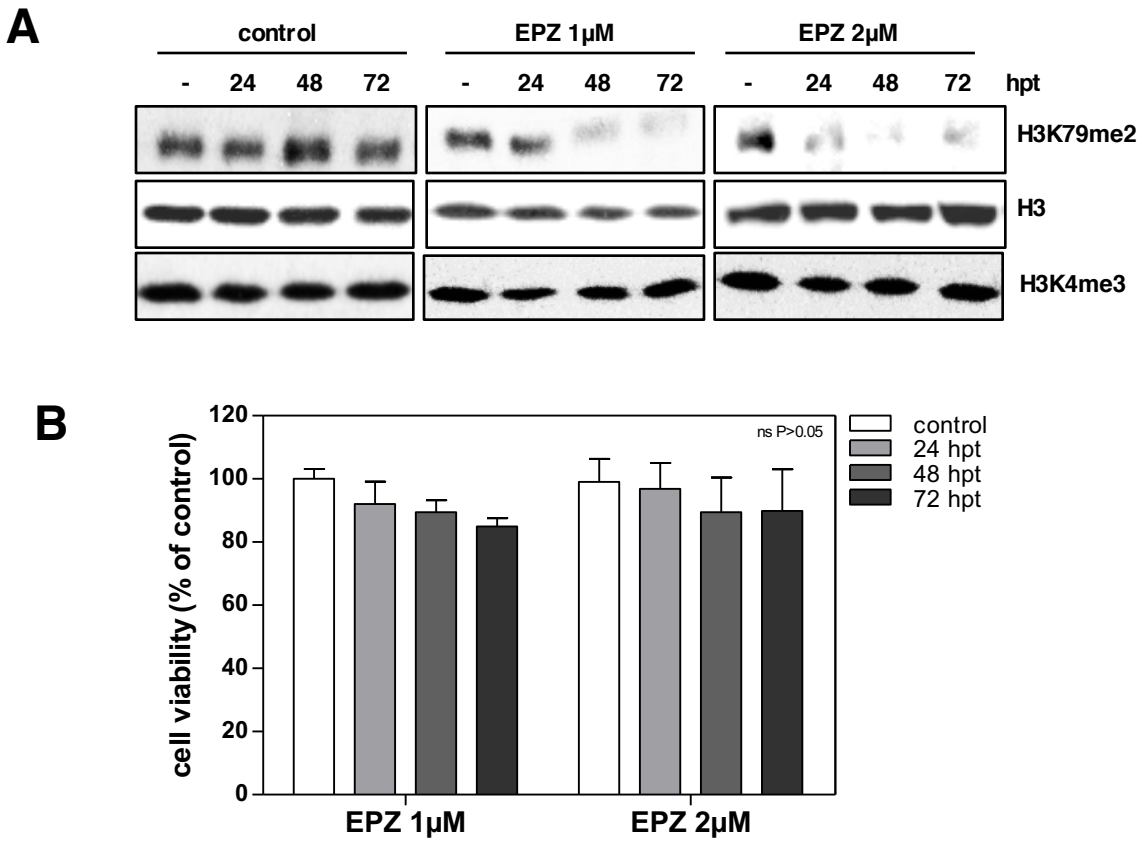
Epigenetic control of influenza virus: role of H3K79 methylation in interferon-induced antiviral response

Laura Marcos-Villar, Juan Díaz-Colunga, Juan Sandoval, Noelia Zamarreño, Sara Landeras-Bueno, Manel Esteller, Ana Falcón and Amelia Nieto

SFig.1



Supplemental Fig. 1. Specificity and cell viability of Dot1L inhibitor treatment. A) Total extracts of A549 cells treated with DMSO, 1 μM or 2 μM Dot1L inhibitor (EPZ) were collected at various times post-treatment; H3 and methylated H3K79 and H3K4 levels were determined by Western blot. B) Viability of A549 cells treated with Dot1L inhibitor was determined by MTT assay to measure cell metabolic activity.

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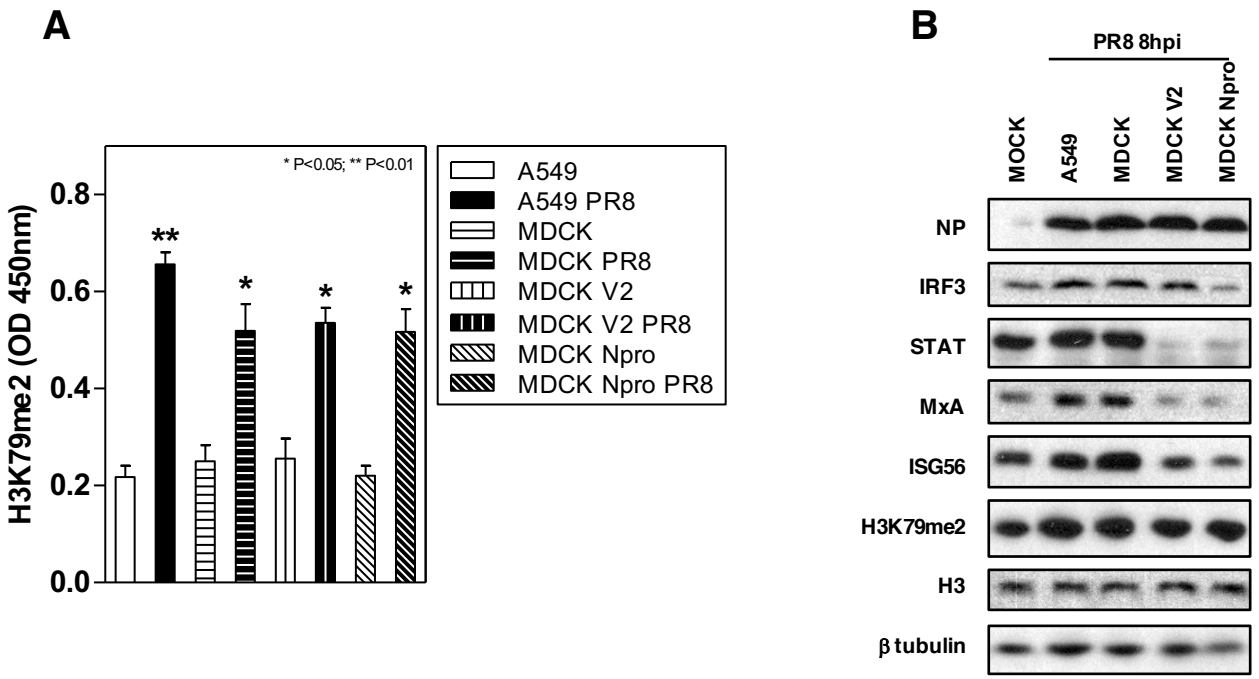
SFig. 2



Supplemental Fig. 2. Accumulation levels of H3K79 and IFN-related protein in normal and cells deficient for IFN response. A) Cells were left uninfected or infected with influenza virus PR8 at 3 m.o.i for 8 h. H3K79 methylation levels, were analyzed by colorimetric assays (B). Uninfected A549 cells (MOCK), or A549, MDCK, MDCK V2 and MDCK Npro cells were infected with PR8hv (3 m.o.i., 8 h), total extracts were obtained and used for detection of the indicated proteins by Western blot assays.