

# Gene therapy may activate stem cells in heart failure patients

22 February 2013



Image courtesy of Blausen Medical

Delivery of an SDF-1 encoding plasmid acts a homing signal for stem cells and improves clinical status in patients with symptomatic heart failure due to ischemic cardiomyopathy, according to a study published online Feb. 21 in *Circulation Research*.

(HealthDay)—Delivery of an SDF-1 encoding plasmid (JVS-100) acts a homing signal for stem cells and improves clinical status in patients with symptomatic heart failure due to ischemic cardiomyopathy (IsCM), according to a study published online Feb. 21 in *Circulation Research*.

Marc Penn, M.D., from Summa Health System in Akron, Ohio, and colleagues conducted a phase I, open-label, dose-escalation study (5, 15, or 30 mg of JVS-100 via endomyocardial injection) with 12 months follow-up in subjects with IsCM. The 17 subjects had New York Heart Association (NYHA) Class III [heart failure](#), with an [ejection fraction](#) ?40 percent on stable medical therapy.

The researchers found that at one month and four months the primary safety end point of no major adverse cardiac events was met. All cohorts demonstrated improvements in six-minute walk distance (6MWd), quality of life (QOL), and NYHA class at four months. Improvements over baseline in 6MWd were seen in the 15-mg and 30-mg dose groups (15 mg: Median [Range]: 41 [3 to 61] m; 30 mg: 31 [22 to 74] m), as were improvements in QOL (15 mg: ?16 [+1 to ?32] points; 30 mg: ?24 [+17 to ?38] points). Improvements in symptoms

were maintained at 12 months.

"These data highlight the importance of defining the [molecular mechanisms](#) of stem cell-based tissue repair and suggest that over-expression of SDF-1 via gene therapy is a strategy for improving heart failure symptoms in patients with IsCM," the authors write.

One author is named as an inventor on [patent applications](#) for the use of SDF-1 to treat cardiovascular disease that have been licensed by Juventas Therapeutics, which funded the study.

**More information:** [Abstract](#)

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APA citation: Gene therapy may activate stem cells in heart failure patients (2013, February 22) retrieved 24 June 2018 from <https://medicalxpress.com/news/2013-02-gene-therapy-stem-cells-heart.html>

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