PPAR Research

Special Issue on
Pathway of PPARg Activation during Pathogen Infection

CALL FOR PAPERS

Recent studies have shed light on the extensive interactions between the immune and metabolic activations of macrophages. In this context, PPAR activation has emerged as a potential target in regulating the processes associated with lipid metabolism and inflammatory response in leukocytes during intracellular pathogen infection.

The crosstalk pathway between innate immune receptor TLR2 and lipid-activated nuclear receptor PPAR has been demonstrated. The distribution of receptors on the membrane allows rapid and efficient coupling with ligands and effectors of the intracellular signaling system on PPAR activation mechanism. There is evidence that different receptors such as TLR2 and CD36 can cooperate on the signaling and divert the host response with increased expression and activation of PPAR through NF-κB-independent pathways, leading to upmodulation of lipid metabolism and downmodulation of macrophage response.

Notwithstanding the fact that different pathogens such as Helicobacter pylori, Pseudomonas aeruginosa, Klebsiella pneumonia, Escherichia coli, Mycobacterium tuberculosis, and Brucella abortus cause infection, they also induce significant morbidity, which requires treatment with multiple antibiotic combinations. The identification of PPAR, a well-characterized drug target, suggests that PPAR inhibitors can be a potential adjunct to antibiotic therapy.

We invite investigators to submit original research and review articles that will contribute to the identification of PPAR as a target of pathogen infection, as well as their potential for therapies.

Potential topics include, but are not limited to:

- Crosstalk between innate immune pathway and lipid-activated nuclear receptor PPAR pathway
- PPAR ligands and macrophage polarization
- PPAR in lipid metabolism and inflammatory response
- PPAR/NF-κB cofactors and signaling pathway
- PPAR as treatment target during pathogen infection

Authors can submit their manuscripts via the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/ppar/api/.

Lead Guest Editor
Patricia E. Almeida, Federal University of Juiz de Fora, Juiz de Fora, Brazil
pat.elaine.almeida@gmail.com

Guest Editors
Adriana R. Silva, Fundacao Oswaldo Cruz, Rio de Janeiro, Brazil
arsilva@ioc.fiocruz.br

Carlos H. Serezani, Indiana University, Indianapolis, USA
hserezan@iu.edu

Murugesan Rajaram, Ohio State University, Columbus, USA
rajaram.3@osu.edu

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