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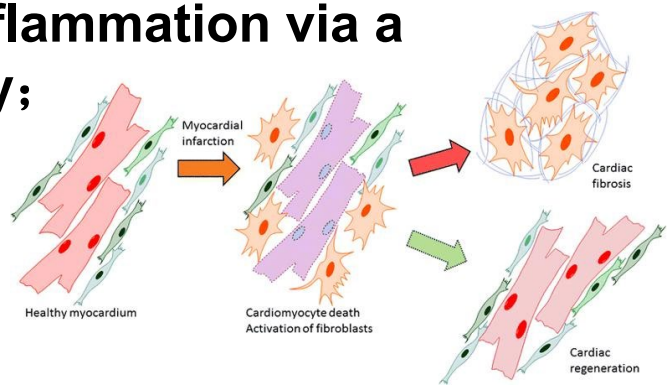
Low-intensity pulsed ultrasound ameliorates angiotension II-induced cardiac fibrosis by alleviating inflammation via a caveolin-1-dependent pathway

Key words: **Low-intensity pulsed ultrasound (LIPUS); Caveolin-1; Cardiac fibrosis; Inflammation; Angiotensin II.**

Research Summary

This article mainly focused on the key role of low-intensity pulsed ultrasound (LIPUS) in regulating angiotensin II (AngII)-induced cardiac fibrosis and the underlying molecular mechanisms:

- **LIPUS irradiation could ameliorate AngII-induced cardiac fibrosis in vitro;**
- **LIPUS irradiation could improve AngII-induced cardiac remodeling in vivo;**
- **LIPUS irradiation alleviates inflammation via a caveolin-1-dependent pathway;**



Innovation points

A series of comprehensive figures were generated to exhibit the anti-fibrotic effects of LIPUS on ameliorating AngII-induced cardiac fibrosis.

Figure 1 | Effects of LIPUS on AngII-induced cardiac fibrosis in vitro.

Figure 2 | Effects of LIPUS on AngII-induced myocardial hypertrophy in vivo.

Figure 3 | Effects of LIPUS on AngII-induced myocardial fibrosis in vivo.

Figure 4 | Role of caveolin-1 in mediating the anti-hypertrophic effects of LIPUS in vivo.

Figure 5 | Role of caveolin-1 in mediating the anti-fibrotic effects of LIPUS in vitro

Figure 6 | Role of caveolin-1 in mediating the anti-fibrotic effects of LIPUS in vivo.

Figure 7 | Relationship between inflammation and caveolin-1 in the anti-fibrotic effects of LIPUS in vivo.

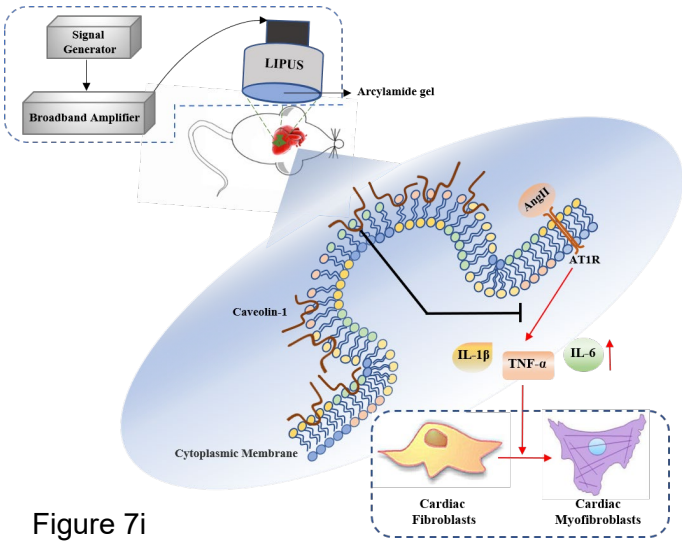


Figure 7i

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