SARM1 INHIBITION AMELIORATES METABOLIC CARDIOMYOPATHY
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Cardiovascular disease (CVD) is the number one cause of death globally and the most common cause of death in patients with metabolic dysregulation as seen with Type I and Type II Diabetes.

Nicotinamide adenine dinucleotide (NAD+) depletion is a hallmark in the pathogenesis of diabetic, aged or pressure-overloaded hearts.

Strategies to activate synthesis pathways and replenish NAD+ levels have shown promises to treat heart diseases.

Novel treatments for heart disease that focus on inhibiting the NAD+ consuming hydrolases, specifically Sterile Alpha and TIR motif-containing 1 (SARM1), are yet to be developed.
Inhibiting NAD+ consuming hydrolases increases NAD+ availability and can ameliorate metabolic CVD.
FINDINGS

SARM1 deficiency maintains NAD+ levels and protects cardiac function in diabetic mice

FIG A: NAD+ levels in diabetic WT (D-WT) and diabetic KO (D-KO) male mice
FIGS B and C: Systolic function (% FS) and the diastolic function (E'/A' ratio) in D-WT and D-KO male mice

FIGS D-F: Myocardial Performance Index (MPI) measurements, e/E’ ratios and the IsoVolumic Relaxation Time (IVRT) in D-WT and D-KO male mice
Dr. Lee has identified a novel mechanism to treat metabolic cardiomyopathy.

He has demonstrated that inhibition of SARM1 elevates NAD+ levels and ameliorates metabolic cardiomyopathy.

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