POSTER SESSION 1

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Assignment of novel functions to Helicobacter pylori 26695's genome and reconstruction of a genome-scale metabolic model

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Helicobacter pylori is a pathogenic organism associated with human gastric diseases. The development of mathematical models of metabolism is now considered a fundamental part of the study of the cell. For the particular case of microorganisms associated with human diseases, information on metabolic and regulatory networks can be used to understand the molecular factors of the microorganism that are likely to interact with the host and cause diseases. The availability of the genome sequence of H. pylori 26695 and its annotation has allowed in the past the construction of a metabolic model for this organism. The first genome-scale metabolic model for H. pylori 26695 was published in 2002 (iCS291) and a corrected reconstruction was published in 2005 (iIT341 GSM/GPR). The main goal of the present work was to update H. pylori's genome-scale metabolic model based on the new information made available. For that purposes, using new annotation methodologies and data available in databases, an assignment of novel functions to H. pylori 26695's genome was performed. For a total of 510 "hypothetical proteins" (almost 1/3 of the genes) identified in the last re-annotation, 137 new functions were attributed. A total of 581 E.C. numbers were assigned to CDS, being 528 complete E.C. numbers. This new information was used as the basis of the model reconstruction. In addition, transport reactions in the model were updated. The biomass equation was reviewed and H. pylori biomass coefficients and composition were adjusted. The obtained model successfully predicted the nutritional requirements and amino acids essentialities, which were experimentally validated. As a result, the present work presents a new H. pylori 26695 genome-scale metabolic model with more accurate and reliable predictions and can be used to identify potential targets for designing more effective drugs for H. pylori inactivation.