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This article was originally published in Volume 24, Number 3, pages 189-196 (http://dx.doi.org/10.3727/096504016X14641336229602). Author Lingfei Han was included in error in the original article. Below is shown the correct author contributors with Lingfei Han removed.

Knockdown of HVEM, a Lymphocyte Regulator Gene, in Ovarian Cancer Cells Increases Sensitivity to Activated T Cells

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Ovarian cancer is highly malignant with a gradually increasing incidence and a high mortality rate. Immunosuppression is induced in ovarian cancer, although the mechanism detail is not clear. It has been indicated that HVEM (herpesvirus entry mediator) B- and T-lymphocyte attenuator (BTLA) negatively regulates the immune responses of T lymphocytes. Here, HVEM mRNA was found to be elevated in ovarian cancer tissue samples and primary ovarian cancer cells in comparison with benign tissue samples. We then knocked down HVEM expression in an ovarian cancer cell line, OVCAR3, by lentivirus-based small hairpin RNA (shRNA). Cell Counting Kit-8 (CCK-8) assay and flow cytometry analysis showed that HVEM-shRNA had no effect on the proliferation, early apoptosis, or cell cycle distribution of OVCAR3. We then isolated activated T cells and performed coculture experiments in Transwell. Remarkably, HVEM-silenced ovarian cancer cells (primary ovarian cancer cells and OVCAR3) increased the number of T cells and the secretion of tumor necrosis factor-α (TNF-α) and interferon-γ (IFN-γ), while activated T cells promoted the apoptosis of HVEM-silenced ovarian cancer cells. The current study partially explains the immune escape mechanism of ovarian cancer cells and provides a possible target for immunotherapy.

Key words: Herpesvirus entry mediator (HVEM); Ovarian cancer; T cells; Immunosuppression

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