

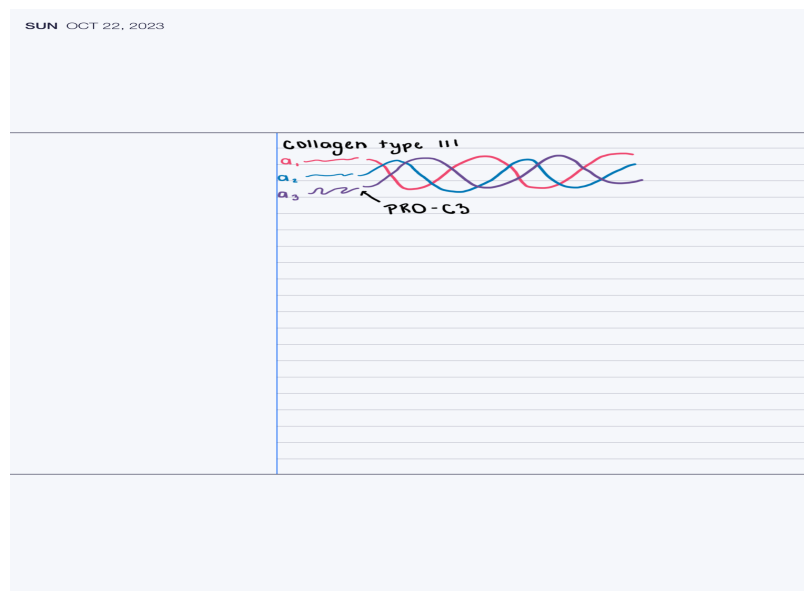
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Scientific Literacy Essay

Background Essay

Squamous cell carcinoma, let's talk about the most common skin cancer. This is a specific type of cancer that starts on the skin but has the potential to grow in size and spread throughout the body if not properly treated. It starts as squamous cells which are the middle and outer layer of our skin. Now when we're taught to apply sun-screen while in the sun especially in the summer, this is why as most of the carcinomas are caused by too much UV radiation. However, according to the Mayo Clinic, those of black and brown skin are more likely to develop carcinomas in areas that aren't directly impacted by the sun. Some physical changes that may indicate a carcinoma are a firm bump on the skin that can range in colors from skin color to pink and red to black and brown; rough patches on the lip that develop into open sores, or raised patches or wart like sores. As I previously stated, this is the most common type of skin cancer so if there are any concerns with changes in the skin that do not heal in a couple months, it is suggested that you make an appointment with your doctor for further evaluation.

What is collagen III and why is it relevant? So to begin with collagen type III is a fibrillar collagen, contrasting most collagens that we associate with hair and nail growth, and healthy skin. This collagen is produced by fibroblasts and other mesenchymal cell types. It's a structural component in the elasticity of our hollow organs including large blood vessels, uterus, bowel, and stretching tissues. It contributes to a variety of inflammation concerns such as lung injuries, a variety of liver diseases, kidney fibrosis, hernias, and vascular disorders.



Your bodies have so many elements that are important to our growth and development, but inversely can contribute to the downfall of our health and collagen III is an example of one of those elements. Collagen, in summary, influences cancer cell behavior. Cancer cells have the ability to reshape collagen, and through adhesion they form cell-collagen loops which will help connect cancer cell receptors. So metaphorically, it is like adding fuel to the fire as collagen aids in cancer growth and migration. In tissues of low oxygen, this progression is intensified.

Essay

To begin, ECM; short for extracellular matrix, is a meshwork of proteins that can anchor tumor cells and provide signals that regulate tumor cell behavior. In metaphorical terms, they are adding fuel to the fire. Studies, research, and experiments have been conducted to get a better understanding of ECM, and how that knowledge can aid in reducing tumor growth. True tumor cell growth, on a cellular level is known as proliferative tumor cell lines. While tumor cells that “don’t grow” are known as dormant tumor cell lines, and they can be defined as cells of reversible growth arrest.

According to Di Martio, there are a few notable architectural differences in the changes in the ECM for dormant and proliferative tumor cell lines. In dormant tumor cell lines, the ECM produced is rich in type III collagen fibers. It has been shown that there is a lower degree of linear organization of the collagen fibers, as it has been described as wavy collagen fibers across the three different dormancy models. In the proliferative tumor cell lines, it has been shown that there is a much higher degree of linear orientation, especially those who are advanced. In table 1 figure a, we can visually view the differentiation in these orientations of fibers. Proliferative is much more linear and has relatively much more organization compared to dormant cell lines. This is consistent with single cells versus metastases, as “the cell cycle progression of these correlates with a shift in ECM organization toward a more aligned ECM.” Within the cell cycle, metastatic tumors show a mixed population of the cancer cells in different phases of the cell cycle. Whereas, single cells were confirmed to be in G0/G1 meaning they were of poor linear orientation. We similarly see this in dormant cells.

Based on previous data provided in the study, I had already formed inferences on the relationship collagen III would have with these proliferative T-HEp3 cells. We see this in the results of the research where there is a direct correlation between collagen III and the tumor size. When the T-HEp3 cell lines were injected with the type III collagen, there was a very considerable difference in the tumor growth size as they were much smaller than when tested against other collagens. When this was tested on nude mice subjects, T-HEp3 cells were still injected with collagen III and still observed that there was significantly slower growth of those tumors. In figure 5a, we can see that there are four different tumors that were injected with four different collagens. It is very evident based on the data table as well as the D6 Cam photos that T-HEp3 injected with collagen resulted in the smallest sized tumor.

To conclude, collagen II was able to successfully decrease the number of cells going through S/G2. In those cells of high amounts of type III collagen, the tumor is evidently driven into a state of dormancy. This controls the number of cells going through those growth phases. In addition, in previous research, it was shown that low amounts of collagen III was correlated with an increase in growth of proliferative breast cancer tumors; and the collagen could aid in preventing relapse.

Sources

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