Corporate Presentation

February 2026

Forward-looking statements

This presentation includes forward-looking statements, including forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. All statements other than statements of historical facts contained in this presentation are forward looking statements, including statements regarding our cash runway, strategy and plans, industry environment, potential growth opportunities, and the therapeutic potential of our programs. The words "believe," "may," "will," "estimate," "continue," "anticipate," "design," "expect," "could," "plan," "potential," "predict," "seek," "should," "would," or the negative version of these words and similar expressions are intended to identify forward-looking statements.

We have based these forward-looking statements on our current expectations and projections about future events and trends that we believe may affect our financial condition, results of operations, strategy, short- and long-term business operations and objectives, and financial needs. These forward-looking statements are subject to a number of risks, uncertainties and assumptions, including but not limited to, our ability to develop and advance our programs and product candidates, our ability to maintain and establish collaborations or strategic partnerships, our regulatory approvals and filings, and other risks, uncertainties and assumptions identified in our filings with the Securities and Exchange Commission (the "SEC"), including our most recent Form 10-K and Form 10-Q filed with the SEC, and any subsequent filings with the SEC.

Moreover, we operate in a very competitive and rapidly changing environment, and it is not possible for our management to predict all risks, nor can we assess the impact of all factors on our business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in any forward-looking statements we may make. In light of these risks, uncertainties and assumptions, the forward-looking statements and circumstances discussed in this presentation may not occur and actual results could differ materially and adversely from those anticipated or implied in the forward-looking statements. You should not rely upon forward-looking statements as predictions of future events. Although we believe that the expectations reflected in the forward-looking statements are reasonable, we cannot guarantee that the future results, levels of activity, performance or events and circumstances reflected in the forward-looking statements will be achieved or occur. Moreover, except as required by law, neither we nor any other person assumes responsibility for the accuracy and completeness of the forward-looking statements. We undertake no obligation to update publicly any forward-looking statements for any reason after the date of this presentation to conform these statements to actual results or to changes in our expectations, unless required by law.

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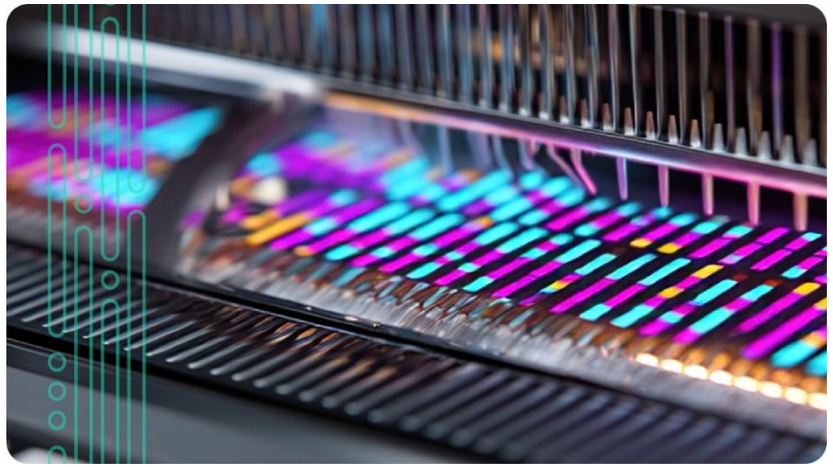
From novel genome editing systems to curative therapies

Metagenomi THERAPEUTICS



An in vivo genome editing company capitalizing on its proprietary technologies to create curative genetic medicines

Focusing on wholly owned programs in hemophilia A and secreted protein disorders, and partnered assets targeting cardiometabolic indications

Overcoming limitations of CRISPR/Cas9

20,000+ signature editing system from Metagenomi's database

N

Nucleases

Enabling gene knockdown, large deletions and large gene integration

BE

Base editors

Enabling single base correction

RIGS

RIGS

Enabling gene correction

CAST

CAST

Enabling large gene insertion

Proprietary genome editing

Attributes demonstrated in preclinical translation:

Higher specificity

No off-target cuts or translocations seen in genome-wide screens

Broad genomic targeting

Increased ability to edit targets of interest

Durable large-gene integration

Overcoming one of the major issues in AAV gene therapies

Multiplexed editing

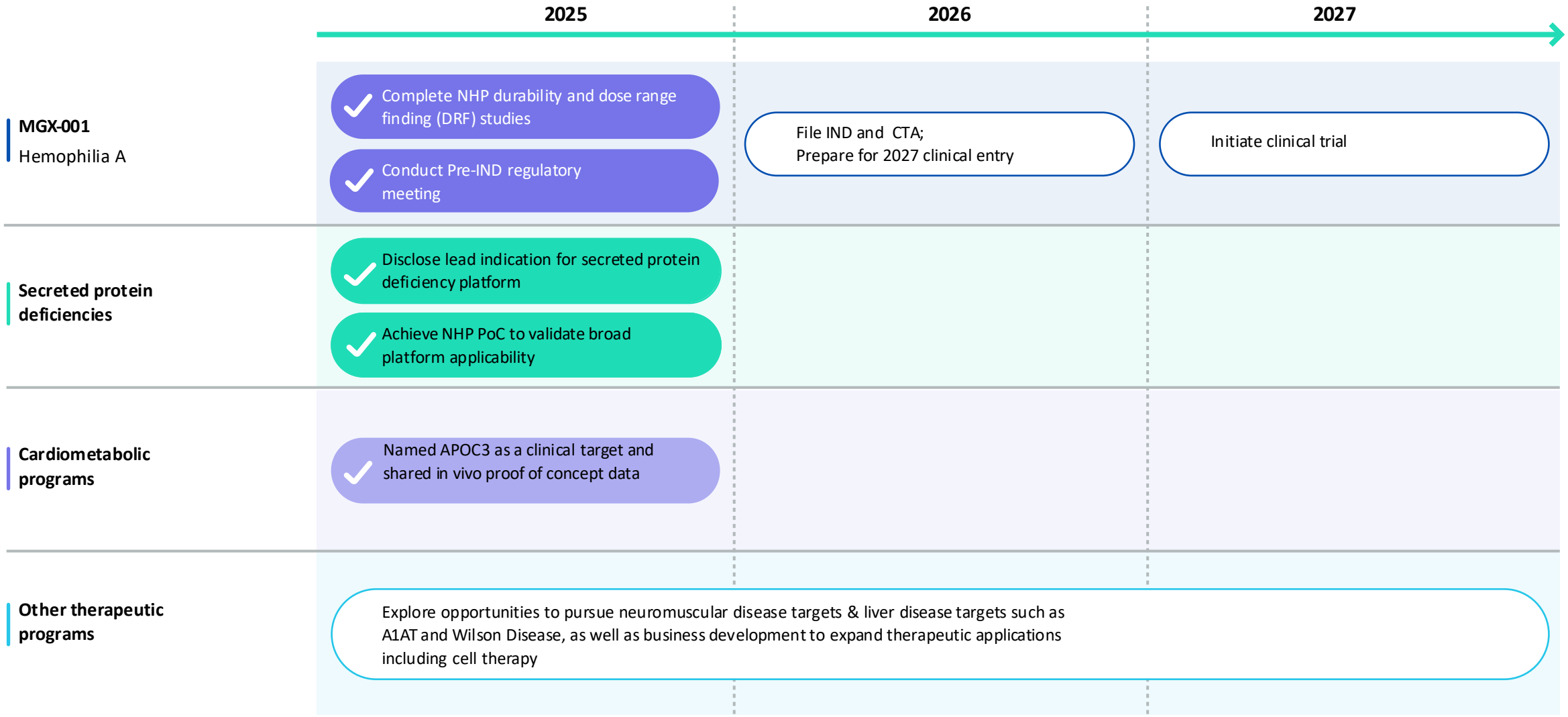
Using multiple editing systems to create new functionality in primary cells without translocations

With novel nucleases and other proprietary systems, we improve editing precision and expand genome targeting and editing functionality beyond CRISPR/Cas9 to effectively address genetically-driven diseases.

Progressing wholly-owned and partnered programs toward the clinic

Metagenomi is exploring opportunities to pursue neuromuscular disease targets & liver disease targets such as A1AT and Wilson Disease, as well as business development to expand therapeutic applications including cell therapy.

Focused on generating clinical data in 2027



MGX-001 in hemophilia A advancing toward clinic

Demonstrated curative Factor VIII activity with best-in-class treatment potential

- Dose-dependent efficacy of both AAV and LNP
- Therapeutically relevant FVIII activity in each animal treated in all but the lowest dose
- Data informs clinical dose regimen strategy

Builds on previous data

- Durable FVIII activity over an approximately 19-month study
- Encouraging safety profile, with minimal steroid use at the time of dosing
- No off-target editing

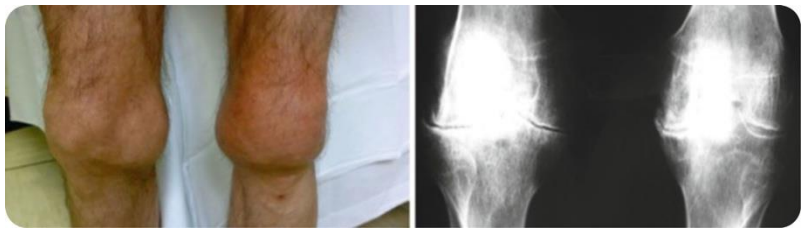
IND submission on track for Q4 2026

- Completed pre-IND meeting in December 2025
- Ongoing KOL and patient advocacy engagement

Potential competitive advantages of MGX-001

- Enables endogenous production of FVIII for hemostatic regulation
- Potential to effectively treat both adults and children
- One-time potentially curative therapy allowing patients a hemophilia free mindset

Hemophilia A: a validated target waiting for a durable cure



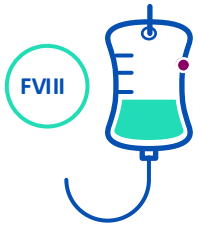
~26,500
patients in the U.S.[1]

~500,000
worldwide[2]

Hemophilia A is the most common X-linked inherited and de novo bleeding disorder, largely affecting males.

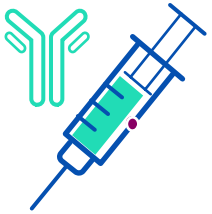
Caused by variety of mutations in the Factor VIII (FVIII) gene leading to loss of functional FVIII protein.

Current SOC:



Factor VIII replacement therapy

- IV typically dosed 1 - 3 times/week
- Significant adherence challenges
- Risk of breakthrough bleeding
- Chronic treatment, non-curative



Bi-specific antibody "mimetic"

- SQ dosed 1, 2 or 4 weeks post loading
- Risk of breakthough bleeding
- Treatment burden, non-curative

Gene therapy

- Variable initial efficacy
- Significant decline in FVIII levels over time
- High risk of prolonged corticosteroid use
- Not suitable for pediatric patients

Annual treatment cost[3]:

~$565K - $750K

Lifetime treatment cost:

~$18M - $24M[4]

One-time treatment cost[3]:

$2.9M[5]

NASDAQ: **MGX**

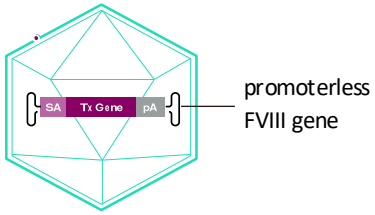
1- Soucie, J.M., et al, 2020. Haemophilia. Vol. 26, no. 3, pp. 487–493.
2 - Stonebraker, J. S., et al, 2010. Haemophilia. Vol. 16, pp. 20–32.
3 - ICER. Gene Therapy for Hemophilia B and A: Final Evidence Report. Dec 22, 2022.

4 - Curtis R et al. Poster presented at: 65th ASH Annual Meeting & Exposition; December 11, 2023; San Diego, CA.
5 - Zemplenyi A et al., Pharmacoeconomics (2024) 42(3):319–328. Hemophilia joint bleeds image: International Hemophilia Training Center.

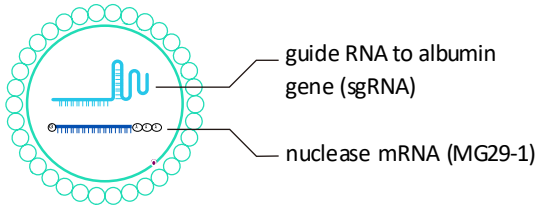
MGX-001 mechanism leverages natural promoter

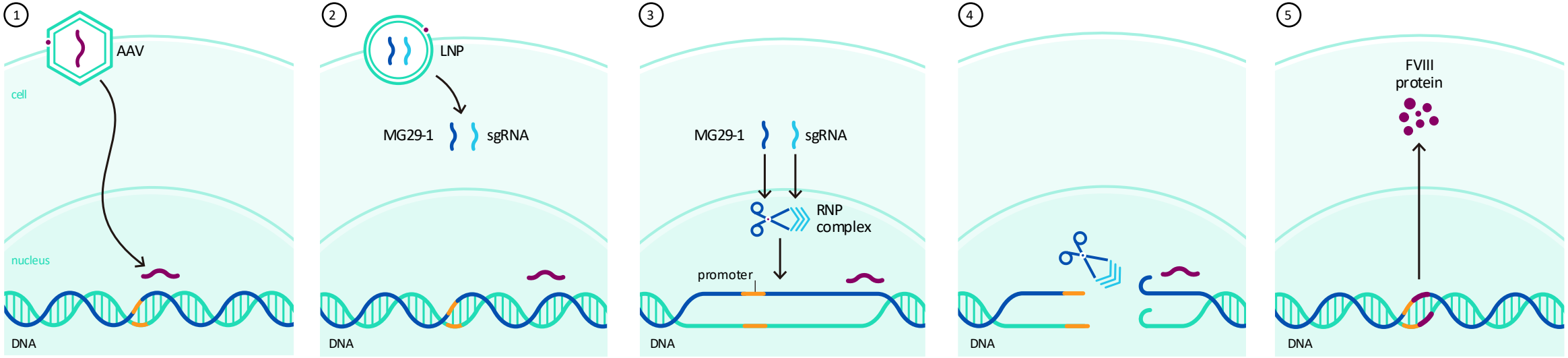
MGX-001 components:

AAV delivers FVIII gene (donor DNA):



LNP delivers nuclease mRNA & sgRNA targeting albumin site:





AAV delivers promoterless FVIII gene. AAV enters hepatocytes and the donor FVIII cassette localizes to the nucleus.

LNP delivers MG29-1 nuclease mRNA + guide targeting albumin promoter. The mRNA is released in the cytoplasm.

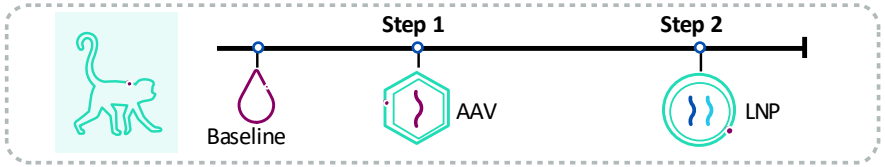
The mRNA is translated into MG29-1 nuclease, which binds the guide RNA to form a ribonucleoprotein (RNP) complex.

The guide pairs with the matching bases and MG29-1 nuclease cuts at the albumin locus.

The FVIII cassette integrates at the albumin cut site via natural end-joining.

The albumin promoter drives the expression of FVIII, which triggers the production of FVIII protein.

NHP study designs



Durability study

N=3

Study duration: **19 months**

	NHP FVIII AAV dose (vg/kg)	NHP FVIII LNP dose (mg/kg)
Animal 1001		
Animal 1002	2.0×10^{13}	1.0
Animal 1003*		

Pre-development candidate

Dose range finding study

N=24

Study duration **3 weeks**

FVIII evaluation: **days 5, 8, 11**

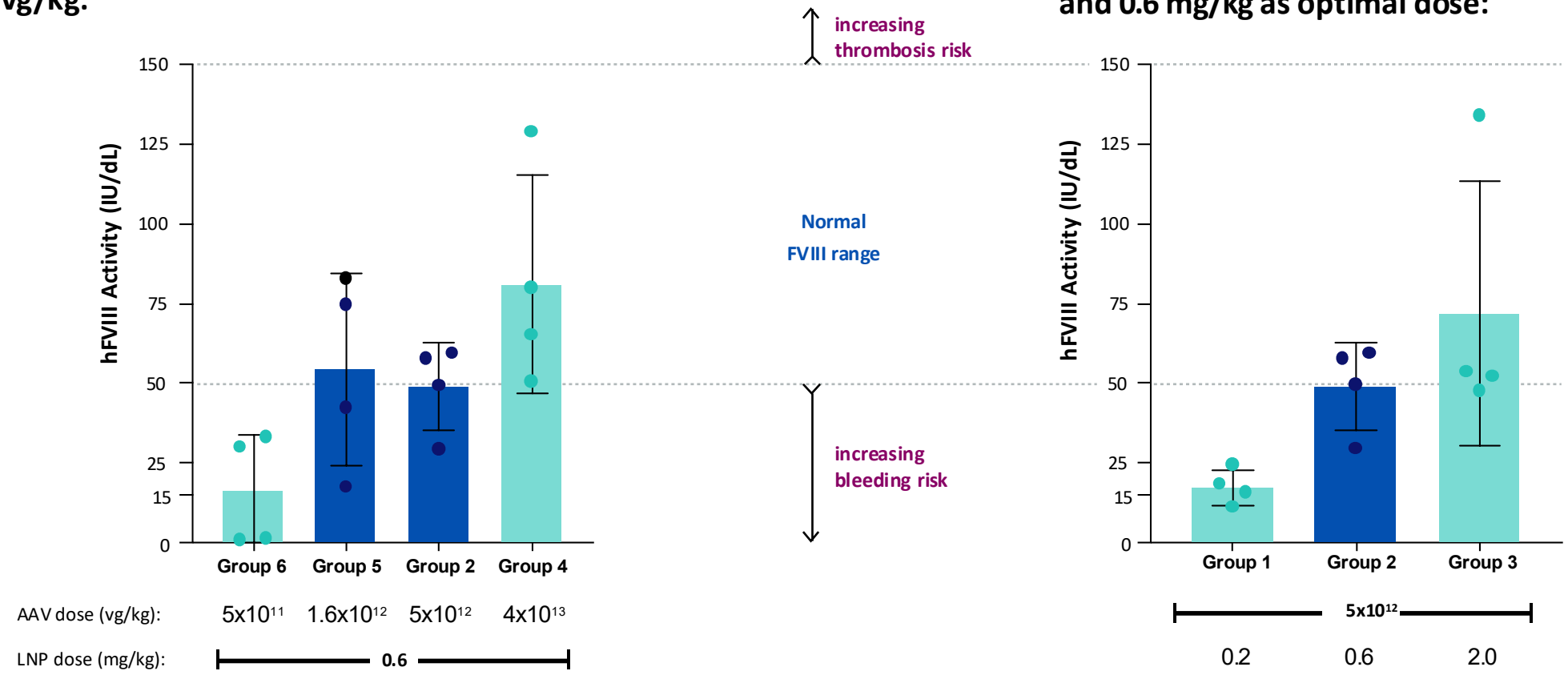
		Human FVIII AAV dose (vg/kg)	Human FVIII LNP dose (mg/kg)
Group 1:	N=4	5.0×10^{12}	0.2
Group 2:	N=4	5.0×10^{12}	0.6
Group 3:	N=4	5.0×10^{12}	2.0
Group 4:	N=4	4.0×10^{13}	0.6
Group 5:	N=4	1.6×10^{12}	0.6
Group 6:	N=4	5.0×10^{11}	0.6

Development candidate

Dose dependent FVIII activity in NHP: minimally efficacious and clinically relevant doses identified

Development candidate

AAV dose response identified the optimal efficacious dose of $5x10^{12}$ vg/kg:



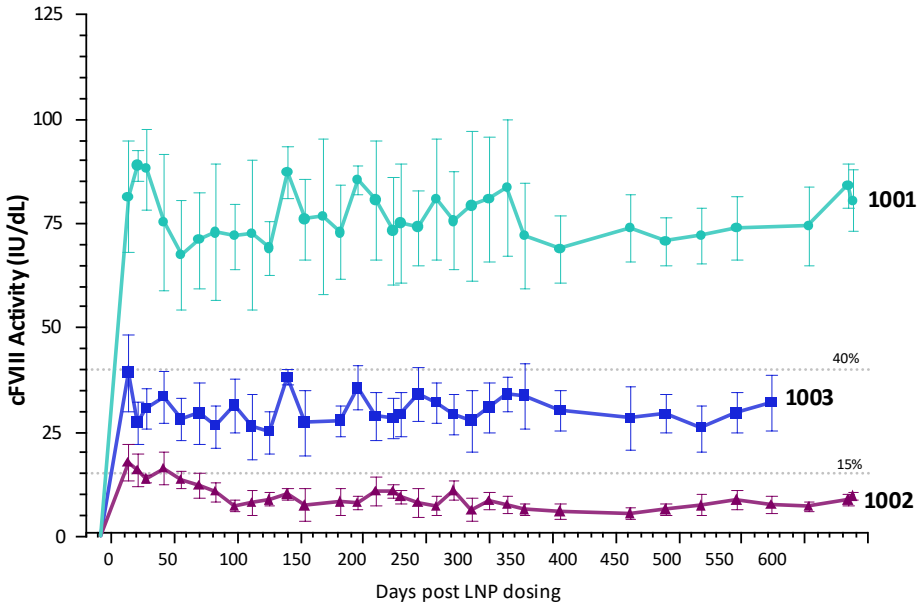
LNP dose response identified 0.2 mpk as the minimally efficacious dose and 0.6 mg/kg as optimal dose:

FVIII activity for each animals is the mean of values on d5, d8, d11 post LNP measured with a capture-chromogenic assay. hFVIII activity was stable from d5 to d11 post LNP. Xyntha was used for the standard curve.
Means and standard deviation per group are shown.

Durable, therapeutic levels of FVIII achieved in NHP

Pre-development candidate

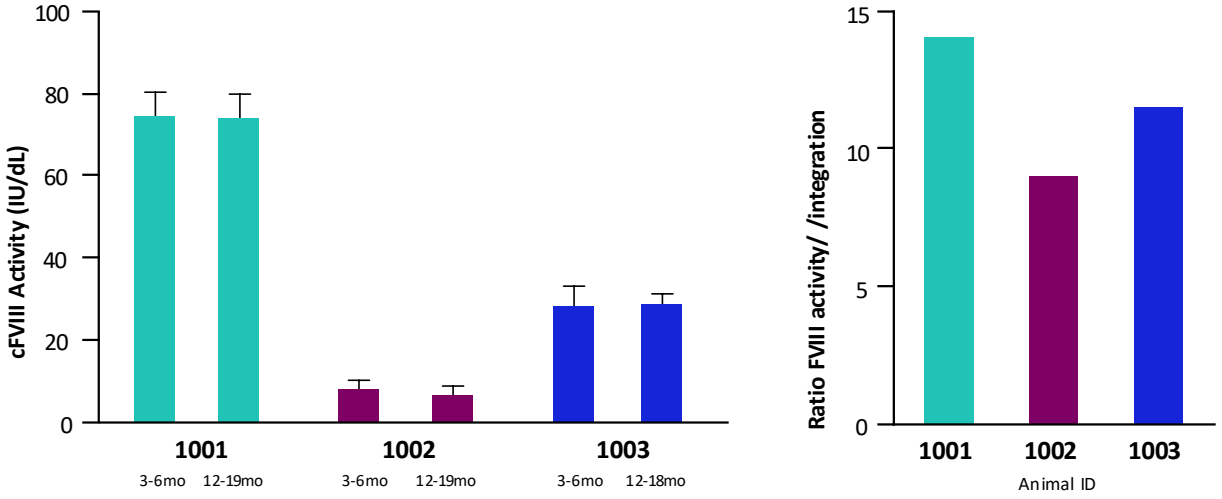
NHP durability study:



FVIII activity values are the mean and standard deviation of at least 3 independent assay runs with each sample run in at least duplicate in each assay.

Animal 1003 died on day 540 (17.8 mo) post LNP, assessed as unrelated to the treatment.

Plasma FVIII activity levels unchanged between 3-6 months and 12-19 months and correlate to integration:



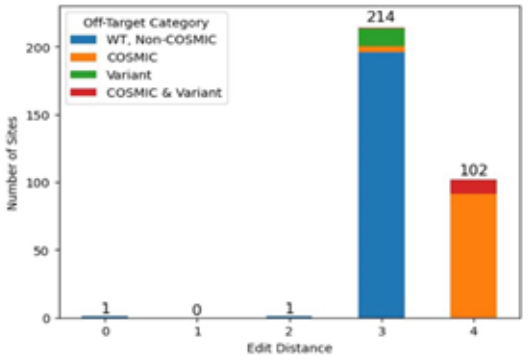
1 - Integration in forward orientation (copies per 100 haploid genomes, average of 5 liver lobes).

No genotoxicity observed with MGX-001

Development candidate

Discovery of potential off-target sites

1. In silico off-target discovery:



2. Biochemical off-target discovery:

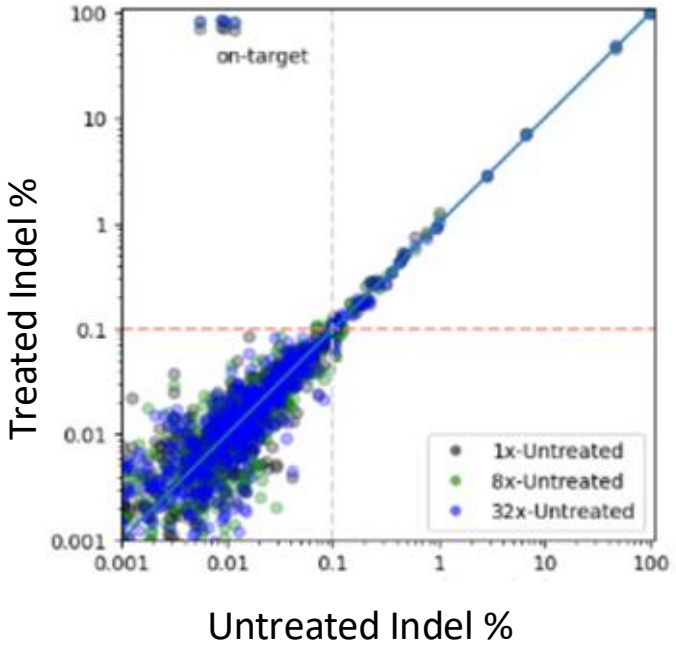
No potential off-targets were discovered
in cell-based assays.

3. In cell off-target discovery:

No potential off-targets were found.

No validated off-target sites observed

Three independent primary human hepatocyte donors:



No validated off-target editing observed.

High genome integrity maintained as observed in off-target editing, and AAV integration assays.

De-risked and clear opportunity for genome editing in hemophilia A

Hemophilia A is an ideal indication for genome editing approach:

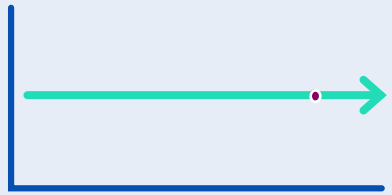
- Monogenic and well-characterized biology with clear biomarker
- Clearly defined target threshold of curative FVIII level & wide safety range
- Robust preclinical models and regulatory familiarity
- Strong advocacy and infrastructure

MGX-001 is uniquely suited for patients of all ages:

Technology:

proprietary Type V nuclease

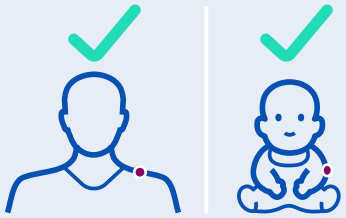
Durability:



Regulatory status:

IND-enabling stage

Pediatric potential:



MGX-001 is a potentially durable, curative approach for adults and children – the population with the most to gain.

Potential cure for adults and children living with hemophilia A

Designed to enable the body's own ability to produce FVIII

Expected durable FVIII activity via gene integration

Established regulatory and clinical pathways for advancement to pivotal study

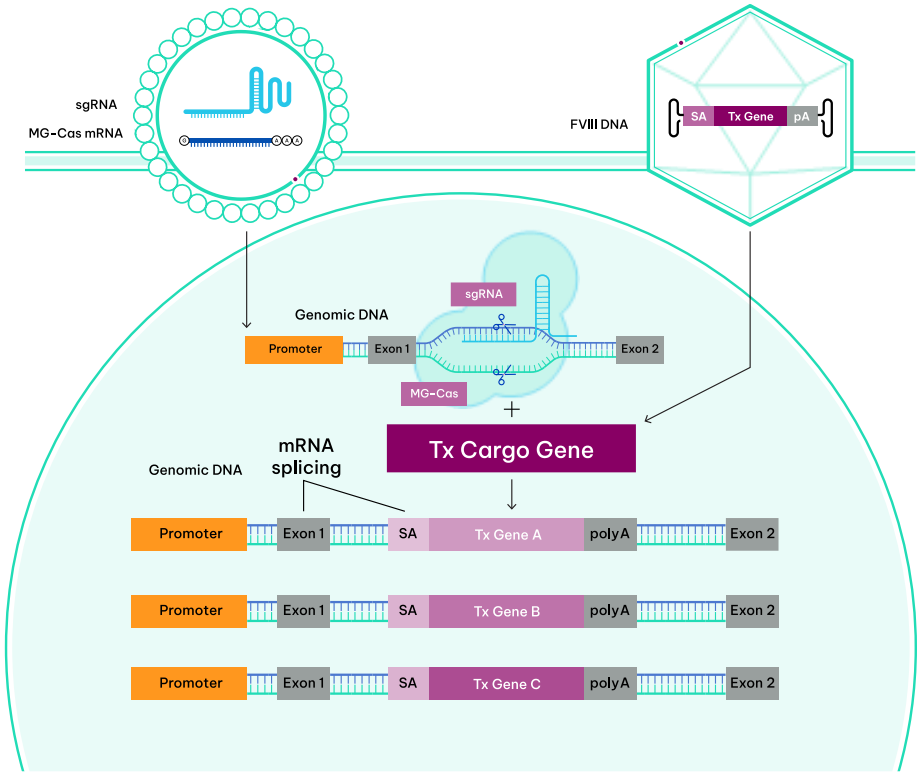
Our goal: to provide patients with a hemophilia-free mind

Beyond Hem A: pursuing secreted protein diseases with site-specific large gene insertion

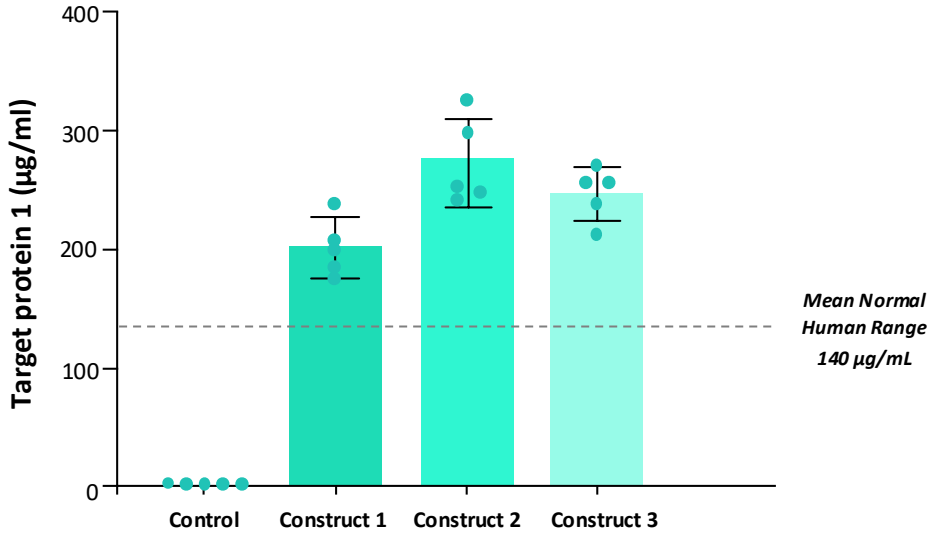
Expanding MGX-001 site-specific gene integration system into additional secreted protein deficiency therapeutics

LNP delivers nuclease mRNA and guide targeting albumin site

AAV delivers Transgene (donor DNA)

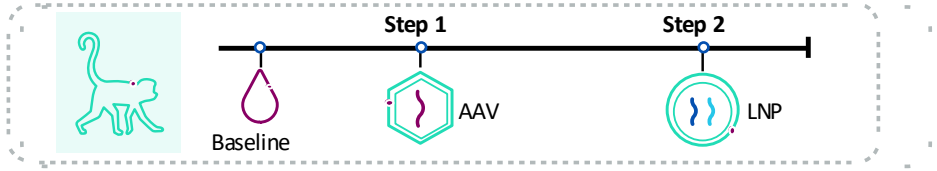


Normal circulating levels of target protein achieved in mice with multiple construct designs



- Above normal human protein expression achieved in mouse plasma
- Insertion assessed with multiple DNA template constructs
- LNP and AAV dose titration can be used to fine tune therapeutic window

In vivo PoC in AT-III achieved with MGX-001 site-specific large gene integration system



N=3

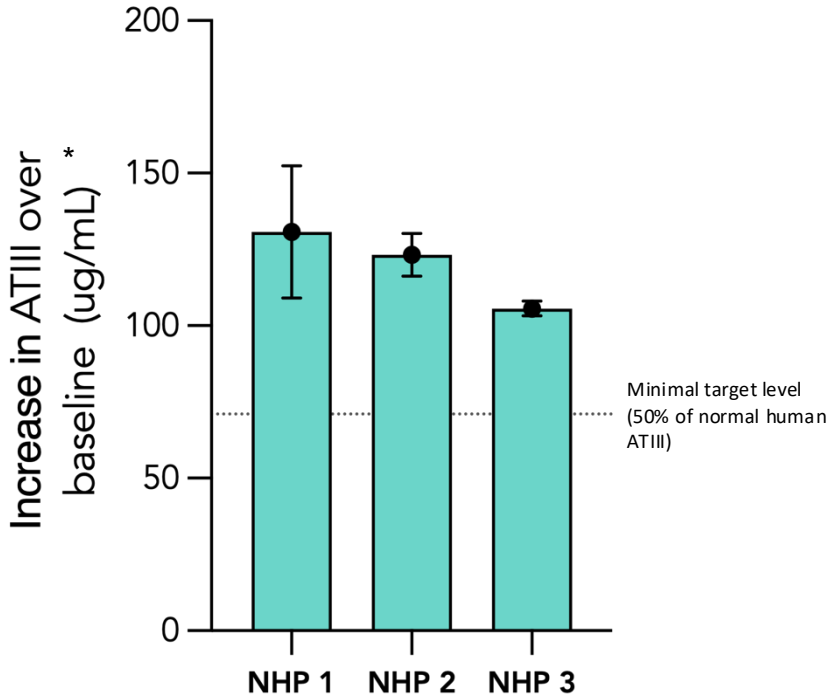
In-life, study

Ongoing at 46 days

	AAV dose (vg/kg)	LNP dose (mg/kg)
Animal 1001		
Animal 1002	1.0×10^{13}	1.0
Animal 1003		

- Severe antithrombin (AT-III) deficiency increases risk of venous thromboembolism (VTE)
- On average patients with severe disease have 50% of the normal amount of AT-III in their blood (70 ug/ml) [1,2]
- Replacing the missing AT-III with at least 50% of normal is expected to be a functional cure

Achieved circulating AT-III protein exceeding curative target of 50% of normal human levels



*Data are the mean of day 8 and 11 post-dosing minus the mean of days 0, 4 and 7 pre-dose.

1. Lane DA, Bayston T, Olds RJ et al. Antithrombin mutation database: 2nd (1997) update. For the Plasma Coagulation Inhibitors Subcommittee of the Scientific and Standardization Committee of the International
2. Conard et al (1983). Molar antithrombin concentration in normal human plasma. Haemostasis.13:363–8

Pursuing cardiometabolic indications in partnership

- MGX's in vivo genome editing complements Ionis leadership in cardiometabolic space.
- 4 targets: two co-development and co-commercialization options.
- Multibillion dollar TAM.

Current indications:

- TTR
- AGT
- APOC3
- Undisclosed

Team

Executive Leadership:

Jian Irish, PhD, MBA
President and CEO

AMGEN Kite A GILEAD Co

Pamela Wapnick, MBA
CFO

AMGEN True North Therapeutics

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Thank you

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