Cancer is the leading cause of death worldwide. The search for optimal, targeted therapies with limited side effects, which would efficiently eliminate the disease or significantly improve the length and quality of cancer patient's life, is urgently needed. Among promising therapeutic directions are those, which exploit the mechanisms of immune defense. Our proposal follows this trend, as we would like to expand the current knowledge about the role of neutrophils in cancer. Specifically, we would like to focus on neutrophil extracellular traps (NETs), 3D structures released by leukocytes in response to stress factors. NETs have been discovered and described as the unique antibacterial mechanisms of neutrophils; however, recent data indicate their much wider function than originally assumed. Their role was only recently noticed in the processes of carcinogenesis as structures displaying significant impact on cancer development and metastases. It is suggested that the reduction of NETs formation might be an important element of effective anticancer therapy. Therefore, in the current project, we propose that proteases can induce the formation of NETs in the tumor environment. Our hypothesis is based on the observation that proteolytic enzymes are commonly identified in cancers. Moreover, their high concentration was documented in breast cancer, which is going to be a model in our study. We plan to identify the most active proteolytic enzymes and describe the signaling pathway, which leads to the formation of NETs. In the next stage, we will determine how the NETs formed in the response to proteases will affect the biology of cancer cells, as well as tumor development and metastasis. We will conduct the above studies *in vitro* and *in vivo* using an animal model of breast cancer. We believe that the results obtained will expand our basic understanding of the biology of cancer development. Furthermore, focusing our work on breast cancer, the most common cancer in women, will bring us closer to improving therapeutic options, especially in the case of its most aggressive forms that are resistant to existing targeted therapies.