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MTA1 promotes cell proliferation via DNA damage repair in epithelial ovarian cancer

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ABSTRACT. We examined whether metastasis-associated gene 1 (*MTA1*) promotes cell proliferation via DNA damage repair in ovarian cancer. *MTA1* was successfully down-regulated using small interfering RNA in the epithelial ovarian cancer cell lines SKOV-3 and OVCAR-3. Cell growth was evaluated through MTT and colony formation assays. Fluorescence-activated cell sorting analysis was used to evaluate the distribution of cells in the cell cycle, and cytotoxicity assays were performed to study cell sensitivity to cisplatin. A neutral comet assay was used to measure levels of ionizing radiation-induced DNA damage in SKOV-3 cells, and Western blot analyses were carried out to examine the expression of key proteins involved in DNA damage repair pathways. *MTA1* knockdown markedly inhibited cell growth and led to S phase cell cycle arrest. In addition, MTA1 depletion conferred sensitivity of ovarian cancer cells to cisplatin. Moreover, MTA1 depletion increased the level of ionizing radiation-induced DNA damage and caused irreparable damage, which was illustrated by a remarkable increase and

persistent existence of a comet tail as well as protein expression levels of γH2AX, pRPA, and pChk1, all of which play critical roles in DNA repair. Thus, MTA1 promotes the proliferation of epithelial ovarian cancer cells by enhancing DNA repair.

Key words: DNA repair; Metastasis-associated gene 1;
Cell proliferation; Ovarian carcinoma

