

A Novel Curcumin Formulation, ASD-Cur, Suppressed the Development of Systolic Dysfunction After Myocardial Infarction in Rats

Hidemichi Takai,¹ Yoichi Sunagawa,^{1,2,3} Masafumi Funamoto,^{1,2} Kana Shimizu,^{1,2} Satoshi Shimizu,^{1,2} Yasufumi Katanasaka,^{1,2,3} Yusuke Miyazaki,^{1,2,3} Atsusi Imaizumi,⁴ Tadashi Hashimoto,⁴ Hiromichi Wada,² Koji Hasegawa^{1,2} and Tatsuya Morimoto^{1,2,3}

1. University of Shizuoka, Shizuoka, Japan; 2. Kyoto Medical Center, Kyoto, Japan;
3. Shizuoka General Hospital, Shizuoka, Japan; 4. Therabiopharma Inc, Japan

Citation: *European Cardiology Review* 2021;16:e69. **DOI:** <https://doi.org/10.15420/ecr.2021.16.P013>

Open Access: This work is open access under the CC-BY-NC 4.0 License which allows users to copy, redistribute and make derivative works for non-commercial purposes, provided the original work is cited correctly.

Objective: It has previously been reported that curcumin prevents the development of heart failure in animal models, demonstrating that the compound is a potential treatment for the disease in humans. Although curcumin is known to be safe, its therapeutic efficiency is limited due to its low bioavailability. To overcome this problem, we developed ASD-Cur, an amorphous formulation of curcumin. In this study, we investigated the effect of ASD-Cur and compared it with Theracurmin®, a colloidal submicron dispersion of curcumin.

Material and methods: Male SD rats were subjected to MI or sham surgery. One week after surgery, the MI rats were randomly assigned to four groups: vehicle, ASD-Cur (0.2 mg/kg curcumin), or Theracurmin (0.2 or 0.5 mg/kg curcumin). Daily oral administration of these compounds was repeated for 6 weeks. After echocardiographic examination, myocardial

cell diameter, perivascular fibrosis, mRNA levels, and the acetylation of histone H3K9 were measured.

Results: Echocardiographic analysis of the rat hearts showed that 0.2 mg/kg ASD-Cur and 0.5 mg/kg Theracurmin significantly improved both MI-induced reduction in fractional shortening (FS) and left ventricular hypertrophy to the same extent. Both treatments significantly suppressed MI-induced increases in myocardial cell diameter, perivascular fibrosis, mRNA levels of hypertrophic markers and cardiac fibrosis, and acetylation of histone H3K9 to the same extent.

Conclusion: These findings indicated that ASD-Cur has greater therapeutic potency towards MI-induced heart failure at a lower dose than Theracurmin. □