Supplementary Figures Legends

Fig. S1. Connectivity hierarchies of GBM1 (**A**) and GBM2 (**B**) hierarchical TR/TF-CGMs networks. The top in the hierarchical pyramid of connectivity is (i) and the lower level is (vi). (**A**) The top regulator TGFβ controls all 11 CGMs of GBM1. The density of connections is maximal for CGMs 1–5 and 7–8 at the levels 2 (ii) and 3 (iii) of GBM1. The CGMs 9 and 11 are involved in regulation of the levels 4 (iv) and 5 (v). (**B**) In GBM2 the CGMs 1, 3, and 4 have the most significant number of TF/TRs connections at the levels 2 (ii) and 3 (iii). CGMs 1–6 have a majority of the regulation connections on the levels 4 and 5.

Fig. S2. TGFβ controls coherent-gene modules (CGMs) of primary glioblastoma (GBM1). Each of statistically-significant CGMs represented as a heat map of its gene functions. Color represents *p*-value, and the size—number of genes involved in the function.

Fig. S3. JUN and TGFβ control coherent-gene modules (CGMs) of secondary glioblastoma (GBM2). Each of statistically-significant CGMs represented as a heat map of its gene functions. Color represents *p*-value, and the size—number of genes involved in the function.

Fig. S4. Correlation of survival by network-associated genes in SurExpress dataset. Kaplan-Meier plots depict survival analyses in patients with specific gene abnormalities. The X-axis denotes period (in months) while Y-axis indicates probability of survival (in percent). High expression in red and low expression is in green. The inset denotes gene expression (Y-axis) in both high-risk (red) and low-risk (green) patient groups for different genes (X-axis). **A.** MAPK8, **B.** SRC, **C.** CCL2, and **D.** MAPK14 expression levels (but not TGFB1, TP53, CTNNB1, FOS, MYC, JUN, RPS27A, MAPK1) were significantly related with patient survival.

Fig. S5. Correlation of survival and disease-free status by network associated genes in cBioPortal dataset. Kaplan-Meier plots depict survival analyses in patients with specific gene abnormalities. The X-axis denotes period (in months) while Y-axis indicates probability of survival (in percent). For each gene, GBM patients were classified as the up-regulated (red), no alternation (blue) and down-regulated (green), based on gene expression levels. **A.**, **C.**, **E.**, and **G.** depict overall survival in patients with abnormal expression of MAPK8, CCL2, SRC, and MAPK14 respectively. Similarly, **B.**, **D.**, **F.**, and **H.** depict progression (disease)-free survival in patients with altered expression levels of MAPK8, CCL2, SRC, and MAPK14 respectively.

Fig. S6. Correlation of molecular subtypes by the selected top level network associated genes in TCGA dataset with overall (sets "i") and progression free (sets "ii") survival**. A.** TGFB1, **B.** MAPK8, **C.** TP53, **D.** CTNNB1, **E.** SRC, **G.** MYC **in the 4** TCGA **subtypes** of GBM. The figures show **Kaplan-Meier plots of survival** in GBM patients with different **expression levels of abovementioned genes in all 4** GBM **subtypes: [*C*] –classical, [*P*] – proneural, [*M*] – mesenchymal, [*N*] - neural.** If we depict the most significant changes in survival we can note that: TGFB higher expression level in the tumor correlates with the decrease of survival in *N* and *P* subtypes and with the increase of survival in *M* subtype (Fig. S6A). MAPK8 higher expression level in the tumor correlates with the decrease of survival in *N* subtype and with the increase of survival in P subtype (Fig. S6B). TP53 higher expression level in the tumor correlates with the increase of survival in *M* and *P* subtypes and with the decrease of survival in *N* subtype (Fig. S6C). CTNNB1 higher expression level in the tumor correlates with the increase of survival in *N*, *M*, and *P* subtypes (Fig. 6D). SRC higher expression level in the tumor correlates with the decrease of survival in *N* subtype (Fig. S6E). MYC higher expression level in the tumor correlates with the decrease of survival in *N* subtype and with the increase of survival in *P* subtype (Fig. S6F).