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Annals of Experimental Biology, 2022, 10 (5): 63-64

(http://www.scholarsresearchlibrary.com)

ISSN:2348-1935

Analysis of VIM Expression Across Cancer Types in Human Cancer Tissues

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***Received:** 10-Sep-2022, Manuscript no. AEB-22-82010; **Editor assigned:** 12-Sep-2022, Pre QC no AEB-22-82010 (PQ); **Reviewed:** 22-Sep-2022, QC no AEB-22-82010 (Q); **Revised:** 24-Sep-2022, Manuscript no. AEB-22-82010 (R); **Published:** 29-Sep-2022*

ABSTRACT

More than 19 million cancer cases are expected to be detected in 2020. Early detection is one of the best routes for patients to more efficient therapy with the potential of being cured. As an intermediate filament protein, vimentin (VIM) is widely expressed in mesenchymal cells. Numerous biological processes, including cellular component assembly and biogenesis, metabolic processes, and biological regulation, are controlled by VIM. A growing body of research suggests that the VIM gene's (VIM) expression is messed up in carcinomas during the epithelial-mesenchymal transition. Here, using data from The Cancer Genome Atlas, we performed an extensive analysis of the VIM gene expression and promoter methylation profile in 19 different cancer types (TCGA).

Keywords: Cancer, Tissue

INTRODUCTION

A single copy of the vimentin gene (VIM) can be found on the short arm of chromosome 10 (10p12) (Rittling and Baserga 1987). Three distinct components that control the expression of the gene make up the VIM promoter. Vimentin, a 57 kDa polypeptide that VIM genes for, is one of type III Intermediate Filament (IF) protein family's most frequently expressed and highly conserved members. Vimentin's major biological role is to protect cells from damage and maintain cellular integrity. Vimentin may also join forces with other adaptor proteins and cell signaling molecules to form a complex.

Being a multifunctional protein with the ability to interact with numerous other proteins, the VIM product has the potential to regulate a variety of physiological processes. Vimentin may relocate outside the cell during pathological conditions such as tissue damage, inflammation, or cancer, according to mounting evidence from recent years. These results show that VIM may be a viable diagnostic biomarker for autoimmune diseases (like Crohn's disease), viral infections (like HIV), and severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) in addition to cancer. Vimentin is a diversely expressed IF protein that is abundantly expressed in a wide range of cells and tissues.

Epithelial-Tomesenchymal Transition (EMT) is one of the primary molecular pathways involved in oncogenesis and the promotion of cancer progression. Cells lose their epithelial features, particularly polarity, during the EMT process and have a migratory behavior. They change in form and show enhanced motility as a result. The interaction between normal and neoplastic cells (such as direct contact, and production of active chemicals) results in a change in the tumor phenotype during EMT as a result of continuous inflammation and hypoxia. The EMT procedure is extensively discussed in the literature and is crucial to the progression of cancer. Cells first develop the capacity to migrate, allowing them to isolate themselves from the rest of the population.

Vimentin may be essential for the growth of cancer and the immune system's subsequent response, according to recent studies. Vimentin may play a role in the death of neutrophils and lymphocytes, according to research. Vimentin has an interesting involvement in lymphocyte apoptosis, which may be used as a serum biomarker for sepsis prognosis. Vimentin has also been suggested

as a potential cellular target for the treatment of COVID-19, a coronavirus disease. Vimentin is thought to act as a co-receptor for the entry of SARS-CoV-2 into cells, hence COVID-19 patients can be treated with medications that reduce vimentin expression. On the one hand, if VIM is overexpressed in particular cancer types, it might be a useful biomarker with diagnostic implications.

On the other hand, we believe that the reduced expression of VIM may be crucial for treatment strategies. Only bioinformatics analyses from online databases were used in this study. Vimentin during carcinogenesis has to be studied further to improve experimental and clinical therapy methods for cancer patients. Evaluation of the expression profiles of many genes in healthy and cancerous tissues may be more useful in the diagnosis and prognosis of tumors. Finding markers that enable quick detection of this process can be a breakthrough in cancer treatment because the spread of cancer cells is thought to be one of the most significant causes of disease progression, treatment failure, and ultimately patient mortality. The development of customized treatments may be made possible by a thorough strategy that considers the relationships between molecular profiles and the spread of cancer cells. Last but not least, researchers might investigate new signaling networks in cancer or other disorders, including viral infections, using the databases revealed in this study

CONCLUSION

In the current work, we carried out a comprehensive examination of VIM expression in particular types of cancer using numerous online bioinformatics platforms and web tools (TCGA UALCAN, GeneMANIA, STRING, and cBioPortal). We established that VIM is overexpressed, particularly in females, in CHOL, GBM, HNSC, and KIRC. The examination of the relationship between VIM expression and gene promoter methylation revealed that promoter methylation may control VIM expression in three different cancer types (KIRC, KIRP, and BRCA). In certain malignancies, our study reveals that VIM overexpression may be a potential new diagnostic biomarker. In addition, additional research into VIM may reveal it to be a possible therapeutic target. In addition to providing access to local blood arteries and lymph nodes, the transition process also permits cells to keep moving away from the initial site and to form micro-metastases. The production of mesenchymal cytoskeletal proteins, such as vimentin, which triggers the formation of focal adhesion complexes and facilitates cell motility, is associated with the development of the mesenchymal phenotype.