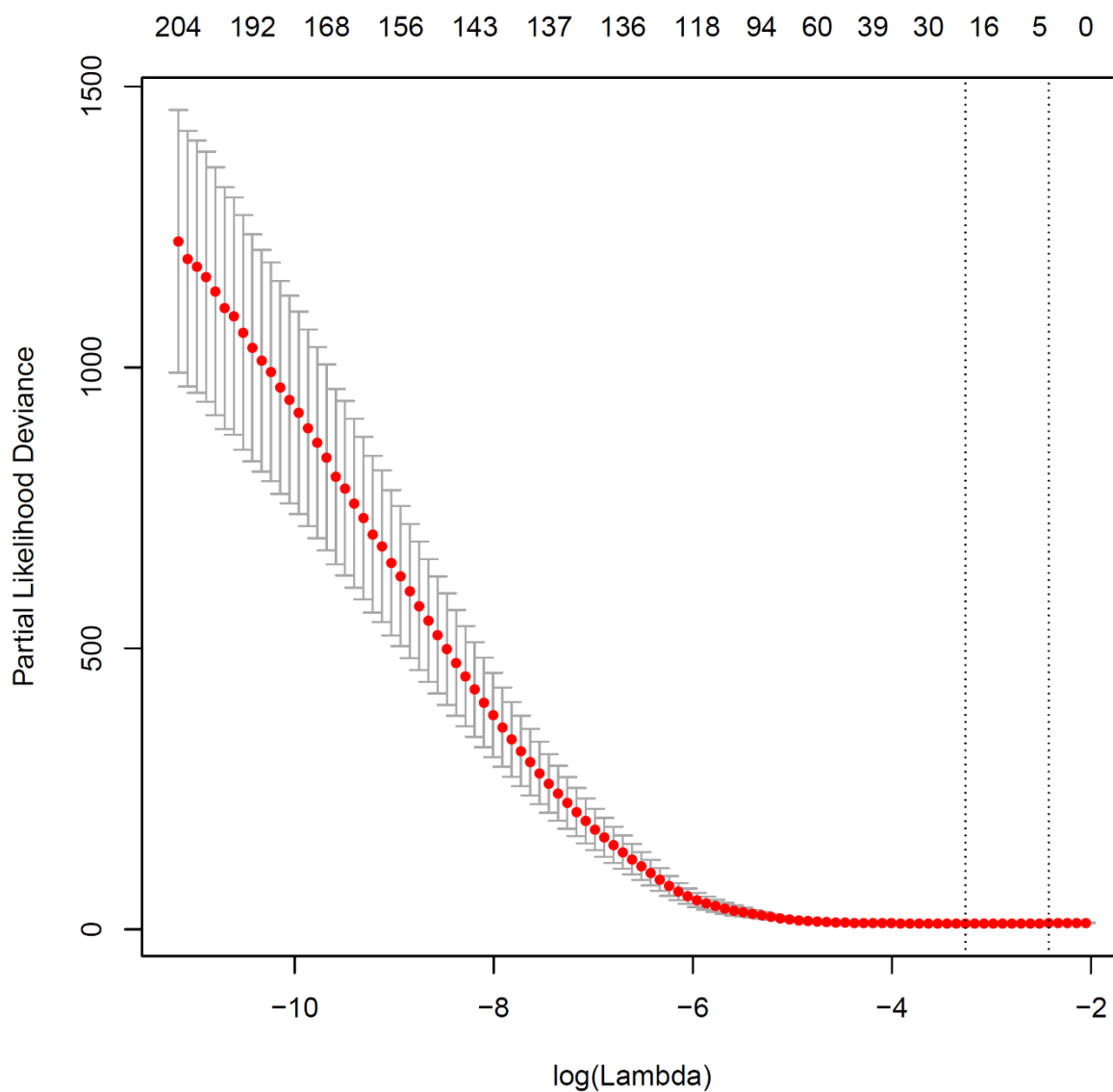


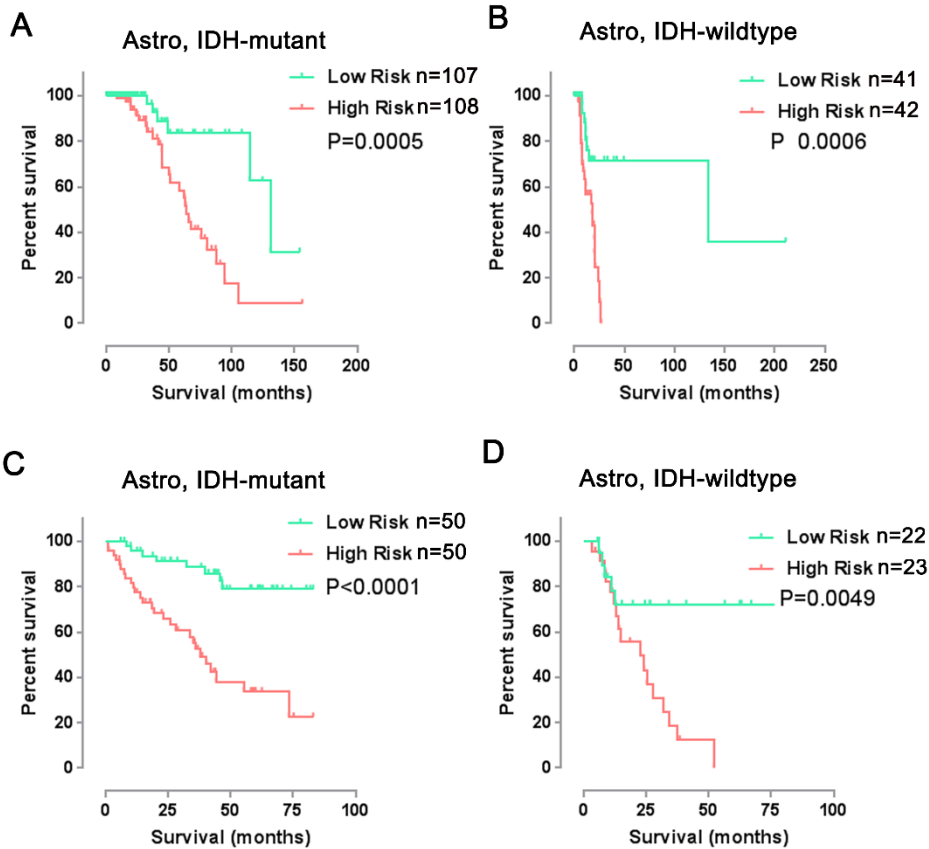
1 **Supplementary Figures:**



2

3 **Supplementary Figure 1.** Ten-fold cross validation for tuning parameter selection in the LASSO  
4 model. LASSO coefficient profiles of the 1203 1p19q genes. The minimum criteria are indicated  
5 by the dashed vertical line (left).

6



1

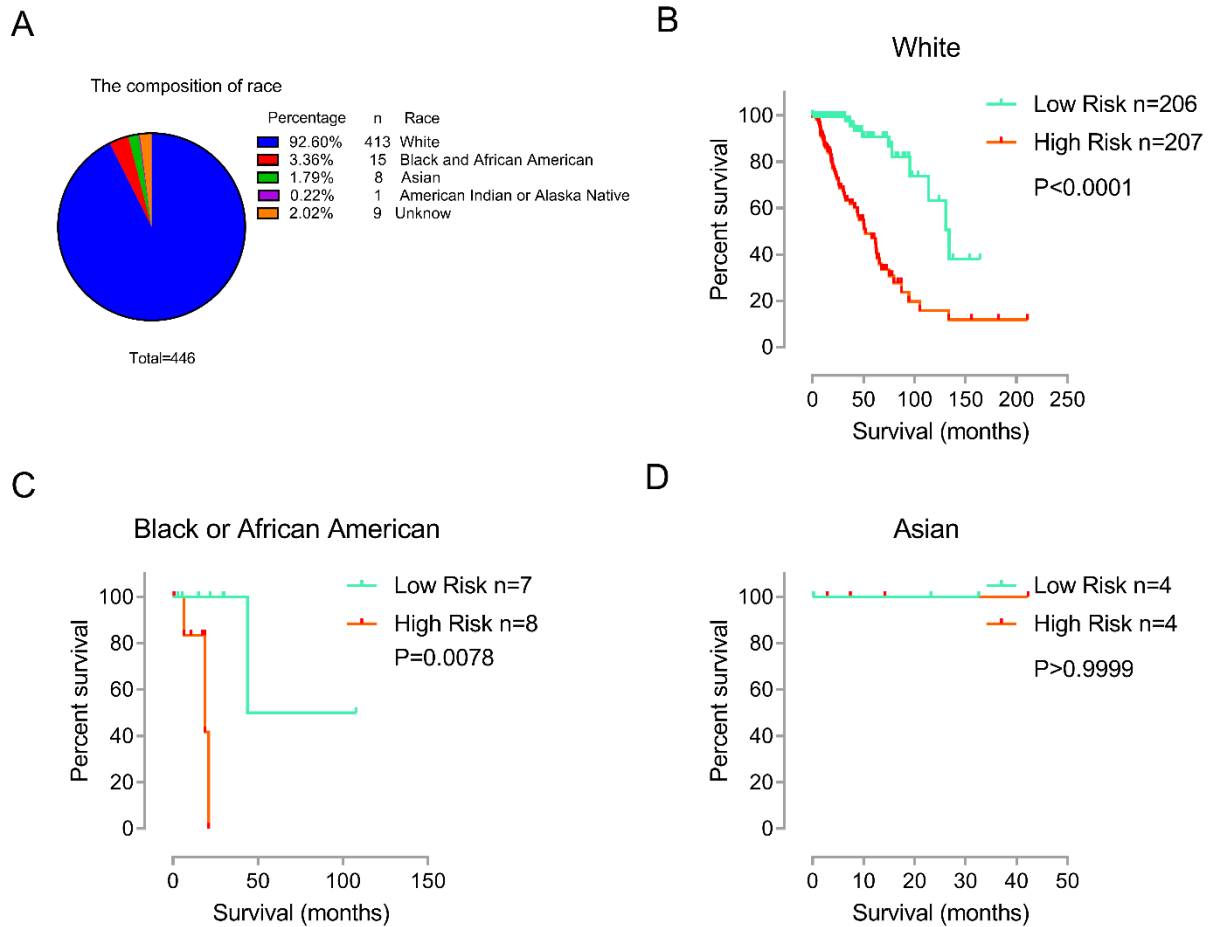
2 **Supplementary Figure 2.** Prognostic value of the risk signature in astrocytoma. Kaplan–Meier

3 overall survival (OS) curves for patients from TCGA (A-B) and CGGA (C-D) datasets with

4 astrocytoma with *IDH*-mutant or *IDH*-wildtype. The P value and sample numbers (n) are labeled

5 on the figure.

6



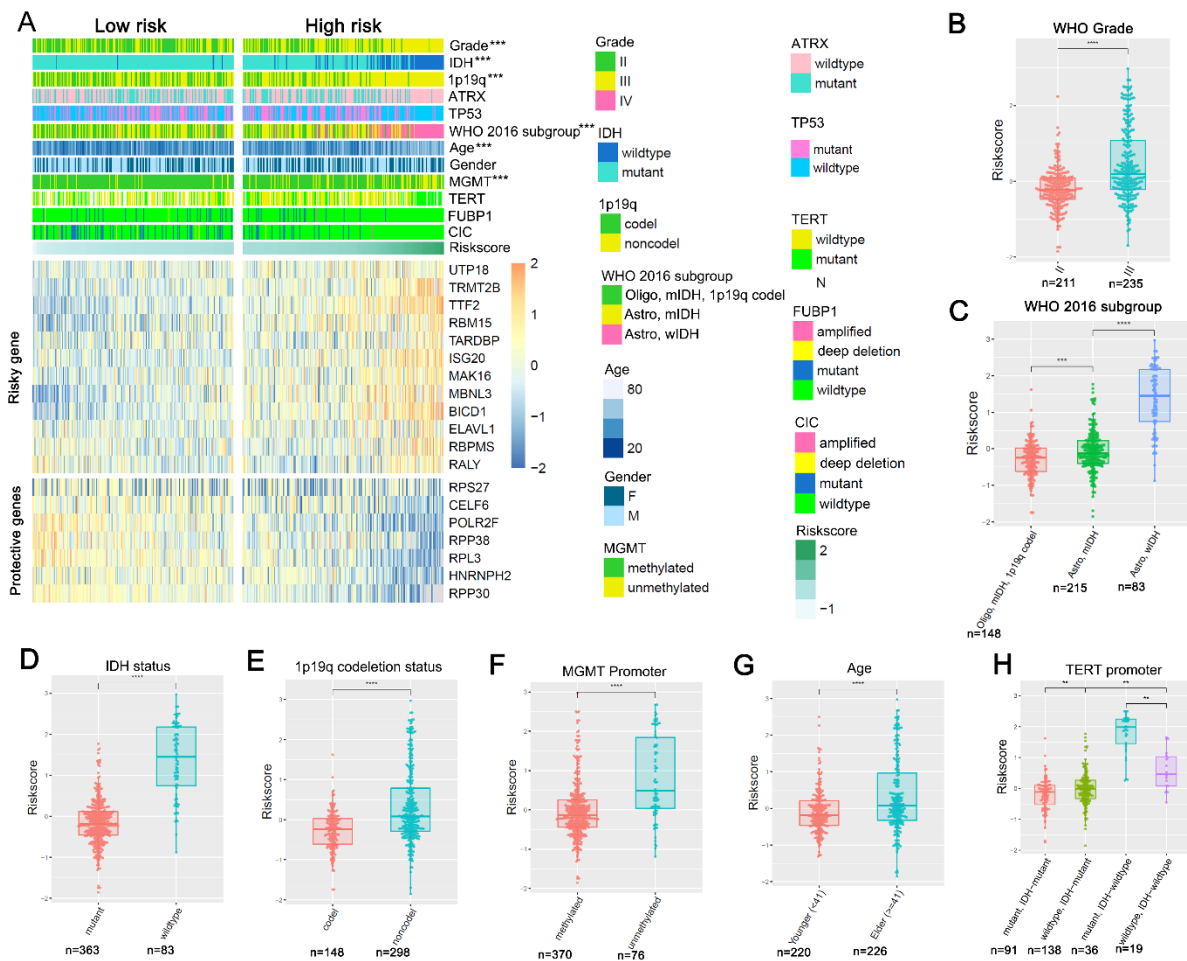
1

2 **Supplementary Figure 3.** Prognostic value of the risk signature in different races of TCGA dataset.

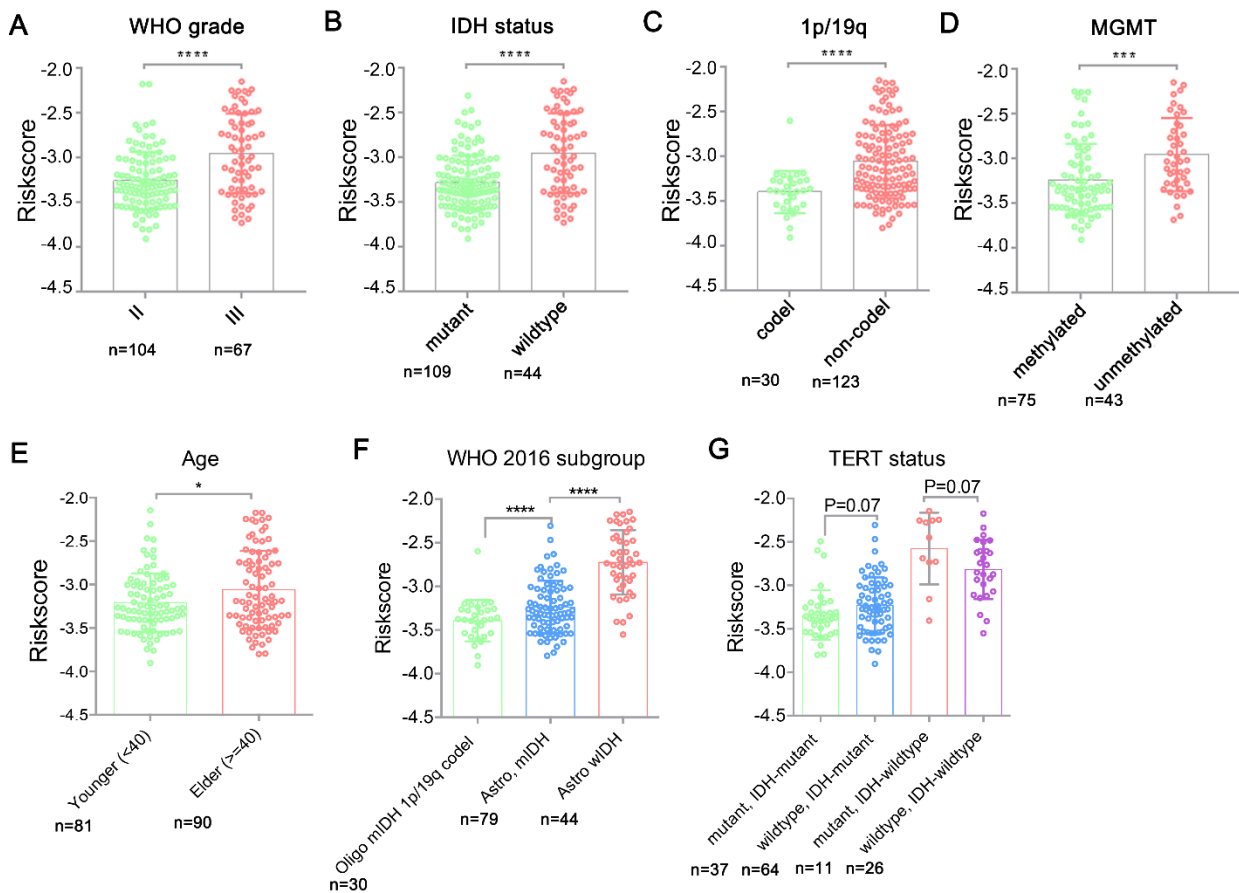
3 (A) The composition of races in TCGA dataset. (B-D) Kaplan–Meier overall survival (OS) curves  
4 for White (B), Black or African American (C), and Asian population in TCGA (D), respectively.

5 The P value and sample numbers (n) are labeled on the figure.

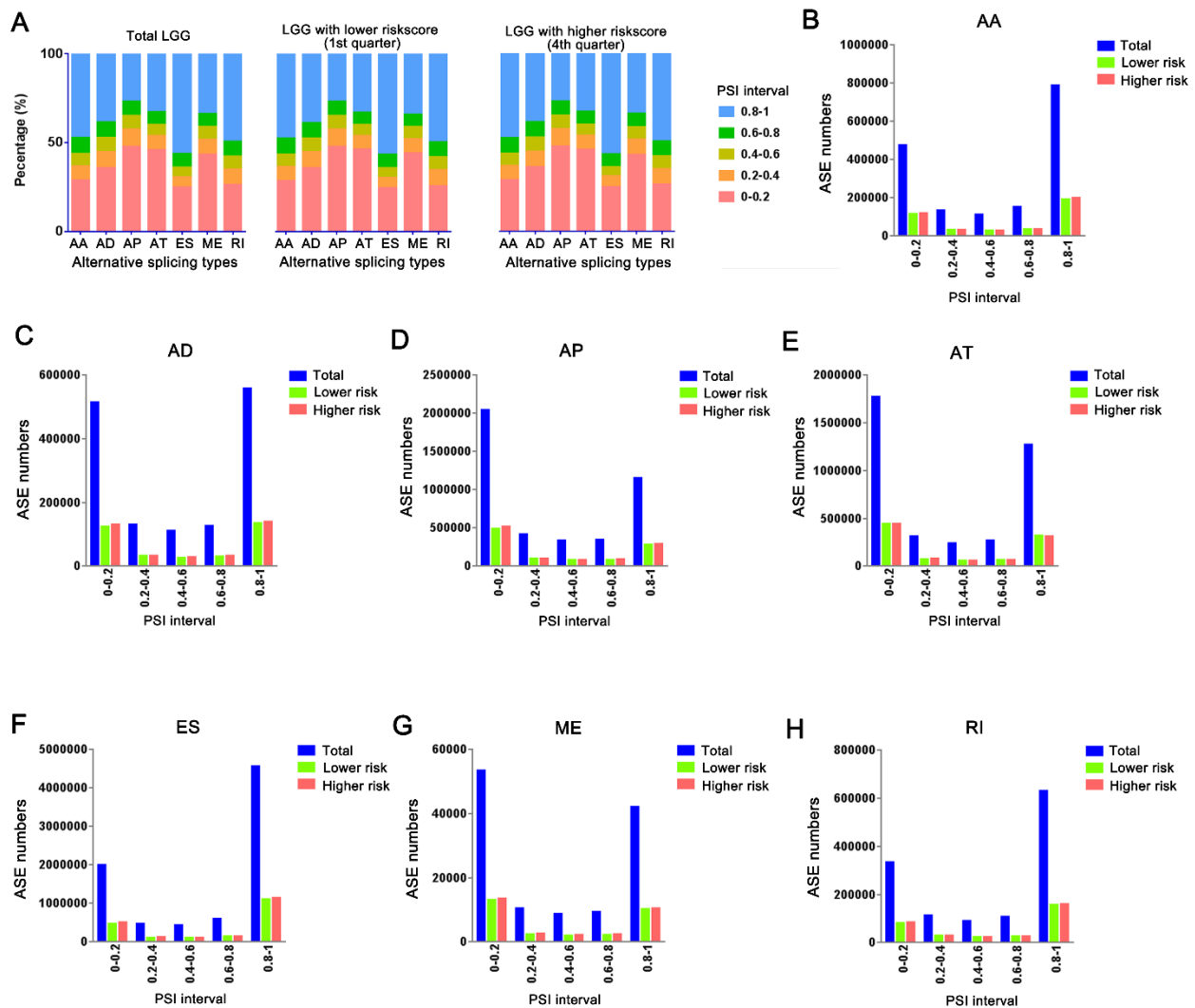
6



**Supplementary Figure 4.** Distribution of risk scores in patients stratified by clinicopathological and gene expression characteristics. (A) Heatmap showing the expression level of the 19 signature genes in low-risk and high-risk LGGs. Expression levels are indicated by the color bar to the right ranging from blue (no/low) to red (high) expression. The distribution of clinicopathological features was also compared between the low- and high-risk groups. (B–H) Distribution of risk scores for patients in TCGA dataset stratified by WHO grade (B) WHO 2016 subgroup (C), *IDH* status (D), 1p/19q codeletion status (E), *MGMT* promoter methylation status (F), age (G), and TERT promoter (H). \*\* $P < 0.01$ , \*\*\* $P < 0.001$ , and \*\*\*\* $P < 0.0001$ .

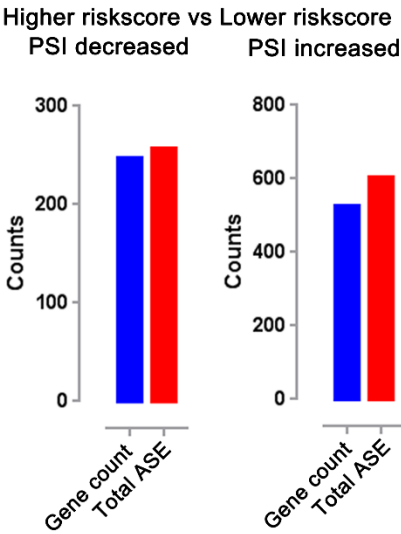


**Supplementary Figure 5.** Distribution of risk score in LGGs stratified by different pathological features in CGGA dataset. (A–G) Distribution of risk scores for patients in the CGGA dataset stratified by WHO grade (A), *IDH* status (B), 1p/19q codeletion status (C), *MGMT* promoter methylation status (D), age (E), WHO 2016 subgroup (F), and TERT promoter (G). \* $P < 0.05$ , \*\* $P < 0.01$ , \*\*\* $P < 0.001$ , and \*\*\*\* $P < 0.0001$ .

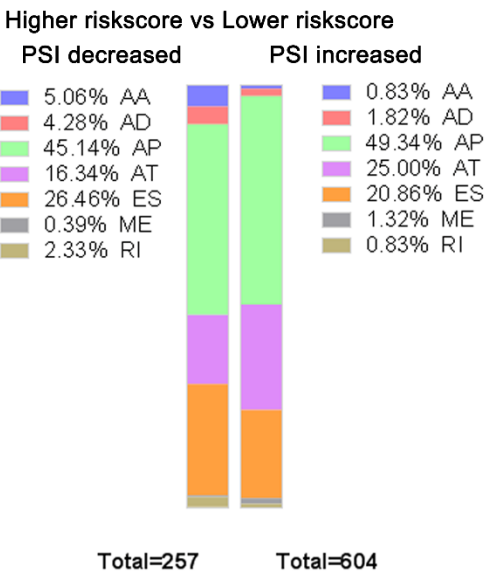


**Supplementary Figure 6.** Distribution of percent spliced in (PSI) levels for AEs in LGGs. (A) Fraction of events of distinct PSI levels in total LGGs, LGGs with lower and higher risk score. (B–H) Numbers of events of distinct PSI in total LGGs ( $n = 446$ ), LGGs with lower ( $n = 111$ ), and higher ( $n = 111$ ) risk score. AA: alternate acceptor site; AD: alternate donor site; AP: alternate promoter; AT: alternate terminator; ES: exon skip; ME: mutually exclusive exons; RI: retained intron.

A



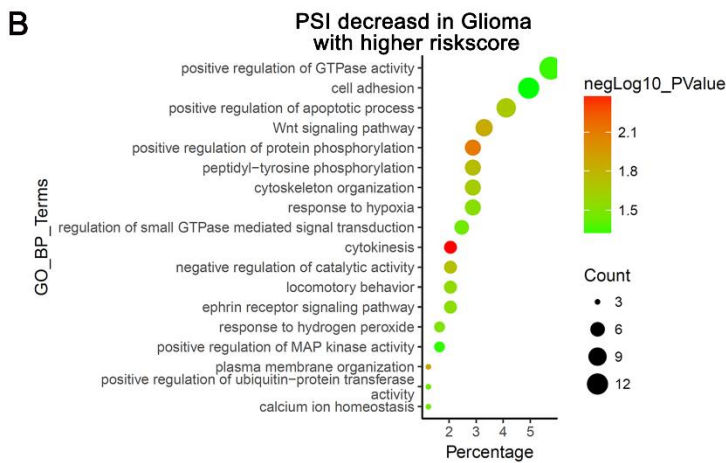
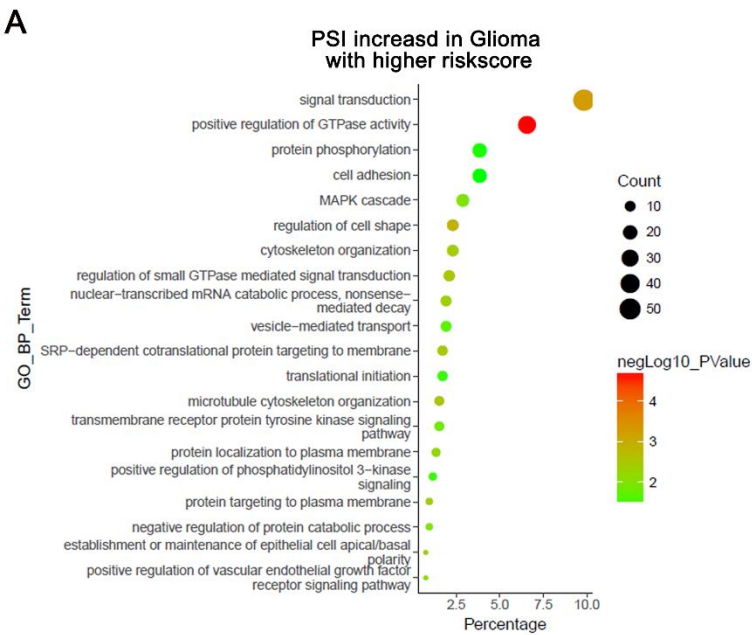
B



1

2 **Supplementary Figure 7.** General information for ASE with differential PSI between LGG with  
3 lower and higher risk scores. (A–B) Total number of changed spliced genes and ASEs (A) and the  
4 fraction of changed ASE types (B) between LGG groups with lower and higher risk scores are  
5 summarized.

6



1

2 **Supplementary Figure 8.** Functions of spliced genes with differentially PSI between gliomas

3 with lower (n=111) and higher (n=111) risk scores. (A–B) functional analysis of spliced genes with

4 decreased PSI (A) or increased PSI (B) in LGGs with higher risk score.