

# **Can the intracellular proteins in platelets and immune cells influence cancer cells ability to metastasize?**

By Kjersti Marie Hjelle | May 2015

Cancer researchers have for decades tried to solve the mysteries associated with the complex nature of tumor development. Traditionally, cancer research has focused on revealing the mechanisms inside the cancer cells. However, cancer research is changing towards regarding tumors as complex tissues consisting of other cell types that influence the progression and behavior of a tumor through cross-talk with the cancer cells. A tumor can be seen as a wound that lacks the normal regulation mechanisms for secreting factors that recruits blood vessels, platelets and other immune cells. Recent research has revealed that the immune cells and platelets, that are supposed to kill abnormal cells and pathogens, actually can be reprogrammed by the cancer cells to facilitate metastasis to other organs through the blood stream. Platelets are thought to form aggregates with cancer cells in the blood stream, producing a protective shield around the cancer cells and facilitating attachment to the blood vessel wall. Upon contact with cancer cells, platelets secrete factors that recruit immune cells to the platelet-cancer cell aggregates. These immune cells secrete factors that can make the blood vessels permeable, facilitating cancer cell migration from the blood stream and into the tissue.

Being both a part of the surrounding tumor tissue, and possibly facilitating metastasis of cancer cells, the factors secreted by platelets and immune cells seem to play an important role in tumor progression and behavior. This knowledge motivated a study on the secretory granules in platelets and immune cells. A protein, called serglycin, functioning as a core for other proteins to attach inside the granules of platelets, immune cells and even cancer cells, is an interesting target to study in the quest of exploring the host-to-cancer cell cross-talk. In this study, blood samples were obtained from tumor bearing mice without serglycin, to see if the loss of serglycin affected the amount of granulocytes, monocytes and platelets in the blood stream. Tumor tissue was also obtained from these animals to investigate the amount of granulocytes, macrophages and platelets in the tumor. No significant difference was found between the mice with and without serglycin. This triggered a study exploring whether the function of these cells were changed instead. A test of monocytes derived from the bone marrow of the mice suggested that two factors, one responsible for blood vessel survival and one responsible for facilitating migration of cells, seemed to be decreased in the mice without serglycin, indicating a changed function of the monocytes.

Despite the fact that no large changes were observed in the amount of immune cells upon loss of serglycin, it would be interesting to further explore the other impacts serglycin may have on immune cells and platelets involved in the formation of platelet-cancer cell aggregates, and on the organization of the matrix surrounding the tumor cells. Understanding the complex nature of how cancer works is the key to develop treatment methods that are better than what is available today for cancer patients. Pursuing tentative mechanisms and interactions like the ones described above contributes to this development.

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