Abstract

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Promoter DNA methylation regulates MHC class II transcription in human ovarian cancer

Authors: Abir Benhissoune[1],✉

Affiliation: [1]School of Medicine, Keele University, UK

Corresponding Author:
x9l73@students.keele.ac.uk

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ABSTRACT

Introduction: Ovarian cancer is a leading cause of gynaecological cancer mortality and frequently evades immune surveillance. Major histocompatibility complex class II (MHC-II) molecules are essential for antigen presentation, yet their expression is often suppressed in tumour cells. Promoter DNA methylation can silence gene transcription and may contribute to reduced MHC-II expression in ovarian cancer. Understanding this mechanism may help improve tumour immune recognition and immunotherapy responses.

Methods: Four human ovarian cancer cell lines with low baseline MHC-II expression (SKOV-3, KURAMOCHI, IGROV-1, and PEO-1) were treated with the DNA hypomethylating agent 5-aza-2′-deoxycytidine (5-AZA) at low (1 μM) and high (10 μM) doses, with dimethyl sulfoxide (DMSO) used as a control. Total RNA was extracted using TRIzol, DNase-treated, and reverse-transcribed to cDNA. Expression of HLA-DRA, HLA-DRB1, and TNF-α was quantified using RT-PCR and RT-qPCR, normalised to GAPDH, and analysed using the ΔΔCt method.

Results: HLA-DRA expression showed minimal response to demethylation across all cell lines. In contrast, HLA-DRB1 demonstrated cell-line-specific regulation, with significant upregulation in IGROV-1 following treatment with 10 μM 5-AZA and a strong dose-dependent increase in PEO-1 cells. TNF-α expression varied across models, with significant induction observed in KURAMOCHI, IGROV-1, and PEO-1 following demethylation treatment.

Conclusion: Promoter DNA methylation contributes to repression of MHC-II-related genes in ovarian cancer in a gene- and cell-line-specific manner, with HLA-DRB1 showing the strongest demethylation-dependent reactivation. These findings suggest that epigenetic modulation may enhance tumour immunogenicity in selected ovarian cancer subtypes and could potentially improve responses to immunotherapy when used in combination strategies.

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