Supplementary Table 1 The detailed clinical features of glioma patients in TCGA RNAseq

Characteristics	Total
Age	
≥60	158(22.87%)
＜60	533(77.13%)
Gender	
Female	296(42.84%)
Male	395(57.16%)
Race	
American indian or alaska native	1(0.14%)
Asian	13(1.88%)
Black or african american	33(4.78%)
Not reported	12(1.74%)
White	632(91.46%)
WHO grade	
G2	257(37.19%)
G3	266(38.49%)
G4	167(24.17%)
Not reported	1(0.14%)
Sample type	
Primary Tumor	660(95.51%)
Recurrent Tumor	31(4.49%)
Radiation therapy	
NO	194(28.08%)
YES	421(60.93%)
Not reported	76(11.00%)
IDH mutation status	
mutation	407(58.90%)
not reported	17(2.46%)
wild-type	267(38.64%)

Supplementary Table 2 The detailed clinical features of glioma patients in CGGA mRNAseq693

Characteristics	Total
Sample type	
Primary Tumor	404(61.49%)
Recurrent Tumor	253(38.51%)
Grade	
WHO II	172(26.18%)
WHO III	248(37.75%)
WHO IV	237(36.07%)
Gender	
Female	283(43.07%)
Male	374(56.93%)
Age	
≥60	80(12.18%)
＜60	576(87.67%)
Not reported	1(0.15%)
Radio status	
Untreated	131(19.94%)
Treated	501(76.26%)
Not reported	25(3.81%)
Chemo status	
Untreated	156(23.74%)
Treated	480(73.06%)
Not reported	21(3.20%)
IDH mutation status	
Mutant	333(50.68%)
Wildtype	276(42.01%)
Not reported	48(7.31%)
1p19q codeletion status	
Codel	137(20.85%)
Non-codel	454(69.10%)
Not reported	66(10.05%)
MGMTp methylation status	
Methylated	304(46.27%)
Un-methylated	218(33.18%)
Not reported	135(20.55%)

Supplementary Table 3 The detailed clinical features of glioma patients in CGGA mRNAseq325

Characteristics	Total
Sample type	
Primary Tumor	222(70.93%)
Recurrent Tumor	58(18.53%)
Secondary Tumor[1]	29(9.27%)
Not reported	4(1.28%)
Grade	
WHO II	98(31.31%)
WHO III	74(23.64%)
WHO IV	137(43.77%)
Not reported	4(1.28%)
Gender	
Female	116(37.06%)
Male	197(62.94%)
Age	
≥60	33(10.54%)
＜60	280(89.46%)
Radio status	
Untreated	62(19.81%)
Treated	241(77.00%)
Not reported	10(3.19%)
Chemo status	
Untreated	110(35.14%)
Treated	190(60.70%)
Not reported	13(4.15%)
IDH mutation status	
Mutant	167(53.35%)
Wildtype	145(46.33%)
Not reported	1(0.32%)
1p19q codeletion status	
Codel	62(19.81%)
Non-codel	243(77.64%)
Not reported	8(2.56%)
MGMTp methylation status	
Methylated	152(48.56%)
Un-methylated	143(45.69%)
Not reported	18(5.75%)

1. A secondary tumor is a cancer that has spread or metastasized to another part of the body from where it first began. In gliomas, a secondary tumor generally refers to a glioma that develops again in patient but in a different location from where the tumor was when it was first treated.

Supplementary Table 4 The detailed clinical features of glioma patients in CGGA mRNAarray301

Characteristics	Total
Sample type	
Primary Tumor	264(87.71%)
Recurrent Tumor	23(7.64%)
Secondary Tumor[1]	11(3.65%)
Not reported	3(1.00%)
Grade	
WHO II	117(38.87%)
WHO III	57(18.94%)
WHO IV	124(41.20%)
Not reported	3(1.00%)
Gender	
Female	121(40.20%)
Male	180(59.80%)
Age	
≥60	29(10.18%)
＜60	254(89.12%)
Radio status	
NO	46(15.28%)
YES	237(78.74%)
Not reported	18(5.98%)
Chemo status	
NO	144(47.84%)
YES	133(44.19%)
Not reported	24(7.97%)
IDH mutation status	
Mutant	134(44.52%)
Wildtype	165(54.82%)
Not reported	2(0.66%)
1p19q Codeletion status	
Codel	16(5.32%)
Non-codel	76(25.25%)
Not reported	209(69.44%)
MGMTp methylation status	
Methylated	99(32.89%)
Not reported	15(4.98%)
Un-methylated	187(62.13%)

1. A secondary tumor is a cancer that has spread or metastasized to another part of the body from where it first began. In gliomas, a secondary tumor generally refers to a glioma that develops again in patient but in a different location from where the tumor was when it was first treated.

Supplementary Table 5 GO and KEGG pathway analysis of up-regulated genes in cluster1 relative to cluster2

Category	Description	Gene Ratio	P value
Biological process	Immune effector process	112/479	9.67E-30
	External encapsulating structure organization	60/479	2.58E-28
	Defense response	131/479	6.44E-28
	Cell activation	116/479	1.50E-27
	Response to cytokine	102/479	1.01E-26
Cellular component	Collagen containing extracellular matrix	79/415	1.34E-41
	External encapsulating structure	90/415	1.40E-41
	Secretory granule	76/415	8.06E-19
	Endoplasmic reticulum lumen	43/415	7.16E-18
	MHC class II protein complex	12/415	5.72E-16
Molecular function	Extracellular matrix structural constituent	37/448	1.57E-22
	Signaling receptor binding	102/448	8.23E-15
	Integrin binding	25/448	2.31E-13
	Growth factor binding	23/448	5.96E-12
	Collagen binding	16/448	5.26E-11
KEGG pathways	ECM-receptor interaction	19/263	4.85E-11
	Cell adhesion molecules (CAMs)	18/263	1.58E-06
	Th1 and Th2 cell differentiation	12/263	4.80E-05
	NOD-like receptor signaling pathway	17/263	0.000102182
	Th17 cell differentiation	12/263	0.000210563

Supplementary Table 6 GO and KEGG pathway analysis of down-regulated genes in cluster1 relative to cluster2

Category	Description	Gene Ratio	P value
Biological process	Synaptic signaling	127/546	1.34E-62
	Cell cell signaling	168/546	5.70E-47
	Regulation of trans synaptic signaling	81/546	3.98E-43
	Neurogenesis	156/546	4.02E-41
	Neuron differentiation	137/546	1.07E-37
Cellular component	Synapse	205/480	4.73E-90
	Neuron projection	179/480	5.79E-65
	Postsynapse	112/480	1.59E-52
	Somatodendritic compartment	124/480	7.16E-48
	Synaptic membrane	81/480	2.18E-43
Molecular function	Gated channel activity	49/491	7.48E-17
	Passive transmembrane transporter activity	55/491	9.65E-15
	Cation transmembrane transporter activity	62/491	1.14E-13
	Metal ion transmembrane transporter activity	49/491	2.38E-13
	Cation channel activity	43/491	2.38E-13
KEGG pathways	cAMP signaling pathway	26/231	6.03E-10
	Calcium signaling pathway	18/231	1.24E-05
	Wnt signaling pathway	10/231	0.018534501
	Alanine, aspartate and glutamate metabolism	4/231	0.02004155
	Regulation of lipolysis in adipocytes	5/231	0.021590081