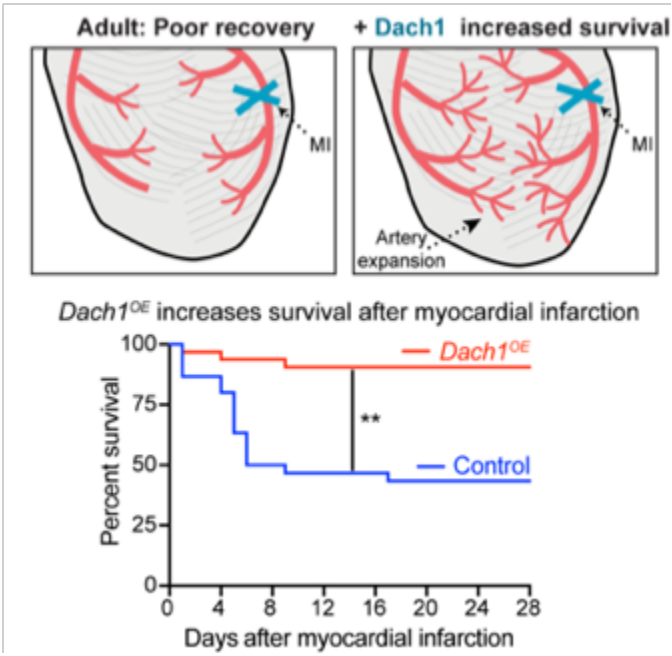
Docket #: S19-329

Regulation of Dach1 for Treatment of Coronary Artery Disease

Stanford researchers have developed a strategy to manipulate the architecture of coronary arteries by upregulating the transcription factor Dach1 in endothelial cells. Mice with overexpressed Dach1 had an increased number of artery cells, as well as longer, more branched, and more robust artery networks in the heart. This expansion to the coronary arteries caused by Dach1 overexpression, improved survival and heart function in adult mice following myocardial infarction (MI). For patients with Coronary Artery Disease (CAD), this offers a non-surgical therapy and avoids commonly ineffective treatments such as blood thinners, vasodilators or cholesterol lowering medication. Increased vasculature around the heart leads to higher survival rates and could help prevent myocardial damage after MI.



Stage of Research

- in vivo studies

Applications

- **Patients with CAD**
- Forming coronary artery networks that protect against myocardial infarction

Advantages

- **Non-surgical approach**
- Strengthened artery networks during early CAD
- Possible prevention of myocardial damage after MI

Publications

- Raftrey et. al. bioRxiv (2020) Dach1 extends artery networks and protects against cardiac injury

Patents

- Published Application: 20220340627

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