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Inhibition of prolyl hydroxylase 3 ameliorates cardiac dysfunction in diabetic cardiomyopathy

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Highlights

1. PHD3 gene silencing ameliorated cardiac dysfunction via alleviating cardiac apoptosis and fibrosis in diabetic cardiomyopathy.

2. ROS mediated high glucose-induced PHD3 overexpression.

3. PHD3 gene silencing reduced high glucose-induced H9c2 cardiomyoblasts apoptosis.

4. MAPKs activation was involved in PHD3 mediated H9c2 cardiomyoblasts apoptosis.

Abstract

Prolyl hydroxylase 3 (PHD3) is a member of the prolyl hydroxylases (PHDs) family and is