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## The tumor suppressor p53 plays an essential role in mediating lens differentiation and heterochromatin protection of RPE cells

The tumor suppressor, p53 is a multi-function molecule regulating transcription, cell proliferation, differentiation and apoptosis. However, how would p53 regulate lens differentiation remains largely known until our recent demonstration. Oxidative stress (OS)-induced retinal pigment epithelium (RPE) cell apoptosis is critically implicated in the pathogenesis of age-related macular degeneration (AMD), one of the leading causes for blindness in the elderly. The highly condensed, repressive heterochromatin is recently found to play critical roles in mediating diverse stress response. How RPE heterochromatin is regulated upon OS exposure is largely unknown. Here in this lecture, I will discuss our recent work using both mouse model and cultured human RPE cells to investigate how p53 mediates the heterochromatin protection of RPE cells against oxidative damage upon OS exposure.

## Biography

David Wan Cheng Li received his PhD degree in Molecular and Cellular Biology from the University of Washington in Seattle, and completed his Postdoctoral training in the Harkness Eye Institute of Columbia Medical Center in New York City. He is currently an elected One-Hundred Talent Professor in the State Key Laboratory of Zhongshan Ophthalmic Center in Sun Yat-Sen University, an elected Lotus Scholar Professor of Cellular and Developmental Biology in Hunan Normal University in China. He made numerous important discoveries in both eye development and ocular diseases as well as cancer research fields, published over 100 articles in PNAS, NAR, Cancer Research, CDD, Oncogene, MBC, JBC, and IOVS, etc. He has trained 30 PhD students and Postdoctoral fellows, and lectured in a dozens of countries including German, England, USA, Japan and China. He receiverd the Outstanding Achievements Award of Cataract Research from the National Foundation for Eye Research, USA in 2006.

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