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Luteolin in Non-Alcoholic Fatty Liver Disease (NAFLD): Bridging Preclinical Promise to Clinical Challenges

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Abstract

Non-Alcoholic Fatty Liver Disease (NAFLD) has emerged as a significant global health issue, necessitating effective therapeutic strategies. Luteolin, a flavonoid abundant in various plants, shows potential in preclinical NAFLD models due to its diverse pharmacological properties. This comprehensive review consolidates current knowledge on Luteolin's potential benefits, acknowledges gaps in research, and proposes strategies for advancing its clinical application. Key areas of focus include Luteolin's impacts on hepatic lipid metabolism, inflammation, oxidative stress, and its pharmacokinetic limitations. The review emphasizes the necessity of rigorous clinical trials to confirm its effectiveness and safety in human NAFLD populations.

Keywords: Luteolin; Non-Alcoholic Fatty Liver Disease (NAFLD); Hepatic lipid metabolism; Autophagy; Apoptosis; Natural compounds; Clinical translation

Introduction

Non-Alcoholic Fatty Liver Disease (NAFLD) has become increasingly prevalent alongside rising rates of obesity and metabolic syndrome. This spectrum of disorders ranges from benign fat accumulation in the liver to more severe conditions such as Non-Alcoholic Steatohepatitis (NASH) and liver cancer. Current treatments are limited, prompting exploration into natural compounds like Luteolin as potential therapeutic options. Luteolin, sourced from various dietary sources, presents a novel approach due to its anti-inflammatory, antioxidant, and lipid-lowering properties [1].

Description

The journey from preclinical promise to clinical application is often fraught with challenges and complexities. In the case of Luteolin, an abundant flavonoid with multifaceted pharmacological properties, the transition from laboratory research to practical therapeutic use in NAFLD patients encapsulates both hope and hurdles. To thoroughly understand the therapeutic potential and limitations of Luteolin, it is crucial to dissect the existing preclinical evidence, scrutinize its pharmacological characteristics, assess its safety profile, and explore strategies for successful clinical translation.

Preclinical evidence

Luteolin's efficacy in preclinical models of NAFLD has been extensively documented. Studies indicate its ability to reduce fat accumulation in the liver by affecting AMP-Activated Protein Kinase (AMPK) and Sterol Regulatory Element-Binding Proteins (SREBPs) [2]. Additionally, Luteolin enhances autophagy and reduces cell death in liver cells, thus mitigating liver damage associated with NAFLD progression.

Pharmacological properties

Despite promising results in preclinical studies, Luteolin faces challenges related to its absorption and metabolism. Its limited solubility and rapid breakdown hinder its effective delivery to target tissues, necessitating improvements in formulation for enhanced clinical efficacy [3].

Safety and toxicity

Animal studies indicate that Luteolin has an overall favorable safety profile, but comprehensive human trials are lacking. Long-term studies are critical to evaluate potential adverse effects, particularly at higher doses or with prolonged use [4].

Clinical translation

Bridging the success of preclinical studies to clinical applications poses significant challenges. Rigorous randomized controlled trials are essential to validate the efficacy of Luteolin in diverse populations with NAFLD and to establish optimal dosing strategies [5].

Research gaps and future directions

Important gaps in research include the need for studies on doseresponse relationships, exploration of combined therapies with existing treatments for NAFLD, and clarification of Luteolin's specific mechanisms of action in living organisms [6]. Addressing these gaps is crucial to advancing Luteolin from experimental research to practical clinical use.

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Conclusion

Luteolin shows promise as a therapeutic option for NAFLD due to its diverse effects on hepatic lipid metabolism, inflammation, and oxidative stress. However, successful clinical implementation requires overcoming challenges related to its absorption and conducting robust clinical trials. Future research should focus on optimizing Luteolin formulations, assessing long-term safety, and confirming its effectiveness in diverse patient populations. With concerted efforts, Luteolin could become a safe and effective treatment for NAFLD, addressing a critical gap in current medical options.

References

Tong J, Gao J, Liu Q, He C, Zhao X, et al. (2020) Resveratrol derivative excited postsynaptic potentiation specifically via PKC-NMDA receptor mediation. Pharmacol Res 152: 104618.

- Liu G, Zhang Y, Liu C, Xu D, Zhang R, et al. (2014) Luteolin alleviates 2. alcoholic liver disease induced by chronic and binge ethanol feeding in mice. J Nutr 144: 1009-1015.
- Liu Y, Tian X, Gou L, Sun L, Ling X, et al. (2013) Luteolin attenuates diabetes-associated cognitive decline in rats. Brain Res Bull 94: 23-29.
- Yang JT, Wang J, Zhou XR, Xiao C, Lou YY, et al. (2018) Luteolin 4. alleviates cardiac ischemia/reperfusion injury hypercholesterolemic rat via activating Akt/Nrf2 signaling. Naunyn Schmiedebergs Arch Pharmacol 391: 719-728.
- Zhu M, Chen D, Li D, Ding H, Zhang T, et al. (2013) Luteolin inhibits 5. angiotensin II-induced human umbilical vein endothelial cell proliferation and migration through downregulation of Src and Akt phosphorylation. Circ J 77: 772-779.
- Xin SB, Yan H, Ma J, Sun Q, Shen L (2016) Protective effects of luteolin on lipopolysaccharide-induced acute renal injury in mice. Med Sci Monit 22: 5173.