The transcriptional effect of histone deacetylase inhibitor Oxamflatin on the E-cadherin expression in gastric cancer cell line

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Introduction & Aim: Gastric cancer is among the leading causes of cancer-related death, and the symptoms are commonly characterized in advanced stages. Histone acetylation is among the most important epigenetic alterations occurring during cancer development. In addition, reduced E-cadherin expression is a major contributor in the process of tumor cell invasion and metastasis. Oxamflatin is a histone deacetylase inhibitor that has been suggested as a promising anti-tumor agent; yet its effect on the viability and invasion of gastric tumor cells is unclear. We aimed to assess the impact of oxamflatin on the viability of gastric tumor cells and expression of E-cadherin as a marker of tumor invasion susceptibility.

Methods: In this study, MKN-45 cells were treated with 1, 2.5 and 5 mM oxamflatin and followed by MTT assay after 24–48 h of incubation. To determine E-cadherin expression in treated cells, total RNA was extracted and reverse transcribed to complementary DNA, followed by quantitative real-time PCR. MTT results showed that the viability of MKN-45 cells declines with increasing concentrations of oxamflatin.

Results: The results of quantitative real-time PCR showed increased expression of E-cadherin following treatment with oxamflatin at the concentration of 2.5 mM compared with 1 mM. The present results showed that oxamflatin can induce E-cadherin expression and also reduce cell viability in the MKN-45 cell line.

Conclusion: On the basis of these findings, oxamflatin can be further considered for the prevention of tumor metastasis.

Key words: Oxamflatin, E-cadherin, MKN-45 cell line, gastric cancer