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Damage-associated molecular patterns in systemic inflammation diseases, friends or enemies

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DAMPs are endogenous structure which is released upon cell death and/or cell activation. Main DAMPs originate from nucleus and mitochondria such as: cell-free DNA (cfDNA, e.g. nucleosomes), DNA-binding proteins (histones) and mitochondrial DNA (mtDNA) respectively. Upon ligation of pattern recognition-receptor (PRR) DAMPs may elicite a host immune response by the activation of cells. DAMPs have been revealed to play a crucial role in host immune response resulting in systemic inflammation. Nucleosomes levels in sepsis correlate with severity and fatality. Interestingly, DAMPs are considered to propagate systemic inflammation in sepsis despite efficient neutralization of the causing microorganism, which may explain the still high mortality of severe sepsis. In analogy to sepsis, same mechanisms are observed in patients infected by SARS-CoV-2 developing severe systemic inflammation. Given the role of DAMPs in diseases characterized by systemic inflammation, therapeutic strategies to prevent release and/or neutralize DAMPs gained more and more interest the last years. The seminar will touch the principles of DAMPs; their role in the pathogenesis of different diseases characterized by systemic inflammation, such as sepsis or Covid-19 and will close with therapeutic strategies to neutralize DAMPs.

Speaker Biography

Tiphaine Ruggeri works in Inselspital, University of Bern. Her current research interests in the immune system modulation in a systemic inflammation context. Her aim is to target the inflammatory response in sepsis model.

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