

Correction to “5-aza-Cytidine Is a Potent Inhibitor of DNA Methyltransferase 3a and Induces Apoptosis in HCT-116 Colon Cancer Cells via Gadd45- and p53-Dependent Mechanisms”

In the above article [Schneider-Stock R, Diab-Assef M, Rohrbeck A, Foltzer-Jourdainne C, Boltze C, Hartig R, Schonfeld P, Roessner A, and Gali-Muhtasib H (2005) *J Pharmacol Exp Ther* **312**:525–536], similar groups of cells appear at different time points in Figs. 2A and 3A that have raised suspicion regarding the original preparation of these figures. Given the length of time since original publication, the exact reason for this discrepancy could not be determined. These experiments have been repeated and the new data clearly support the main message of the manuscript on the proapoptotic effects of 5-aza-cytidine (5-aza-CR).

Corrected figure panels are reprinted below with new legends. A *Methods* for the repeated experiments is also provided.

The authors regret this error and any inconvenience it may have caused.

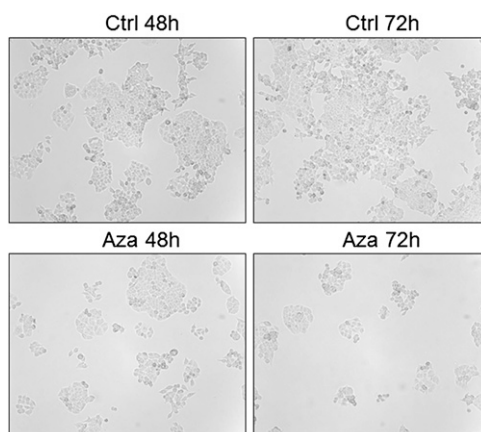


Fig. 2A. Induction of growth arrest by 5-aza-CR (Aza) in HCT-116 (+/+) human colon cancer cells. Cells were seeded and treated with 1 μ M 5-aza-CR for 48 and 72 hours. Representative bright field images were taken of two separate experiments. Ctrl, control.

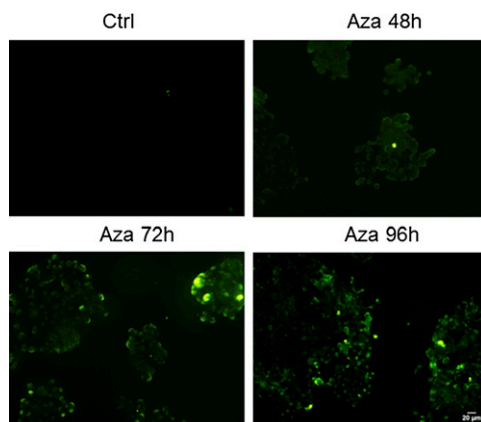


Fig. 3A. The apoptotic effects of 5-aza-CR (Aza) in HCT-116 (+/+) cells. Cells treated for 48, 72, and 96 hours with 1 μ M 5-aza-CR and the extent of DNA fragmentation determined by terminal deoxyribonucleotidyl transferase-mediated dUTP nick-end labeling assay (green) in fixed cells using fluorescence microscopy. Scale bar, 20 μ m. Ctrl, control.

Methods

Cell Growth and Treatment. The human colon cancer HCT-116 cells were cultured in RPMI 1640 medium. Cells were grown at 37°C in an atmosphere of 5% CO₂ supplemented with penicillin (100 U/ml), streptomycin (100 mg/ml), and 10% fetal calf serum. For experiments, cells were seeded on six-well plates at a density of 40,000 cells per well. Cells were treated with 1 μ M of freshly prepared 5-aza-CR (Sigma-Aldrich, St. Louis, MO) 24 hours after plating, and treatment was replenished every 48 hours. Randomly selected bright field microscopic fields were acquired on an inverted Leica DMI1 microscope (Wetzlar, Germany) using 4 \times and 10 \times air objective lenses.

Apoptosis: Terminal Deoxyribonucleotidyl Transferase-Mediated dUTP Nick-End Labeling Assay. Apoptosis was scored by estimating the extent of DNA fragmentation using the terminal deoxyribonucleotidyl transferase-mediated dUTP nick-end labeling (TUNEL) assay. Briefly, cells were plated on autoclaved glass coverslips in six-well culture plates and treated with 1 μ M 5-aza-CR. The medium was then aspirated and cells were washed twice with warm phosphate-buffered saline. Cellular DNA was stained with the In Situ Cell Death Detection kit (Roche Diagnostics, Mannheim, Germany), and the assay was performed according to the recommendations of the manufacturer. For each of three replicated experiments, four randomly selected microscopic fields were acquired on an inverted Nikon eclipse Ti-U microscope using a 20 \times air objective lens (Nikon, Tokyo, Japan). The same microscopy setup was used for all representative images. Scale bar, 20 μ m.