THE ANATOLIAN JOURNAL OF CARDIOLOGY

Official journal of the TURKISH SOCIETY OF CARDIOLOGY

Critical Appraisal of Isorhamnetin as a Candidate for Pulmonary Arterial Hypertension Treatment

LETTER TO THE EDITOR

To the Editor,

The recent study by Shao et al[1] titled "Bioinformatic Analysis and Molecular Docking Identify Isorhamnetin as a Candidate Compound in the Treatment of Pulmonary Artery Hypertension," was read with great interest, suggesting a potential role for isorhamnetin in pulmonary arterial hypertension (PAH) through computational modeling.[1] While this study lays a foundation for future exploration, it is believed that certain critical aspects warrant further investigation.

Flavonoids, including isorhamnetin, have been reported to possess vasodilatory properties, yet their efficacy in PAH remains unverified. Prior studies emphasize the necessity of *in vivo* validation to confirm their mechanistic role in modulating pulmonary vascular resistance.[2] Further experimental models, including animal studies and human endothelial assays, are imperative to establish translational relevance.

A significant limitation of flavonoid-based therapies lies in their bioavailability. Evidence suggests that isorhamnetin undergoes rapid metabolism, resulting in poor systemic absorption, thereby limiting its therapeutic potential.[3] To ascertain its viability as a PAH treatment, detailed pharmacokinetic profiling and formulation strategies aimed at enhancing its bioavailability are essential.

In conclusion, Shao et al[1] provide an intriguing preliminary framework; however, additional experimental validation and pharmacokinetic studies are crucial before isorhamnetin can be considered a viable therapeutic option for PAH. Further research in these domains is encouraged to substantiate these promising findings.

Declaration of Interests: The authors have no conflicts of interest to declare.

Funding: The authors declare that this study received no financial support.

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Cite this article as: Iftikhar MK, Iftikhar Q. Critical appraisal of isorhamnetin as a candidate for pulmonary arterial hypertension treatment. *Anatol J Cardiol.* 2025; 29(7):375.

DOI:10.14744/AnatolJCardiol.2025.5303

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