UF Author Rights Policy

Faculty Senate Meeting
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Perry Collins, M.A., M.L.I.S.
Associate University Librarian

Josepha A. Cheong, M.D.
Professor, College of Medicine
Chair, University Libraries Committee

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, 20192021 (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

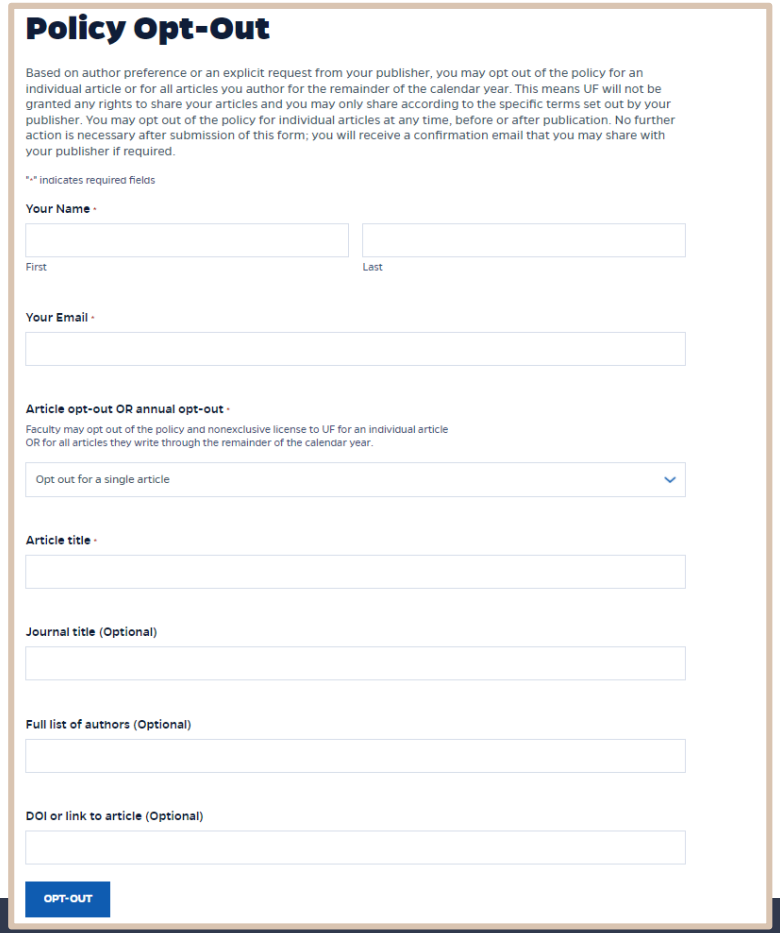
How does it work?

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.



Policy Opt-Out

Based on author preference or an explicit request from your publisher, you may opt out of the policy for an individual article or for all articles you author for the remainder of the calendar year. This means UF will not be granted any rights to share your articles and you may only share according to the specific terms set out by your publisher. You may opt out of the policy for individual articles at any time, before or after publication. No further action is necessary after submission of this form; you will receive a confirmation email that you may share with your publisher if required.

"*" indicates required fields

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Faculty may opt out of the policy and nonexclusive license to UF for an individual article OR for all articles they write through the remainder of the calendar year.

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Article title *

Journal title (Optional)

Full list of authors (Optional)

DOI or link to article (Optional)

OPT-OUT

Policy Opt - Out online form

Hippocampal Stratum Radiatum, Lacunosum and Moleculare Sparing in Mild Cognitive Impairment

Li Su[a,b,*], Lawrence Hayes[a,*], Soteris Soteriades[a], Guy Williams[c], Susannah AE Brain[d], Michael J Firbank[e], Giulia Longoni[a], Robert J Arnold[a], James B Rowe[c,g,+], John T O'Brien[a,+]

a. Department of Psychiatry, University of Cambridge School of Clinical Medicine, Cambridge, CB2 0SP.

b. China-UK Centre for Cognition and Ageing Research, Faculty of Psychology, Southwest University, Chongqing, China.

c. Wolfson Brain Imaging Centre, University of Cambridge, School of Clinical Medicine, Cambridge Biomedical Campus, Cambridge CB2 0QQ.

d. Oxford University Hospitals NHS Trust, Windmill Road, Oxford OX3 7LD.

e. Institute of Neuroscience and Newcastle University Institute for Ageing, Newcastle University Campus for Ageing and Vitality, Newcastle upon Tyne, NE4 5PL.

f. Department of Clinical Neurosciences, Cambridge University, CB2 0SZ.

g. MRC Cognition and Brain Sciences Unit, Cambridge, CB2 7EF.

* These authors contributed equally. + Joint senior authors

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

Correspondence to: Dr Li Su, Department of Psychiatry, School of Clinical Medicine

Abstract: (246 words)

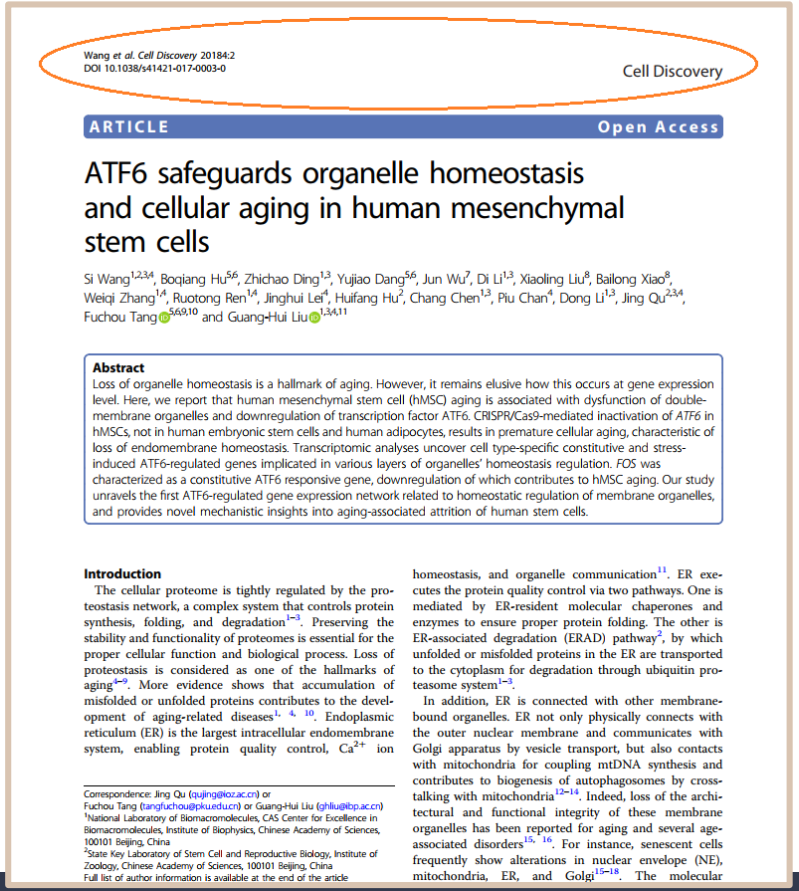
Background: Alzheimer's disease (AD) is associated with atrophy in entorhinal cortex (ERC), the hippocampus, and its subfields Cornu Ammonis 1 (CA1) and subiculum, which predict conversion from Mild Cognitive Impairment (MCI) to clinical AD. The stratum radiatum, lacunosum and moleculare (SRLM) are also 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. T1 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 measurements 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 neurobiology in AD.



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Cell Discovery

ARTICLE Open Access

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

Si Wang[1,2,3,4], Boqiang Hu[5,6], Zhichao Ding[1,3], Yujiao Dang[5,6], Jun Wu[7], Di Li[1,3], Xiaoling Liu[8], Bailong Xiao[8], Weiqi Zhang[1,4], Ruotong Ren[1,4], Jinghui Lei[4], Huifang Hu[2], Chang Chen[1,3], Piu Chan[4], Dong Li[1,3], Jing Qu[2,3,4], Fuchou Tang[5,6,9,10] and Guang-Hui Liu[1,3,4,11]

Abstract

Loss of organelle homeostasis is a hallmark of aging. However, it remains elusive how this occurs at gene expression level. Here, we report that human mesenchymal stem cell (hMSC) aging is associated with dysfunction of double-membrane organelles and downregulation of transcription factor ATF6. CRISPR/Cas9-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 analyses uncover cell type-specific constitutive and stress-induced ATF6-regulated genes implicated in various layers of organelles' homeostasis regulation. *FOS* was characterized as a constitutive ATF6 responsive gene, downregulation 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[1–3]. 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[4–9]. More evidence shows that accumulation of misfolded or unfolded proteins contributes to the development of aging-related diseases[1, 4, 10]. Endoplasmic reticulum (ER) is the largest intracellular endomembrane system, enabling protein quality control, Ca^{2+} ion homeostasis, and organelle communication[11]. ER executes 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[2], by which unfolded or misfolded proteins in the ER are transported to the cytoplasm for degradation through ubiquitin proteasome system[1–3].

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[12–14]. Indeed, loss of the architectural and functional integrity of these membrane organelles has been reported for aging and several age-associated disorders[15, 16]. For instance, senescent cells frequently show alterations in nuclear envelope (NE), mitochondria, ER, and Golgi[15–18]. The molecular

Correspondence: Jing Qu (qujing@ioz.ac.cn) or Fuchou Tang (tangfuchou@pku.edu.cn) or Guang-Hui Liu (ghliu@ibp.ac.cn)
[1]National Laboratory of Biomacromolecules, CAS Center for Excellence in Biomacromolecules, Institute of Biophysics, Chinese Academy of Sciences, 100101 Beijing, China
[2]State Key Laboratory of Stem Cell and Reproductive Biology, Institute of Zoology, Chinese Academy of Sciences, 100101 Beijing, China
Full list of author information is available at the end of the article

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Thank you!

Questions? Feedback?

Perry Collins
perrycollins@ufl.edu