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- 일시: 4/4 화요일 오후 2시
- 장소: 생명과학관 8101호

Maintenance of mature neuron morphology

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Mature neurons maintain their distinctive morphology for extended time in adult life. Compared to developmental neuronal process outgrowth, guidance, and target selection, relatively little is known of mechanisms that maintain mature neuron morphology. Loss of function in *C. elegans* DIP-2, a member of the conserved lipid metabolic regulator Dip2 family, results in progressive overgrowth of neurites in adults. We find that *dip-2* mutants display strong and specific genetic interactions with *sax-2*, the *C. elegans* ortholog of mammalian FRY. Combined loss of DIP-2 and SAX-2 results in drastic disruption of neuronal morphology in adults, accompanied by increased release of neuronal extracellular vesicles (EVs). Screening suppressors of *dip-2 sax-2* double mutant defects, we identified gain-of-function (*gf*) mutations in the conserved Dopey family protein PAD-1 and its associated phospholipid flippase TAT-5. In *dip-2 sax-2* double mutants carrying either *pad-1(gf)* or *tat-5(gf)* mutation, EV release is reduced and neuronal morphology is restored to largely normal. The domain containing *pad-1*(*gf*) is essential for PAD-1 function, and PAD-1(*gf*) protein displays increased association with the plasma membrane and inhibits EV release. We propose that DIP-2 and SAX-2 maintain morphology of neurons and other types of cells through PAD-1 and TAT-5-dependent regulation of membrane trafficking. Our work sheds insight on the mechanistic basis of neurological disorders where human orthologs of DIP-2, SAX-2, PAD-1, and TAT-5 have been implicated.

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