2 via one of several redundant Lig dehydrogenases. GP-2 and GP-1, as Hibberts ketones, can spontaneously interconvert. The LigL dehydrogenase reduced GP-2 to 1-(4-hydroxy-3-methoxyphenyl)propane-1,2-diol (GD). GD is then metabolized by an unknown enzyme to produce vanillin.