

Seminars in Biotechnology BTEC 591 & BTEC 691

“NOD-like Receptors in shaping adaptive and innate immune responses”

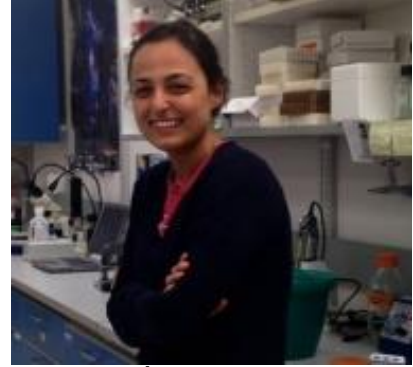
Thursday, October 14, 2021

13:30

Sürekli Eğitim Merkezi (SEM), Management Conference Room

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Ceren Çıracı received her PhD from Iowa State University and completed her post-doctoral studies at the University of Iowa Inflammation Program. She is currently an Associate Professor in the Molecular Biology Department at Istanbul Technical University. Dr Çıracı has an interest in innate and adaptive immunity, in particular the study of Nod-like Receptors (NLRs). Her lab currently focuses on the regulation of adaptive immune responses through the components of innate immunity based on the preliminary results that utilized in vivo mouse models of airway inflammation.

Abstract

The precise context in which the innate immune system is activated plays a pivotal role in the subsequent instruction of CD4⁺ T helper (Th) cell responses. Ever since their discovery, NLRs have drawn considerable attention for their ability to form multiprotein complexes called inflammasomes and also for their roles as NLRs, independent of inflammasome complexes. This talk will first report the finding from our recent publication about NLRP11 which is a member of the PYD domain containing, nucleotide-binding oligomerization (NOD)-like receptor (NLR) family. The true stimuli of NLRP11 is still remain unclear to date, so the current study built upon NLRP11 induction via adenosine stimulation and that activation can shape adaptive immune responses in a caspase-1 independent manner. Second part of the talk will focus on the preliminary findings we obtained from the population of knockout mice that are deficient in a member of inflammasome complex; NLRC4 (also known as IPAF). NLRC4 is composed of an N terminal CARD, a central NACHT domain and C-terminal LRRs domains. Recent studies on NLRC4 suggest that NLRC4 inflammasome complex can recognize numerous pathogens including *Salmonella*, *Shigella*, *Legionella*, *Listeria* and *Pseudomonas* resulting in the activation of caspase-1 and release of IL-1 β . Hence, we aim to examine the roles of NLRC4 inflammasome complex in the regulation of human eosinophils using in vitro systems to verify our in vivo findings.