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Apigenin Attenuates Transverse Aortic Constriction-Induced Myocardial Hypertrophy: The Key Role of miR-185-5p/SREBP2-Mediated Autophagy [Letter]

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Dear editor

We have read the article by Yan et al entitled Apigenin Attenuates Transverse Aortic Constriction-Induced Myocardial Hypertrophy: The Key Role of miR-185-5p/SREBP2-Mediated Autophagy.[1] The purpose of this study was to clarify the apigenin anti-myocardial hypertrophy(MH) specific to the changes in miR-185-5p-mediated autophagy. Apigenin (4', 5, 7-trihydroxyflavone) is a natural flavonoid compound commonly found in vegetables and fruits and has been used traditionally for hypertension by locals in many countries.

Yan et al conducted research on transverse aortic constriction (TAC) in rats to induce Myocardial Hypertrophic symptoms. TAC is a widely used method to mimic cardiovascular disease in humans and to elucidate the fundamental signaling processes involved in the cardiac hypertrophic response and the development of heart failure. Other experimental models of heart failure, such as complete occlusion of the left anterior coronary artery (LAD) when compared with TAC, show that TAC provides a more reproducible model of cardiac hypertrophy and a more gradual time course in the development of heart failure.[2] They found that TAC surgery caused weight increase, structure damage, and collagen deposition in the hearts of model rats. While the addition of apigenin can improve cardiac function, attenuated cardiac hypertrophy which induced by TAC surgery.

Previously Gao et al, found that apigenin improves hypertension and cardiac hypertrophy in SHRs which are associated with the down-regulation of NADPH oxidase-dependent ROS generation and inflammation in the PVN. Previously, Gao et al demonstrated that hypertension and cardiac hypertrophy in SHRs can improved using apigenin by down-regulating NADPH oxidase-dependent ROS production and inflammation in the PVN.[3] Moreover, the cardioprotective effect of apigenin was identified by detecting changes in miR-185-5p/SREBP2 axis. MicroRNA (miRNA) is a type of non-coding RNA that either inhibits RNA translation or induces the destruction of target mRNAs. Mostly, miRNAs alter cellular signaling by targeting multiple genes within the pathway. Specifically, miR-185 was consistently detected in four different cardiac hypertrophy signaling pathways and was highly expressed in the heart, brain, and kidney indicating that miR-185 may have a potential role in these organs. In the heart of pressure overload-induced cardiac hypertrophy models miR-185 is expressed differentially. Moreover, miR-185 has 22 high-score targets in the cardiac hypertrophy signaling pathway, as determined by cross-species target predictions.[4] Yan et al found that TAC induced the expression level of miR-185-5p and decreased the expression level of SREBP2, while the addition of apigenin reserved this effect.[1] Kim et al 2015 has found that miR-185 significantly inhibited cardiac hypertrophy signaling through numerous targets, making it a potential drug target for diseases such as heart failure.

We acknowledge and appreciate the findings obtained by this research. We agree that apigenin had anti-MH effects where they could increase the autophagy effect by inhibiting miR-185-5p and activating SREBP.

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Disclosure

All authors report no conflicts of interest in this communication.

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