CDB SEMINAR

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16:00~17:00 A7F Seminar Room

X chromosome inactivation initiated by dysfunctional Xist RNA

* This seminar is a part of the Epigenetics Seminar Series 2016.

Summary

In female mammals, one of the two X chromosomes becomes transcriptionally silenced during early development to compensate for the imbalance of the X-linked gene dosage between the sexes. X-liked long noncoding Xist RNA plays a pivotal role in gene silencing and heterochromatinization of the X chromosome to be inactivated. It has been shown that the A-repeat, one of the conserved repeats present in Xist RNA among many mammalian species, is essential for the silencing function of the RNA in differentiating ES cells. We previously attempted to explore the role of the A-repeat in vivo by targeted deletion of the corresponding genomic sequence in the mouse. This unexpectedly abolished transcriptional upregulation of the mutated Xist allele, precluding the further analysis for the behavior of the Xist RNA lacking the A-repeat in vivo. Here, we introduced a new Xist allele lacking the A-repeat under the control of a constitutively active promoter in the mouse. When this allele was paternally transmitted, the mutated Xist RNA was successfully expressed and coated the paternal X chromosome, inducing apparent heterochromatinization albeit defective in silencing in the embryo. A detail analysis of transcriptional state of the mutated X is currently underway. I will discuss about the behavior of the mutated Xist RNA lacking the A-repeat in vivo and biological significance of X chromosome inactivation.

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