

Lipid accumulation drives cellular senescence in dopaminergic neurons

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A new research perspective titled Lipid accumulation drives cellular senescence in dopaminergic neurons has been published in *Aging*.

As highlighted in the Abstract of this perspective, Parkinson's disease (PD) is an age-related movement disorder caused by the loss of dopaminergic (DA) neurons in the [substantia nigra](#) pars compacta

(SNpc) of the midbrain. However, the underlying causes of this DA neuron loss in PD are unknown, and there are currently no effective treatments to prevent or slow neuronal loss or the progression of related symptoms.

In their perspective, researchers Taylor Russo and Markus Riessland from Stony Brook University found that artificially inducing GluCer accumulation leads to cellular senescence of DA neurons. This suggests that [lipid](#) aggregation plays a crucial role in the pathology of PD by driving senescence in these vulnerable neurons.

"Here, we discuss the relevance of the age-related aggregation of lipids as well as the direct functional link between general lipid aggregation, cellular senescence, and inflammaging of DA neurons," the researchers state.

Additionally, they propose that the expression of a [cellular senescence](#) phenotype in the most vulnerable neurons in PD can be triggered by lysosomal impairment and lipid aggregation.

"Importantly, we highlight additional data that perilipin (PLIN2) is significantly upregulated in senescent DA neurons, suggesting an overall enrichment of lipid droplets (LDs) in these cells," they add.

More information: Taylor Russo et al, Lipid accumulation drives cellular senescence in dopaminergic neurons, *Aging* (2024). [DOI: 10.18632/aging.206030](https://doi.org/10.18632/aging.206030)

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