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Interaction of Tetraspanin KAI1/CD82 and Tetraspanin CD151 in Hepatocyte Growth Factor c-Met Receptor Regulation in Prostate Cancer Cell Lines

Marina Ibarra
Grand Valley State University

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Marina Ibarra
McNair Scholar

Suganthi Sridhar
Faculty Mentor

Cancer is a complex disease that is not well understood and alarmingly, according to a recent UK study published in 2015 in the British Journal of Cancer, one in two people will be diagnosed with the tragic disease. Understanding the types and stages of cancer leads the way toward generating effective and efficient treatments. The goals of standard cancer treatments today are incredibly aggressive in order to rid the body of the cancerous cells. These forms of treatment initially appear ideal; however, after further investigation, those methods come with unforeseen consequences and do not take other factors into consideration. In order for a treatment plan to be successful, it must deviate from the "one size fits all" mentality and consider the fact that cancer varies with each individual in regard to genetic mutations, specific protein expression, severity, location, type and etiology of the disease. Today's standard treatment methods include surgery, chemotherapy, radiation and immunotherapy. A problem that is common in patients receiving these aggressive treatments is that their healthy cells are compromised causing adverse effects. The least recognized yet potentially the most effective of all methods is targeted drug therapy. Targeted drug therapies work by attacking specific cellular signals used by cancer to spread, grow and communicate without affecting healthy cells. For example, through previous studies the cellular signaling pathway of the gene CD82 was discovered to suppress cancer tumor metastasis per regulation of proteins: c-Met and Src kinases. During the studies, CD82 interacted with the gene CD151 and was found to have the opposite effects on metastasis compared to CD82. This pathway could ideally be targeted for the use of drug therapy; therefore, the research on this pathway is imperative.

The research focuses on observing changes in protein interactions involved in metastatic prostate cancer cells: CD151's association with C-met in the presence and absence of CD82. The results indicate that the overexpression of CD82 in a metastatic prostate cancer cell line significantly reduces the association and regulation between CD151 and C-met and thus inhibits metastasis at the molecular level. The effectiveness of the study was tested using two techniques that measure the changes in protein level expression called immunofluorescent staining and gel electrophoresis. Immunofluorescent staining allows for the protein expression on the cell surface to be visualized through fluorescent biomarkers. Three separate sets of two proteins were stained with antibodies to be evaluated through immunofluorescent staining. Set 1: CD82 with C-met; Set 2: CD151 with C-met; and Set 3: CD82 with CD151, cellular surface expressions were studied. Understanding the relationship between these three proteins propose a greater potential for understanding the mechanism of metastasis. Analysis of the specific molecules contributing to metastasis could orchestrate the development of successful targeted drug therapies as treatment for cancer. The uniqueness of this study allows for other researchers to piece the discovered information with known information to create a broader understanding in the field of cancer biology.