Restoring aging genes in rats

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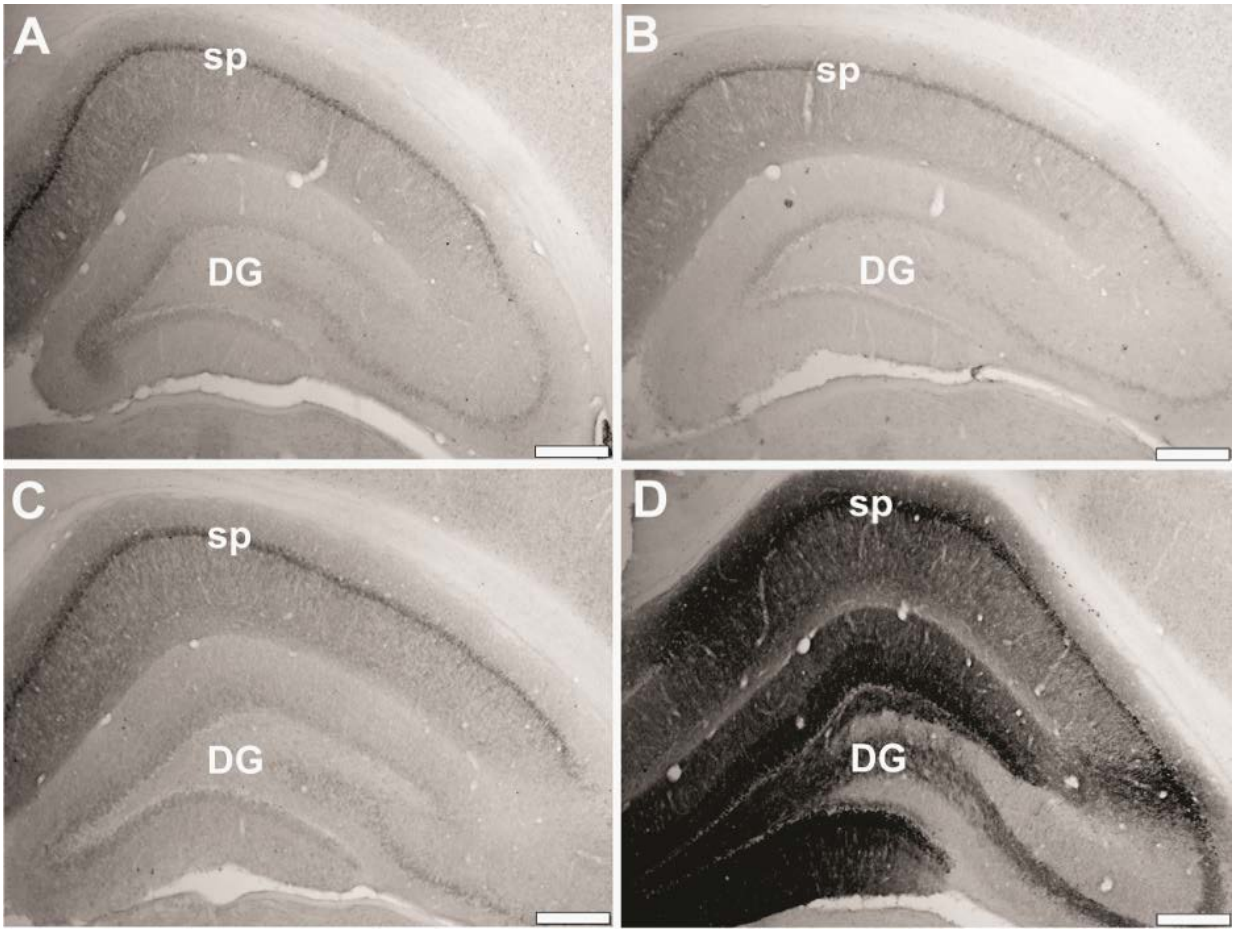

Representative photomicrographs from (A) young control, (B) aged control, (C) aged short-term FKBP1b and (D) aged long-term FKBP1b. Note the substantial increase in FKBP1b expression at both the mRNA and protein levels, particularly in the LT-FKBP1b group. (sp- stratum pyramidale; DG- dentate gyrus; calbar- 500 uM) Credit: Gant et al., *JNeurosci* (2017)

Overexpression of a protein that regulates calcium homeostasis in hippocampal neurons can safely and effectively reverse and prevent age-related memory impairments in rats while restoring altered gene expression, finds new research published in the *Journal of Neuroscience*.

Philip Landfield, John Gant, Eric Blalock and colleagues found that long-term and short-term treatment of aging rats that induces overexpression of FK506-Binding Protein 12.6/1b (FKBP1b) restored the expression of more than 800 genes affected by aging to levels comparable to those of young untreated rats, in addition to improving performance on a water maze task.

The restored genes represent a new genomic network that regulates the integrity of neuronal structure in the hippocampus and is targeted by aging.

These results suggest that addressing FKBP1b deficiency may represent a new avenue for countering age-related memory loss.

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