Drosophila protease ClpXP specifically degrades DmLRPPRC1 controlling mitochondrial mRNA and translation

廣藤, 雄太

https://doi.org/10.15017/1928629

出版情報:九州大学, 2017, 博士(歯学), 課程博士

バージョン: 権利関係:

氏 名	廣藤 雄太
論 文 名	<i>Drosophila</i> protease ClpXP specifically degrades DmLRPPRC1 controlling mitochondrial mRNA and translation (ショウジョウバエのプロテアーゼ ClpXP はミトコンドリア mRNA
	と翻訳を調節する DmLRPPRC1 を特異的に分解する)
論文調査委員	主 査 九州大学 教授 中西 博
	副 査 九州大学 教授 山下 喜久
	副 査 九州大学 教授 自見 英治郎

論文審査の結果の要旨

ClpXP is the major protease in the mitochondrial matrix in eukaryotes, and is well conserved among species. ClpXP is composed of a proteolytic subunit, ClpP, and a chaperone-like subunit, ClpX. Although it has been proposed that ClpXP is required for the mitochondrial unfolded protein response, additional roles for ClpXP in mitochondrial biogenesis are unclear. In this study, *Drosophila* leucine-rich pentatricopeptide repeat domain-containing protein 1(DmLRPPRC1) was found to be a specific substrate of ClpXP. Deletion or introduction of catalytically inactive mutation of ClpP increased DmLRPPRC1 and caused non-uniform increases of mitochondrial mRNAs, accumulation of some unprocessed mitochondrial transcripts, and modest repression of mitochondrial translation in *Drosophila* Schneider S2 cells. Moreover, DmLRPPRC1 over-expression induced the phenotypes similar to those observed when ClpP was depleted.

These results suggest that ClpXP regulates mitochondrial gene expression by changing the protein level of DmLRPPRC1 in *Drosophila* Schneider S2 cells. Therefore, the thesis is worthy of being defended for the Doctor of Philosophy (Dental Science).