Prognostic value and their clinical implication of 89-gene signature in glioma

Supplementary Materials



Supplementary Figure S1: Hierarchical clustering analysis of gene expression data from the training set. Genes with an expression level that had at least 2-fold difference relative to median value across 30 samples were selected for hierarchical clustering analysis (1,009 gene features). Expression profiles (red, relative high expression; green, relative low expression) between high and low risk groups in the training set.



Supplementary Figure S2: Differentially expressed genes between high and low risk groups in the training set. Genes were selected by two-sample t-test with permutation test and stringent cut-off (p < 0.001 and 2.5-fold difference) was applied to retain genes whose expression was significantly between the two groups (129 genes). Expression profiles (red, relative high expression; green, relative low expression) between high and low risk groups in the training set.



Supplementary Figure S3: Kaplan-Meier survival analysis of histology grades in the training set. Patients showed a significant difference according to the histology grades in the training set. The *p* values were computed by the log-rank test.



Supplementary Figure S4: Prognostic significance of the 89-gene signature in RNA-seq TCGA data set. Kaplan-Meier survival plots of overall survival (OS) of the two groups in the RNA-seq data. The *p* values were computed by the log-rank test.



Supplementary Figure S5: Kaplan-Meier survival analysis of age in the training and validation data sets. (A–B) Patients under and over 40 years of age groups in the training data set. **(C–D)** Patients under and over 40 years of age groups in the validation data sets. Each group was classified by the 89-gene signature into high and low risk groups. The *p* values were computed by the log-rank test.



Supplementary Figure S6: Kaplan-Meier survival analysis of grades III and IV in the training and validation data sets. (A–B) Patients in grades III and IV in the training data set. (C–D) Patients in grades III and IV in the validation data sets. Each group was classified by the 89-gene signature into high and low risk groups. The *p* values were computed by the log-rank test.



Supplementary Figure S7: Kaplan-Meier survival analysis of adjuvant chemotherapy and radiation therapy in the training by EUMC and validation by TCGA data sets. (A–B) Patients with radiotherapy in the EUMC and TCGA data sets. (C–D) Patients with chemotherapy in the EUMC and TCGA data sets. (E–F) Patients with combined therapies in the EUMC and TCGA data sets. Each group was stratified according to chemotherapy, radiotherapy, and combined therapies. The *p* values were computed by the log-rank test.



Supplementary Figure S8: Protein interaction network analysis in the 89-gene signature. Interaction map was generated using the STRING database.

Supplementary Figure S9: NF-KB, AP-1, and STAT3 networks from Ingenuity pathway analysis. Gene networks from IPA showed upstream genes significantly associated with NF-KB, AP-1, and STAT3 pathways.

Supplementary Table S1: Annotation of the 89-gene signature. See Supplementary_Table_S1 and S2

Supplementary Table S2: Gene ontology (GO) analysis of statistically significant genes in the 89-gene signature in glioma. See Supplementary_Table_S1 and S2