Crocin alleviates vascular endothelial senescence induced by diabetes through regulating mitochondrial homeostasis at YAP1/FOXO1/GPX1 axis

Reporter: Yue Luxiao

Instructor: Professor Yang Huike

01

Background And Significance Of The Topic

01 Background And Significance Of The Topic

Vascular complication

- **Vascular complications of diabetes** are a difficult problem in the prevention and treatment of diabetes. The pathological mechanism of vascular complications is vascular endothelial aging. However, the molecular mechanism and key targets of regulating vascular endothelial aging caused by diabetes remain to be clarified.

Crocin

- Previous studies of our research group have confirmed that **crocin** can improve mitochondrial function and inhibit vascular endothelial damage in diabetes mellitus, but the effect and mechanism of crocin in anti-diabetic vascular endothelial aging are not clear.

YAP1

- **YAP1 (Yes-associated protein 1)** is the core molecule of Hippo signaling pathway, which can regulate biological processes such as cell proliferation, apoptosis and stem cell self-renewal. However, the regulatory role of YAP1 in vascular aging is not fully clear.

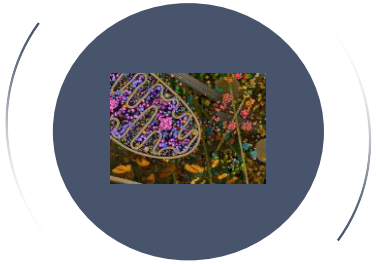
02

Research Methods And Processes

02 Research Methods And Processes

First, Western blot assay was used to detect P53, P16 and other aging signature proteins as well as age-related β-galactosidase (SA-β-gal) staining to determine the induction effect of high glucose culture on endothelial cell aging.

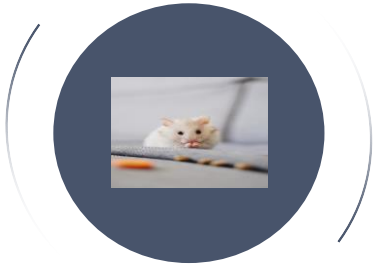
Then, transcriptome sequencing was performed on young endothelial cells and high-glucose-induced senescent endothelial cells to screen potential regulatory pathways and key regulatory genes, and Western blot, q-PCR and other methods were used for verification.



The senescence and mitochondrial function of endothelial cells were detected by Western blot, SA-β-gal, reactive oxygen species (ROS), Mito-tracker, mitochondrial JC-1 and other fluorescent probes.

02 Research Methods And Processes

The downstream target genes of key genes were selected by Aging Atla, MitoCarta, GeneCard and other public databases, and verified by immunofluorescence co-localization and chromatin co-immunoprecipitation.



The effects of crocin on endothelial cell senescence induced by high glucose and mitochondrial function, as well as the regulation of key genes, were investigated by in vivo and in vitro experiments.

By promoting the expression of YAP1 and GPX1, crocin improves mitochondrial function and delays the aging of vascular endothelial cells.

03

Summary Of The Paper

03 Summary Of The Paper

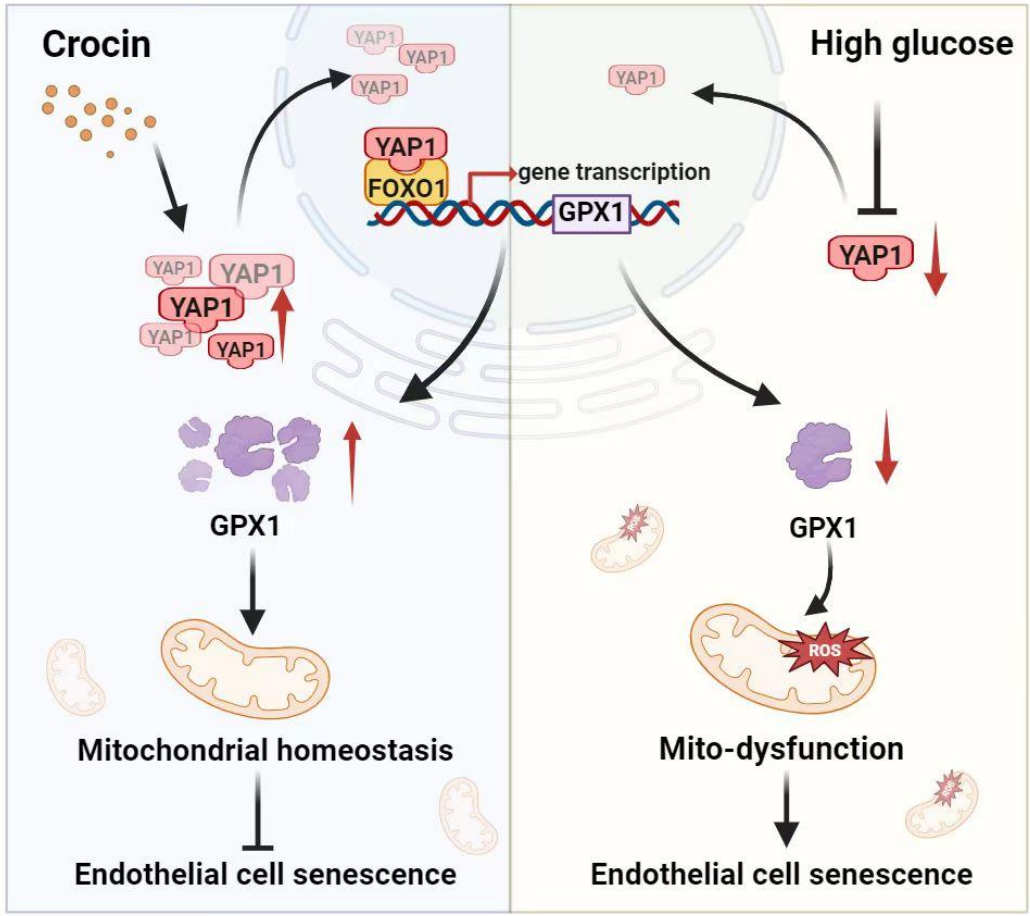
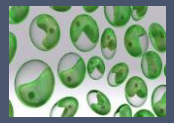


Figure 20. Molecular mechanism of crocin inhibits vascular endothelial aging induced by diabetes.

03 Summary Of The Paper

- In this study, it was confirmed that **high glucose** environment can induce vascular endothelial cell senescence.
- Subsequently, we found that high glucose may damage the mitochondrial function of ECs and induce senescence by down-regulating the expression of **YAP1**.



- Then, the regulation pathway of **YAP1/FOXO1/GPX1** signaling axis in endothelial aging was elucidated.
- In addition, this study found that **crocin** may regulate mitochondrial homeostasis through the YAP1/FOXO1/GPX1 axis and inhibit vascular endothelial aging induced by diabetes.

Crocin provides a new therapeutic target for the prevention and treatment of diabetic vascular complications.

Thanks To Correct Me