Heart Failure (HF) is a chronic, progressive condition in which the heart muscle is unable to pump sufficient quantity of blood through the arterial system to meet the body's needs for blood and oxygen.

There is an unmet need to develop new antifibrotic therapies to treat HF since fibrosis is present even in the heart of those HF patients treated according to the current clinical practice guidelines, and it is associated with a bad prognosis.

A novel target to treat myocardial fibrosis in HF has been identified (from biobank and patients data).
- It is over-stimulated in the myocardium of HF patients
- There is a related biomarker measurable in blood
- A proof of concept has been developed: siRNA and chemical probe.

Primary Indication: Heart Failure.

Scope of the problem
- In the US, over 5.7 million people are currently living with HF. An estimated 400,000 to 700,000 new cases of HF are diagnosed each year.
- About one in five people who have HF die within one year from diagnosis despite being treated in accordance with the standard guidelines.
- Global HF therapeutic market reached 4,068.5M USD in 2010 and it has been predicted to reach 5, 104.1M in 2018.
- HF is the cause for 12-15 million medical visits per year and 6.5-7 million days of hospitalization per year.

Patient need addressed
To prevent the development of myocardial fibrosis that is associated with a detrimental impact on cardiac function and on clinical outcome in HF patients.

New target
- A new potential anti-fibrotic target over-stimulated in the myocardium of HF patients and associated with myocardial fibrosis (Figures A and B).
- Target activity correlates with a biomarker measurable in blood (Figure C).

Proof of Concept
In vitro. Target inhibition prevents TGF-β induced collagen production in human fibroblasts.

Safety
Homoyzgous constitutive knock-out mice are viable, with absence of gross abnormalities and no myocardial defects described.

Intellectual Property
Patent application to be filed.

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