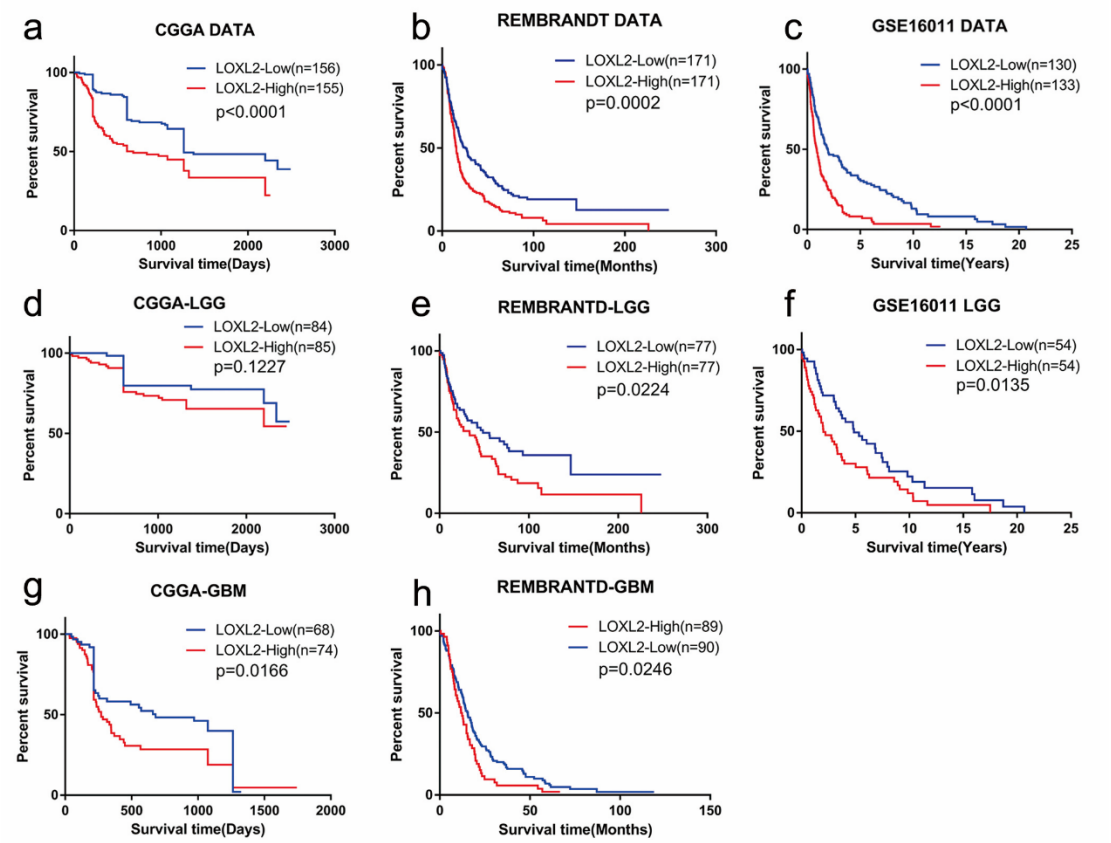
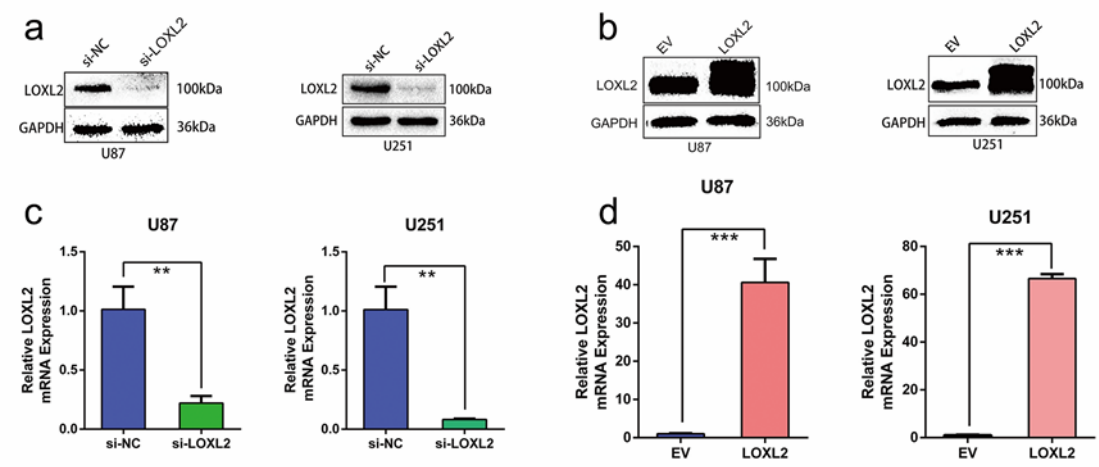
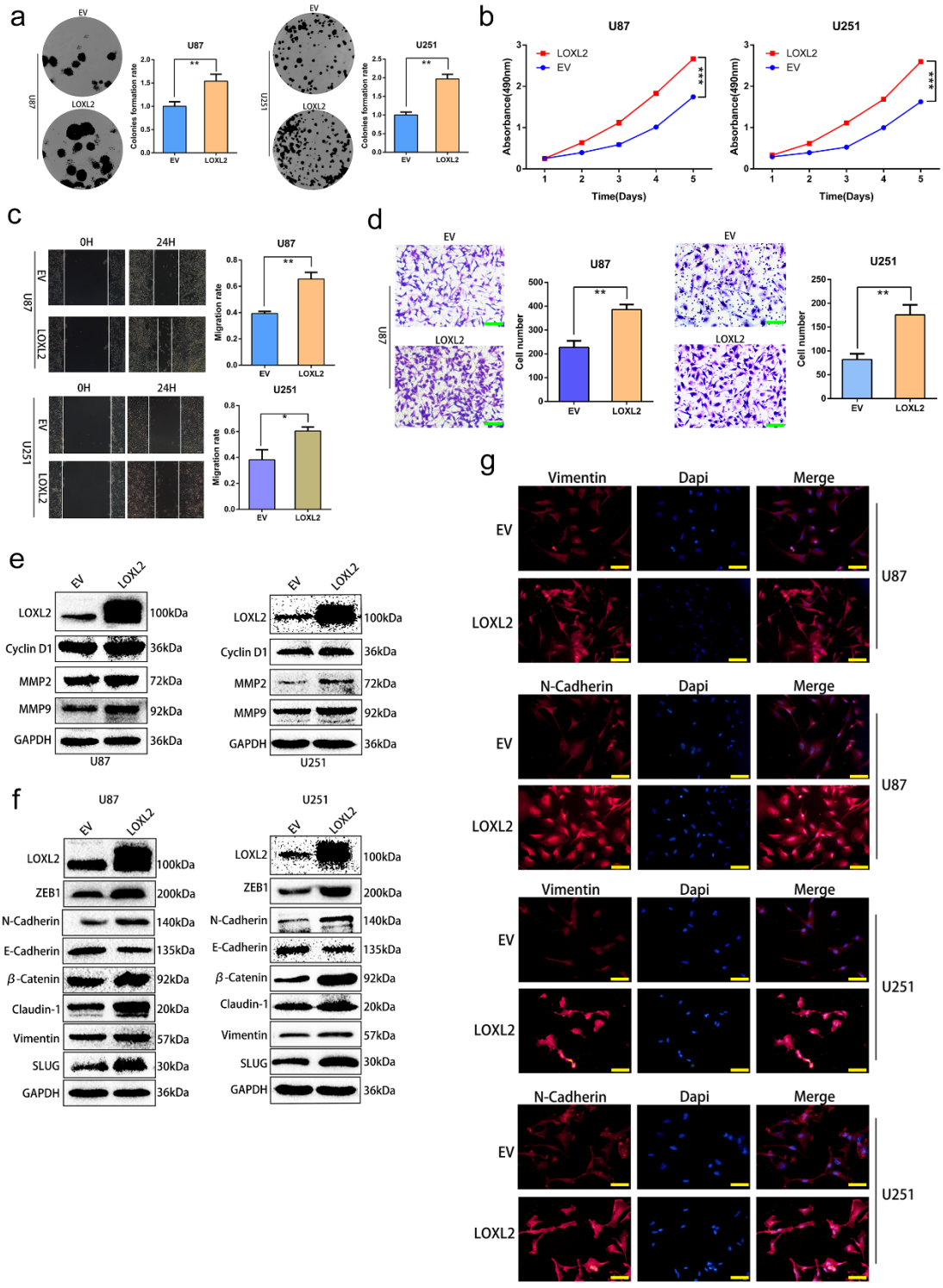
Supplementary figures



Supplementary Figure 1. The relationships between LOXL2 expression and survival of glioma patients. Kaplan-Meier survival curves were used to examine the relationships between LOXL2 expression and survival of glioma patients in CGGA (**a**), REMBRANDT (**b**) and GSE16011 (**c**) cohorts. Kaplan-Meier survival curves were used to examine the relationships between LOXL2 expression and survival of patients with LGG in CGGA (**d**), REMBRANDT (**e**) and GSE16011 (**f**) cohorts. Kaplan-Meier survival curves were used to examine the relationships between LOXL2 expression and survival of patients with GBM in CGGA (**g**) and REMBRANDT (**h**).

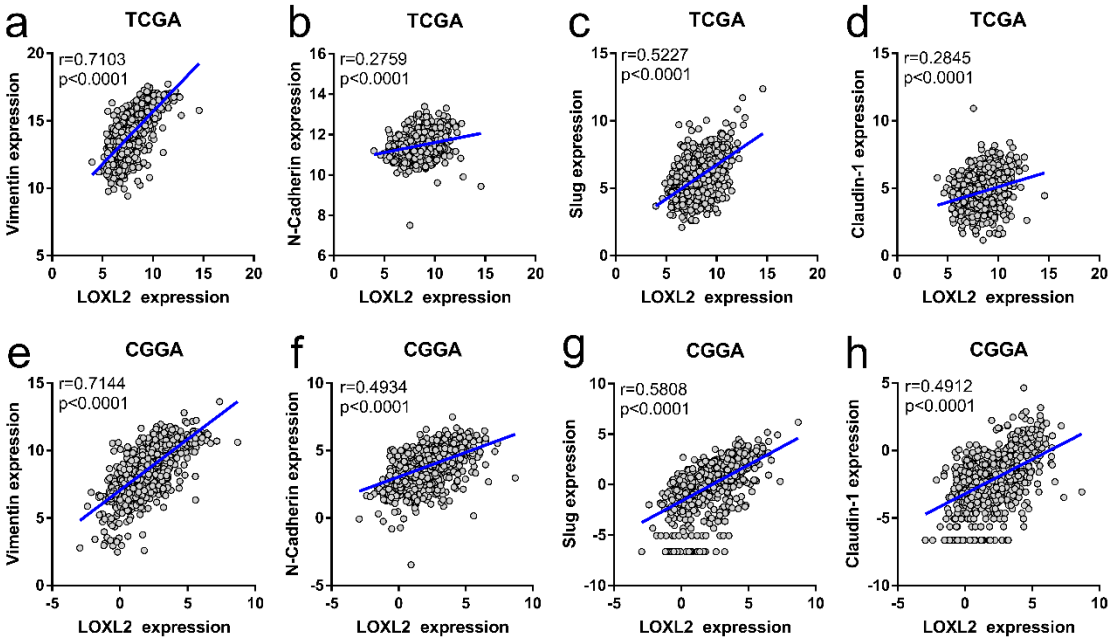


Supplementary Figure 2. Efficiency of LOXL2 overexpression and knockdown. (**a**)(**c**) Confirmation of LOXL2 silence by qRT-PCR and western blotting. (**b**)(**d**) Confirmation of LOXL2 overexpression by qRT-PCR and western blotting.

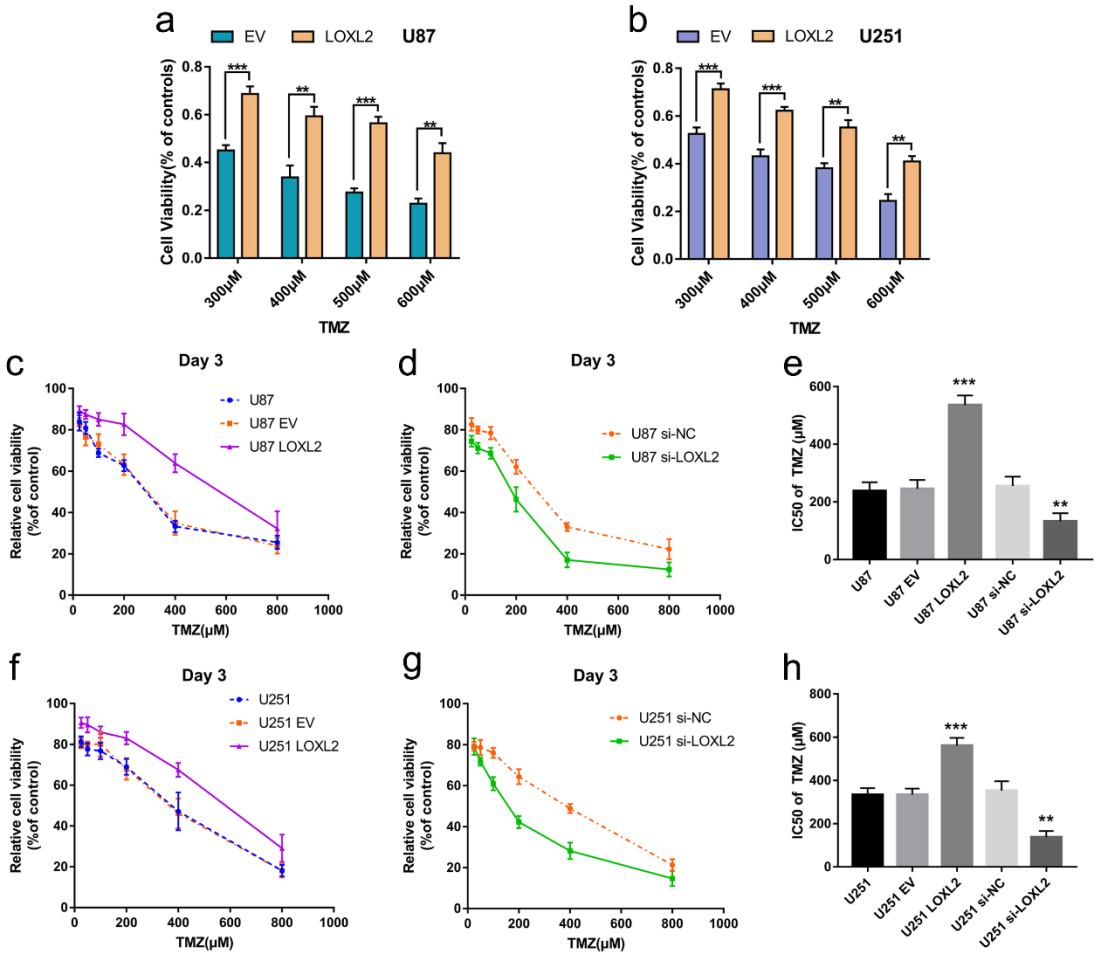


Supplementary Figure3. LOXL2 overexpression promoted glioma proliferation, migration, invasion, and EMT in vitro. (**a**) (**b**), U87 and U251 cell viability increased after LOXL2 overexpression as measured using colony formation and MTS assays. (**c**) (**d**), U87 and U251 cell motility increased after LOXL2 overexpression, measured by wound healing and transwell assays; scale bar = 100 μm. (**e**) Western blotting showed the regulation of cell cycle-regulated proteins cyclin D1 and invasion-related proteins

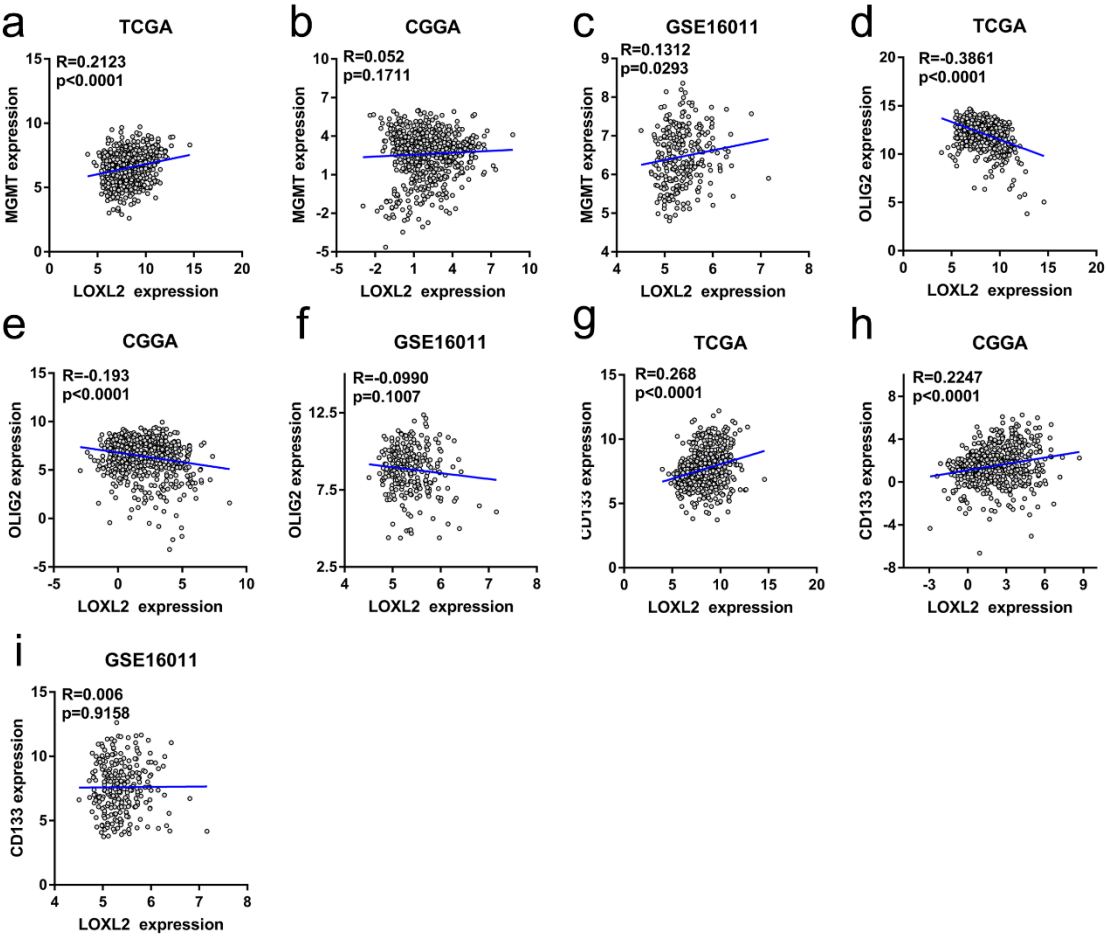
MMP-2 and MMP-9 after LOXL2 overexpression in U87 and U251 cells. (**f**) Western blotting showed regulation of EMT-related proteins after LOXL2 overexpression in U87 and U251 cells. (**g**) Immunofluorescence assays demonstrated expression of vimentin and N-cadherin after LOXL2 overexpression in U87 and U251 cells; scale bar = 100 μm. All data are presented as the mean ± S.D. (3 experiments). *P < 0.05; **P < 0.01; ***P < 0.005 (t-test).



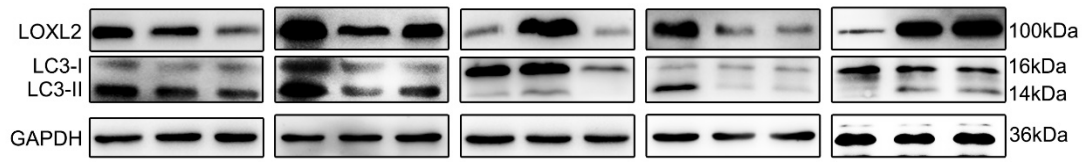
Supplementary Figure 4. The relationship between LOXL2 and EMT markers. (a–d) The relationship between the expression of LOXL2 and EMT markers (Vimentin, N-Cadherin, Slug, Claudin-1) in TCGA dataset. (e–h) The relationship between the expression of and EMT markers (Vimentin, N-Cadherin, Slug, Claudin-1) in CGGA, dataset.



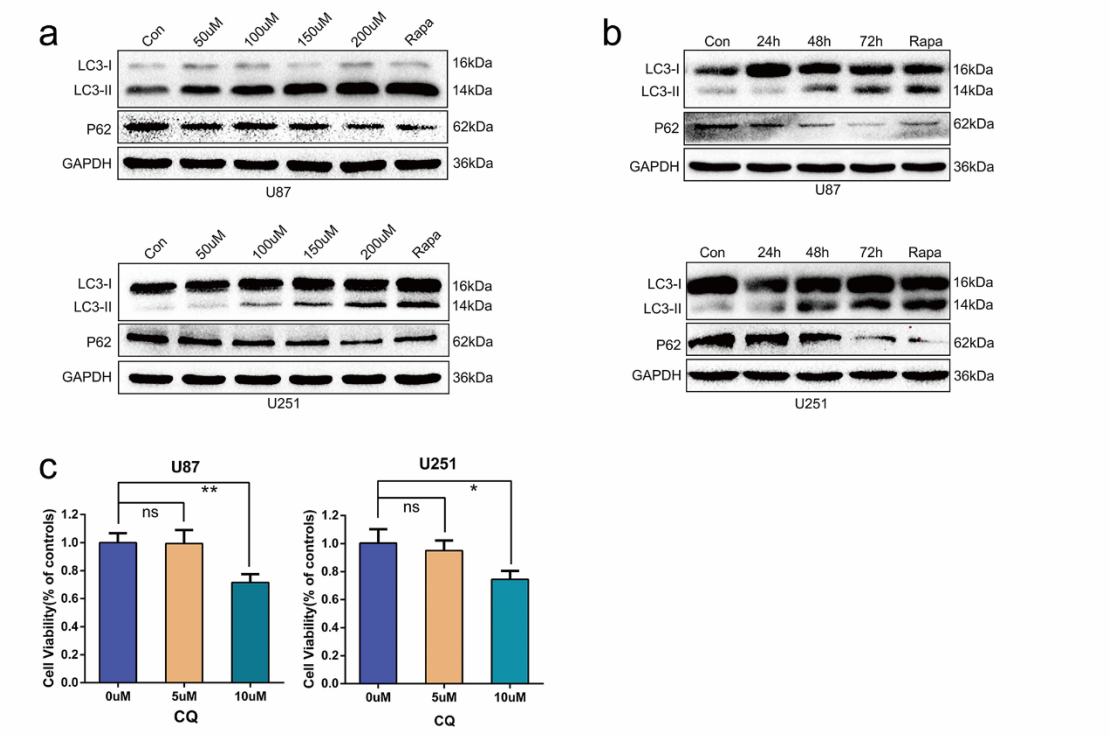
Supplementary Figure 5. Dose-effect curves and IC50 for TMZ of different transfected glioma cells. (a, b) U87 cells overexpressing or silencing LOXL2 were treated with varying concentrations of TMZ. (c) The IC50 value for TMZ of differently transfected U87 cell. (d, e) U251 cells overexpressing or silencing LOXL2 were treated with varying concentrations of TMZ. (f) The IC50 value for TMZ of differently transfected U251 cell.



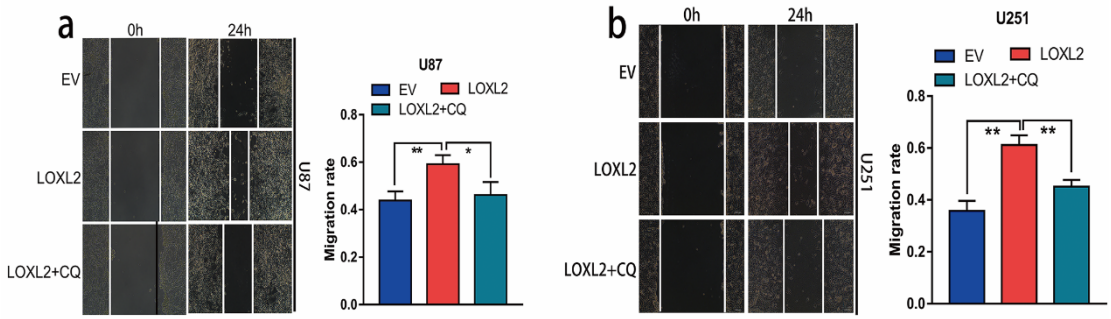
Supplementary Figure 6. The relationship between LOXL2 and MGMG, LOXL2 and glioma stem cell markers. (a–c) The relationship between the expression of LOXL2 and MGMT in TCGA, CGGA, and GSE16011 datasets. (d–f) The relationship between the expression of LOXL2 and OLIG2 in TCGA, CGGA, and GSE16011 datasets. (g–h) The relationship between the expression of LOXL2 and CD133 in TCGA, CGGA, and GSE16011 datasets



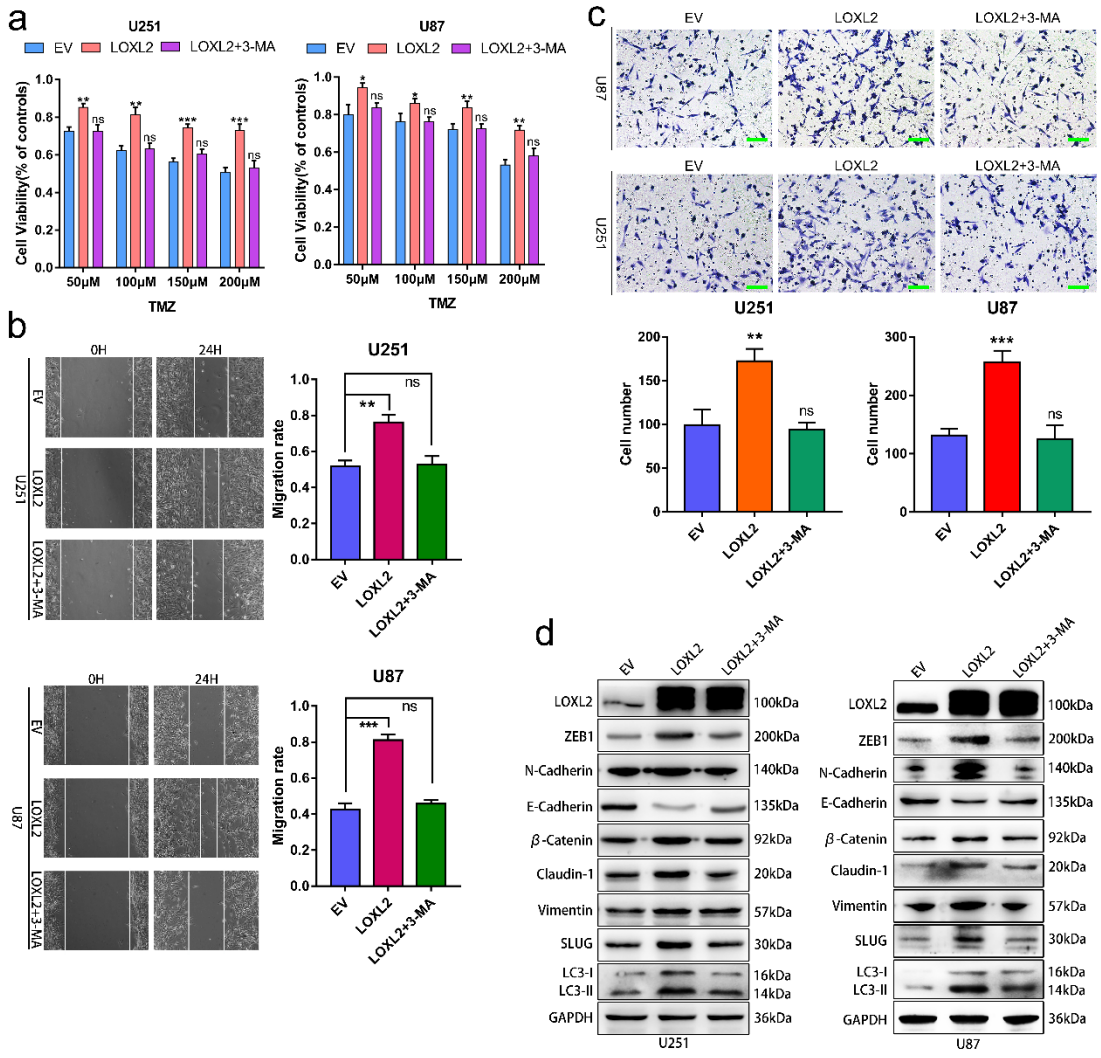
Supplementary Figure 7. The relationship between the expression level of LOXL2 and the intensity of autophagy in glioma samples. 15 glioma samples were randomly selected from 56 glioma samples for detection of LOXL2 and LC3 expression.



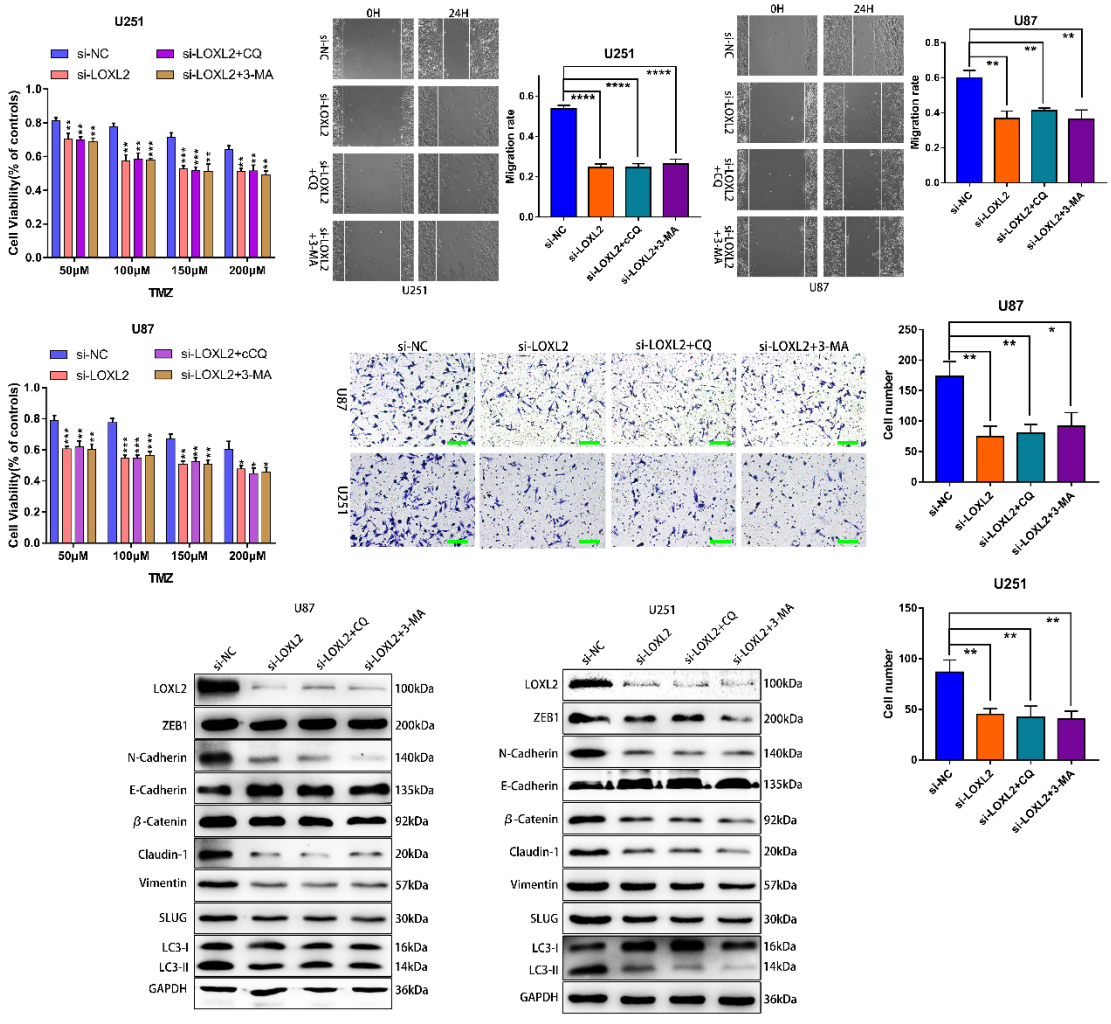
Supplementary Figure 8. TMZ treatment induced autophagy in glioma cells. (**a**) Western blotting of LC3 and P62 in U251 and U87 cells with indicated doses of TMZ for 72 h. (**b**) Western blotting analysis of LC3 and P62 in U251 and U87 cells with TMZ (200 uM) treatment at each indicated time. Rapa(rapamycin) (50 nM), was the positive control. Con, control with DMSO instead of TMZ. (**c**) High concentrations of chloroquine (CQ) affected glioma cell survival. All data are presented as the mean ± S.D. (3 experiments). *P < 0.05; **P < 0.01; ***P < 0.005 (t-test).



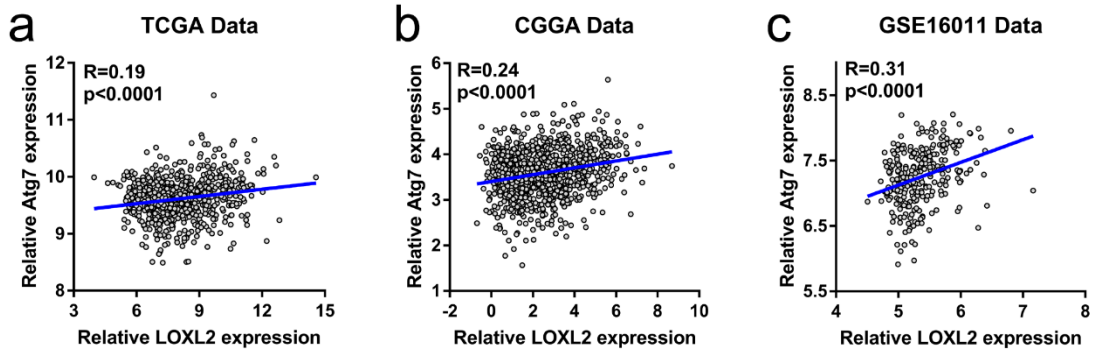
Supplementary Figure 9. CQ reversed the role of LOXL2 in promoting glioma cell migration. (**a**) In U87 cells, CQ (5um, 48h) inhibited the role of LOXL2 in promoting glioma cell migration; (**b**) In U251 cells, CQ (5um, 48h) inhibited the role of LOXL2 in promoting glioma cell migration.



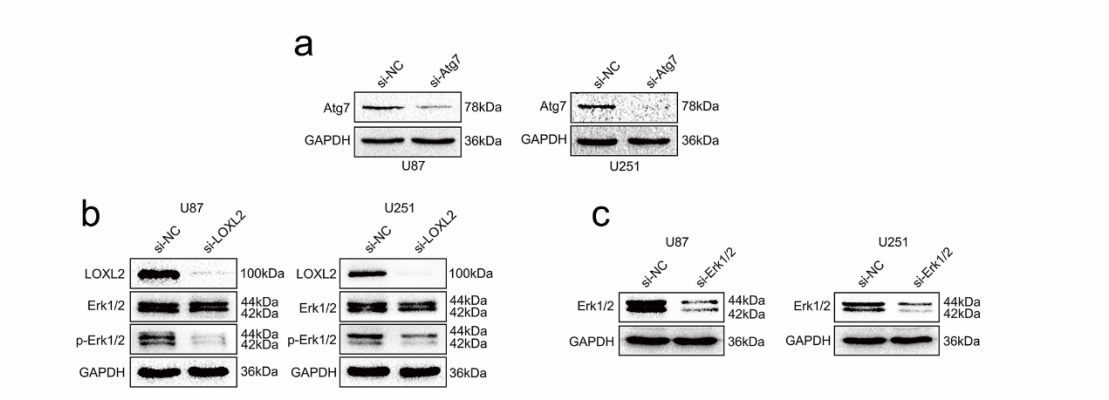
Supplementary Figure 10. The effect of 3-MA on the overexpression of LOXL2 in glioma cells. (a) Viability of control cells or LOXL2-overexpressing cells with or without 3-MA (10 mM, 72 h) with indicated TMZ doses. (b) 3-MA (10 mM, 48h) inhibited the role of LOXL2 in promoting glioma cell migration. (c) Invasion ability of glioma cells treated with EV, LOXL2, LOXL2+3-MA (10 mM, 48 h); scale bar = 100 μm. (d) Expression of EMT-related proteins and LC3 in glioma cells treated with EV, LOXL2 and LOXL2+3-MA (10 mM, 48 h).



Supplementary Figure 11. The effect of 3-MA and CQ on the silencing of LOXL2 in glioma cells. (a) Viability of control cells or LOXL2- silencing cells with or without 3-MA (10 mM, 72 h) or CQ (5 μM, 72 h) with indicated TMZ doses. (b) 3-MA (10 mM, 48h) and CQ (5 μM, 48h) had no effect on the role of LOXL2 silencing in regulating glioma cell migration. (c−e) 3-MA (10 mM, 48h) and CQ (5 μM, 48h) had no effect on the role of LOXL2 silencing in regulating glioma cell invasion; scale bar = 100 μm. (f) 3-MA (10 mM, 48h) and CQ (5 μM, 48h) had no effect on the role of LOXL2 silencing in regulating EMT related markers and LC3 expression.



Supplementary Figure 12. LOXL2 regulates the expression of Atg7 in glioma. The expression of Atg7 was significantly correlated with that of LOXL2 in TCGA(**a**), CGGA(**b**), and GSE16011(**c**) gliomas.



Supplementary Figure 13. Efficiency of Atg7 and Erk1/2 knockdown. (**a**) Confirmation of Atg7 silencing with Atg7-specific si-RNA by western blotting. (**b**) LOXL2 knockdown inhibited Erk1/2 phosphorylation. (**c**) Confirmation of Erk1/2 silencing with Erk1/2-specific si-RNA by western blotting.