UF Author Rights Policy

Faculty Senate Meeting
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Why do we need it?

Traditional publishing model requires authors to sign away copyright with limited or no options to share.

The proposed policy would enable sharing a peer reviewed version of journal articles in noncommercial venues online upon publication.

This will support:

- Access for peers at international institutions, government agencies, etc.
- Increased citation impact
Where did it come from?

By-and-for faculty initiative developed by the University Libraries Committee, 2019-2021 (Chairs Czerne Reid, Angelos Barmpoutis, Josepha Cheong)

Adapted from similar policies at 75+ US institutions, including University of California and UNC-Chapel Hill.

Timeline:
2019-2020: Research, peer discussion, drafting
2020-2021: Listening tour and revisions
How does it work?

By default, faculty will share (not transfer!) rights in journal articles with UF. Even if faculty sign a publisher agreement, the license to UF remains in place and authors may rely on this license to share.

In practice, this means faculty will be able to share the peer-reviewed accepted manuscript in noncommercial venues (UF institutional repository, arXiv, PubMed Central) upon publication.

The policy is NOT a mandate to share or to publish in specific journals.

Any faculty may opt out via a simple form.
Hippocampal Stratum Radiatum, Lacunosum and Molecular Sparing in Mild Cognitive Impairment

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Running title: Preservation of SRLM in MCI but not in AD

Abstract: (246 words)

Background: Alzheimer’s disease (AD) is associated with atrophy in entorhinal cortex (ERC), the hippocampus, and its subfields Cornu Ammonis 1 (CA1) and subiculum, which predict conversion from Mild Cognitive Impairment (MCI) to clinical AD. The stratum radiatum, lacunosum and molecular (SRLM) are also important gateways involving ERC and CA1, which are affected by early AD pathology.

Objective: To assess whether the SRLM is affected in MCI and AD.

Methods: In this proof-of-concept study, 27 controls, 13 subjects with AD and 22 with MCI underwent 3T MRI. TI maps were used for whole-hippocampal volumetry. T2 maps were segmented for hippocampal subfield areas, entorhinal cortex and subiculum thickness, and evaluated for SRLM integrity.

Results: Significant CA1 atrophy and subiculum thinning were found in both AD and MCI compared to similarly aged controls. However, SRLM integrity was only significantly reduced in AD but not in MCI compared to controls. There were no significant differences in other hippocampal subfields (CA2, CA3/Dentate Gyrus) or ERC thickness between the groups. Finally, CA1 and CA3/DG areas and SRLM clarity were correlated with clinical and cognitive measures of disease severity.

Conclusion: Although this study was cross-sectional, it suggests a progression of specific subfield changes from MCI to established AD that is associated with the reduced integrity of SRLM, which may reflect more widespread hippocampal involvement as the disease progresses and the relative preservation of SRLM in MCI. These results provide new MRI biomarkers for disease staging and understanding of the neuropathology in AD.

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ATF6 safeguards organelle homeostasis and cellular aging in human mesenchymal stem cells

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Abstract
Loss of organelle homeostasis is a hallmark of aging. However, it remains elusive how this occurs at the gene expression level. Here, we report that human mesenchymal stem cell (hMSC) aging is associated with dysfunction of double-membrane organelles and down-regulation of transcription factor ATF6. CIRBP/Ca2+-mediated inactivation of ATF6 in hMSCs, not in human embryonic stem cells and human adipocytes, results in premature cellular aging, characteristic of loss of endomembrane homeostasis. Transcriptomic analysis uncovers cell type-specific constitutive and stress-induced ATF6-regulated genes implicated in various layers of organelle homeostasis regulation. ATF6 was characterized as a constitutive ATF6 responsive gene, down-regulation of which contributes to hMSC aging. Our study unravels the first ATF6-regulated gene expression network related to homeostatic regulation of membrane organelles and provides novel mechanistic insights into aging-associated attrition of human stem cells.

Introduction
The cellular proteome is tightly regulated by the proteostasis network, a complex system that controls protein synthesis, folding, and degradation. Preserving the stability and functionality of proteomes is essential for the proper cellular function and biological process. Loss of proteostasis is considered as one of the hallmarks of aging. More evidence shows that accumulation of unfolded or misfolded proteins contributes to the development of aging-related diseases. Endoplasmic reticulum (ER) is the largest intracellular endomembrane system, enabling protein quality control. Ca2+-ion homeostasis, and organelle communication. ER exerts the protein quality control via two pathways. One is mediated by ER-resident molecular chaperones and enzymes to ensure proper protein folding. The other is ER-associated degradation (ERAD) pathway, by which unfolded or misfolded proteins in the ER are transported to the cytoplasm for degradation through ubiquitin proteasome system.

In addition, ER is connected with other membrane-bound organelles. ER not only physically connects with the outer nuclear membrane and communicates with Golgi apparatus by vesicle transport, but also contacts with mitochondria for coupling mtDNA synthesis and contributes to biogenesis of autophagosomes by cross-talking with mitochondria. Indeed, loss of the architectural and functional integrity of these membrane organelles has been reported for aging and several age-associated disorders. For instance, senescent cells frequently show alterations in nuclear envelope (NE), mitochondria, ER, and Golgi[16]. The molecular
Sharing scholarly research supports collaboration and enhances impact. A proposed faculty author rights policy offers a legal option to ensure broader dissemination of academic journal articles.

LEARN ABOUT THE POLICY

What it is
A policy protecting faculty rights to share our scholarly research, specifically academic journal articles.

What it is not
A requirement to publish in specific journals or to share your research against your wishes.

How it works
The policy lets you share your work widely by granting a nonexclusive license to the University. It is not a transfer of copyright, and you can opt out for any reason, no questions asked.

https://digitalpartnerships.uflib.ufl.edu/scholarly - communications/uf-author-rights-policy/
Thank you!

Questions? Feedback?

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