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02-2789-9621

Evolutionary Genetics and Genomics

Genetic Conflicts Drive Rapid Evolution of Protamines in *Drosophila*

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Time: 2023. 02. 17 Fri. 15:00

Venue: Auditorium, 1st Floor
Interdisciplinary Research Building
跨領域科技研究大樓1樓演講廳

Host: Dr. Isheng Jason Tsai 蔡怡陞研究員

~Attendee must wear mask~
~與會者請配戴口罩~

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Abstract

My research program aims to understand how genetic conflicts can shape rapid evolution of even essential chromosomal proteins like protamines. Many animal species employ protamines to package sperm genomes tightly. Despite their essential requirements for male fertility in many animal species, protamines vary across animal lineages and evolve rapidly in mammals. Their rapid evolution is unexplained and could result from relentless competition between sperm for fertilization success or genetic conflicts with selfish genetic elements in the male germline. I used a phylogenomic approach to investigate protamine diversification in *Drosophila* species. I found that most protamine genes in Drosophila melanogaster evolve under positive selection. Unexpectedly, evolutionarily young protamine genes are more likely to be critical for fertility than ancient, conserved protamine genes. For example, I found that ancient CG30056 is dispensable for male fertility despite being one of three protamine genes universally retained in *Drosophila* species, whereas both 'young' protamine genes Prtl99C and Mst77F are essential for fertility. I found 19 independent protamine gene amplification events that occurred preferentially on sex chromosomes. Conversely, the montium group of *Drosophila* species lost otherwise-conserved protamine genes, coincident with an X-Y chromosomal fusion. My observations lead to the hypothesis that autosomal protamine genes suppress meiotic drive, whereas sex-chromosomal protamine expansions lead to meiotic drive. X-Y fusions in the montium group render autosomal protamines dispensable by making X-versus-Y meiotic drive obsolete or costly, resulting in their loss. Thus, genetic conflicts between sex chromosomes may drive protamines rapid evolution during spermatogenesis in *Drosophila* species. Using gene-swap analyses I am currently testing this hypothesis. I found that replacing fertility-essential Mst77F-mel with a distantly related species Mst77-ana leads to sterility, whereas replacement with closely related species Mst77F-sim or Mst77F-yak leads to recovery of fertility but with male-biased progeny. My findings support the hypothesis that evolution of Mst77F is driven to suppress X-versus-Y sperm killing. Thus, genetic conflicts explain the paradoxically rapid evolution of fertility-essential protamine genes.