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Exosomal *let-7a-5p* derived from human umbilical cord mesenchymal stem cells alleviates coxsackievirus B3-induced cardiomyocyte ferroptosis via the SMAD2/ZFP36 signal axis

Key words: Exosome, *let-7a-5p*, SMAD2, CVB3, ferroptosis

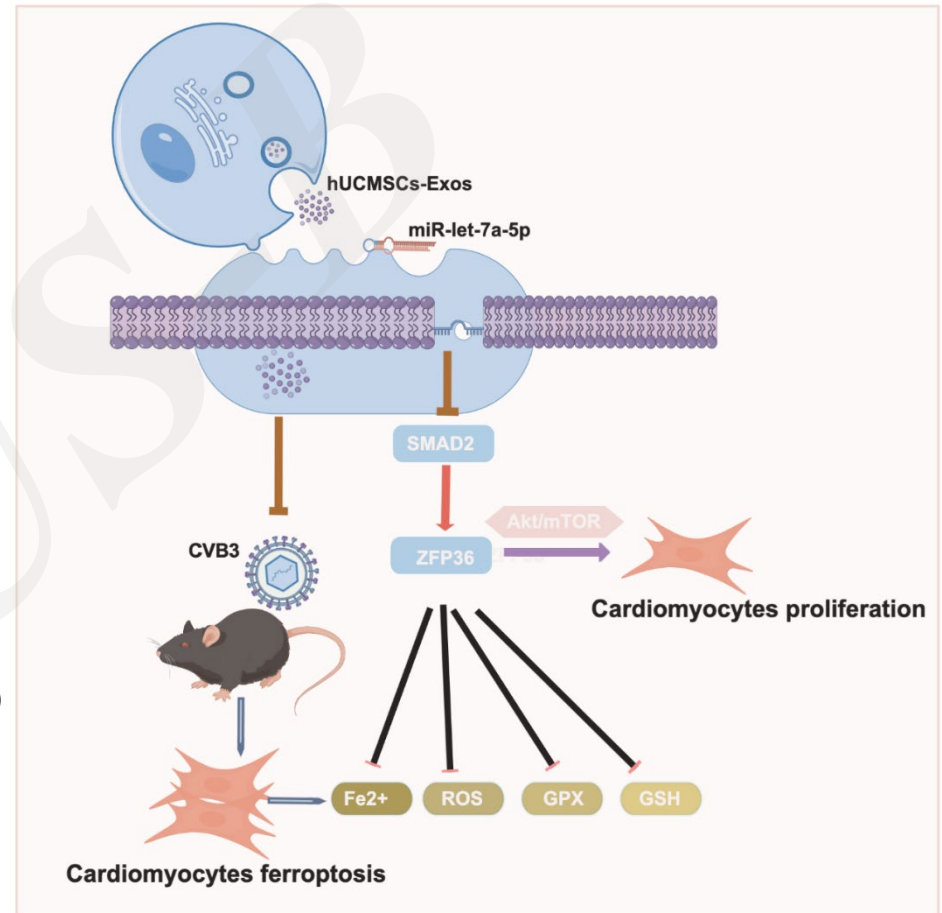
Research Summary

This study aims to investigate the regulatory pathway by which exosomes alleviate ferroptosis in cardiomyocytes (CMCs) induced by coxsackievirus B3 (CVB3), and summarize from the following aspects:

- **CVB3-induced viral myocarditis is subjected to ferroptosis of CMCs**
- **Exo-let-7a-5p derived from hucMSCs suppresses CVB3-induced ferroptosis in CMCs**
- **SMAD2 can target let-7a-5p and participate in let-7a-5p inhibition of CVB3-induced CMCs ferroptosis *in vitro***
- **SMAD2/ZFP36 signaling axis inhibits ferroptosis in CMCs**

Innovation points

- Determination of CVB3-induced ferroptosis in CMCs
- Validation of the alleviation of ferroptosis in CMCs by Exo-let-7a-5p derived from hucMSCs
- Pathway analysis of Exo-let-7a-5p regulating ferroptosis in CMCs through SMAD2/ZFP36 signaling axis



Supplementary Figure S1