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Transition of autophagy and apoptosis in fibroblasts depends on dominant expression of HIF-1α or p53

Key words: Hypertrophic scar, HIF-1α, p53, Autophagy, Apoptosis

Research Summary

This research mainly focused on the mechanism of scar hyperplasia and regression. The present study identified a teeterboard-like conversion relationship between HIF-1α and p53.

The research consists of the following parts：

- HIF-1α expression and autophagy are upregulated in HS tissue at the proliferative stage.
- Expression of HIF-1α, autophagy, and proliferation are upregulated, and p53 expression and apoptosis are downregulated in HDFs under moderate hypoxia.
- There is a teeterboard-like conversion relationship between HIF-1α and p53, and the transition of autophagy and apoptosis in fibroblast is dependent on the dominant expression of HIF-1α or p53.
- HIF-1α and p53 compete for binding to transcription cofactor p300.

Research Summary

	sh-HIF-1α	sh-p53	$p53^{OE}$	Moderate Hypoxia
HIF-1α	↓	↑	↓	↑
p53	↑	↓	↑	↓
Autophagy	↓	↑	↓	↑
Apoptosis	↑	↓	↑	↓
Proliferation	↓	↑	↓	↑
Collagen III	↓	↑	↓	↑

The present study identified a teeterboard-like conversion relationship between HIF-1α and p53. The results also showed that changes in autophagy and apoptosis are dependent on the dominant expression of HIF-1α and p53, which may play a significant role in the mechanism of scar hyperplasia and regression.

Innovation points

●Simulation of moderate hypoxic environment for hypertrophic scars in vitro.

● Respective regulation of HIF-1α and p53 gene expression.

● HIF-1α and p53 compete for binding to transcription cofactor p300.