



OUR SOLUTION · THE EHT MODEL

Human engineered heart tissue you can load, injure, perturb, and measure

hiPSC-based 3D engineered heart tissue (EHT) with automated functional characterization and FDA-validated*: one platform, every relevant cardiac phenotype, from foundational biology to a closed target-discovery loop.

Scalable

24 EHTs in 90 seconds

Predictive

human, robust & reproducible

Closed system

sterile, controlled analysis

Automated

GLP-compliant acquisition

Services across discovery, safety and efficacy – we perform clinical trials in a cell culture dish for you

Disease Modelling

- EHTs from control, patient-derived or isogenic gene-edited iPSC lines (individualized phenotypes)
- **Phenotypes on demand:** hypoxia/reoxygenation (post MI), HFpEF with diastolic dysfunction, pathological hypertrophy

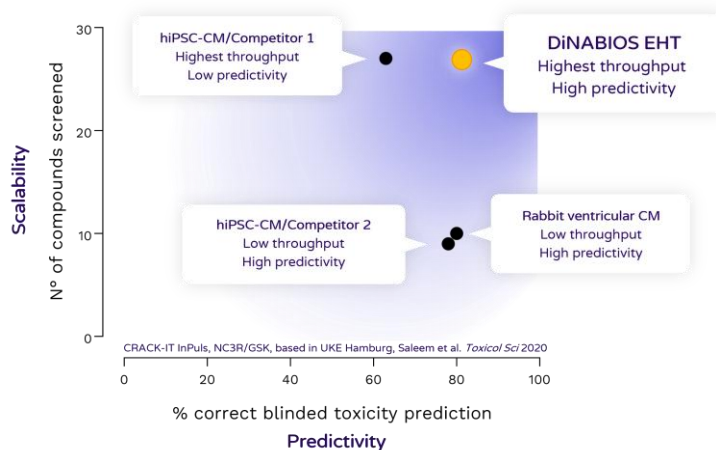
Discovery

- **Lead identification & optimization:** acute and chronic treatment of control or diseased EHTs
- Medium-throughput screening for advanced modalities: ASOs, anti-miRs, siRNA, and viral vectors
- CRISPR/Cas9-based target validation and functional genomics

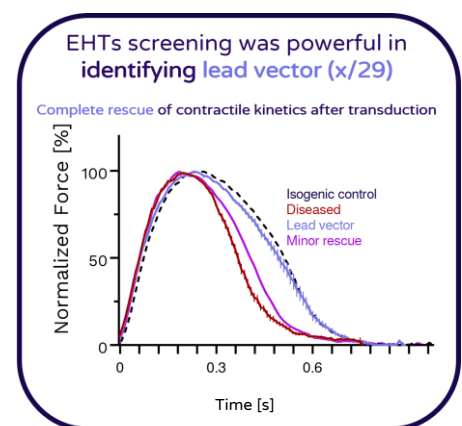
Efficacy & Safety

- **Preclinical testing, toxicology studies, risk assessment**
- Concentration–response curves, bolus application, chronic treatment
- Full diastolic panel: force, frequency, contraction and relaxation times, velocities, rhythm

Screening of 27 compounds with/without toxicological potential



Screening of 29 gene therapy candidates for lead identification



Human atrial trabeculae (hAT): still the gold standard, but a dying preparation**

Force runs down from the start. Significant decline after 20 min; time controls lose 4% at 5 min and 24% at 55 min.

Only 5 min per concentration step. Slow-acting drugs are missed: digoxin showed no inotropic effect in hAT, but did in EHT.

Material is scarce and fragile. 18 of 88 trabeculae were unusable before dosing; supply depends on cardiac surgery.

Usable window ≈ 30 min. EHTs show no run-down and stay stable for up to 240 min.

Wide scatter. Intra-batch CV of baseline force 49% (hAT, n = 52) versus 17% (EHT, n = 107).

Atrial, no ventricular phenotype, and no option for gene editing, AAV transduction or patient-specific lines.

EHT: same drug effects, a stable preparation, low scatter, ventricular-like phenotype and an unlimited supply.**

GLP & quality assurance

FACS, cell counter, SOPs, ELN, GLP-compliant acquisition & processing

Downstream analyses

RT-qPCR, Western blot, mass spectrometry, imaging ELISA biomarkers, sequencing

Consulting & support

Customized training, tailored study plans, technical and scientific platform support

How a programme runs: induce the phenotype → perturb it → measure 24 tissues in 90 s at any paced rate → integrate with omics and biomarkers in DiNALYTIQS™ → rank by restored phenotype and re-validate in independent donor lines or perform simple toxicology assessment/drug characterization on contractility

*Arefin et al. 2023, **Mannhardt et al. 2017