Title: Function of lipid storage droplet-1 and 2 in development of *Drosophila melanogater*

Abstract

Lipid droplets comprise neutral lipids that are enclosed by a monolayer of phospholipids. The surface of a lipid droplet is decorated with several types of proteins such as structural proteins, lipid-synthesis enzymes, lipases, and membrane-trafficking proteins. Notably, *Drosophila* and mammals share these lipid metabolism-related genes and basic metabolism function. Perilipins are known to be involved in regulating the accessibility of lipases; e.g., mammalian adipose triglyceride lipase (ATGL) and hormone-sensitive lipase (HSL), by either recruiting these enzymes or preventing their access to lipid droplets. Perilipins regulate ATGL activation through the interaction with comparative gene identification 58 (CGI-58), which is an ATGL coactivator. Also, the role of perilipin 1 locus was identified as obesity risk factors, and perilipin 2 participates in the pathogenesis of diet-induced insulin resistance. *Drosophila* encodes two perilipin family member, *Lsd-1*/*perilipin-1* and *Lsd-2*/*perilipin-2.* LSD-1 indiscriminately labels lipid droplets of varying size whereas LSD-2 localizes to smaller lipid droplets. LSD-1 exclusively locates on lipid droplets while LSD-2 presents both in the cytoplasm and on lipid droplets. However, the roles of these genes in development remain unclear. This present study, we found that LSD-1 expresses not only in wing pouch but also detection in notum of wing imaginal disc. Specific knockdown of *Lsd-1* by *pannir*-GAL4 driver in wing disc leads to split thorax phenotype, suggesting that an essential role of *Lsd-1* in development of *Drosophila* thorax. This role of *Lsd-1* is dependent on the activity of the *Drosophila* c-Jun N-terminal kinase, as overexpression of *basket* suppresses split thorax induced by *Lsd-1* knockdown. Besides these events, depletion of *Lsd-1* causes an enhancement apoptosis cell death in wing notum. Moreover, we also demonstrated that knockdown of *Lsd-2* led to dramatic cell death and developmental defects in the wing blade. Also, *Lsd-2* knockdown-induced apoptosis cell death was shown to be mediated by dFoxO. Taken together, these data indicate *Lsd-1* and *Lsd-2* have main role in *Drosophila* development.