CDB SEMINAR

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Title: " The steroid deficiency gene *ecdysoneless* "

Date: Wednesday, March 16
Time: 16:00 P.M.～17:00 P.M.
Place: 7F Conference room of Building A, CDB

Summary

Insects require the steroid hormone ecdysone for molting, metamorphosis, and reproduction. *Drosophila* mutants deficient in steroid production are therefore useful for understanding the hormonal regulation of development and physiology. Among these mutants, *ecdysoneless[1]* (*ecd[1]*) has been extensively used since its discovery in 1977, although the gene and its defect remained unknown. We have cloned *ecdysoneless*, which encodes a novel but well-conserved protein. A proline to serine mutation is responsible for the steroid deficiency in *ecd[1]* larvae. Unlike these larvae, null *ecd[2]* mutants surprisingly could be rescued neither by ecdysone feeding nor by Ecd expression in the ecdysone-producing ring gland. Mosaic analyses show that Ecd functions cell-autonomously, because clones of *ecd(-/-)* cells fail to survive in proliferating imaginal discs, or cause abnormal fusions of adjacent egg chambers in the ovary. Loss of Ecd in the germline causes mutant egg chambers to degenerate before vitellogenesis. Interestingly, mosaic egg chambers in which only half of the cells lack Ecd while the others carry the female sterile *ovo[D1]* mutation mature. Together, these results indicate that in addition to its effect on the ring gland, Ecd plays an essential cell-autonomous role independently of the circulating hormone.

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