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Computational insights to high dimensional data from Clostridium difficile infection

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In recent years, new technology has allowed us to achieve measurements of hundreds of metabolites and the expression of thousands of genes. With this large scale, all-encompassing, 'omics' data comes a critical need to reduce these datasets to the most functional elements so that we can discover key components driving disease pathogenesis. Current techniques to analyze omics data are not geared towards supporting parsimonious mechanistic models of bacterial pathogenesis. Specifically, networks are common tools for analyzing these data, however, at times graphical networks can be overwhelming due to the complexity of the information. In this work, we use sparse graphical networks to understand correlations between multiple high dimensional data sets. We apply these method to metabolomics and transcriptomics data from a recent animal model for *Clostridium difficile* infection in which mice were antibiotic treated with cefoperazone and challenged with *C. difficile* 2 days following treatment. We find significant changes in the *C. difficile* transcriptome at later time points compared to earlier time points; while, most of the amino acid changes occur in the first 24 hours. We find taurine, proline, 3-(4-hydroxyphenyl)lactate, 5-aminovalerate, 5-oxoproline, thioproline are the main amino acids contributing to the changes across all time points. Utilizing the key components of the sparse graphical model we've developed and analyzed an ordinary differential equation model to better understand the specific mechanisms leading to *C. difficile* colonization.

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