Supplementary Materials

Figure S1

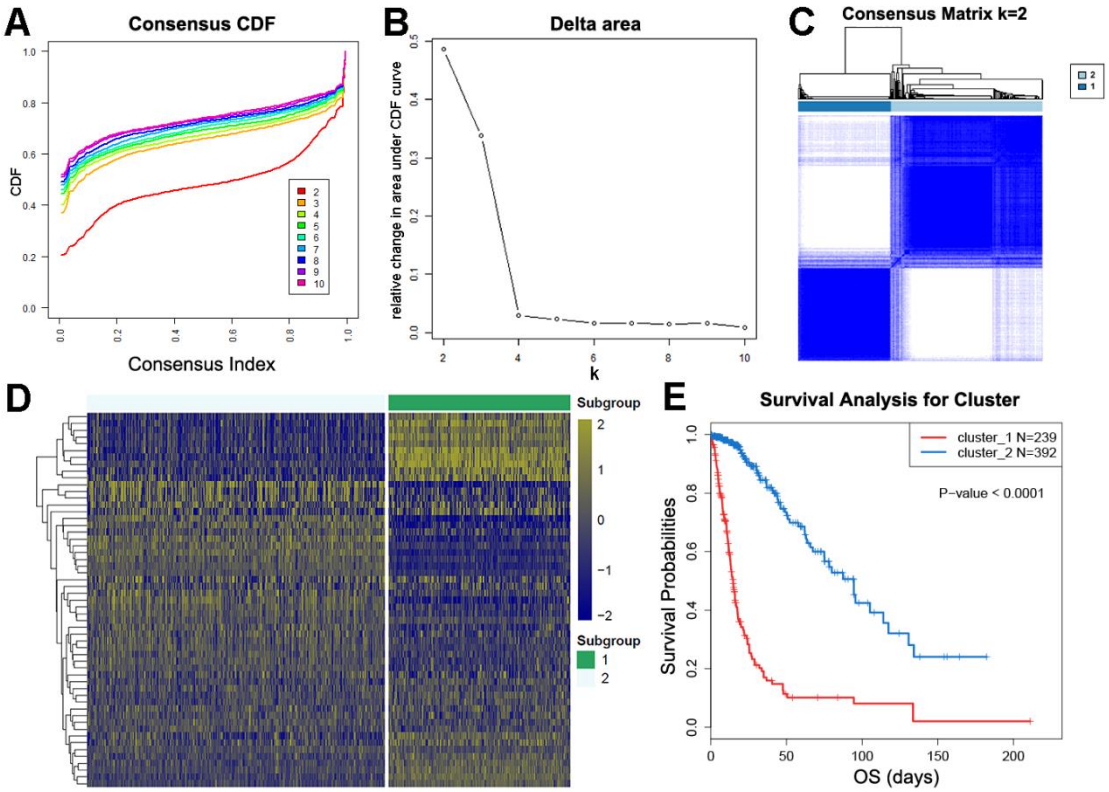

Figure S1. Consensus clustering for ATP metabolism-related genes in glioma patients from TCGA dataset. (A) Consensus clustering CDF for k = 2 to k = 10. (B) Relative change in area under CDF curve for k = 2 to k = 10. (C) Consensus clustering matrix of 631 samples from TCGA dataset for k = 2. (D) Heat map of two clusters defined by the top 55 variable expression genes. (E) K-M survival curve of patients in two clusters.

Figure S2

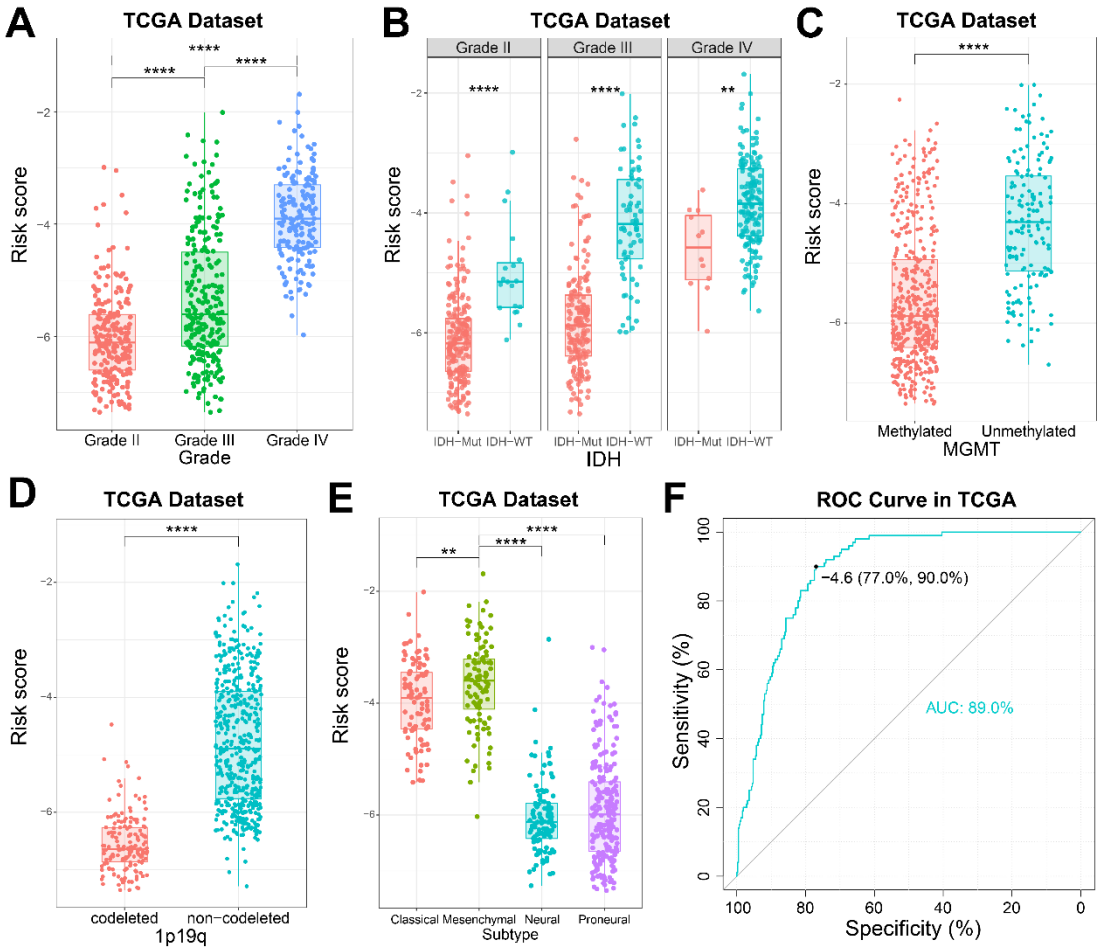

Figure S2. Association between pathologic characteristics and the ATP metabolism-related signature in TCGA dataset. (A-E) Distribution of the risk score in stratified patients by WHO grade, TCGA molecular subtype, IDH mutation status, MGMT promoter methylation, 1p/19q co-deletion status and TCGA molecular subtypes. (F) ROC curves predicted ATP metabolism as a biomarker of mesenchymal subtype glioma. ** p <0.01, **** p <0.0001.

Figure S3

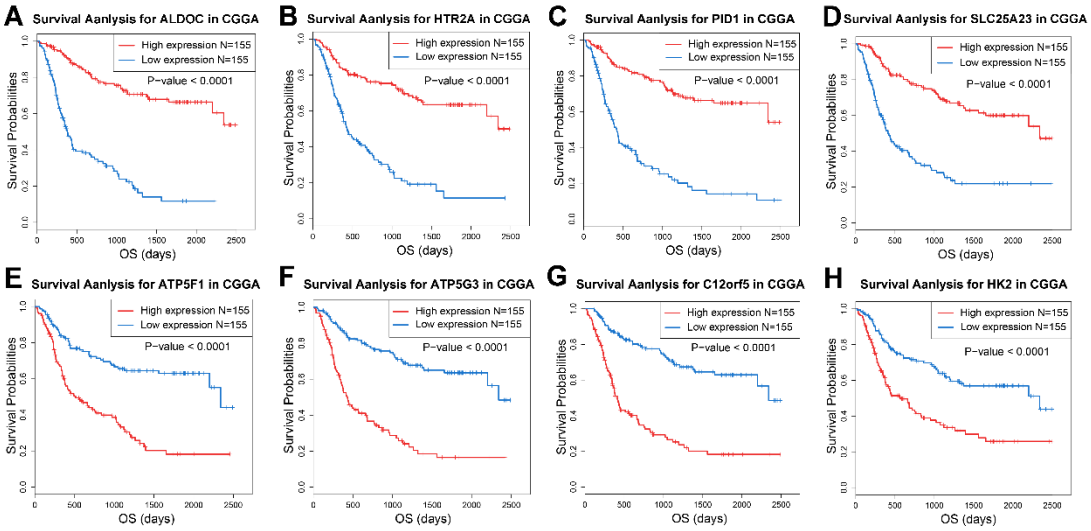

Figure S3. Kaplan-Meier survival curves of OS based on the expression of each independent gene in the ATP metabolism-related signature in CGGA dataset.

Figure S4

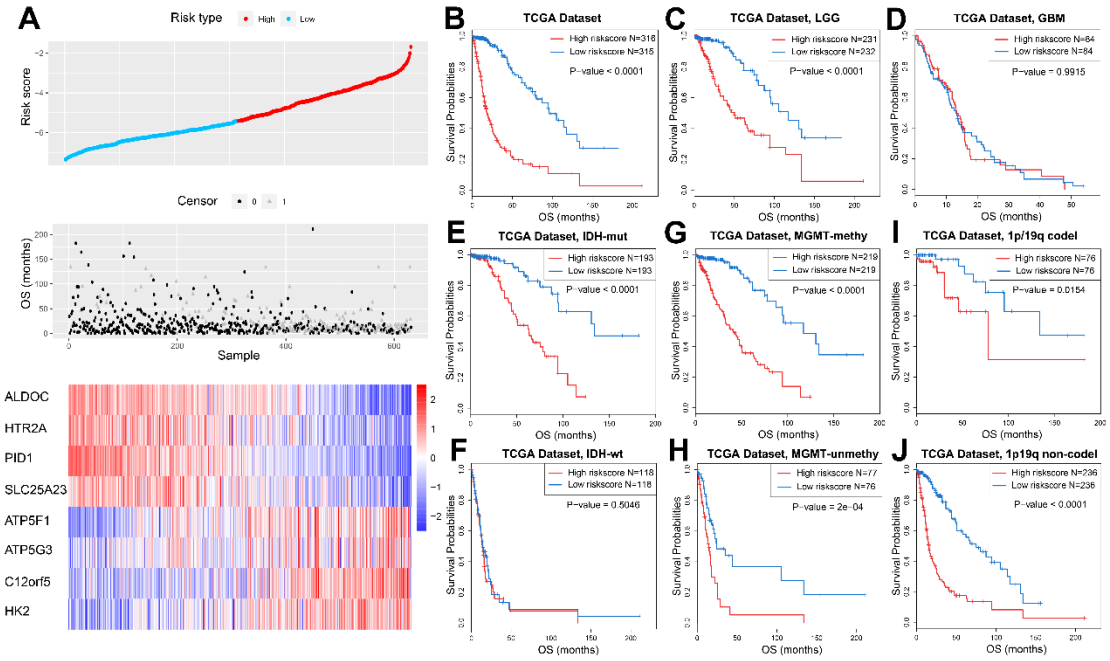

Figure S4. The prognostic value of the risk signature in TCGA dataset. (A) Distribution of the risk score, OS and expression level of 8 genes in the risk signature. (B-J) K-M survival analysis of the risk signature in glioma patients stratified by WHO grade, IDH mutation status, MGMT promoter methylation and 1p/19q co-deletion status.

Figure S5

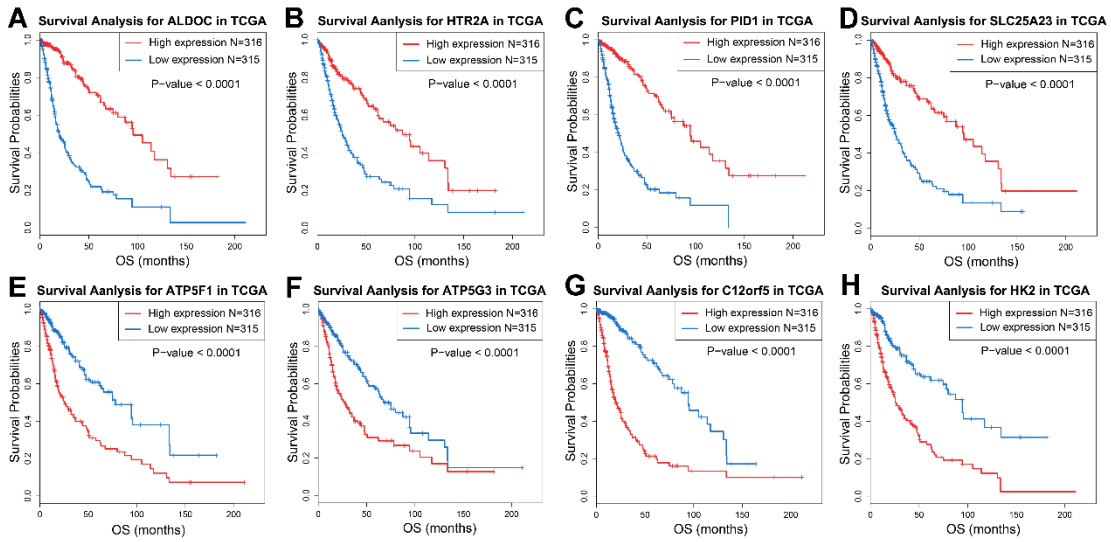

Figure S5. Kaplan-Meier survival curves of OS based on the expression of each independent gene in the ATP metabolism-related signature in TCGA dataset.

Figure S6

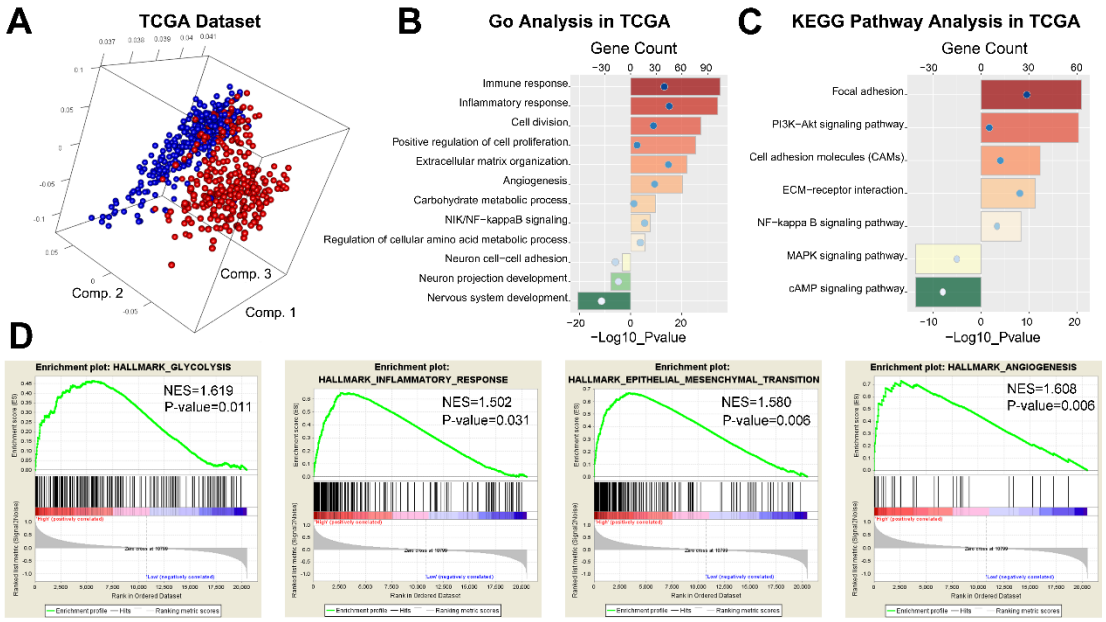

Figure S6. Functional analysis of the ATP metabolism-related signature in TCGA dataset. (A) Principal components analysis of whole gene expression data between high-risk and low-risk group. (B, C) GO and KEGG pathway analyses were performed via the DAVID website to explore the functional annotation of the risk signature. (D) GSEA analysis was used to explore the biological functions and pathways that were significantly enriched in patients with high risk score.

Figure S7

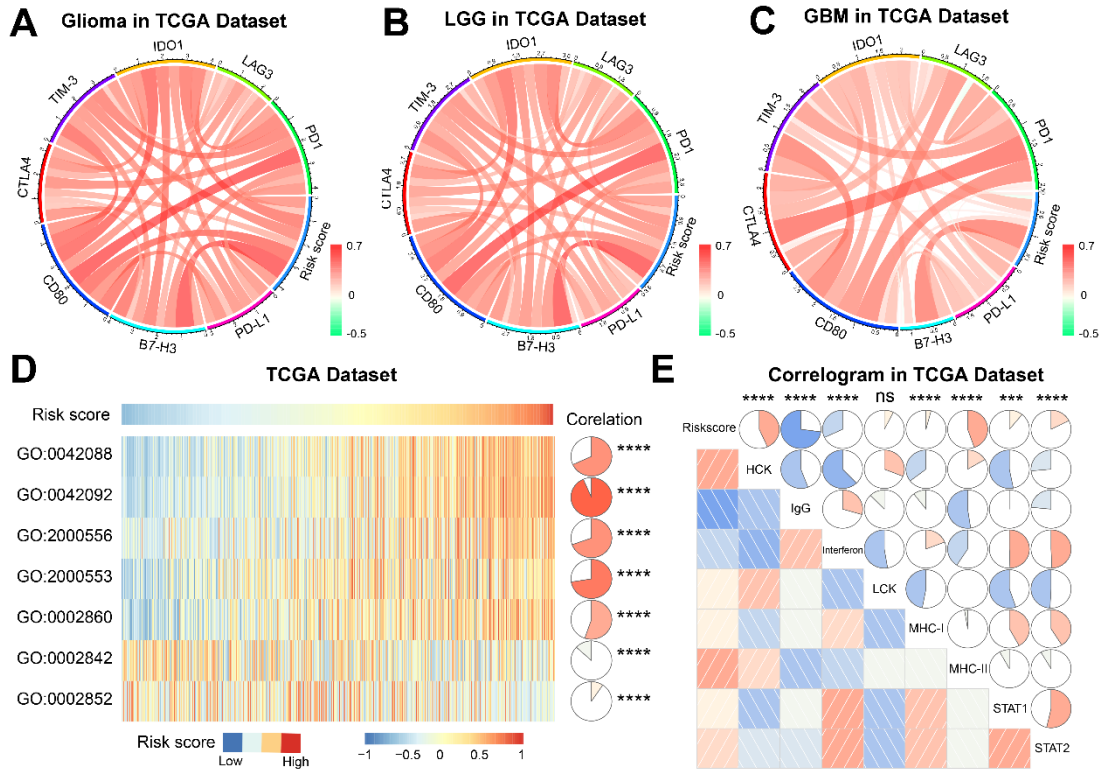

Figure S7. The 8-gene signature related immune responses and inflammatory activities in gliomas in TCGA dataset. (A-C) Correlation analysis between immune checkpoint members and risk signature in whole gliomas, low-grade gliomas and GBM, respectively. (D) The relationship between the risk signature and T cell related immunity. GO:0042088: T-helper 1 type immune response, GO:0042092: T-helper 2 type immune response, GO:2000556: positive regulation of T-helper 1 cell cytokine production, GO:2000553: positive regulation of T-helper 2 cell cytokine production, GO:0002860: positive regulation of natural killer cell mediated cytotoxicity directed against tumor cell target, GO:0002842: positive regulation of T cell mediated immune response to tumor cell, GO:0002852: regulation of T cell mediated cytotoxicity directed against tumor cell target. (E) The relationship between CMTM6 and inflammatory activities. *** p <0.001, **** p <0.0001, ns: no statistically significant.

Figure S8

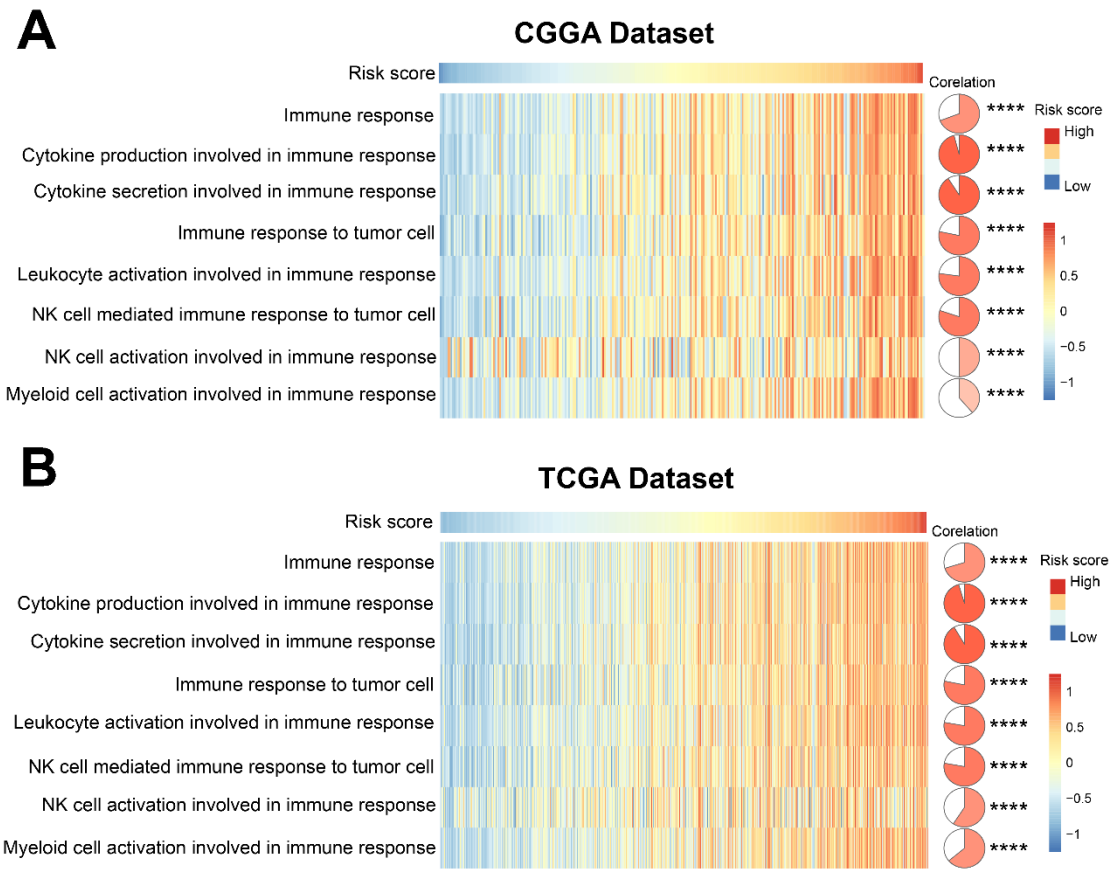

Figure S8. Association between the risk signature and immune response mediated by various immune cells in CGGA dataset (A) and TCGA dataset (B). **** p <0.0001.

Figure S9

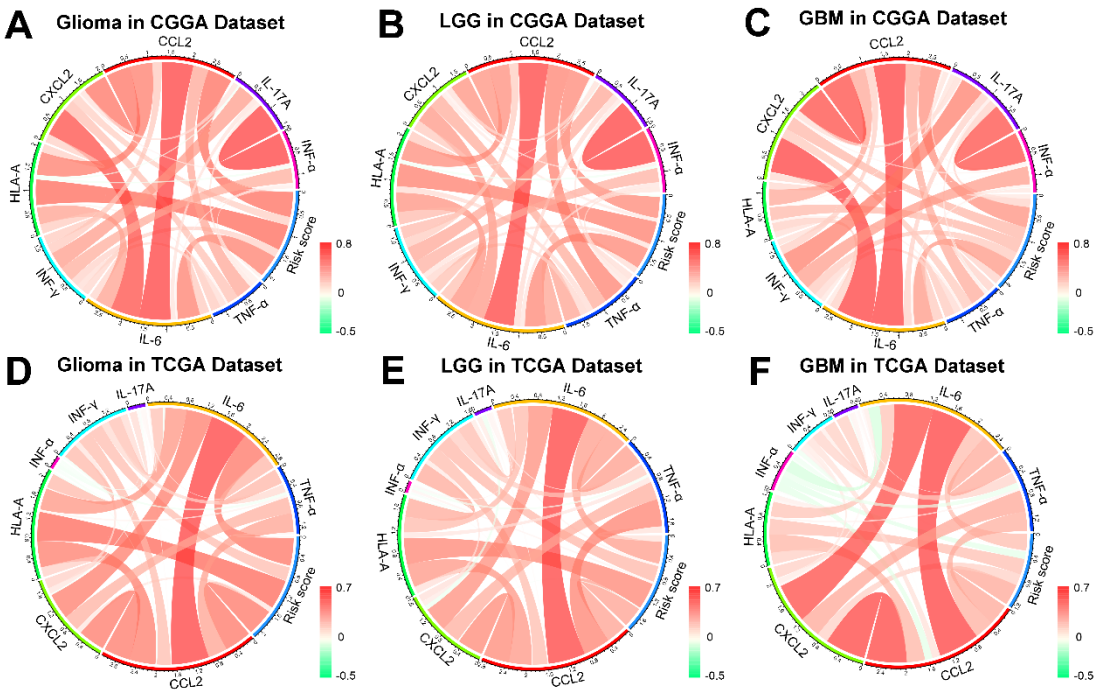

Figure S9. Correlation analysis between inflammatory genes and risk signature in whole gliomas, low-grade gliomas and GBM in CGGA dataset (A-C) and TCGA dataset (D-F).

Table S1

ATP metabolism related gene list				
ALDOC	DDIT4	INSR	ATP5G1	C11orf83
PID1	ENO2	PGAM4	IGF1	ENO4
HDAC4	NCOR1	ATP6V0A4	ENO3	ATP5H
SLC25A23	ATP5A1	PRKAA2	TPI1	PFKFB4
DHTKD1	ATP5O	GPD1	GADD45GIP1	GPI
HTR2A	ALDOB	APOC3	GALK1	ENO1
PFKM	ATP5G2	CHCHD10	MYOG	PGAM2
PPIF	PRKAG2	ENTPD5	PARK7	VCP
ATP5S	HKDC1	ALDOA	ATP5L	ATP5E
LDHA	OGT	PGK2	OGDH	STAT3
PPARGC1A	ATP5B	DNAJC15	BPGM	ATP5F1
PINK1	ATP5D	ATP5J	PFKL	FBP1
ATP6V0A1	SURF1	HK1	ATPIF1	GAPDH
PPARA	PFKFB2	ATP5L2	ATP5EP2	TSPO
P2RX7	PFKFB1	ATP5I	PRKAA1	PGK1
OGDHL	PRKAG1	LDHC	SIRT6	ATP5G3
PGAM1	ACTN3	C1orf177	DNM1L	HK2
SNCA	CBFA2T3	GCK	ATP6V0A2	TGFB1
SLC25A33	SLC25A13	GAPDHS	FLCN	ADPGK
PFKP	AKD1	PFKFB3	PGM1	C12orf5
ECD	ATP5C1	ARNT	ATP7A	TCIRG1
GBAS	PKLR	INS	HIF1A	HK3

Table S2. Characteristics of patients in cluster 1 and cluster 2 in TCGA database

Characteristic	N	Cluster 1	Cluster 2	p value
Total cases	631	239	392	
Gender				0.6031
Male	364	141	223	
Female	267	98	169	
Age (years)				<0.0001
≤40	244	31	213	
> 40	387	208	179	
Grade				<0.0001
II	221	10	211	
III	242	64	178	
IV	168	165	3	
Subtype				<0.0001
Classical	89	86	3	
Mesenchymal	100	92	8	
Proneural	226	24	202	
Neural	104	8	96	
IDH Status				<0.0001
Mutation	386	20	366	
Wildtype	236	212	24	
MGMT Promoter				<0.0001
Methylation	438	88	350	
Unmethylation	154	113	41	
1p19q				<0.0001
Codel	152	2	150	
Intact	472	231	241	

Table S3. Univariate and multivariate analysis of OS in TCGA sequencing database

Variables	Univariate analysis		Multivariate analysis	
	HR (95% CI)	p value	HR (95% CI)	p value
Risk score	1.845 (1.670-2.041)	< 0.001	1.313 (1.062-1.624)	0.012
Age at Diagnosis	1.073 (1.061-1.084)	< 0.001	1.064 (1.047-1.080)	< 0.001
Gender	1.111 (0.840-1.469)	0.462	-	-
WHO Grade	4.794 (3.798-6.051)	< 0.001	1.803 (1.266-2.567)	0.001
TCGA Subtype	2.004 (1.765-2.275)	< 0.001	1.074 (0.860-1.342)	0.527
IDH mutation status	0.117 (0.085-0.161)	< 0.001	0.948 (0.474-1.899)	0.881
MGMT methylation	0.337 (0.247-0.461)	< 0.001	0.735 (0.487-1.108)	0.141
1p19q co-deletion	0.228 (0.139-0.376)	< 0.001	0.590 (0.318-1.095)	0.094