

A fly lamin gene is both like and unlike human genes

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Mitch Dushay and colleagues at Uppsala University in Sweden announce the publication of their paper, "Characterization of lamin Mutation Phenotypes in *Drosophila* and Comparison to Human Laminopathies" in the June 13th issue of the online, open-access journal PLoS ONE.

Lamins are intermediate filament proteins that make up a matrix underlying the nuclear membrane. Mammals have two types of lamins; A-type lamins are expressed in differentiating cells, while B-type lamins are expressed ubiquitously.

Mutations in the gene coding for human lamin A cause a range of diseases collectively called laminopathies, including forms of muscular dystrophy and premature aging diseases. The fruit fly *Drosophila melanogaster* has 2 lamin genes that are expressed in A- and B-type patterns, and it has been assumed that similarly expressed lamins perform similar functions.

Yet, Dushay and his colleagues, among others, have shown that the fly lamin genes are more closely related to each other than to mammalian lamin genes. While the independent evolution of similar expression patterns must have been driven by similar vital lamin gene functions, Dushay et al. found that mutations in the ubiquitously expressed *Drosophila* lamin gene cause larvae to move less and show subtle muscle defects, while surviving lamin adults walk poorly and can't fly – like aged wild type flies.

This suggests that lamin mutations might cause neuromuscular defects, premature aging, or both. The resemblance of *Drosophila* lamin phenotypes to human laminopathies provides an interesting case of gene expression and function diverging through evolution, and promises greater insight into lamin function, and possibly into laminopathic diseases and aging.

Source: Public Library of Science

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