Multiple myeloma (MM) is a type of cancer originating from blood cells called plasma cells. Their physiologial function is to fight infections by producing antibodies which recognize pathogens, like bacteria or viruses. Typically, plasma cells live in the bone marrow. When they become mutated and divide too quickly, they start destroying their environment, causing bone damage. They also secrete extremely large amounts of antibodies, which start forming deposits in multiple organs, damaging them as well. For these reasons, this aggressive disease is often fatal and new treatment methods are urgently needed.

Many reports suggest that the interactions between multiple myeloma cells and the vessels in the bone marrow are necessary for the development of the disease. They are both able to communicate with each other and exchange certain intercellular signals through various proinflammatory and vessel growth-promoting proteins (called cytokines) produced by both MM and the vessel cells (also known as endothelial cells). We have found that both MM and endothelial cells produce large amounts of a possible drug target – PIM kinases. PIM kinases are responsible for coordination of the cytokine-mediated communication between cells. They are produced by cells after receiving signals telling them to divide, avoid cell death, and form more blood vessels. Therefore, an inhibitor of PIM kinases could be beneficial for patients suffering from MM. We hypothesize that blockade of PIM kinases could disrupt the interactions between MM and endothelial cells and slow down the progression of the disease.

First, we will try to observe how often PIMs are present in the blood vessels of bone marrow samples from patients with multiple myeloma. To do that, we will use a special microscope which will allow us to see PIM kinases inside endothelial cells with the help of fluorescent staining. Then, we will analyze the effect of PIM inhibitor on basic processes needed for efficient vessel formation using cells cultured *in vitro*. In addition, we will simulate the bone marrow environment by culturing MM and endothelial cells together. In this experimental setup, we will try to dissect what kind of changes in intercellular communication are caused by PIM inhibitor. Finally, we will check if application of this potential drug is able to stop formation of vessels induced by MM cells. We hope that this study will shed new light on the mechanisms of development and progression of multiple myeloma, and identify a new method of treating this disease.