Electronic Supplementary Material (ESI) for Integrative Biology

Cancer develops, progresses and responds to therapies through restricted perturbation of the protein-protein interaction network

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Supplementary file legends

Supplementary file 1. Table S1: Numbers of differentially expressed genes at each stage of breast cancer development and progression, and the corresponding number of proteins mapped in the interactome network (HPRD dataset). The sets correspond to expression probes with FDR-adjusted *p* values < 0.05. The numbers of proteins (shown in parentheses) correspond to the main component of the interactome network.

Supplementary file 2. Figure S1: Breast cancer-related conditions associate with topological robustness in the IntAct-based interactome network. For breast cancer development and progression, and different values of α, the graphs show the number of node failures (random - observed) across τ values. Black or colored lines (for under- and over-expressed sub-sets) indicate significant differences (empirical $p < 0.05$).

Supplementary file 3. Figure S2: KEGG pathway sets that do not reveal topological robustness when selected in the HPRD-based interactome network. Results are shown for 136 sets. The limits of the main graph axis are equivalent to those shown in the rest of figures. Dashed-line boxes highlight protein sets associated with an opposite topological impact to those of cancer conditions.

Supplementary file 4. Figure S3a: KEGG pathway sets that reveal topological robustness when selected in the HPRD-based interactome network. Results are shown for 35 sets. The limits of the main graph axis are equivalent to those shown in the rest of figures. Dashed-line boxes highlight protein sets associated with a similar topological impact to those of cancer conditions. Figure S3b: Results from the analysis of the protein sets corresponding to the Gene Ontology terms "Immune Response" and "Intracellular Protein Kinase Cascade".

Supplementary file 5. Figure S4: Results of the topological study of kinases in the HPRD-based interactome network. Both sets (differentially and non-differentially expressed kinases in the N-IDC comparison) reveal topological robustness when selected in the interactome network.

Supplementary file 6. Figure S5: Colorectal cancer associates with topological robustness. For the comparison between normal tissue (N) and adenomas (A), and adenomas and carcinomas (C), and different values of α, the graphs show the number of node failures (random - observed) across τ values. Black or colored lines (for under- and over-expressed sub-sets) indicate significant differences (empirical $p < 0.05$).

Supplementary file 7. Table S2: Differential expression between MCF7 and MCF7-*SNCA* cells. Results correspond to a $>|2\text{-fold}|$ change between two clones of each cell line. Raw data has been deposited at GSE31180.

Supplementary file 8. Table S3: Average shortest path values for each KEGG pathway set in the HPRD-based interactome network. Values are shown for sets with a different or similar topological impact to those of cancer conditions.