

Summary

It is widely postulated that tissue aging could be, at least partially, caused by reduction of stem cell number, activity, or both. However, the mechanisms of controlling stem cell aging remain largely a mystery. Here, we use *Drosophila* ovarian germline stem cells (GSCs) as a model to demonstrate that age-dependent decline in the functions of stem cells and their niche contributes to overall stem cell aging. BMP signaling activity from the niche significantly decreases with age, and increasing BMP signaling can prolong GSC life span and promote their proliferation. In addition, the age-dependent E-cadherin decline in the stem cell-niche junction also contributes to stem cell aging. Finally, overexpression of SOD, an enzyme that helps eliminate free oxygen species, in either GSCs or their niche alone can prolong GSC life span and increase GSC proliferation. Therefore, this study demonstrates that stem cell aging is controlled extrinsically and intrinsically in the *Drosophila* ovary.

Author Keywords: STEMCELL; DEVBIO; HUMDISEASE

Article Outline

Introduction

Results

The Number and Functions of GSCs and Niche Cells Undergo Age-Related Decline

The Compromised BMP Signal Production from the Aged Niche Contributes to GSC Aging

Increased BMP Signaling Activity in Aged GSCs Can Enhance Their Life Span and Proliferation

Strengthening Physical Interactions between GSCs and Their Niche Can Prolong GSC Life Span

Overexpression of SOD in Either the Niche or GSCs Can Prolong GSC Life Span

Discussion

The Declined Niche Function Contributes to Stem Cell Aging
Stem Cells Also Undergo Intrinsic Aging

Experimental Procedures

Drosophila Stocks

Generation of Transgenic *UASp* Lines

Experimental Genotypes

BrdU Labeling of GSCs

Quantification of Cap Cells, GSCs, and Cysts

Quantification of *Dad-lacZ* and E-Cadherin Expression in GSCs

Immunohistochemistry

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References

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