

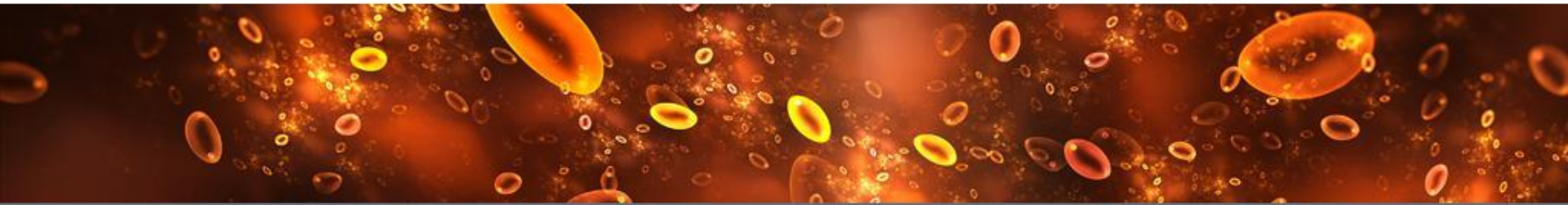
Corporate Presentation



Tamid**Bio**, Inc.



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Tamid Bio, Inc. Pipeline

	Product Candidate	Indication	Stage of Development				
			Preclinical	Phase 1	Phase 2	Phase 3	Commercial
Tamid Bio, Inc.	IDUA Enzyme Gene Therapy	MPS I Blindness					
	Dysferlin Gene Therapy	Dysferlinopathy					
	HLA-G Gene Therapy	Corneal Transplant Rejection / Anti-Vascularization					



Novel adeno-associated virus (AAV)-based gene therapies for improving various orphan diseases

Focus	Developing treatments towards towards rare diseases
Market Size	Potential disease targets: Mucopolysaccharidosis type 1 (MPS1) (prevalence of ~1-9/1,000,000) Corneal transplant rejection (~33,000 transplants per year in the US, failure rate up to 50%) Dysferlinopathies (LGMD2B: ~1-9/1,000,000 people, approximately 1,000-10,000 pts in the US & EU)
Product Candidates	IDUA Enzyme Gene Therapy for MPS1 Blindness HLA-G Gene Therapy for Corneal Transplant Rejection Dysferlin Gene Therapy for Dysferlinopathies
Development Stage	Preclinical

MPS1 Blindness Market Opportunity

Patient Population

MPS1 (aka Hurler and Scheie syndrome) is a rare lysosomal storage disorder, which has severe cardiac, orthopedic, developmental and ophthalmologic manifestations (90% lose vision, 50% progress to blindness).

Enzyme replacement therapy does not treat the ocular manifestations of MPS1. Bone marrow transplant (BMT), if a match is found, also does not treat the ocular manifestations of the disease well.

Corneal transplant is not used frequently (20% of patients), because it is cumbersome, high rate of rejection and does not have good durability.

Gene Therapy Target

Lack of the IDUA enzyme leads to the accumulation of glycosaminoglycans (GAGs) in the eye. The buildup of GAGs causes glaucoma, corneal clouding and subsequent retinal degeneration, amblyopia and optic atrophy.

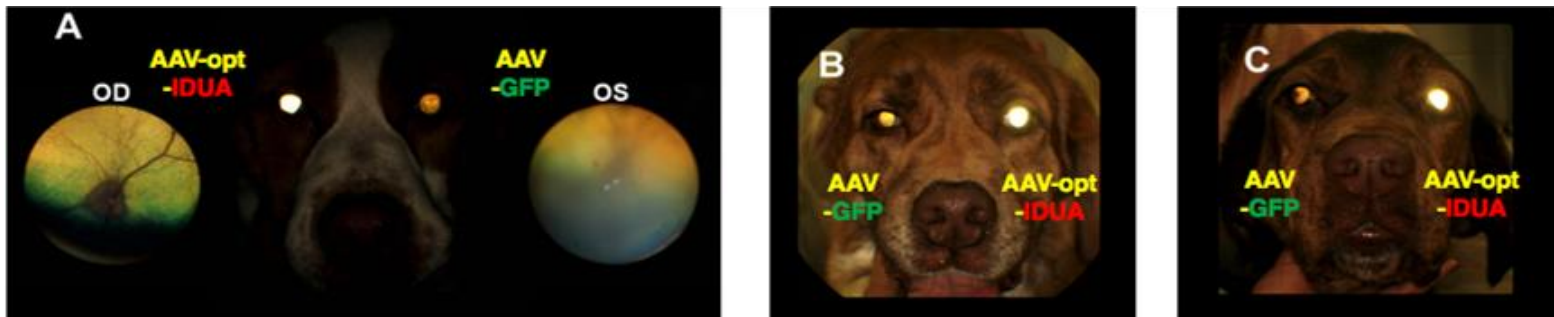
This gene therapy aims to constitutively replace the enzyme in the eye to metabolize GAGs leading to a decrease in accumulation.



Preclinical Data: MPS1 Dog Model

4 MPS1 dogs were treated with AAV Gene Therapy.

7 days post injection there was clearing of corneal clouding showing reversal of GAG buildup.



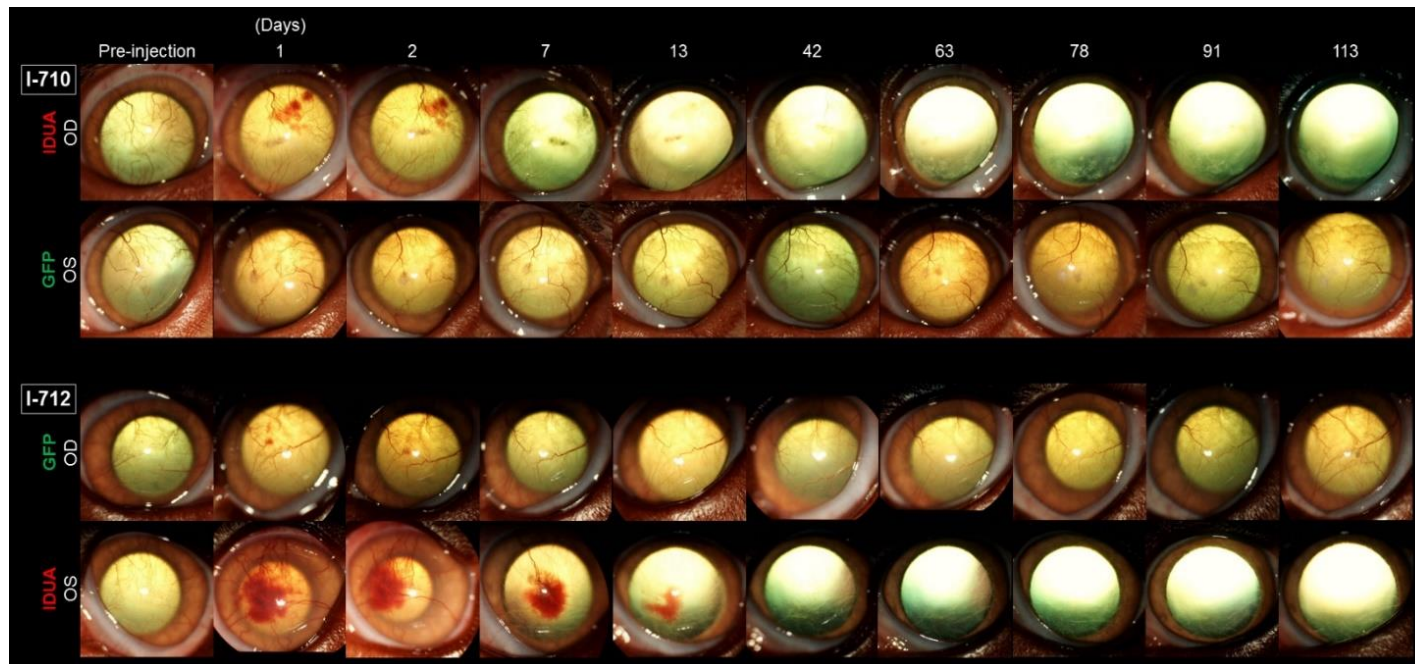
(shine in eye represents light being able to pass through clear ocular fluid, each photo is an individual dog with GFP eye acting as its own control. Photos were taken at 7, 8 and 11 weeks respectively)



Preclinical Data: MPS1 Dog Model

Two of the MPS1 dogs are represented below – both dogs were injected at 13 months with advanced disease.

Corneal clouding started to reverse by day 7 and maintain throughout the 25 week observation period





Pipeline Programs

Corneal Transplant Rejection

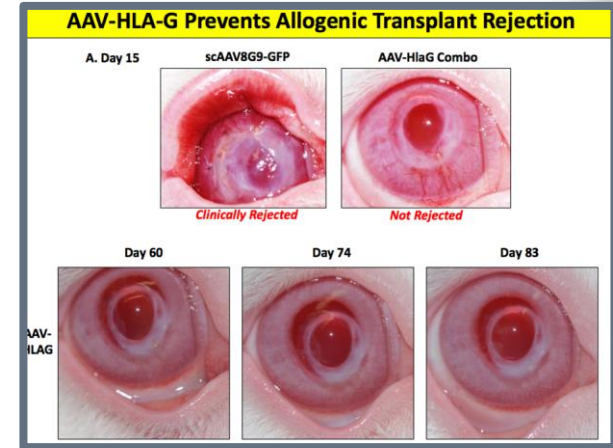
Approximately 33,000 Americans undergo corneal transplants per year. The failure rate in high risk patients, rejection rate can be greater than 50 percent.

HLA-G is a known immunomodulatory and anti-inflammatory molecule discovered in the human placenta where it plays a role in preventing the maternal immune system from rejecting the “foreign” fetus.

Preclinical Data:

Intrastromal injection in a rabbit disease model.

Injected corneas demonstrated the near complete absence of vasculature while control corneas became heavily vascularized. This result was coupled with decreased immune cell infiltration and fibrosis of the cornea.



Ex vivo HLA-G therapy prevents allogeneic corneal transplant rejection

Dysferlinopathies

Examples of dysferlinopathies include limb-girdle muscular dystrophy type 2B (LGMD2B) and Miyoshi myopathy (MM). They are caused by mutations in the dysferlin gene. In both dysferlinopathies patients usually become wheelchair bound as a result of muscle weakness.

The large size of the dysferlin protein dysferlin precludes its packaging into a single AAV capsid. Therefore, using 3D modeling a dysferlin-like molecule amenable to single AAV vector packaging was engineered (termed Nano-Dysferlin).

Preclinical Data: Improvement in motor function and muscle structure in LGMD2B mouse model

