

PRMT1 arginine methyltransferase accumulates in cytoplasmic bodies that respond to selective inhibition and DNA damage

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Supplementary Figures

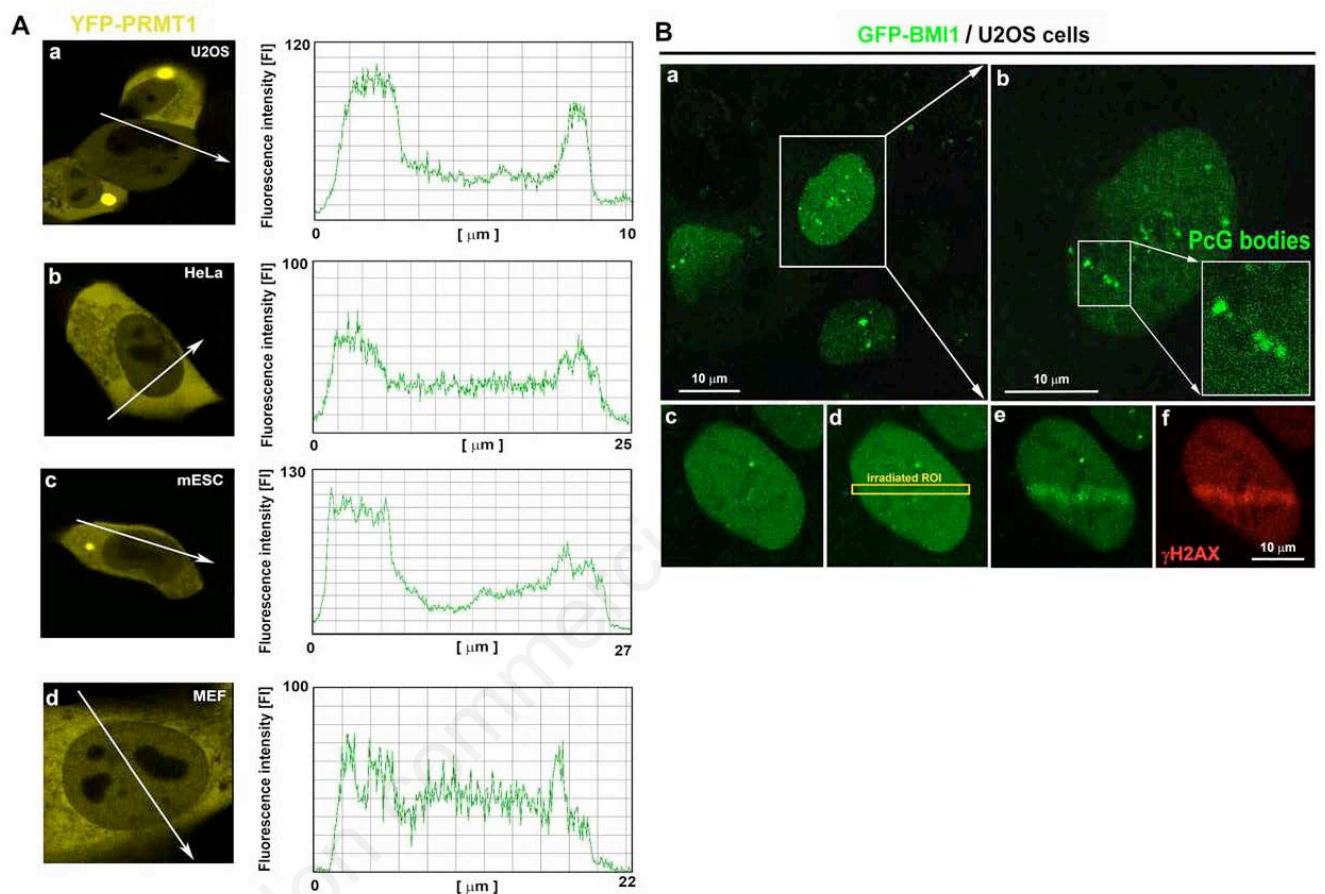


Figure 1. Protein recruitment to UV-A induced DNA lesions. A) Levels of YFP-PRMT1 (yellow) analyzed by Leica software (LEICA LAS AF, version 2.1.2) in (a) U2OS cells, (b) HeLa, (c) mESCs and (d) MEFs. Profiles of fluorescence intensity are shown in graphs (green lines). (Ba-b) Nuclear localization of GFP-BMI1 protein (green) and its accumulation into Polycomb group (PcG) bodies. (Bc-f) BMI1 (green) recruitment to DNA lesions and γ H2AX (red) in UV-A irradiated ROI (green) were monitored in live U2OS cells stably expressing GFP-BMI1 protein. γ H2AX was visualized by immunofluorescence. GFP-BMI1 accumulated at DNA lesions within 15 s after microirradiation with a UV-A laser. Similar recruitment of BMI1 to DNA lesions was shown for both exogenous and endogenous BMI1 protein.²⁶

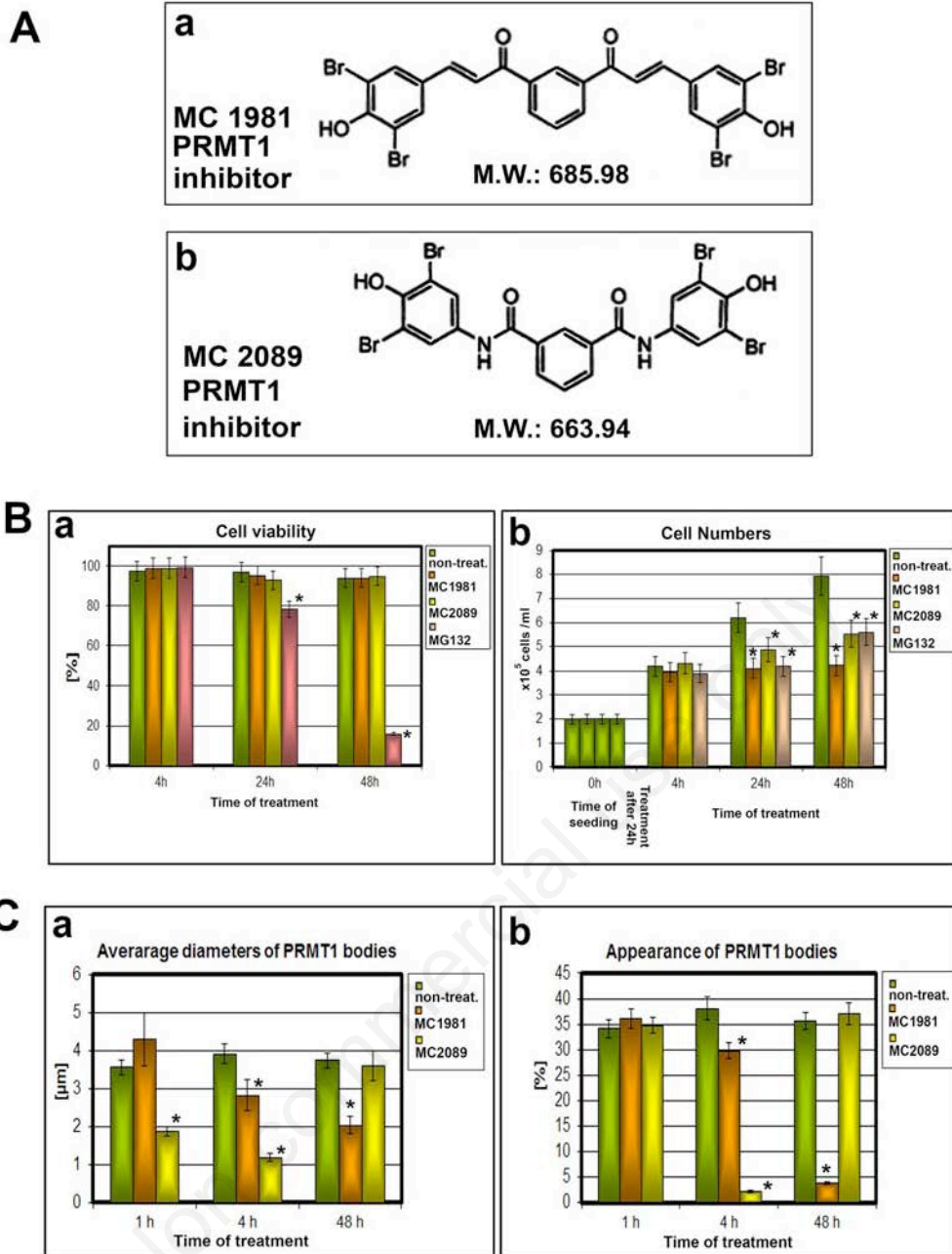


Figure 2. Effects of PRMT1 inhibitors. A) Chemical formulas of (a) MC 1981 and (b) MC 2089. B) Cell numbers before and after selected experimental treatments: cell viability after the cell treatment (Ba); in panel (Bb) initial cell number (time 0) is shown, then cells were cultivated 24 h and treated by inhibitors; cell numbers were calculated 4, 24 and 48 h after the cell treatment; cell numbers were calculated using the TC10 (BioRad) automated cell counter; data are shown as a mean \pm standard errors; Student *t*-test was performed and asterisks showed statistically significant differences ($P \leq 0.05$) from control values for 4, 24 and 48 h of the cell treatment; non-treat., no treatment by inhibitors. Average diameters of PRMT1 nuclear bodies (Ca) and an appearance of PRMT1 cytoplasmic bodies (Cb) in transfected cells (shown as percentage of the cells from the cell population).