微生物学免疫学教室特別セミナー

“Noc4L dictates Treg activation in vivo”

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（要旨）Regulatory T cells (Treg) are a specialized CD4+ T cell lineage that plays a central role in controlling self-immune tolerance. It is well documented that antigen triggered Tregs activation is crucial for their suppressive function; however, the underlying molecular mechanism is poorly understood.The ribosome plays a universally conserved role in catalyzing the translation of all mRNAs in every cell across all kingdoms of life. It is therefore not surprising that the ribosome is one of the most complex and elaborately formed “molecular machines” in the cell, whose biogenesis is extraordinarily orchestrated. Although ribosome biogenesis is considered as a conserved housekeeping process, emerging evidence support a new hypothesis that different cell types might actually have variety in the repertoire of ribosome biogenesis factors (RBFs), which are required to generate specialized ribosome and confer the translation of selected mRNAs. Here, we focused on the conserved ribosome protein Noc4L based on the observations that Noc4L was highly expressed in activated CD4+CD25+ T cells compared with the resting Treg cells in vitro. Treg specific knockout Noc4L mice developed a lethal autoimmune phenotype, resembling Treg-deficient "scurfy" mice. Interestingly, Noc4L defection did not globally affect the overall protein translation in Tregs, but a rather select detriment of the expression of the genes related to Treg activation. Together, these results demonstrated a central role for the ribosome biogenesis factor Noc4L in controlling Tregs activation. The ribosome-mediated tranlational regulation is a new layer in the regulation of gene expression of Tregs.

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