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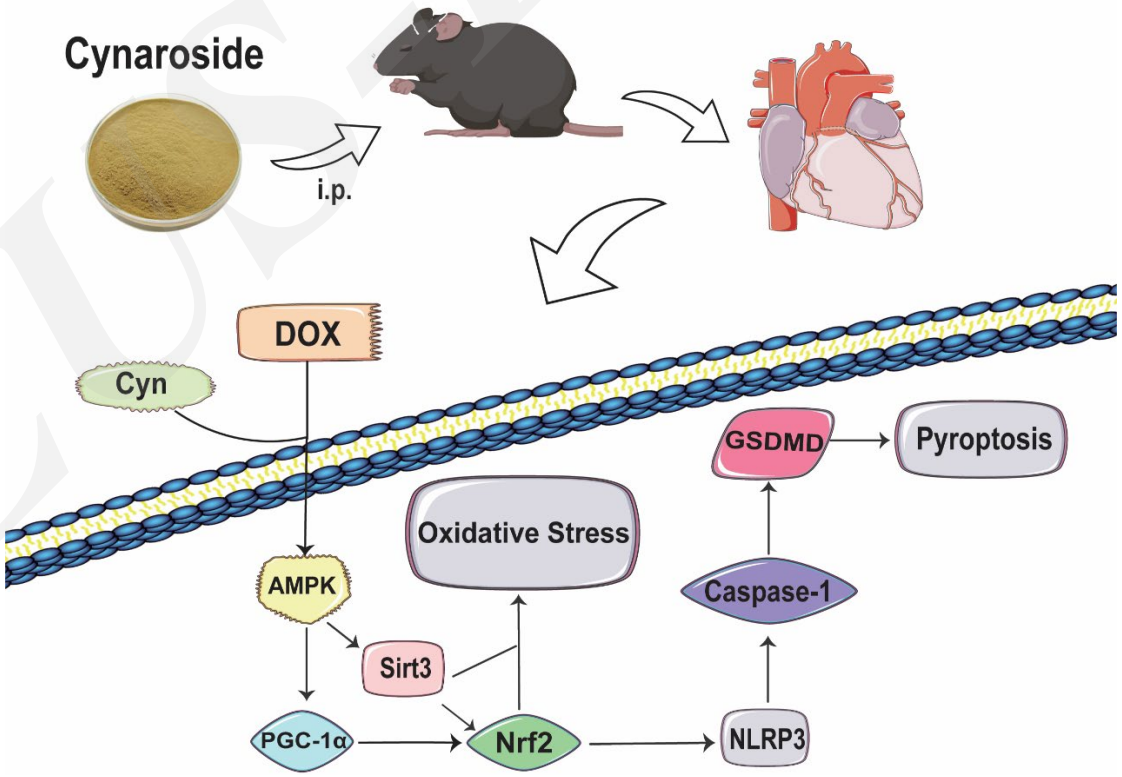
Cynaroside regulates the AMPK/SIRT3/Nrf2 pathway to inhibit doxorubicin-induced cardiomyocyte pyroptosis

Key words: Cynaroside, Doxorubicin, Pyroptosis, Cardiotoxicity, Oxidative stress

Research Summary

This article confirms the therapeutic potential of Cyn in DIC by regulating the AMPK/SIRT3/Nrf2 pathway.

- **The significant benefits of Cyn treatment in mitigating DIC**
- **Effectively alleviate oxidative stress to a certain extent**
- **Maintain the equilibrium of cell apoptosis**
- **Enhance the cardiac function**
- **The mediation of AMPK/Sirt3/Nrf2 pathway**



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Innovation points

- ✓ **Cyn is pivotal in alleviating DOX-induced NLRP3-mediated cardiomyocyte pyroptosis through its regulatory effect on energy metabolism and the enhancement of the antioxidant capacity of cardiac cells.**
- ✓ **The underlying mechanism involves the activation of AMPK/Sirt3/Nrf2 pathway by Cyn, effectively countering the detrimental effects of DOX on cardiac cells.**

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Innovation points

- **Cyn emerges as a compelling candidate for further investigation as a potential cardioprotective agent against DIC and other chemotherapy-related cardiac complications.**
- **This paper provides valuable insights into future considerations for treating DIC, and suggest the potential for Cyn to serve as an alternative therapy in specific contexts.**
- **This research contributes to expanding the knowledge base dedicated to enhancing the well-being and quality of life for cancer patients undergoing chemotherapy, promising a brighter future regarding the addressment of cardiac complications associated with cancer treatment.**

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