

# Cardio-metabolic Models

Fully validated and customizable in vivo and in vitro cardio-metabolic models to allow preclinical drug development for different heart failure etiologies

The complex nature of cardiac dysfunction severely complicates drug development. Our validated cardio-metabolic models are specifically developed to represent one or multiple relevant heart failure disease comorbidities and etiologies, resulting in data of high translational value. Our models mimic key pathophysiological mechanisms of both heart failure with reduced and preserved ejection fraction (HFrEF and HFpEF).

With flexible study designs and rapid start times, InnoSer experts take a collaborative approach when it comes to your research needs. Our models are executed by highly skilled surgeons using cutting edge techniques to deliver reliable and reproducible study results.

## Choose the right model for your research

	Ischemic		Hypertrophic		Cardio-metabolic
	Myocardial Infarction	Ischemia/Reperfusion	Pressure Overload Hypertrophy	Hypertension	Diabetic Rat Model (Cardiomyopathy)
	Permanent LAD	Transient LAD	Transverse Aortic Constriction	Angiotensin II Osmotic Pump	High Fat + High Sugar
Ischemia	✓	✓			
Cardiomyocyte Loss	✓	✓			
Inflammation	✓	✓	✓	✓	✓
Fibrosis	✓	✓	✓	✓	✓
Hypertension			✓	✓	✓
Renal Complications				✓	✓
HFrEF	✓	✓	✓	✓	
HFpEF			✓	✓	✓

To benefit from a complete drug development solution these models can be integrated or combined with conventional drug discovery and preclinical research services aimed to assess your drugs pharmacological and toxicological profile.

**Get in touch**

info@innoserlaboratories.com



# Evaluate the efficacy of your compound through key endpoints

Thoroughly explore your compound's mode of action with tailored and specific readout options that provide the most relevant insights to advance your research.

## Echocardiography and functional readouts:

- Ultrasound left and right ventricular function
- Speckle tracking, myocardial motion and strain analysis
- Cardiac remodeling and hypertrophy
- Infarct size & area at risk
- Coronary flow
- Hemodynamics analysis

## Ex vivo cell shortening and contraction kinetics

### Metabolic profiling

### Glucose tolerance testing (OGTT, IPGTT)

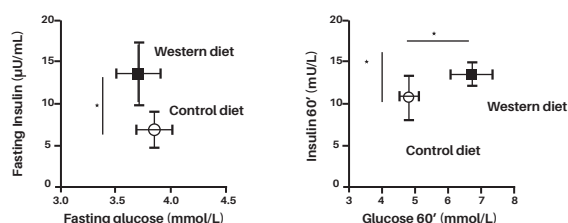
### Blood biochemistry:

- Blood cholesterol
- Triglyceride
- Insulin
- Glucagon

**A**

Echocardiography	Control Diet	Western Diet
Ejection fraction (%)	74 ± 2	69 ± 3
Anterior wall thickness (mm)	1.46 ± 0.02	1.71 ± 0.04 *
Left ventricle pressure (mmHg)	99 ± 3	105 ± 2
End-diastolic pressure (mmHg)	6.46 ± 0.61	10.65 ± 1.90 *

**B**



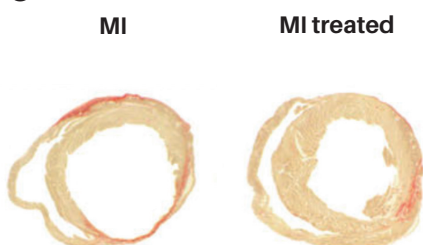
**A.** Echocardiography healthy rats vs after 18 weeks Western diet. **B.** OGTT measurements in healthy rats vs after 18 weeks Western diet

## Histopathology readouts:

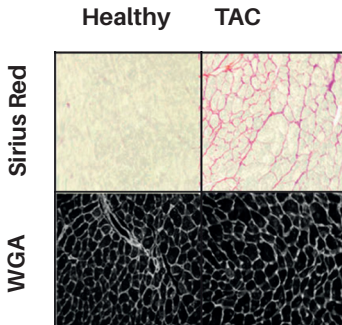
Tailored application of clinically relevant markers for translational results.

Indications	Blood Markers	Histology Biomarkers
Fibrosis		Masson's trichrome, Sirius Red, Collagen-I Collagen-III, $\alpha$ -SMA
Apoptosis	Cytochrome C, BCL2, BCLX	TUNEL assay, Caspase 3, P53
Heart Muscle Damage	TnT, Tni, BNP, ANP	Histologic assessment - H&E
Angiogenesis		VEGF, CD34, CD31
Repair		Ki-67
Inflammation	CRP	IL-6, TNF- $\alpha$
Kidney Function	BUN, creatinine	Lcn2 (NGAL)

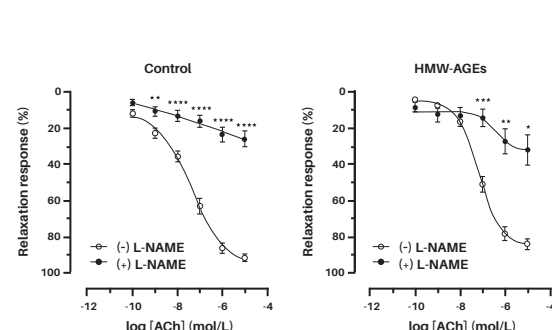
**C**



**D**



**E**



**C.** Infarct size after MI with and without treatment via sirius red. **D.** Fibrosis and cardiomyocyte hypertrophy histopathology in healthy and TAC induced mice. **E.** Ex vivo vascular reactivity of aortic rings of rats treated for 6 weeks with and without HMW-AGEs and their response to L-NAME.

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