Mediators of Inflammation

Hindawi

Special Issue on

Toll-Like Receptor Activation in Experimental and Clinical Heart Failure

CALL FOR PAPERS

Patients with heart failure (HF) are characterized by systemic inflammation, as evidenced by increased levels of a variety of cytokines, while the degree of elevation is related to the severity of the disease. Although the exact mechanism of systemic inflammation is unknown, a solid body of evidence shows that inflammation plays a role in both the development and the progression of HF. There is a wealth of information about the role of inflammatory cells and pathways during acute injury and the reparative processes that are subsequently activated.

However, the innate immune system can detect the highly conserved, relatively invariant structural motifs of pathogens. The most important innate immune receptors, Toll-like receptors (TLRs), characterize first line defense against infectious pathogens and play a pivotal role in initiating and shaping innate and adaptive immune responses.

TLRs are expressed not only in immune cells, but also in cardiovascular cells. TLRs can also recognize endogenous ligands and play a role in mediating cardiomyocyte cell death and myocardial functioning. Moreover, endogenous TLR ligands are often mentioned as alarmins and serve as initial warning signals to innate and adaptive immune systems. The activation of the innate immune system not only is based on the recognition of pathogen-associated molecular patterns (PAMPs) but also relies on the existence of danger signals or danger-associated molecular patterns (DAMPs), released by injured cells. The endogenous alarmins and exogenous PAMPs are subgroups of the larger category of DAMPs.

Furthermore, TLRs could be a link between cardiovascular diseases and the immune system. There exists solid evidence that TLR activation contributes to development and progression of acute cardiac injury and heart failure.

This special issue was initiated to gain a deeper insight into the link between the activation of the immune system and advanced HF. We invite investigators to contribute original research articles as well as review articles that will help in understanding the interplay of the immune system and advanced HF in various experimental models and in the clinical setting. Both experimental and clinical papers are welcome.

Potential topics include but are not limited to the following:

- Novel role of the myeloid differentiation factor 2 (MD-2) and its direct cell specific stimulation of the TLR4/ NFkB pathway in experimental and clinical heart failure (myocardial infarction, dilated cardiomyopathy, and hypertensive HF)
- Validation of inhibition or substitution of the TLR-MD2 complex as a potential therapeutic strategy in inflammatory and ischemic HF phenotypes
- Specific impact of innate immune receptors (e.g., Toll-like receptors, pattern recognition receptors (DAMPs, PAMPs)) in experimental heart failure models in different species and in the clinical setting
- Functional role of endogenous molecules, which activate TLR signaling and induce sterile inflammatory responses in HF: the majority of endogenous TLR ligands are extracellular matrix components such as fibronectin, heparan sulphate, biglycan, fibrinogen, oligosaccharides of hyaluronan, and hyaluronan breakdown fragments
- Identification of novel immunomodulatory targets in experimental and clinical heart failure and their therapeutical potential
- Analysis of expression patterns of innate and adaptive immune receptors in experimental and clinical heart failure settings (e.g., time course analyses)
- Interplay of TLR-mediated immune response and adverse cardiac remodeling in different heart failure models and in the clinical setting

Authors can submit their manuscripts through the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/mi/traec/.

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