Sepsis or systemic inflammatory response is a reaction of the body to pathogens such as bacteria, viruses or fungi. Sepsis is characterized by septic shock and multi-organ failure which ultimately lead to high mortality in up to 30-55% cases. The lack of specific therapy and severe course of this infection make systemic inflammation one of the serious health problems world-wide. One of the most important cells of the immune system involved in sepsis are neutrophils. They have the ability to eliminate pathogens, by release of bactericidal proteins outside the cell, intracellular killing, or by ejection of neutrophil extracellular traps (NETs). NETs consist of decondensed DNA to which proteins/enzymes with bactericidal properties are attached. Presence of NETs in the early stages of sepsis favors the immobilization and killing of pathogens. But NETs are not the only structures that are released by neutrophils, as the cells can also secrete extracellular vesicles (EVs). They are microstructures transporting numerous proteins and genetic material (nucleic acids) to target cells which may result in target cell transformation. The release of EVs is observed in both physiological and pathological conditions, including sepsis, but their impact on systemic inflammation is not fully understood. Namely, it is not clear if they suppress or stimulate the inflammatory response. Moreover, EVs can be released by numerous cells, including also other types of leukocytes. Importantly, both EVs and NETs are anti-bacterial and can be released in similar pathological circumstances, and additionally EVs are also found in NETs. Therefore, the aim of the current project is to explain if the two structures can induce and/or interact with each other. And if so, how can this impact sepsis? Would it help or augment the course of systemic inflammation? The project will address these questions by studying EVs and NETs *ex vivo* (on isolated cells, outside of the body) and *in vivo* (in blood vessels of mice). Diverse techniques will be employed and they will include intravital microscopy, and flow cytometry and NTA (nanoparticle tracking analysis). The results of the project will allow to determine the interaction of the two key but poorly understood mechanisms in the course of sepsis - EV and NET secretion. Expanding the current state of knowledge on mechanism of sepsis will allow a deeper understanding of its complex nature. Furthermore, it will be verified if EVs and NETs could be used as potential early markers of sepsis, and if their manipulation could enrich the current therapeutic strategies.