Supplementary Figure 1. FOXM1 associated with β -Catenin in the cytoplasm and the nucleus.

(A) Increased FOXM1 and β -Catenin levels were identified in both the cytoplasm and the nucleus. The cytoplasmic or nuclear extracts from hFOB1.19, U2OS, MG63, Saos-2 and HOS cells were analyzed to determine the levels of FOXM1 and β -Catenin. Tubulin and TFIIb were used as controls for cytoplasmic and nuclear fractions, respectively. (B) Wnt3a treatment enhanced the nuclear translocation of FOXM1 and β -Catenin. U2OS cells were treated with Wnt3a (20 ng/ml) for 0, 30, 60 and 90 min, then the cytoplasmic or nuclear extracts were analyzed to determine the levels of FOXM1 and β -Catenin.

Supplementary Figure 2. The mRNA levels of *c-Myc* and *Cyclin D1* in osteosarcoma cells upon knockdown of *FOXM1* or β -*Catenin*.

(A-D) Knockdown of *FOXM1* in hFOB1.19, U2OS and MG63 cells decreased the expression of *c-Myc* and *Cyclin D1*. The mRNAs from cells used in Figure 3A were analyzed to determine the expression of *c-Myc* and *Cyclin D1* by qRT-PCR. Expression was normalized against β -Actin in each cell line, and the resulting ratios in cells transfected control-shRNA were arbitrarily defined as 1-fold. (E-H) Knockdown of β -Catenin in U2OS and MG63 cells decreased *c-Myc* and *Cyclin D1* expression. The mRNAs from cells used in Figure 3A were analyzed to determine the expression of *c-Myc* and *Cyclin D1* expression. The mRNAs from cells used in Figure 3A were analyzed to determine the expression of *c-Myc* and *Cyclin D1* by qRT-PCR. Expression was normalized against β -Actin in each cell line, and the resulting ratios in cells transfected control-shRNA were arbitrarily defined as 1-fold. Representative data from three independent experiments are shown. ***P*<0.001.

Supplementary Figure 3. Effect of pharmacological treatments on the levels of proteins of the Wnt/β-Catenin signaling pathway.

(A) Wnt3a treatment activated the expression of proteins of the Wnt/ β -Catenin signaling pathway. Cells used in Figure 5A were analyzed to determine the protein levels of FOXM1, β -Catenin, c-Myc and Cyclin D1. (B-D) Treatment with FDI-6 and PKF118-310, but not treatment with 10058-F4, inhibited the expression of proteins of the Wnt/ β -Catenin signaling pathway. Cells used in Figures 5B-5D were analyzed to determine protein levels of FOXM1, β -Catenin, c-Myc and Cyclin D1.

Supplementary Figure 4. AZA treatment inhibited the colony formation ability.

The hFOB1.19, U2OS and MG63 cells were seeded onto 6-well plates, and cultured with 0.1 ml DMEM medium supplemented with AZA (1 μ M) for two weeks. Then, cells were stained with 0.5% crystal violet and pictures were taken.

Supplementary Figure 1





Supplementary Figure 2

Supplementary Figure 3



Supplementary Figure 4

