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Title	STAT3 Inhibition Overcomes Temozolomide Resistance in Glioblastoma by Downregulating MGMT Expression
Author(s)	Kohsaka, Shinji; Wang, Lei; Yachi, Kazuhiro et al.
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Supplementary Figure legend

Supplementary Fig. 1

Growth curve of seven glioblastoma cell lines are cultured in DMEM containing 10% FBS for 5 days in the presence of indicated concentration of TMZ. BrdU incorporation assays were performed to assess the anti-proliferative effects of TMZ to these cell lines. Error bars represent SD of three independent experiments.

Supplementary Fig. 2

(A) Growth curve of U87 and U87 TMZ-resistant cells cultured in DMEM containing 10% FBS for 5 days in the presence of indicated concentration of TMZ. BrdU incorporation assays were performed to assess the anti-proliferative effects of TMZ to these cell lines. Error bars represent SD of three independent experiments. (B) Clonogenic assay of U87 and U87 TMZ-resistant cells. Clonogenic survival assays were performed by seeding 500 cells in six well plates and exposing them to TMZ (10–1000 μ M) for 48 h, followed by further observation for 7 days. Cell density or colonies were assessed using crystal violet staining. Colonies of more than 50 cells were counted. Error bars represent SD of three independent experiments.

Supplementary Fig. 3

(A) Immunoblot analysis of T98G treated with indicated dose of Src-family kinase inhibitor (Dasatinib) for 24 hours. Dose of the Dasatinib is indicated at the top. The level of pSRC, pFAK, pSTAT3 and MGMT were evaluated. Actin is shown as a loading control. (B) Immunoblot analysis of T98G treated with indicated dose of MET inhibitor (PHA665752) for 24 hours. Dose of the PHA665752 is indicated at the top. The level of pMET, pSTAT3 and MGMT were evaluated. Actin is shown as a loading control. (C) Immunoblot analysis of

T98G treated with indicated dose of MEK inhibitor (U0126) for 24 hours. Dose of the U0126 is indicated at the top. The level of pERK1/2, pSTAT3 and MGMT were evaluated. Actin is shown as a loading control.