

CORRECTION

Correction: *PRDM6* promoter methylation as a potential epigenetic biomarker in BRCA-associated ovarian cancer

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Following correspondence with the authors, several issues were identified in the original article [1]. After careful review and confirmation by both the authors and the Editorial Office, the following corrections have been made. The scientific conclusions of the original article remain unchanged.

1. Some references were replaced with more appropriate citations. Minor textual and formatting corrections were also performed throughout the manuscript.

Original sentence (Introduction):

“However, existing studies provide insights into its potential involvement in cancer biology through its functions in cell cycle regulation and the epithelial-to-mesenchymal transition (EMT) [10, 12, 13].”

Revised sentence:

“Although the role of *PRDM6* in OC remains poorly characterized, previous studies in different cancer models have highlighted the importance of EMT-related pathways and cell cycle regulation in tumor progression [10, 12, 13].”

Original sentence (Discussion):

Chronic inflammation is a known contributor to neoplastic progression through mechanisms that promote cellular proliferation and resistance to apoptosis. These inflammatory processes may also drive DNA methylation changes in benign ovarian tissues [39]. In addition to inflammatory signaling, hormonal regulation plays a pivotal role in shaping the epigenetic profile of ovarian tissue. Hormonal cycles influence gene expression via methylation modifications, and disruptions in hormonal homeostasis may increase susceptibility to neoplastic transformation, even in benign neoplasms [39]. For instance, in benign mucinous cystadenomas, the complexity of methylation regulation is compounded by histopathological heterogeneity and microenvironmental influences such as localized inflammation [40].

Revised sentence:

Chronic inflammation is a known contributor to neoplastic progression through mechanisms that promote cellular pro-

liferation and resistance to apoptosis. These inflammatory processes may also drive DNA methylation changes in benign ovarian tissues [38]. In addition to inflammatory signaling, hormonal regulation plays a pivotal role in shaping the epigenetic profile of ovarian tissue. Hormonal cycles influence gene expression via methylation modifications, and disruptions in hormonal homeostasis may increase susceptibility to neoplastic transformation, even in benign neoplasms [34]. For instance, in benign mucinous cystadenomas, the complexity of methylation regulation is compounded by histopathological heterogeneity and microenvironmental influences such as localized inflammation [39, 40].

Original sentence (Discussion):

PRDM6 has been shown to regulate transcriptional programs associated with cell fate, and its methylation-induced silencing could disrupt standard control over the Wnt/ β -catenin pathway, which is known to sustain stemness and promote chemoresistance in cancers, including breast tumors [47–49].

Revised sentence:

PRDM6 has been implicated in transcriptional regulation associated with cell fate determination, while dysregulation of Wnt/ β -catenin signaling has been widely associated with tumor progression, stemness, and therapy resistance in multiple cancer types [47–49].

Original sentence (Discussion):

Furthermore, the loss of *PRDM6* function through methylation may derepress EMT-promoting transcription factors, such as snail family transcriptional repressor 1 (*SNAIL1*) and twist family BHLH transcription factor 1 (*TWIST1*), thereby facilitating cellular plasticity and invasiveness [29]. These molecular events could represent adaptive tumor strategies to overcome therapeutic pressures, supported by compensatory activation of Wnt and EMT pathways in BRCA-mutant malignancies [29, 52].

Revised sentence:

Furthermore, the loss of PRDM6 function through methylation may derepress EMT-promoting transcription factors, such as snail family transcriptional repressor 1 (SNAIL) and twist family BHLH transcription factor 1 (TWIST1), thereby facilitating cellular plasticity and invasiveness [12]. These molecular events could represent adaptive tumor strategies to overcome therapeutic pressures, supported by compensatory activation of Wnt and EMT pathways in BRCA-mutant malignancies [12, 52].

The revised references are listed below:

[12] Kim K, Ryu TY, Jung E, Han TS, Lee J, Kim SK, *et al.* Epigenetic regulation of SMAD3 by histone methyltransferase SMYD2 promotes lung cancer metastasis. *Experimental & Molecular Medicine*. 2023; 55: 952–964.

[13] Matthews BG, Bowden NA, Wong-Brown MW. Epigenetic mechanisms and therapeutic targets in chemoresistant high-grade serous ovarian cancer. *Cancers*. 2021; 13: 5993.

[29] Duan X, Xing Z, Qiao L, Qin S, Zhao X, Gong Y, *et al.* The role of histone post-translational modifications in cancer and cancer immunity: functions, mechanisms and therapeutic implications. *Frontiers in Immunology*. 2024; 15: 1495221.

[38] Vezzani B, Carinci M, Previati M, Giacobazzi S, Della Sala M, Gafà R, *et al.* Epigenetic regulation: a link between inflammation and carcinogenesis. *Cancers*. 2022; 14: 1221.

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[51] Arend RC, Londoño-Joshi AI, Straughn JM III, Buchsbaum DJ. The Wnt/ β -catenin pathway in ovarian cancer: a review. *Gynecologic Oncology*. 2013; 131: 772–779.

[53] Wu Z, Lepcha TT, Zhou D, He Z, Fiches GN, Park Y, *et al.* Analysis of head and neck cancer scRNA-seq data identified PRDM6 promotes tumor progression by modulating immune gene expression. *Frontiers in Immunology*. 2025; 16: 1596916.

[54] Davis CA, Haberland M, Arnold MA, Sutherland LB, McDonald OG, Richardson JA, *et al.* PRISM/PRDM6, a transcriptional repressor that promotes the proliferative gene program in smooth muscle cells. *Molecular and Cellular Biology*. 2006; 26: 2626–2636.

2. Corrections in Section 3.2 (Patient Demographics)

This revision was made to clarify that the BRCA-related

methylation analyses were performed using the evaluable cohort consisting of 375 ovarian cancer patients rather than the total cohort of 387 patients.

Original sentence:

“Among OC patients, 23.8% (n = 92) were found to carry a BRCA mutation (either BRCA1 or BRCA2), while 76.2% (n = 295) were BRCA mutation-negative.”

Revised sentence:

“Among OC patients with available PRDM6 methylation data, 24.3% (n = 91) were BRCA mutation-positive, whereas 75.7% (n = 284) were BRCA mutation-negative.”

3. Corrections in Section 3.3 (PRDM6 methylation analysis)

Similarly, the PRDM6 methylation frequency was incorrectly reported as 52.0% based on the total cohort of 387 patients. Using the correct evaluable methylation cohort of 375 patients, the updated frequency is 53.90% (202/375).

Original sentence:

The methylation frequency of the PRDM6 gene was: 52.0% in OC patients (n = 375, the total number of OC patients was 387; however, 12 individuals were excluded from the study due to unavailable expression data), 60.0% in the individuals with benign ovarian disease group (n = 50), and 37.0% in healthy controls (n = 100) (Fig. 1).

Revised text:

“The methylation frequency of the PRDM6 gene was: 53.90% in OC patients (n = 375, the total number of OC patients was 387; however, 12 individuals were excluded from the study due to unavailable expression data), 60.0% in the individuals with benign ovarian disease group (n = 50), and 37.0% in healthy controls (n = 100) (Fig.1).”

The authors sincerely apologize for these errors and any inconvenience caused to readers. The corrections have been made to improve the accuracy of citations and clarify the study cohort used for specific analyses.

REFERENCES

- [1] Comert N, Sukruoglu Erdogan O, Celik Demirbas B, Kilic Erciyas S, Dinc A, PasinÖ, *et al.* PRDM6 promoter methylation as a potential epigenetic biomarker in BRCA-associated ovarian cancer. *European Journal of Gynaecological Oncology*. 2025; 46: 21–30.