ASHBi / CiRA JOINT SEMINAR

Distinct phospho-variants of STAT3 regulate developmental pace *in vivo*

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Date Monday, 4 December 2023

Time 17:00 – 17:20 [JST]

Venue CiRA Auditorium
1F, Center for iPS Cell Research and Application Bldg. #1

\ Register here /

This seminar will be preceded by the ASHBi/CiRA Joint Distinguished Seminar by Dr. Jennifer Nichols at 5:20 pm.

Abstract

STAT3 has been studied extensively in the context of self-renewal of naïve pluripotent mouse embryonic stem cells (mESCs). Although STAT3 is required to maintain inner cell mass lineages when maternal-zygotically eliminated, the role of STAT3 during gastrulation is unclear as the original knockout of Stat3 develops until E6.5. In this study, we observed that on CD1 genetic background zygotic loss of Stat3 leads to consistent developmental retardation from implantation to mid-gestation, beginning with a significant reduction in the number of epiblast cells by the time of implantation. Remarkably, mutants appear to scale normally and resemble non-affected embryos from the previous day at all postimplantation stages examined. We attribute this phenotype to loss of the active serine phosphorylated (pS727) form of STAT3, required for neural differentiation of mESCs, which is also implicated in growth defects during organ expansion in mice and humans. Bulk RNA-seq analysis showed transcriptional developmental retardation in Stat3 null embryos. Single cell chimera RNA-sequence analysis revealed specific exclusion of Stat3 null cells in rapidly proliferating erythroid lineage. Our study demonstrates the role of the STAT3 in the temporal control of embryonic progression and metabolic mechanisms.

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