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An essential role for TAGLN2 in phagocytosis of lipopolysaccharide-activated macrophages

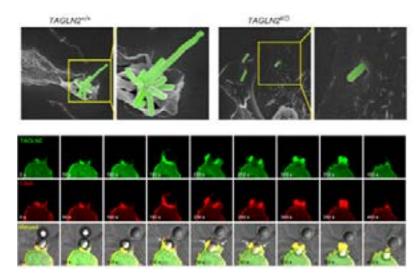
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A ctivated macrophages have a greater ability of phagocytosis against pathogens that is mediated by large-scale actin rearrangement. However, molecular machineries that conduct this task have not been fully identified. Here, we demonstrate an unanticipated role of TAGLN2, a 22-kDa actin-binding protein, in Toll-like receptor (TLR)-stimulated phagocytosis. TAGLN2 was greatly induced in macrophages in response to lipopolysaccharide (LPS), a ligand for TLR4, partly *via* the NF-κB pathway. TAGLN2-deficient macrophages (TAGLN2-f) showed defective phagocytic functions of IgM- and IgG-coated sheep red blood cells as well as bacteria. Cell signaling pathways involved in actin rearrangement-PI3 kinase/AKT and Ras-ERK were also down-regulated in LPS-stimulated TAGLN2-deficient macrophages. Moreover, TAGLN2-f mice showed higher mortality after bacterial infection than wild-type littermates. Thus, our results revealed a novel function of TAGLN2 as a molecular armament required for host defense.

Recent Publications

- 1. Na B R, Jun C D et al. (2015) TAGLN2 regulates T cell activation by stabilizing the actin cytoskeleton at the immunological synapse. J Cell Biol. 209(1): 143-162.
- 2. Kim H R, Jun C D et al. (2011) IGSF4 is a novel TCR z-chain-interacting protein that enhances TCR-mediated signaling. J Exp Med. 208(12): 2545-2560.
- 3. Kim Y D, Jun C D et al. (2011) NSrp70 is a novel nuclear speckle–related protein that modulates alternative premRNA splicing *in vivo*. Nucleic Acids Res. 39(10): 4300-4314.



Biography

Chang Duk Jun discovered several crucial molecules involved in the immunological synapse (IS). Among them, TAGLN2 localizes at the distal area of supramolecular activation clusters (d-SMAC) in IS and stabilizes actin structure that prolongs the duration of IS. In macrophages, TAGLN2 plays an important role by enhancing the phagocytic function of macrophages. He is also interested in mutual communication between T cells and antigen-presenting cells in the process of immune synapses and kinapses.

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