# Supplementary Materials for

NUSAP1 potentiates chemoresistance in glioblastoma through its SAP domain to stabilize ATR

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# This PDF file includes:

Figures. S1 to S5

# Supplementary Fig. S1



#### Figure. S1.

High level of NUSAP1 predicts poor prognosis. (a) Kaplan–Meier analysis of progression-free survival using data from 4 different glioma databases. (b) The level of NUSAP1 in grade (II–IV)

gliomas from TCGA GBMLGG database. (c) The expression of NUSAP1 in grade (I–IV) gliomas from French 284 database.



# Figure. S2.

Knocking down of NUSAP1 inhibits cell viability and DNA synthesis. (a) Viability of NUSAP1-

knockdown GBM cells. (b) BrdU-positive GBM cells after knocking down of NUSAP1.

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# Figure. S3.

NUSAP1 stabilizes ATR in GBM cells. (a) mRNA level of NUSAP1 and ATR in indicated cells. (b) The level of indicated proteins in NUSAP1-overexpressed GBM cells treated with CHX for 0 h, 1 h, 2 h, 4 h, or 8 h. (c) The expression of sumoylated ATR in GBM cells knocking down of NUSAP1.



# Figure. S4.

NUSAP1 contributes to chemoresistance of GBM cells. (a) Caspase3/7 activity of GBM cells depleting of NUSAP1 in response to TMZ and DOX respectively. (b) Caspase3/7 activity of NUSAP1 as well as NUSAP1- $\triangle$  SAP overexpressed GBM cells treated with TMZ and DOX respectively.

# Supplementary Fig. S5



# Figure. S5.

NUSAP1 participates in apoptosis, DNA damage and chemoresistance through ATR. (a) The level of  $\gamma$ -H2AX and cleaved-caspase9 in GBM cells overexpressed ATR after knocking down of NUSAP1. (b) Caspase3/7 activity of GBM cells overexpressed ATR after depleting of NUSAP1 in response to TMZ.