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LETTER

A Comparative Discussion on the Selection of Cardiac Hypertrophy Models: TAC Surgery Vs Ang II Infusion [Letter]

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Dear editor

We reviewed the article by Yang et al titled "Apigenin Attenuates Transverse Aortic Constriction-Induced Myocardial Hypertrophy: The Key Role of miR-185-5p/SREBP2-Mediated Autophagy" published in your esteemed journal.[1] This study offers new insights into the treatment of cardiac hypertrophy, demonstrating that targeting specific miRNAs and autophagy pathways could lead to the development of novel therapeutic strategies. We sincerely appreciate the rigorous efforts and valuable contributions made in this research.

However, we noticed that the study employed two different models for investigating in vivo and in vitro cardiac hypertrophy mechanisms. Specifically, the in vivo experiments utilized Sprague-Dawley (SD) rats to create a Transverse Aortic Constriction (TAC) model, while the in vitro experiments involved Angiotensin II (Ang II)-induced hypertrophy in cardiomyocytes. In contrast, the study by Ye et al used chronic Ang II infusion to establish an in vivo cardiac hypertrophy model.[2] We have a few thoughts regarding the decision to use different modeling approaches in this study:

First, Ang II injection is relatively simple, requiring no complex surgical procedures, while TAC surgery is more complicated, demanding proficient surgical skills and careful postoperative care to avoid complications. Second, Ang II injection affects multiple systems, potentially causing non-cardiac-related side effects such as vasoconstriction and increased renal burden, making it difficult to focus solely on cardiac structural remodeling.[3] In contrast, TAC surgery primarily targets the heart, allowing for more precise study of cardiac functional changes with reduced interference from other systems. Additionally, the dosage and duration of Ang II injection must be carefully controlled to avoid systemic effects that introduce variability. On the other hand, the TAC model achieves precise control of pressure overload through surgery, leading to more reproducible experimental results.[4] Moreover, the systemic effects of Ang II result in greater pharmacokinetic variability between individuals, which may increase the variability of experimental outcomes. In contrast, the TAC model relies less on endocrine and other systems due to its mechanical nature, improving the reliability of results. Lastly, a meta-analysis on the TAC model indicated that specific mouse strains, gender, and age significantly influence the quality of the model, but these confounding factors can be controlled through experimental design.[5]

In conclusion, although TAC surgery is more complex, we believe that through careful optimization of procedures and experimental design, the TAC model can provide more reliable results compared to direct Ang II injection.

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