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**The Effect of Glycosylation Inhibitor; Deoxynojirimycin (DNJ) and Deoxymannojirimycin (DMJ) on Osteosarcoma Invasiveness**

1,2Mustafa, S. H\*., 2Muhamad, M. and 2Ab-Rahim, S.

*1Institute of Medical Molecular Biotechnology (IMMB), Selangor; 2Faculty of Medicine, Universiti Teknologi MARA, Selangor.*

**ABSTRACT**

Osteosarcoma (OS) is a primary malignant bone tumor that is observed to be resistant to currently used therapy leading to disease recurrence and metastasis. Metastasis process is mediated by the cell surface receptor, called the integrin that allows the interaction between cells and extracellular matrix (ECM). Recently, integrin glycosylation has been shown to play a crucial role in cancer progression. Deoxynojirimycin (DNJ) and deoxymannojirimycin (DMJ), are glucose analogs that are likely inhibits the activity of α-glucosidase I and II, and class I α-1-2 mannosidase respectively which are the very first phase of protein glycosylation. Thus; manipulation of cell-ECM by interfering with the glycosylation process of integrin may become a potential target for the regulation of OS. In this study, glycosylation alterations of integrin were carried out by inducing osteosarcoma cell line (MG-63) with 0.5 mM DNJ and 0.5 mM DMJ separately for 24 hours. Then, the invasion activity of induced cells was determined using cell invasion assay kit. The induction of MG-63 cell line with 0.5 mM DNJ led to a decrease in cell invasion capacities by 12.5%. However, the induction of MG-63 cell line with 0.5 mM DMJ causing the invasion capacities of the cells to increase by 287.5%. Inhibiting glycosylation in MG-63 cell lines to regulate OS invasiveness by DMJ and DNJ resulted in different invasion capacities of the cells. This study showed that the interference of class I α-1-2 mannosidase activity by 0.5 mM DMJ in the glycosylation process of OS cell lines did not able to decrease the invasion rate through the extracellular matrix layer. However, targeting α-glucosidase I and II activity by 0.5 mM DNJ may help in reducing the invasion capacity of the cells, thus preventing further formation of integrin that can cause abnormal behavior of tumor cells.