Supplementary Material

Integrating Proteomic, Transcriptional, and Interactome Data Reveals Hidden Components of Signaling and Regulatory Networks

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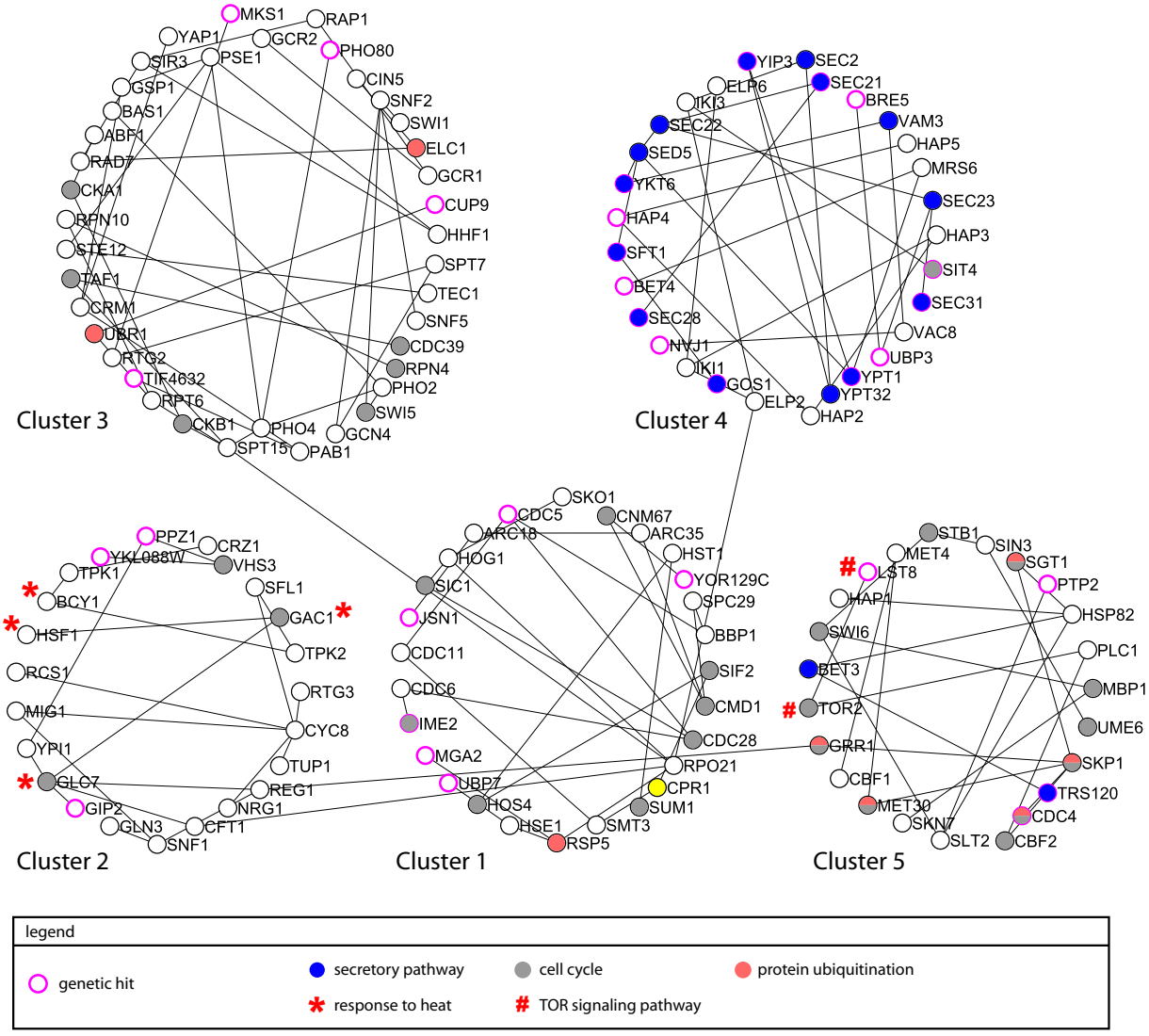

Fig. S1. The clusters in the protein-protein interaction part of the α-syn PCST solution. Nodes are colored or marked by GO biological process. TOR: target of rapamycin.

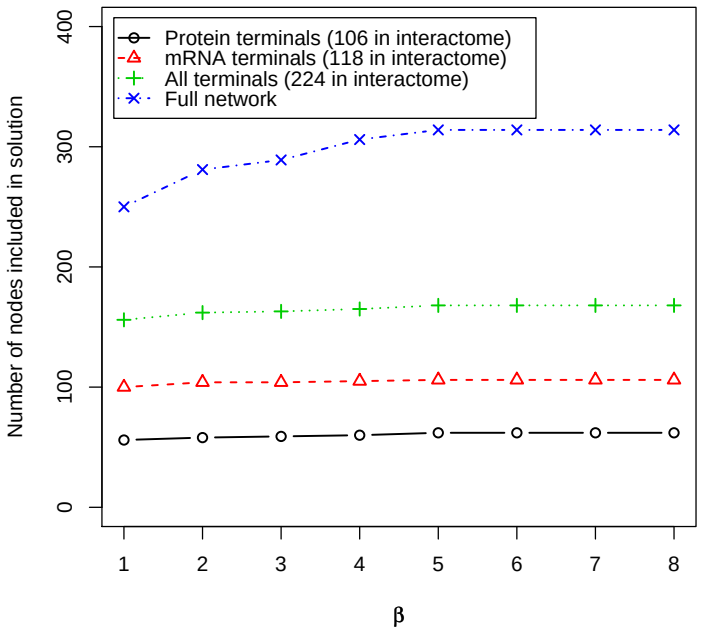

Fig. S2. Statistics of the yeast pheromone PCST solution network for different values of β. The PCST solution constructed from the pheromone response datasets is relatively stable with respect to the parameter β, as measured by the number of terminal nodes included in the solution that represent proteins with differentially phosphorylated sites (protein terminals) and genes that are differentially transcribed (mRNA terminals). The number of terminals indicated in the figure legend counts only the ones present in the interactome.

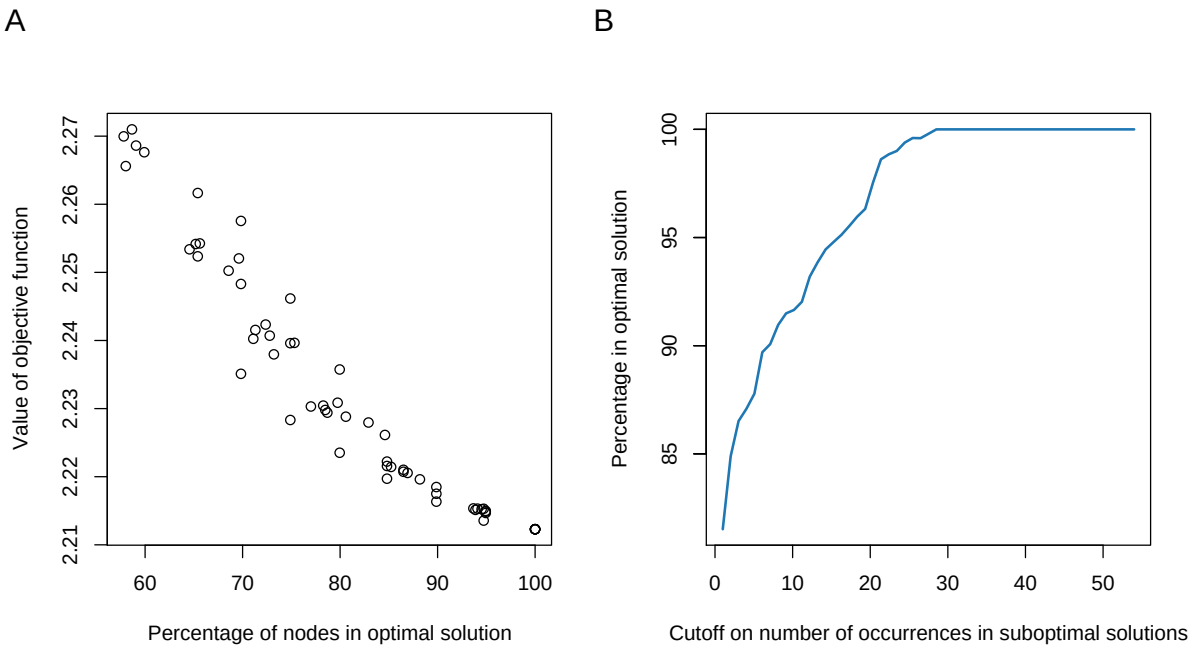

Fig. S3. Alternative or suboptimal solutions to the yeast pheromone response data set. Because we use an optimization approach to analyze inherently noisy data, we asked whether the network we obtained was stable - are there very different networks that explain the data almost as well? For this, we compared the optimal solution network to a set of alternative solution networks obtained by finding networks that are different from the optimal one by at least a specific percentage of nodes. **(A)** No alternative solutions in the neighborhood of the optimal solution achieves the same objective function value. **(B)** Of the nodes that appear at least once in the 54 suboptimal solutions, at least 80% also appear in the optimal solution.

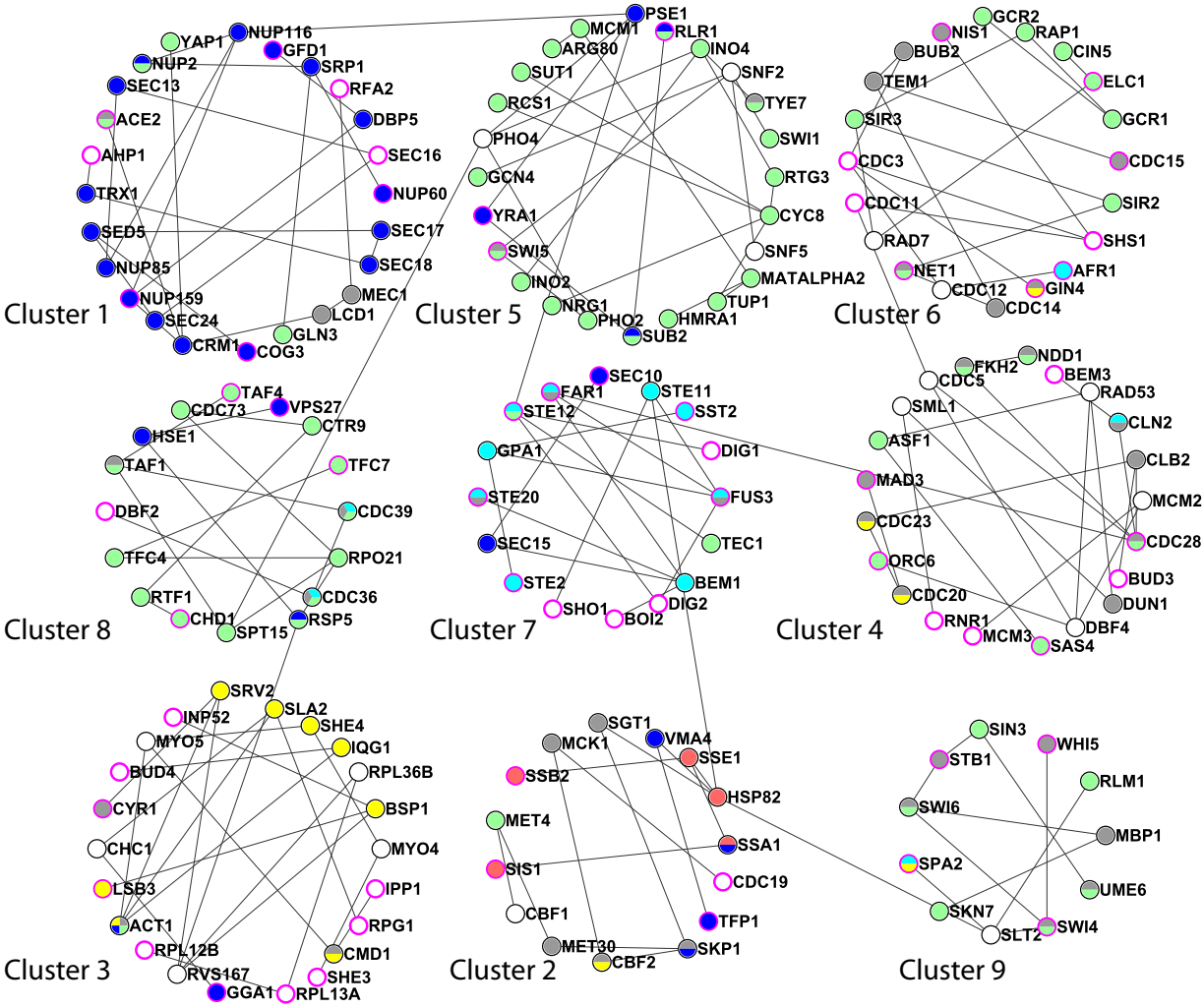

Fig. S4. The clusters in the protein-protein interaction part of the yeast pheromone response PCST solution. Nodes are colored by GO biological process. Cluster labels correspond to those in Table 1.

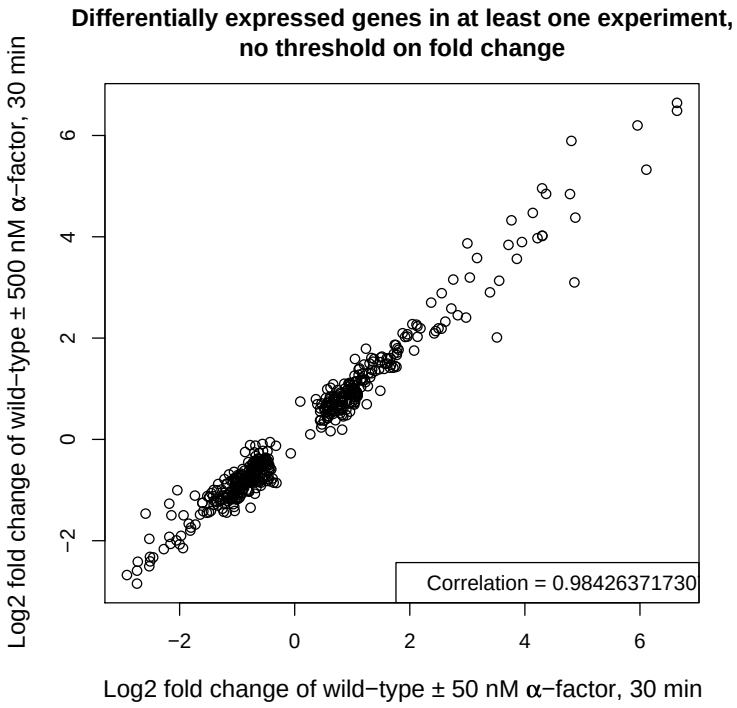

Fig. S5. Scatter plot of gene expression changes after 50 and 500 nM α-factor treatment. Wild-type yeast cells were treated with 50 nM and 500 nM α-factor for 30 minutes (*1*). Fold changes were calculated with respect to wild-type, untreated cells.

References

1. C. J. Roberts, B. Nelson, M. J. Marton, R. Stoughton, M. R. Meyer, H. A. Bennett, Y. D. He, H. Dai, W. L. Walker, T. R. Hughes, M. Tyers, C. Boone, S. H. Friend, Signaling and circuitry of multiple MAPK pathways revealed by a matrix of global gene expression profiles. *Science* **287**:873–880 (2000).