Predicting effects of structural stress in a genome-reduced model bacterial metabolism

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Mycoplasma pneumoniae is a human pathogen recently proposed as a genome-reduced model for bacterial systems biology. The response of its metabolic network to different forms of structural stress, including removal of individual and pairs of reactions and knockout of genes and clusters of genes with correlated coexpression, reveals a network architecture as robust as that of other model bacteria, despite its reduced genome and its greater metabolic linearity. Interestingly, metabolite motifs associated to reactions can predict the propagation of individual failures and strong non-linear damage amplification effects that may arise in double breakdowns. We also detect a significant correlation between gene essentiality and the damages produced by single gene knockouts, and find that genes controlling high-damage reactions tend to be expressed independently of each other, a functional switch mechanism that simultaneously acts as a genetic firewall to protect metabolism. Control of failure propagation is crucial for metabolic engineering or disease treatment.

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[1] Güell O, Sagués F, Serrano MA, 2012. Predicting effects of structural stress in a genome-reduced model bacterial metabolism. *arXiv:1202.5931v1 [q-bio.MN].*