Suhyeon Kim, J Clin Cell Immunol 2017, 8:7 (Suppl)
DOI: 10.4172/2155-9899-C1-046

Role of *Pellino 1* in the development of chronic inflammatory disease

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Pellino (PELI1) 1 is highly conserved in the various species and contains C3HC4 RING motif in its C-terminal domains and catalyzes ubiquitin chains of several key molecules linked to lysine 48 (K48) or 63 (K63) in cell signaling pathway. *PELI1* has been recently found to regulate Toll-like receptor (TLR) and T-cell receptor (TCR) signaling to NF-κB and thus contributes to the maintenance of self-tolerance and the production of pro-inflammatory cytokines. In addition, activation of B cell and T cell receptor-mediated signaling induces *PELI1* expression and activation. However, their physiological roles in other cell types and cell signaling pathways remain unclear. We developed transgenic mice models to examine the molecular lesion caused by *PELI1* gain-of-function. Constitutive expression of *PELI1* resulted in ligand-independent hyperactivation of B cells and facilitated the development of a wide range of lymphoid tumors, with prominent B cell infiltration observed across multiple organs. *PELI1* directly interacted with the oncoprotein B cell chronic lymphocytic leukemia (BCL6) and induced K63-mediated BCL6 polyubiquitination. Furthermore, *PELI1* expression levels positively correlated with BCL6 expression, and *PELI1* overexpression was closely associated with poor prognosis in DLBCLs. Recently, we generated doxycycline-inducible human *PELI1* transgenic mice. A major advantage of our mouse model is that the disease is inducible and can be induced at any stage of life, and the degree as well as the state of inflammation (acute, chronic, or batch wise) can be fine-tuned. We observed that the acute and chronic induction of *PELI1* resulted in abnormal development and activation of peripheral immune system and could developed autoimmune disease and chronic inflammatory disease.

Biography

Suhyeon Kim has completed her Master's degree from Sungkyunkwan University and is currently a PhD candidate at the Sungkyunkwan University School of Medicine.

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