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Epigenetic modulation of Aurora kinases A and B by 5-aza-2'-deoxycytidine in breast cancer cells

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Introduction: Aurora kinases A (AURKA) and B (AURKB) play crucial roles in cell cycle regulation and are frequently over-expressed in various types of cancer, including breast cancer. Epigenetic alterations, such as deoxyribonucleic acid (DNA) methylation, may contribute to the dysregulation of these genes. **Objective:** This study aimed to evaluate the modulatory effects of 5-aza-2'-deoxycytidine (5-aza-dC), a DNA methylation inhibitor, on the expression of AURKA and AURKB genes in luminal (MCF7) and human epidermal growth factor receptor-type 2-positive (HER2+) (BT474) breast cancer cell lines treated with different concentrations of the demethylating agent. **Methods:** Cells were cultured under standard conditions (37°C, 5% CO₂) and treated with 5-aza-dC at various concentrations (10, 20, 30, and 50 µM) for 24, 48, and 72 hours. After treatment, total ribonucleic acid (RNA) was extracted and converted into complementary (c)DNA, followed by gene expression analysis via reverse transcription-quantitative polymerase chain reaction (RT-qPCR). Normalization was performed using housekeeping genes, and data were statistically evaluated using analysis of variance (ANOVA), with Tukey's post hoc test, and with $p < 0.05$ considered significant. **Results:** The experiments revealed a dose-dependent response profile in both cell lines. In MCF7, a significant reduction in AURKA expression was observed at 30 µM and 50 µM, mainly after 48 hours and 72 hours ($p < 0.01$). For AURKB, a significant reduction was observed starting at 20 µM at 72 hours ($p < 0.05$). In the BT474 cell line, both AURKA and AURKB showed a significant decrease in expression at 50 µM, especially at 72 hours ($p < 0.001$). **Conclusion:** Treatment with 5-aza-dC negatively modulated AURKA and AURKB expression in a dose- and time-dependent manner, indicating a possible antitumor epigenetic effect. These findings suggest the potential of 5-aza-dC as an adjuvant therapeutic agent in breast cancers characterized by Aurora kinase overexpression.

Keywords: breast neoplasms; epigenetic; AURKA.