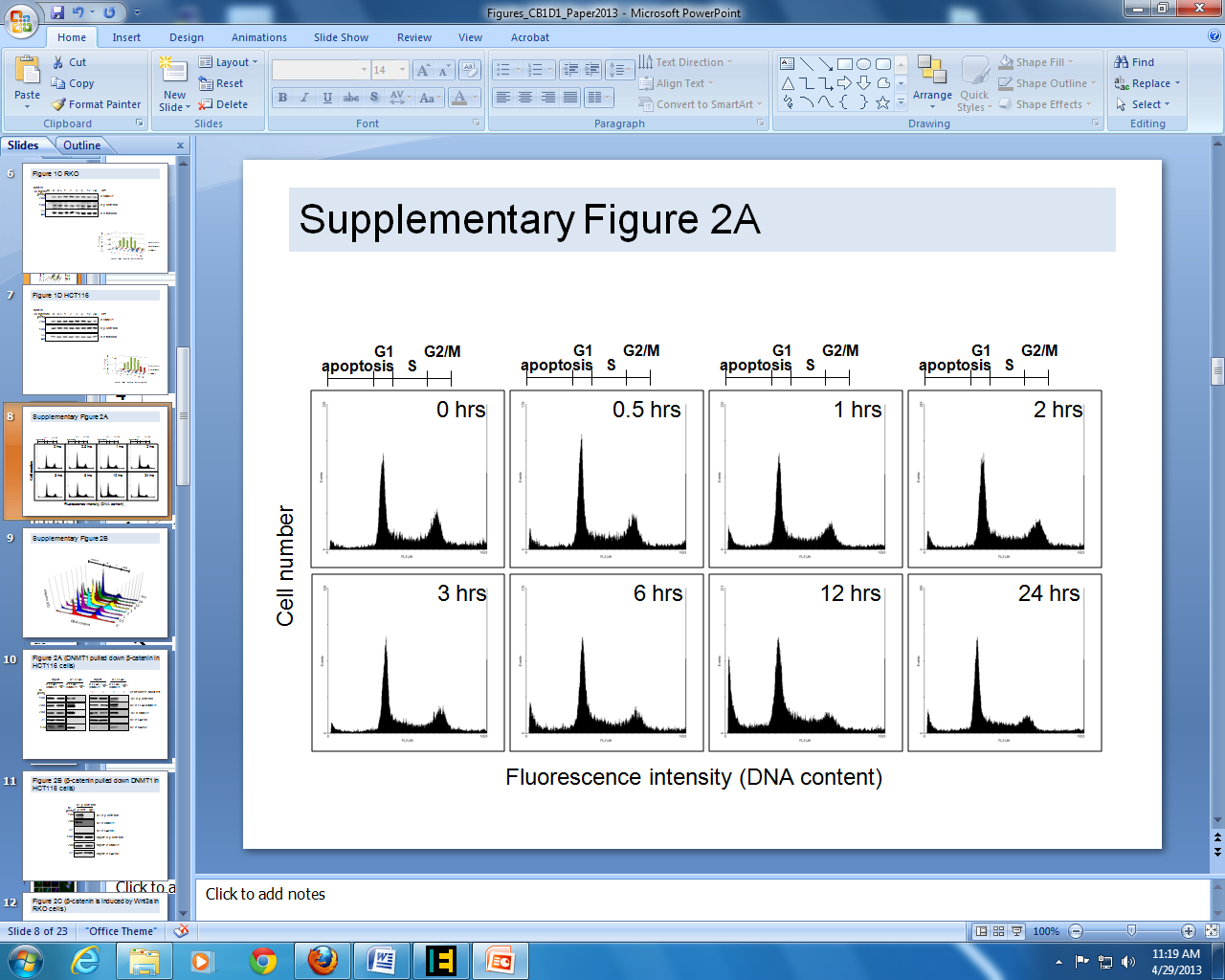
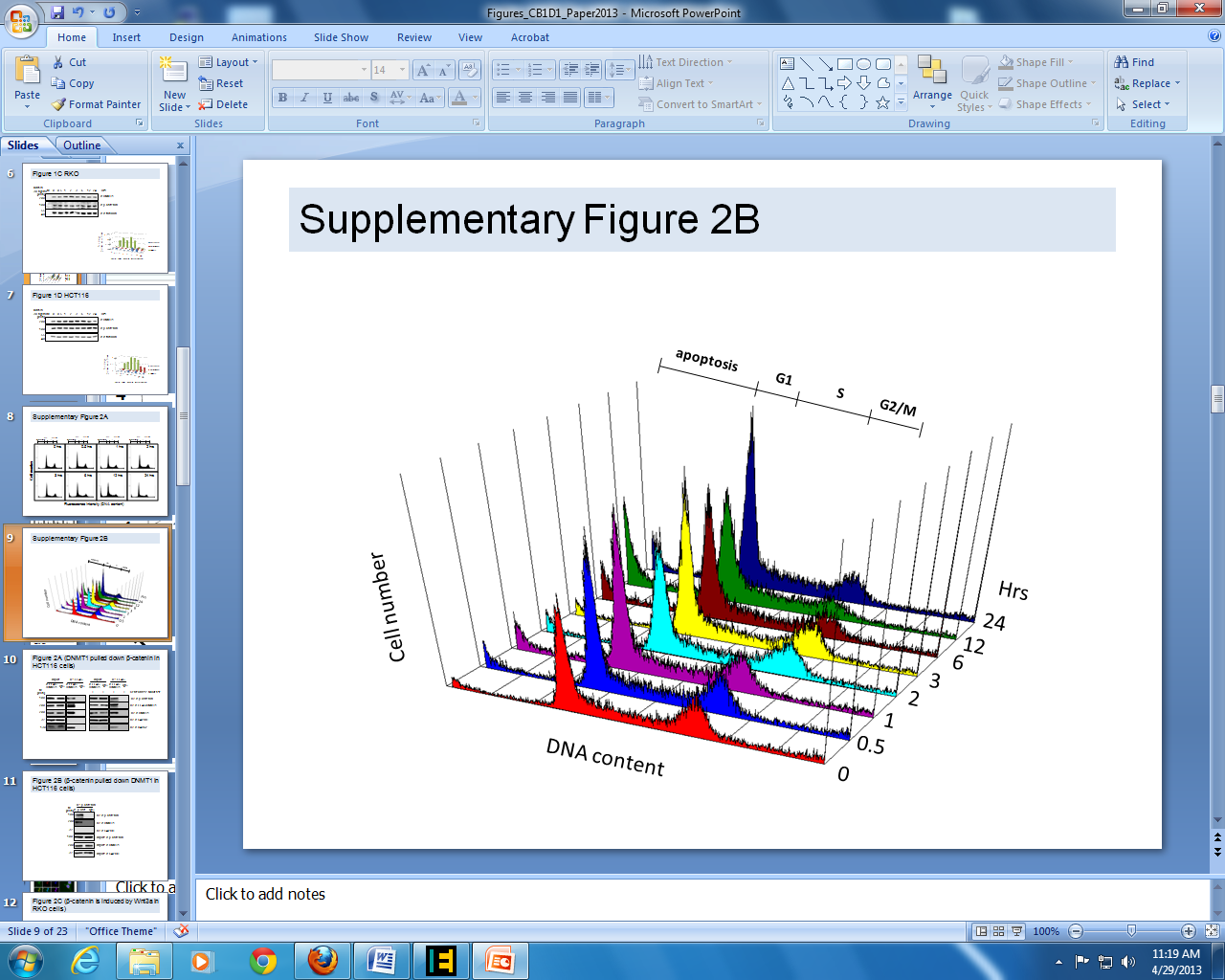
**Supplementary Figure 1**

**A**

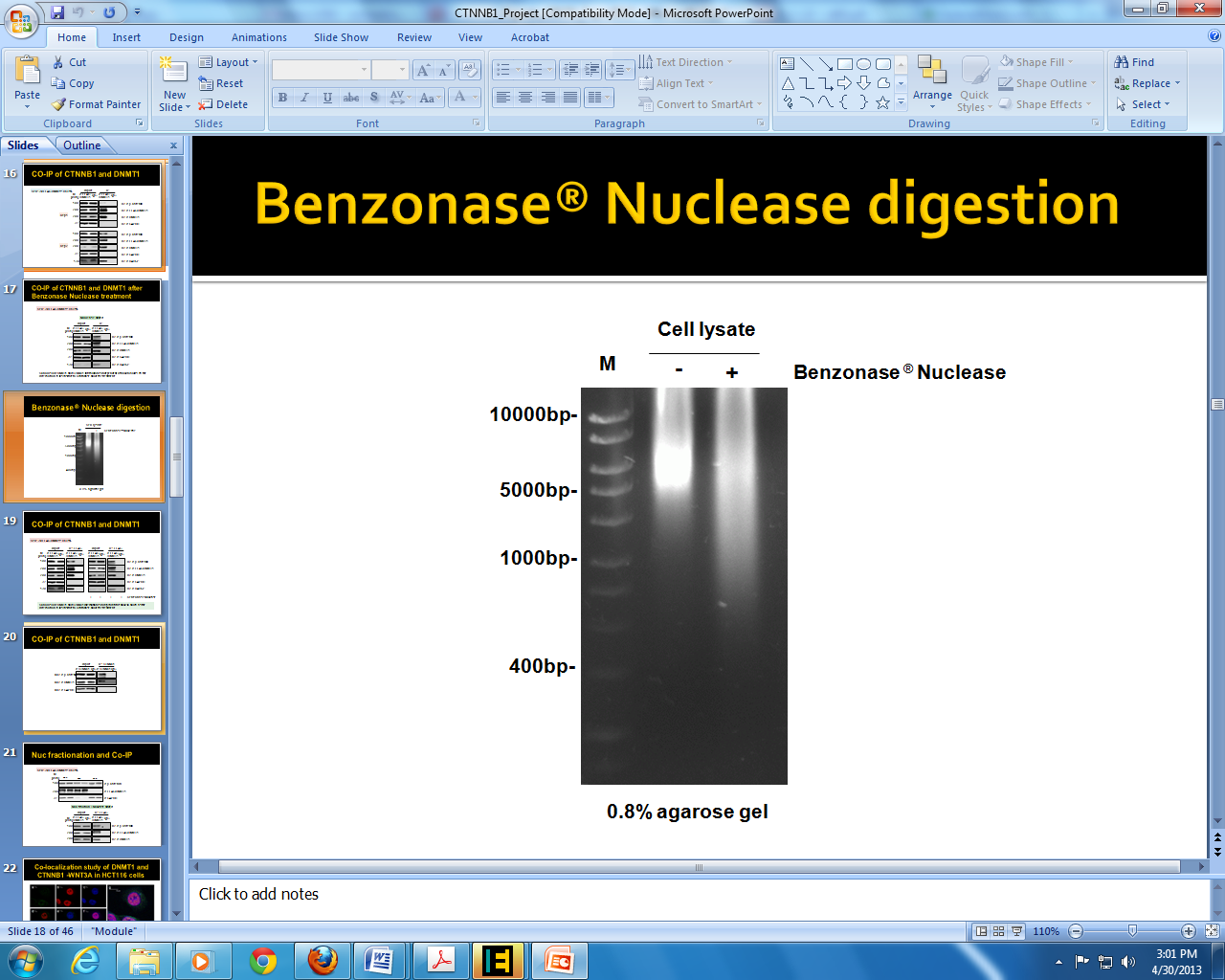
****

**B**

****

**Supplementary Figure 1.Cell-cycle profiles of HEK293T cells following treatment with Wnt3a.**(A) Individual cell-cycle profiles of cells collected at eight time points (0, 0.5, 1, 2, 3, 6, 12, 24 hours post stimulation). Cells that were harvested as the indicated time points were fixed with methanol. Cells were then washed with PBS and treated with 5mg/ml RNase in PBS buffer at 37ºC for 30 min. Propidium iodide (100 µg/ml) was used to stain cells at 4ºC for one hour. Cells were analysed on an Epics XL flow cytometer and percentages of G1, S and G2/M populations were determined by histograms generated by software WinMDI2.9. (B) Overlaid cell-cycle profiles of cells collected at eight time points.

**Supplementary Figure 2**



**Supplementary Figure 2**. Protein lysate samples used in the immunoprecipitation or immunoblots analyses were treated with Nuclease. DNA from samples treated with (+) or without (-) Benzonase Nuclease were run on a Bio-safe DNA-stained 0.8% agarose gel.

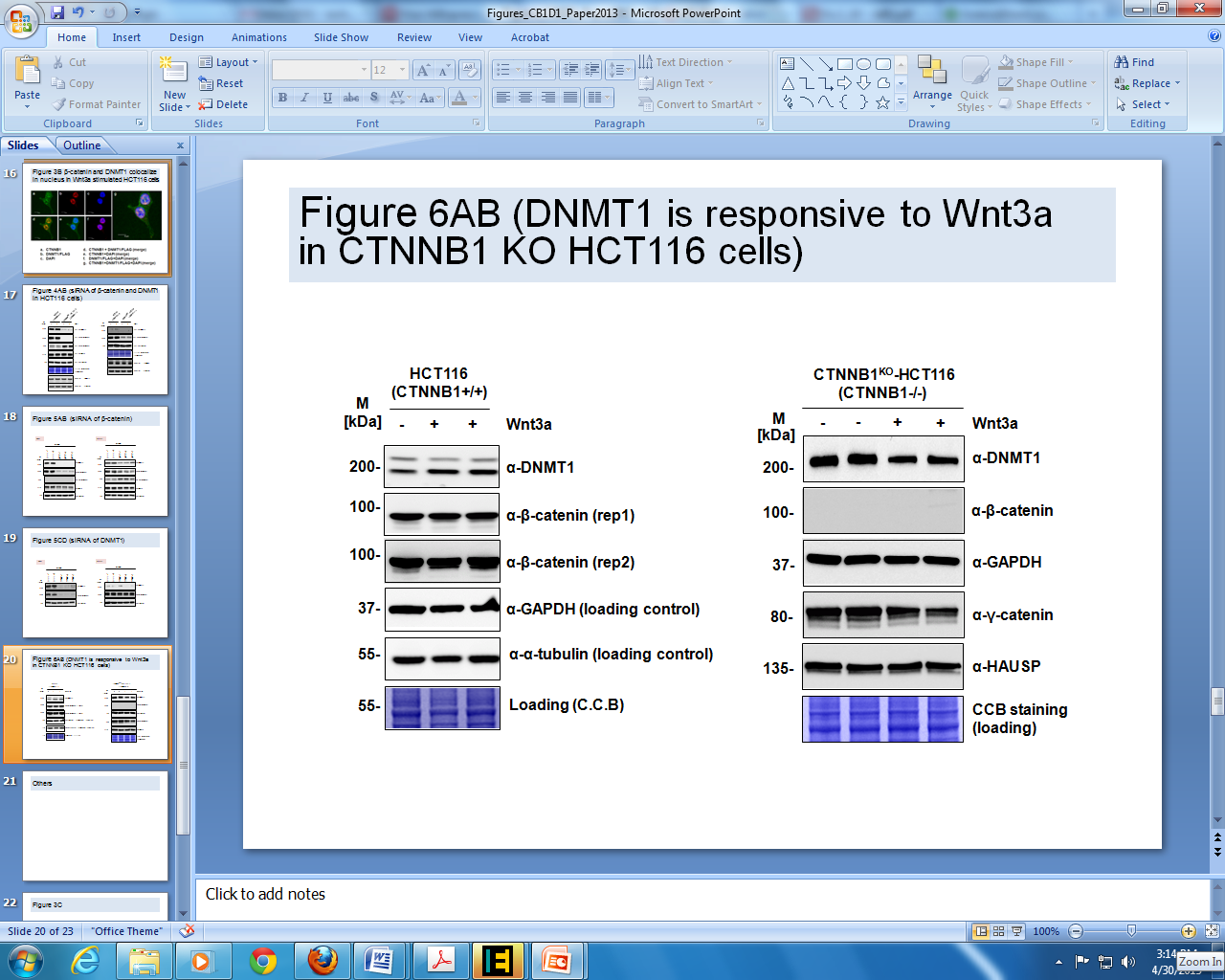
**Supplementary Figure 3**



**Supplementary Figure 3**. Dnmt1 (native antibody) immuno-precipitates β-catenin in parent HCT116 cells.

**Supplementary Figure 4**

**A B**

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**Supplementary Figure 4. DNMT1 protein is stabilized by treatment with Wnt3a in HCT116 but not in CTNNB1KO-HCT116 cells.**

**(A)** Endogenous DNMT1 abundance is increased in Wnt3a stimulated (+) vs. non-stimulated (-) HCT116 cells. **(B)** Endogenous DNMT1abundance is not increased in Wnt3a stimulated CTNNB1 knockout HCT116 cells. Immunoblots were used to quantify proteins withanti-DNMT1, anti-β-catenin, anti-ɣ-catenin, anti-HAUSP sera. GAPDH and α-tubulin were used as loading controls. C.C.B. is coomassie blue stained gel. M refers to standard protein marker.



**Supplementary Figure 5. Degradation profiles for β-catenin and Dnmt1 in HCT116, DNMT1KO-HCT116 or CTNNB1KO-HCT116 cells following cycloheximide treatment**. Degradation rate constants were quantified by measuring the relative intensity of each protein by quantitative Western blotting at 0, 3, and 6 hours after cycloheximide treatment. The intensity data were fit to a first-order decay function to estimate the degradation rate constant, which then was used to calculate the half-life.

**A**



**B**



**Supplementary Figure 6. Abundance of β-catenin and Dnmt1 in DNMT1KO-HCT116 (A) or CTNNB1KO-HCT116 (B) cells treated with MG-132 proteasome inhibitor.**



**Supplementary Figure 7. RT-PCR analysis of H19 expression in HCT116 and CTNNB1KO-HCT116 cells shows increase H19 transcript levels in the KO cells**