Conference Proceedings

2nd World conference on Ageing and Gerontology

July 14-15, 2025 | Rome, Italy

Ying Qin[1,2], Haoxin Liu[3], Sunuwar Janak[1], Taegun Kwon[1], Hongli Wu[1,2]

[1]University of North Texas Health Science Center at Fort Worth, Texas, U.S.A
[2]North Texas Eye Research Institute at Fort Worth, Texas, U.S.A
[3]New York University Langone Hospital, Brooklyn, New York, U.S.A

Grx1/Grx2 Deficiency Leads to Low-grade Inflammation, resulting in Accelerated Senescence in Mouse Lens Epithelial Cells

This study investigates how glutaredoxin1 and glutaredoxin2 (Grx1/Grx2) deficiency promote premature senescence in mouse lens epithelial cells (LECs), aiming to clarify the molecular links between oxidative stress, chronic inflammation, and accelerated lens aging. Primary LECs were isolated from wild-type (WT) and Grx1/Grx2 double knockout (DKO) mice at 1 and 12 months of age. Senescence markers, including senescence-associated β-galactosidase (SA-β-Gal), p53, p21, p16, and phospho-RB, and inflammatory cytokines such as IL-6, were assessed by SA-β-gal staining, western blot, RNA sequencing, and qPCR. DKO LECs showed significantly elevated β-Gal activity in the WT 12-month and DKO groups, with markedly stronger staining in the DKO 12-month group. Age-related increases in senescence were observed in WT 12-month LECs, but to a lesser extent than in DKO counterparts. Western blot results revealed increased p21 expression alongside reduced phospho-RB levels in 12-month DKO LECs ($p < 0.001$), indicating persistent cell cycle arrest. IL-6 protein and mRNA levels were significantly upregulated in DKO lenses ($p < 0.05$ and $p < 0.001$, respectively), suggesting heightened inflammatory signaling. RNA-sequencing analysis further revealed the enrichment of genes involved in oxidative stress responses, immune activation, extracellular matrix remodeling, and inflammation ($p < 0.001$), indicating that chronic inflammation, rather than oxidative stress alone, is a critical and previously underappreciated contributor to cataract pathogenesis. These findings demonstrate that Grx1/Grx2 deficiency disrupts redox balance, enhances chronic inflammation, and accelerates lens aging by driving early-onset and sustained cellular senescence in LECs. Elevated IL-6 supports a role for inflammaging, a persistent, low-grade, sterile inflammation, in contributing to lens dysfunction. This model offers a valuable platform for investigating aging and assessing therapeutic targets to delay cataract formation and age-related tissue decline.

Keywords: Oxidative stress, Chronic inflammation, Senescence, Lens

Biography

Ying Qin is a licensed pharmacist in Texas, U.S.A., and a second-year Ph.D. student at the University of North Texas Health Science Center at Fort Worth. Her research uses cataracts as a model to investigate the molecular mechanisms of aging and age-related diseases. Inspired by clinical cases where medicine fell short, she shifted from pharmacy to biomedical research to help develop new therapeutic strategies. Ying brings a unique blend of clinical and research experience, including work in global clinical trials. Her goal is to advance science and improve outcomes for aging populations through innovative, translational research.

ISBN: 978-1-917892-07-0

doi.org/10.51219/URForum.2025.Ying-Qin