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Aerobic glycolysis in colon cancer is repressed by naringin via the *HIF1A* pathway

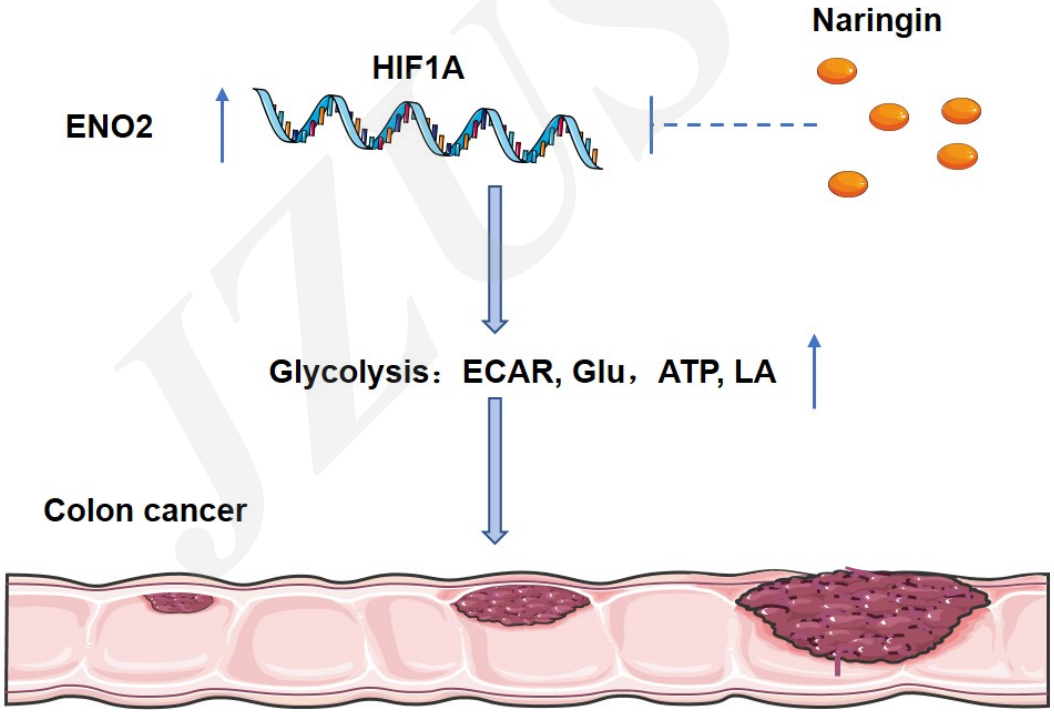
Key words: Colon cancer; Naringin; HIF1A; ENO2; Glycolysis; Metabolic reprogramming

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Research Summary

HIF1A is thought to play an important role in aerobic glycolysis.

In this study, we verified the regulatory effects of HIF1A overexpression on glycolysis, proliferation and migration of colon cancer cells by analysing the extracellular acidification rate and cell function. We demonstrated the inhibitory effect of naringin on the transcriptional activity of HIF1A, which in turn decreased aerobic glycolysis in colon cancer cells.



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Innovation points

- **Hypoxia inducible factor 1 subunit alpha (HIF1A) promotes the expression of enolase 2 (ENO2) in colon cancer.**
- **Overexpression of HIF1A promotes the increase of glycolysis in colon cancer cells.**
- **Naringin inhibits the transcriptional activity of the transcription factor HIF1A.**
- **Naringin inhibits the proliferation and invasion of colon cancer.**

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Innovation points

Fig. 1 | Naringin reduces the Warburg effect of colon cancer cells.

Fig. 2 | HIF1A is necessary for naringin to inhibit colon cancer progression.

Fig. 3 | Co-expression of glycolytic gene ENO2 and HIF1A in colon cancer.

Fig. 4 | HIF1A mediates the transactivation of ENO2 in colon cancer.

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