Whole-Genome Transcriptional Response of *Desulfovibrio vulgaris* to Nitrite

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Sulfate reducing bacteria are capable of reducing soluble metal oxyanions to insoluble forms, a process of great potential in the bioremediation of toxic heavy metals and radionuclides. Nitrate, a common contaminant in DOE sites, however is suggested to inhibit sulfate reduction activity via nitrite. Therefore, we used *Desulfovibrio vulgaris* Hildenborough as a model organism to investigate the inhibition of sulfate reduction by nitrite.

Whole-genome microarrays were developed to study nitrite stress response at the transcriptional level. Our results indicated that *D. vulgaris* was capable of rapid nitrite reduction and the exposure to nitrite induced genes involved in nitrogen metabolism, iron homeostasis, and oxidative stress. The nitrite reductase operon was among the most highly up-regulated genes and the induction of the HcpR regulon further suggested the direct involvement of these genes in electron transport during nitrite reduction. Another group of highly up-regulated transcripts consisted of genes related to iron transport, which were found to contain a consensus up-stream *fur* binding motif, indicating iron uptake and metabolism were strongly associated with the response to nitrite. It is likely that the oxidation of Fe(II) by nitrite resulted in the derepression of the Fur regulon. In addition, many of the up-regulated genes require iron as a cofactor, which may contribute to the demand for iron uptake. We also observed up-regulation of oxidative stress related genes, such as the PerR regulon, which could be a side effect from the oxidative nature of nitrite. On the other hand, down regulated genes included genes encoding for ATP synthase subunits and ribosomal proteins, showing a shift in energy flow and an interruption in biosynthesis.

Overall in response to nitrite, *D. vulgaris* shifted the flow of reducing equivalents from energy conservation and anabolism to the reduction of nitrite. The presence of nitrite also impacted iron homeostasis which led to the increase in iron uptake and metabolism.