Dear Editor:

We read the article by Prado-Uribe et al. (1) “Role of Thyroid Hormones and mir-208 in Myocardial Remodeling in 5/6 Nephrectomized Rats” with great interest. The authors demonstrated that myocardial miR-208 expression was dramatically decreased but cardiac fibrosis was markedly increased in 5/6 nephrectomized rats when compared to sham rats. In thyroidectomized group, myocardial TGF-β expression was significantly reduced, whereas miR-208 was notably augmented. Treatment with thyroxine in 5/6 nephrectomized rats could induce upregulation of miR-208 and downregulation of TGF-β expression. Together we may draw a conclusion that upregulation of miR-208 could prevent cardiac fibrosis in 5/6 nephrectomized rats induced by thyroxine supplementation and miR-208 may have an inverse relationship with cardiac fibrosis.

However, existing evidence has shown that miR-208 has a positive correlation with cardiac fibrosis indicated by myocardial collagen volume fraction in human dilated cardiomyopathy (2). Inhibition of miR-208 expression using antagomiR208 could reduce cardiac fibrosis in response to a high-salt diet in rats (3). There was no fibrosis in miR-208 lacking mice heart compared with wild-type mice induced by thoracic aortic banding (4). Further study revealed that prevention of miR-208 could reduce endoglin and collagen I expression, and overexpression of miR-208 could obviously increase endoglin and collagen I expression in rat cardiac myoblasts induced by mechanical stretch, which may result in cardiac fibrosis (5). Moreover, cardiac fibrosis was apparently augmented resulting from overexpression of miR-208 in aorta-caval (AV) shunt rats, but distinctly attenuated after using antagomiR208. MiR-208 could induce myocardial fibrosis in AV shunt rats (6). These data argued that downregulation of miR-208 may prevent cardiac fibrosis.

In summary, these conflicting findings may suggest that the role of miR-208 in cardiac fibrosis is not clearly elucidated. The reason for these differences cannot be explained only by different animal models. Whether miR-208 can prevent or promote cardiac fibrosis is still to be determined. Further evidence is needed to identify the function of miR-208 in cardiac fibrosis.

Conflict of Interest

The authors declare no conflict of interest.

References


YING HUANG a,b,c
TAO XU a,b
JUN LI a,b,c

aSchool of Pharmacy
Anhui Key Laboratory of Bioactivity of Natural Products
Anhui Medical University
Hefei, 230032, China
bKey Laboratory of Anti-inflammatory and Immune Medicine
Anhui Medical University, Ministry of Education
PR China
cDepartment of Cardiology
The First Affiliated Hospital of Anhui Medical University
Hefei, 230032, China

Address reprint requests to: Jun Li, Professor
School of Pharmacy, Anhui Medical University
Mei Shan Road, Hefei
Anhui Province
230032, China
Phone: +86 551 65161001
FAX: +86 551 65161001
E-mail: kaoyan110@126.com, lj@ahmu.edu.cn

Received for publication February 12, 2014; accepted March 28, 2014 (ARCMED-D-14-00090).