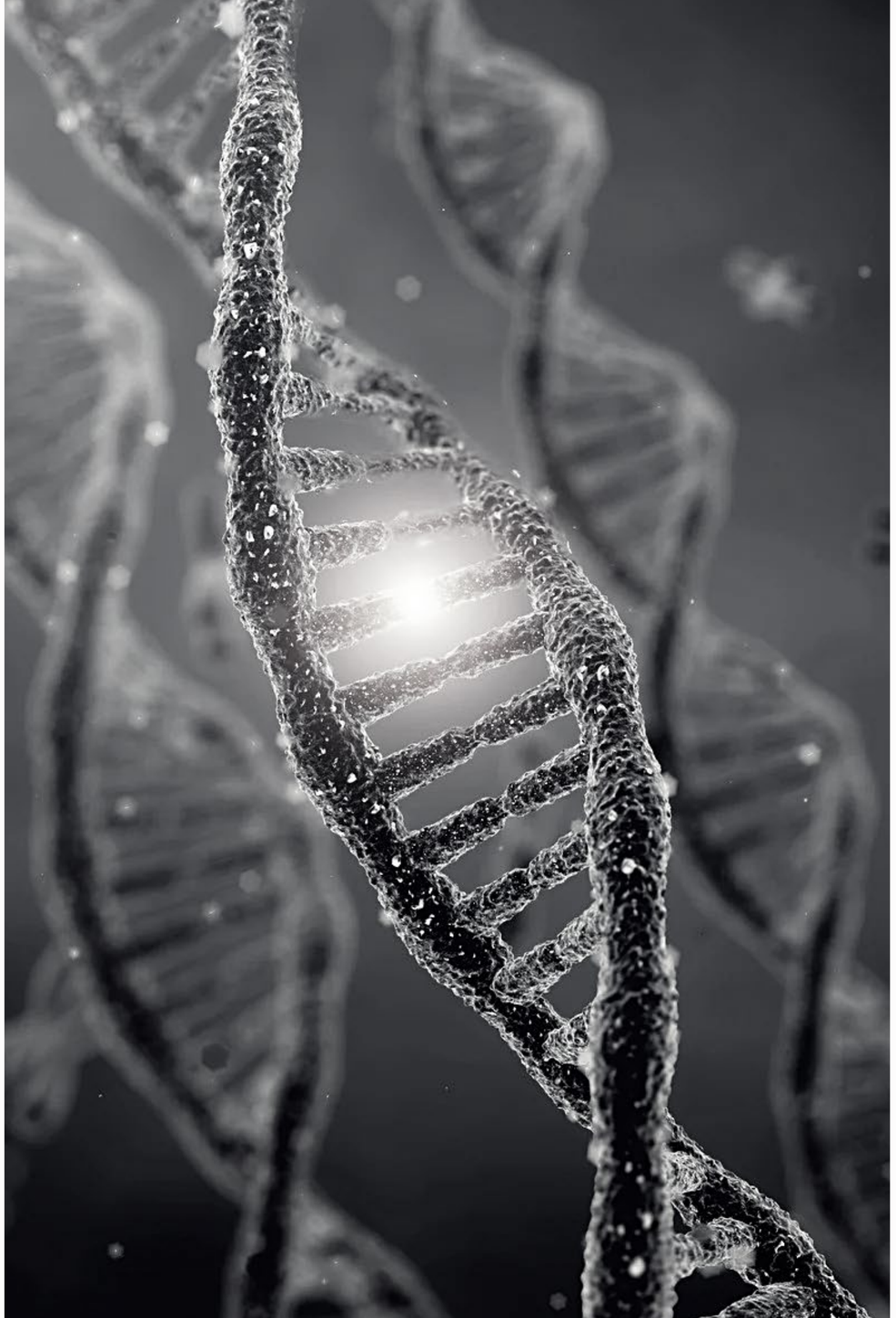

AMERICAN HEART ASSOCIATION'S
ATTR-CM WEBINAR SERIES:
WEBINAR #1

Understanding Amyloidosis & Emerging Therapeutic Frontiers

JUNE 18, 2025

Meeting Reminders

Please Note:

- This webinar is being recorded.
- All participants will be muted upon entry.
- Recordings of today's sessions will be enduring resources in a few weeks on www.heart.org

Questions?

- We encourage an open, conversational discussion, so please engage and share your thoughts!
- Q&A is scheduled at the end of the webinar.
- Submit your questions in the chat anytime—they will be addressed during the designated Q&A.

If you are having issue with audio, please call in using the appropriate number below.

Dial by your location:

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+1 (346) 248-7799	(Houston)
+1 (669) 900-6833	(San Jose)

Meeting ID: 813 5849 3759
Passcode: 704828

Welcome & Introductions

Devin Marie Keating
Director of Operations, Clinical Studies
American Heart Association

Thank you to

Intellia Therapeutics

for being a proud supporter of the

American Heart Association's

ATTR-CM Awareness and Education

AHA Science Advisory - November 2024
Gene Therapy in Cardiovascular Disease

"Gene therapy holds immense promise as a paradigm-shifting approach in the management of CVD [Cardiovascular Disease], offering the prospect of disease prevention, long-lasting cures, and alleviation of lifelong pharmacotherapy."

AHA's ATTR-CM Educational Webinar Series

The ATTR-CM Educational Webinar Series is designed to advance awareness, early diagnosis, and equitable access to care for transthyretin amyloid cardiomyopathy (ATTR-CM). Through a series of expert-led sessions, the series aims to:

Educate | Elevate | Engage | Equip

Welcome & Introductions

Andrew Landstrom, MD, PhD, FAHA
Associate Professor of Pediatrics and Cell Biology
Duke University School of Medicine

Joshua Hare, MD, FACC, FAHA
Louis Lemberg Professor of Medicine/Director,
Interdisciplinary Stem Cell Institute
University of Miami Miller School of Medicine

Disclosures

Andrew Landstrom, MD, PhD, FAHA:

- No Disclosures or RWI
- Research & salary support from NIH, Doris Duke Charitable Foundation, Le Ducq Foundation, Additional Ventures, Hartwell Foundation

Joshua Hare, MD, FACC, FAHA:

- Patents: Listed inventor on patents owned by the University of Miami and Longeveron
- Consultant: Vestion
- Equity: Kardia, Vestion, Heart Genomics, Longeveron, Inc.(Equity Owner)
- Company Co-Founder: Vestion, Inc., Heart Genomics, LLC,
  - Longeveron, Inc. - Inventor of technologies licensed from UM to Longeveron; The University of Miami is an equity owner in Longeveron (LGVN), which has licensed intellectual property from the University of Miami.

Educational Objectives:

1. **CURRENT LANDSCAPE OF GENE THERAPY**
   Explore the expanding role of gene therapy in cardiovascular care, with a focus on ATTR-CM.

2. **RESEARCH & TRIALS**
   Highlight key studies and landmark clinical trials shaping the future of treatment.

3. **AMYLOIDOSIS 101**
   Understand disease mechanisms, types, and clinical presentation

4. **ATTR THERAPEUTICS**
   Explore transthyretin amyloidosis, its cardiac impact, and emerging treatments, including gene therapies.

5. **WHAT'S NEXT?**
   Preview upcoming education tools and innovations in care.

American Heart Association®

Current Landscape of Gene Therapy

Andrew Landstrom, MD, PhD

Associate Professor of Pediatrics and Cell Biology

Duke University School of Medicine

GENE THERAPY:
Genetic modification
of cells to produce
a therapeutic effect

Gene Therapy Development Landscape

33 Gene Therapies Approved*

35 RNA Therapies Approved

72 Non-Genetically Modified Cell Therapies Approved

NEARLY ALL are for cancer, skeletal myopathies, metabolic disease, hematologic disease, among others

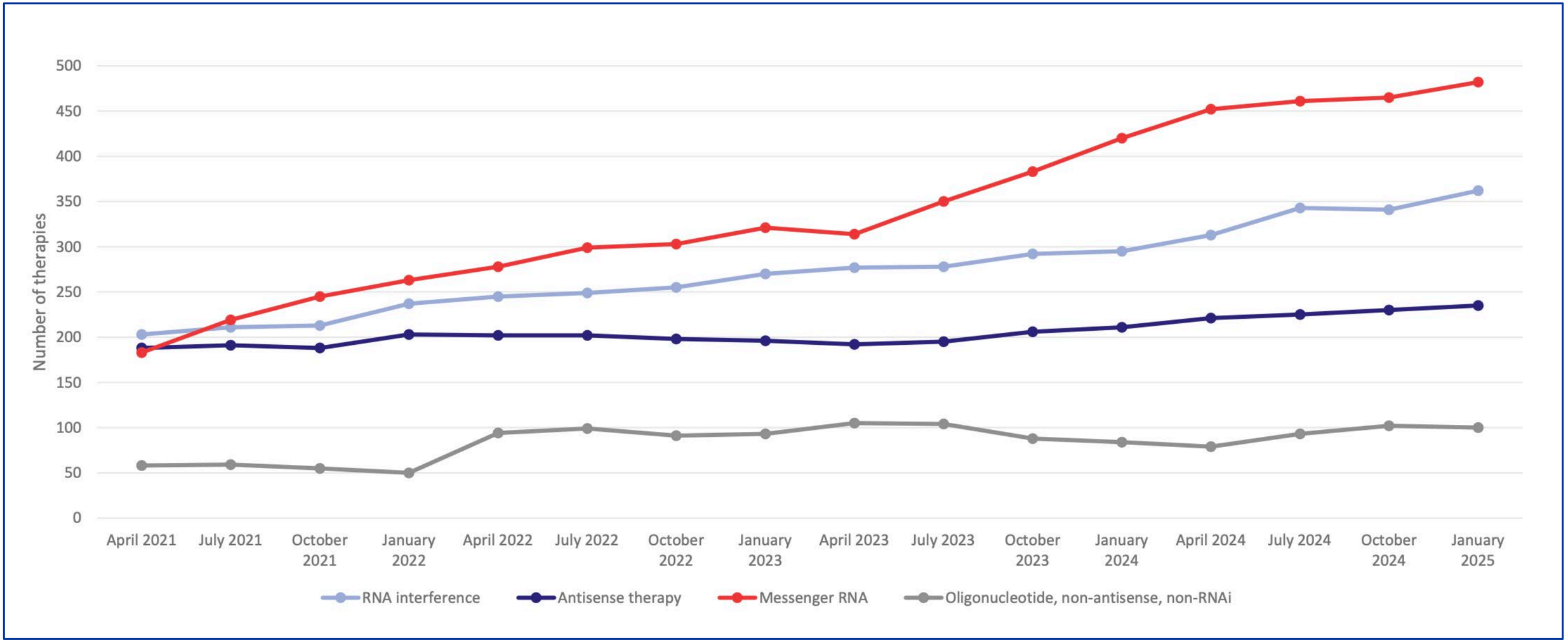
* FDA and other non-US based approvals
American Society for Gene and Cell Therapies, latest from 04/2025

The Development Pipeline

4,238	2,117	1,053	8 of Top 10
Gene, Cell, & RNA Therapies	**Gene Therapies** *Pre-clinical through pre-registration*	**Therapies for Rare Disease** *Pre-clinical through pre-registration*	**Are Oncologic Indications**

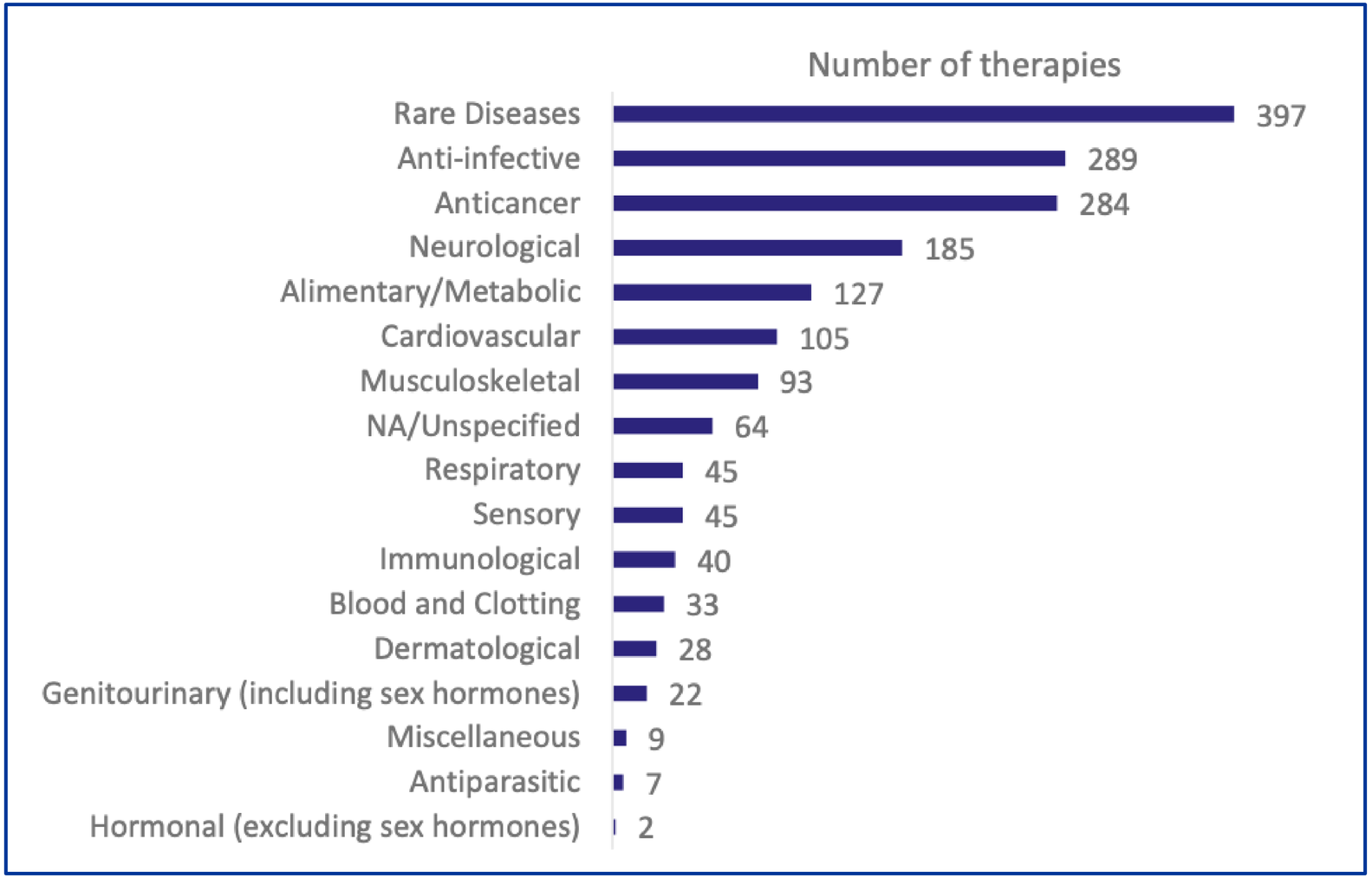
American Society for Gene and Cell Therapies, latest from 04/2025

RNA Therapies in the Development Pipeline



Pharmaprojects | Citeline, January 2025
American Society for Gene and Cell Therapies, latest from 04/2025

RNA Therapies in the Development Pipeline



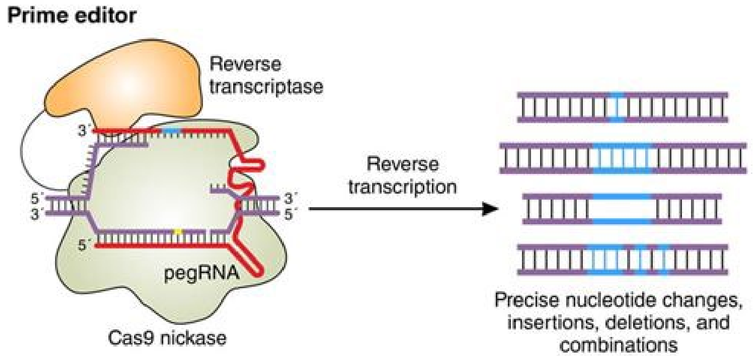
Pharmaprojects | Citeline, January 2025
American Society for Gene and Cell Therapies, latest from 04/2025

AHA Science Advisory:

Gene Therapy in Cardiovascular Disease

November 2024

“Gene therapy holds immense promise as a paradigm-shifting approach in the management of CVD [Cardiovascular Disease], offering the prospect of disease prevention, long-lasting cures, and alleviation of lifelong pharmacotherapy.”




Circulation

AHA SCIENCE ADVISORY

Gene Therapy in Cardiovascular Disease: Recent Advances and Future Directions in Science: A Science Advisory From the American Heart Association

Yuri Kim, MD, PhD, Chair; Andrew P. Landstrom, MD, PhD, FAHA; Svati H. Shah, MD, MHS, FAHA; Joseph C. Wu, MD, PhD, FAHA; Christine E. Seidman, MD, FAHA, Vice Chair; on behalf of the American Heart Association

ABSTRACT: Cardiovascular disease remains the foremost cause of morbidity and mortality globally, affecting millions of individuals. Recent discoveries illuminate the substantial role of genetics in cardiovascular disease pathogenesis, encompassing both monogenic and polygenic mechanisms and identifying tangible targets for gene therapies. Innovative strategies have emerged to rectify pathogenic variants that cause monogenic disorders such as hypertrophic, dilated, and arrhythmogenic cardiomyopathies and hypercholesterolemia. These include delivery of exogenous genes to supplement insufficient protein levels caused by pathogenic variants or genome editing to correct, delete, or modify mutant sequences to restore protein function. However, effective delivery of gene therapy to specified cells presents formidable challenges. Viral vectors, notably adeno-associated viruses and nonviral vectors such as lipid and engineered nanoparticles, offer distinct advantages and limitations. Additional risks and obstacles remain, including treatment durability, tissue-specific targeting, vector-associated adverse events, and off-target effects. Addressing these challenges is an ongoing imperative; several clinical gene therapy trials are underway, and many more first-in-human studies are anticipated. This science advisory reviews core concepts of gene therapy, key obstacles, patient risks, and ongoing research endeavors to enable clinicians to understand the complex landscape of this emerging therapy and its remarkable therapeutic potential to benefit cardiovascular disease.

Key Words: AHA Scientific Statements ■ cardiovascular diseases ■ gene editing ■ gene silencing ■ gene transfer techniques ■ genetic therapy

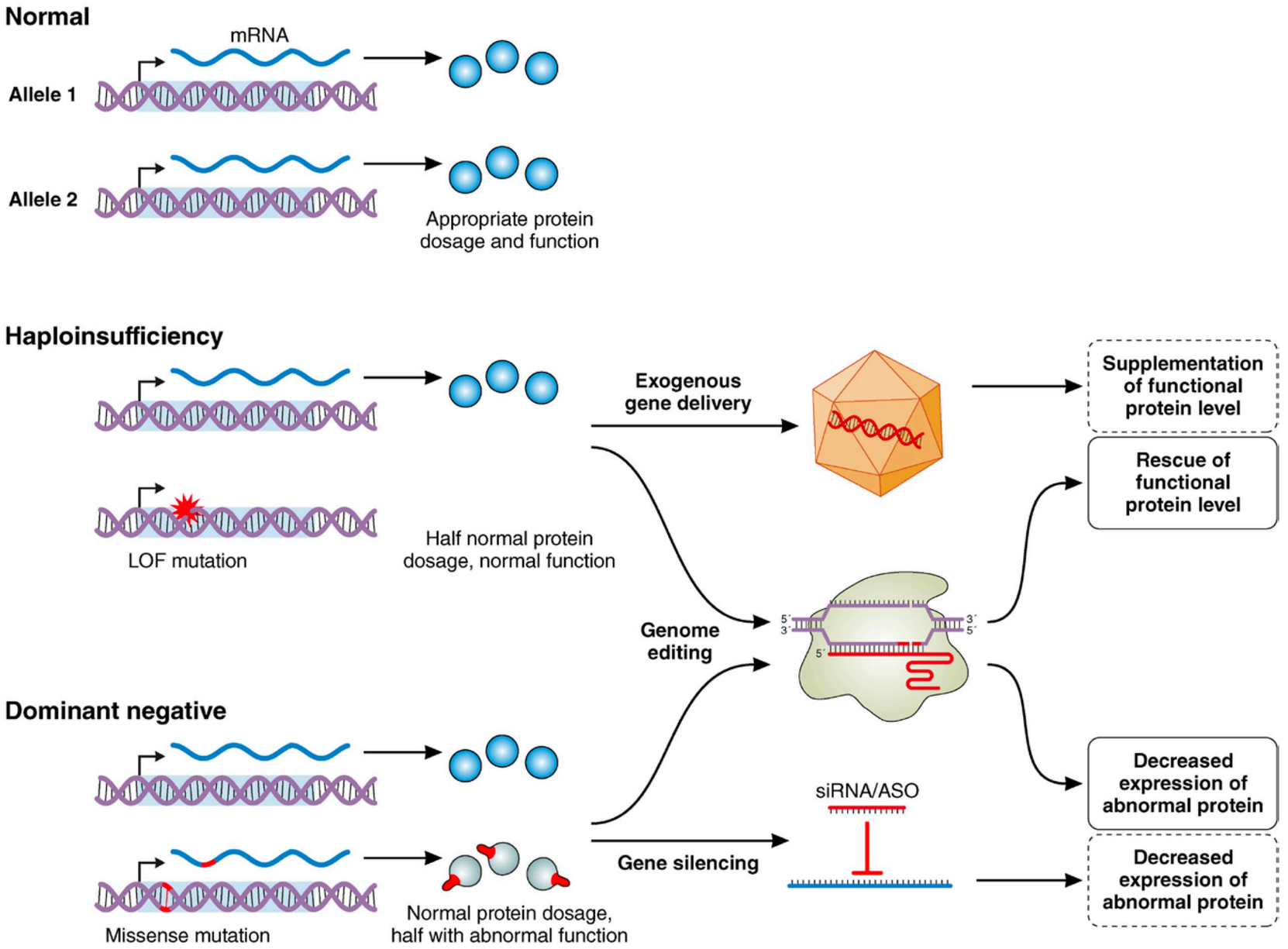
Cardiovascular disease (CVD) affects 28.6 million Americans >20 years of age[1] with enormous annual US health care expenditures and a profound impact on patients and families. Existing treatments and interventions are costly, typically require lifelong administration, and mitigate but rarely cure disease. The identification of genetic causes for many CVD enables an alternative approach: gene therapy to precisely target the molecular driver of disease pathogenesis.

Monogenic disorders arise from rare pathogenic … the structure, function, or nor- … of the gene-encoded protein. … usually cause prominent clinical … creased morbidity and mortal- … ic CVD results from PVs in 1 … h as dilated, hypertrophic, and arrhythmogenic cardiomyopathy, channelopathies, and aortopathies.[2–5] Recessive monogenic CVD arises from PVs in both gene copies such as homozygous familial hypercholesterolemia. PVs that cause monogenic disorders are identified in ≈1.7% of patients undergoing cardiac catheterization; they contribute substantially to the overall burden of CVD.[6] Clinical genetic testing is readily available that assesses for the presence of causal monogenic variants and enables early accurate diagnosis and interventions.[7]

Polygenic disorders arise from multiple genetic variants that occur in healthy populations and individually convey small effects but collectively increase disease susceptibility. Most polygenic variants alter nonprotein coding, regulatory sequences that influence gene expression, and are associated with prevalent CVD,

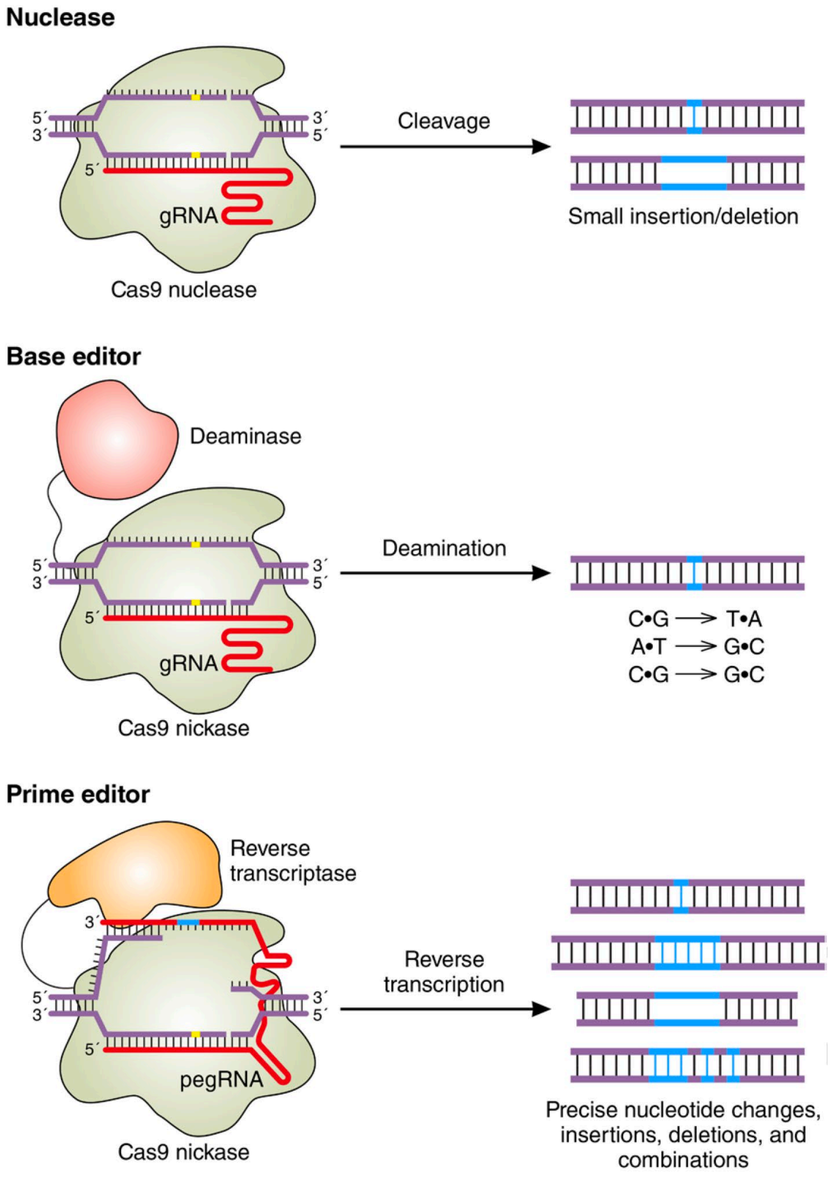
SCAN HERE ⟶
TO READ THE FULL ARTICLE

Mechanistic Approach to Gene Therapy



Kim Y, et al, *Circulation*, 2024

Gene Therapy by Genome Editing



Nuclease cuts both DNA strands

Small insertion/deletion (KO allele)

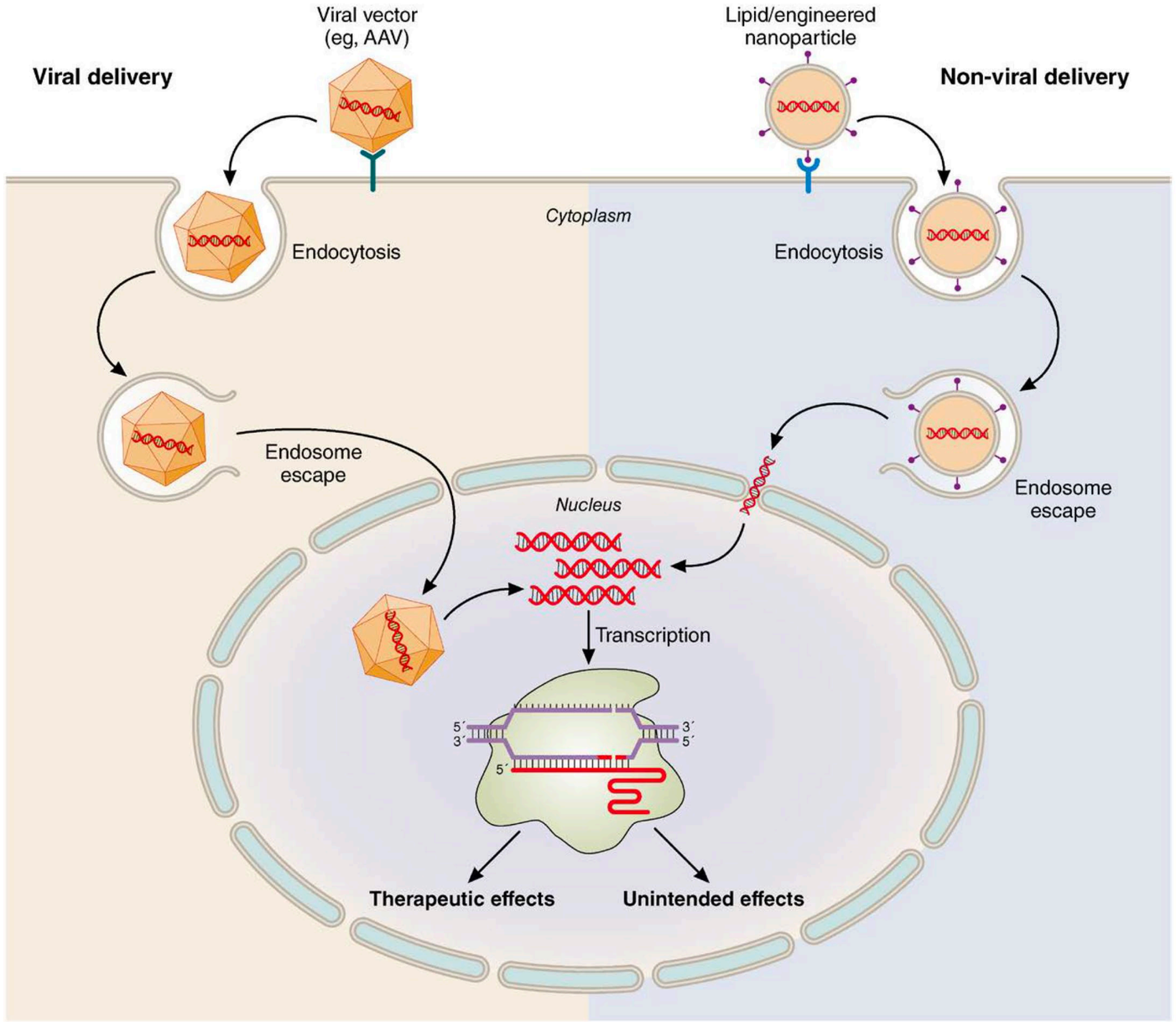
Nickase cuts one DNA strand

Deaminase converts nucleotide (cytosine > thymine, adenine > guanine)

Prime editing gRNA introduce precise nucleotide changes

Kim Y, et al, *Circulation*, 2024

Delivery Methods for Gene Therapy



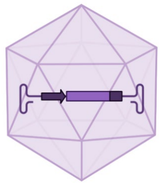
Kim Y, et al, *Circulation*, 2024

Major Themes for Cardiovascular Gene Therapy

Heritable cardiac disease

Defined genetic mechanism

Gene replacement strategy

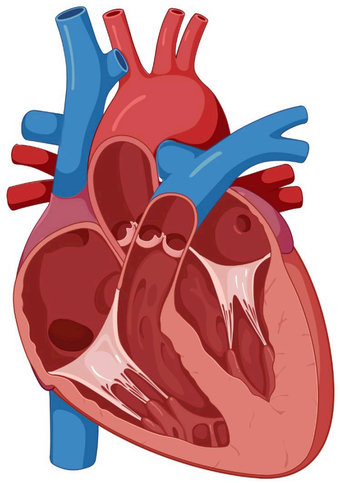


Adeno-Associated Virus (AAV) delivery

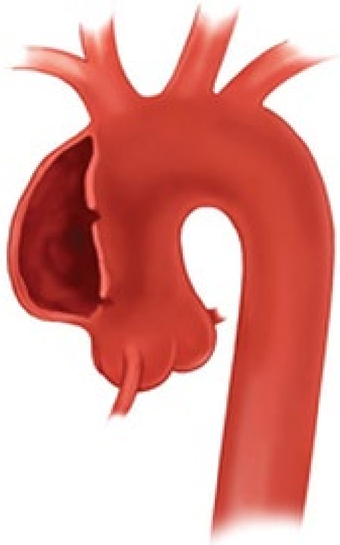
Early phase (1, 2)

Kim Y, et al, *Circulation*, 2024

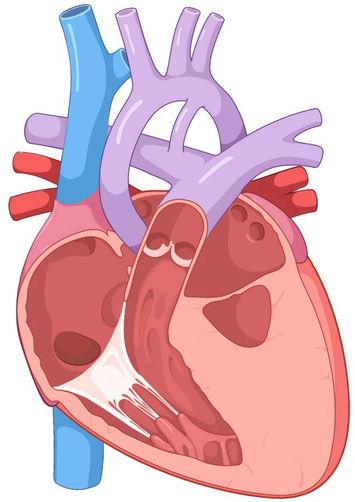
Heritable Cardiovascular Diseases



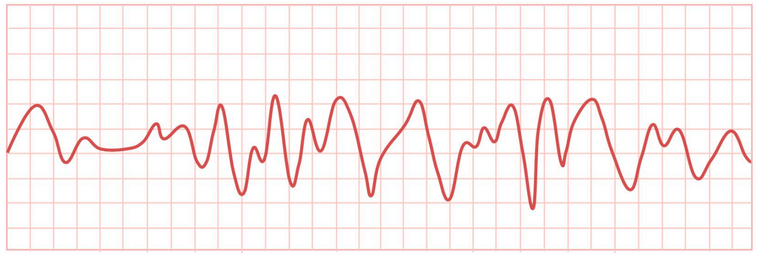
Cardiomyopathies



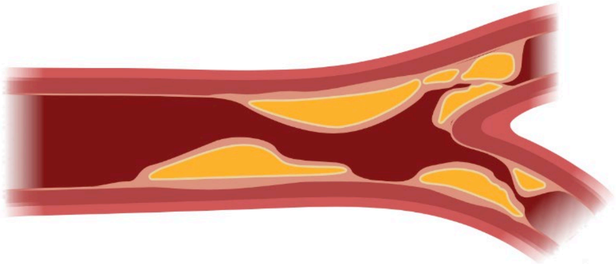
Aortopathies



Structural Heart Disease

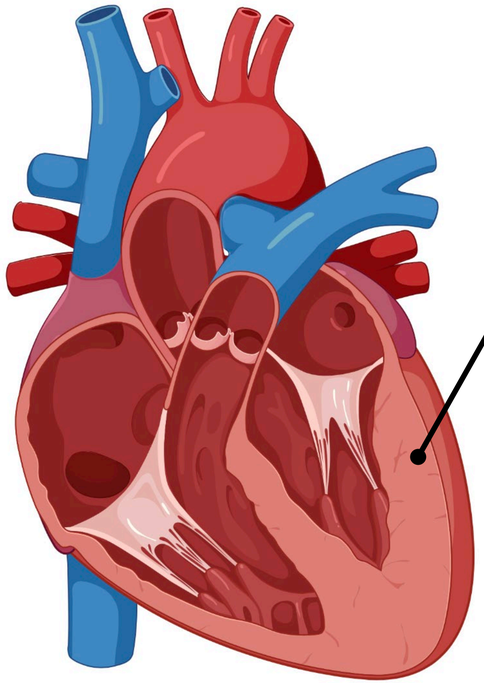


Cardiac Channelopathies



Familial Dyslipidemia

Cardiomyopathy Gene Therapy Trials



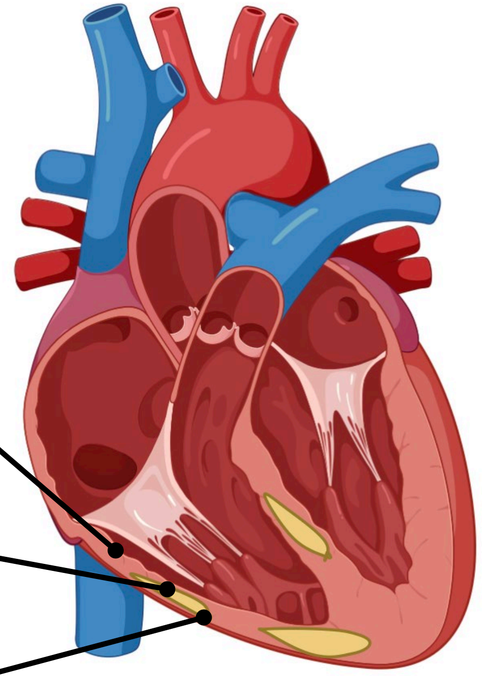
HCM

MyPEAK-1 *MYBPC3* Gene Therapy Trial[1]
Phase 1b Trial Currently Enrolling
>18 years old

RIDGE-1 *PKP2* Gene Therapy Trial[1]
Phase 1b Trial Currently Enrolling
>18 years old

***PKP2*-mediated ACM[2]**
Phase 1 Trial Currently Enrolling
>18 years old

HEROIC *PKP2* Gene Therapy Trial[3]
Phase 1 Trial Currently Enrolling
>18 years old



ACM

[1]Tenaya Therapeutics, [2]Rocket Pharmaceuticals, [3]Lexeo Therapeutics

LAMP2 Replacement for Danon Disease

RP-A501

AAV9.LAMP2B replacement therapy

Phase 1 demonstrated safety of a single infusion with durable expression of LAMP2 in the myocardium

Completed enrollment for Phase 2 Trial in Sept 2024

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Phase 1 Study of AAV9.LAMP2B Gene Therapy in Danon Disease

B. Greenberg, M. Taylor, E. Adler, S. Colan, D. Ricks, P. Yarabe, P. Battiprolu, G. Shah, K. Patel, M. Coggins, S. Carou-Keenan, J.D. Schwartz, and J.W. Rossano

ABSTRACT

BACKGROUND

Danon disease is a rare, X-linked, monogenic cardiomyopathy caused by mutations in the lysosomal-associated membrane 2 gene (*LAMP2*), which encodes the LAMP2 protein. In male patients, the predominant phenotype is progressive cardiac hypertrophy, cardiac dysfunction, and early death. There are no directed therapies for the disease.

METHODS

In this phase 1 study, we evaluated the safety and efficacy of a single infusion of RP-A501, a recombinant adeno-associated virus serotype 9 containing the transgene *LAMP2B*, which encodes an isoform of LAMP2. The primary outcomes were the safety and toxic effects of RP-A501, myocardial LAMP2 transduction and protein expression, stabilization of or reduction in heart-failure symptoms, and stabilization of or improvement in cardiac structure and function. Key secondary outcomes were sustained reduction in or stabilization of symptoms, immunologic response to RP-A501, end-stage heart failure, and overall survival. Exploratory outcomes included improvement in serologic markers of cardiac disease, patient-reported outcomes, and quality-of-life assessments.

RESULTS

RP-A501 infusion was administered to seven male patients with Danon disease: five who were 15 years of age or older and two who were between 11 and 14 years of age. All the patients received a transient immunomodulatory regimen of prednisone, tacrolimus or sirolimus, and rituximab. Phase 1 data over 24 to 54 months, including interim data from a long-term follow-up study, are reported here. One patient had complement-mediated thrombotic microangiopathy (grade 4) with thrombocytopenia and acute kidney injury. Three patients had glucocorticoid-related exacerbation (grade 3) of Danon disease–related skeletal myopathy. One patient with left ventricular systolic dysfunction at baseline had progressive heart failure and underwent transplantation 5 months after infusion. In the six patients with normal left ventricular ejection fraction at baseline, we observed cardiac LAMP2 protein expression and a reduction from baseline in or stabilization of the left ventricular mass index, preservation of left ventricular ejection fraction, and reduction in or stabilization of the levels of cardiac troponin I and N-terminal pro–B-type natriuretic peptide. At 24 to 54 months, all the patients were alive, with complete resolution of side effects.

CONCLUSIONS

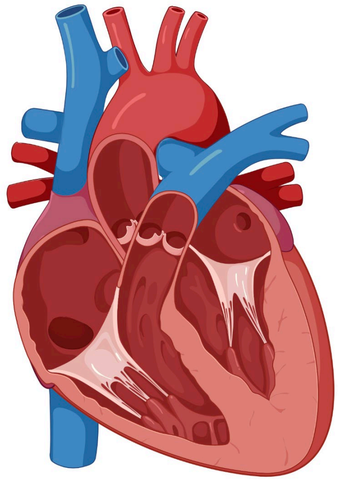
A single infusion of RP-A501 appeared to be safe and was associated with cardiac LAMP2 expression and evidence of clinical improvement over a period of 24 to 54 months. (Funded by Rocket Pharmaceuticals; ClinicalTrials.gov number, NCT03882437.)

The authors' full names, academic degrees, and affiliations are listed in the Appendix. Dr. Greenberg can be contacted at bgreenberg@ucsd.edu or at the Department of Cardiology, University of California, San Diego, 9500 Gilman Dr., La Jolla, CA 92093.

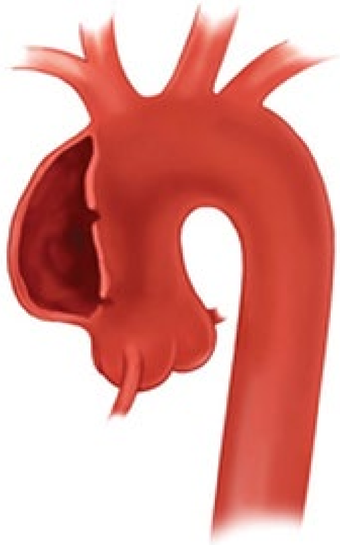
This article was published on November 18, 2024, at NEJM.org.

DOI: 10.1056/NEJMoa2412392

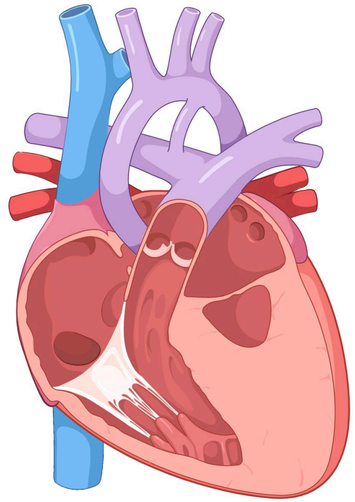
Heritable Cardiovascular Diseases



Cardiomyopathies



Aortopathies



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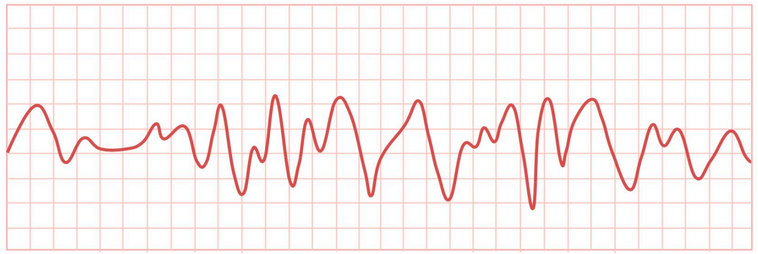

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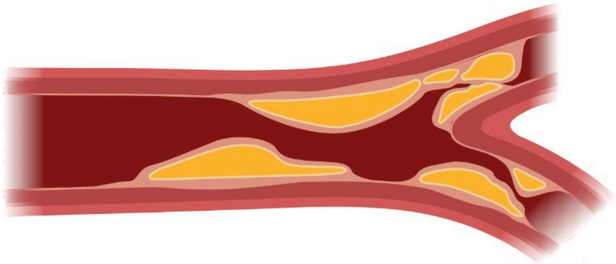
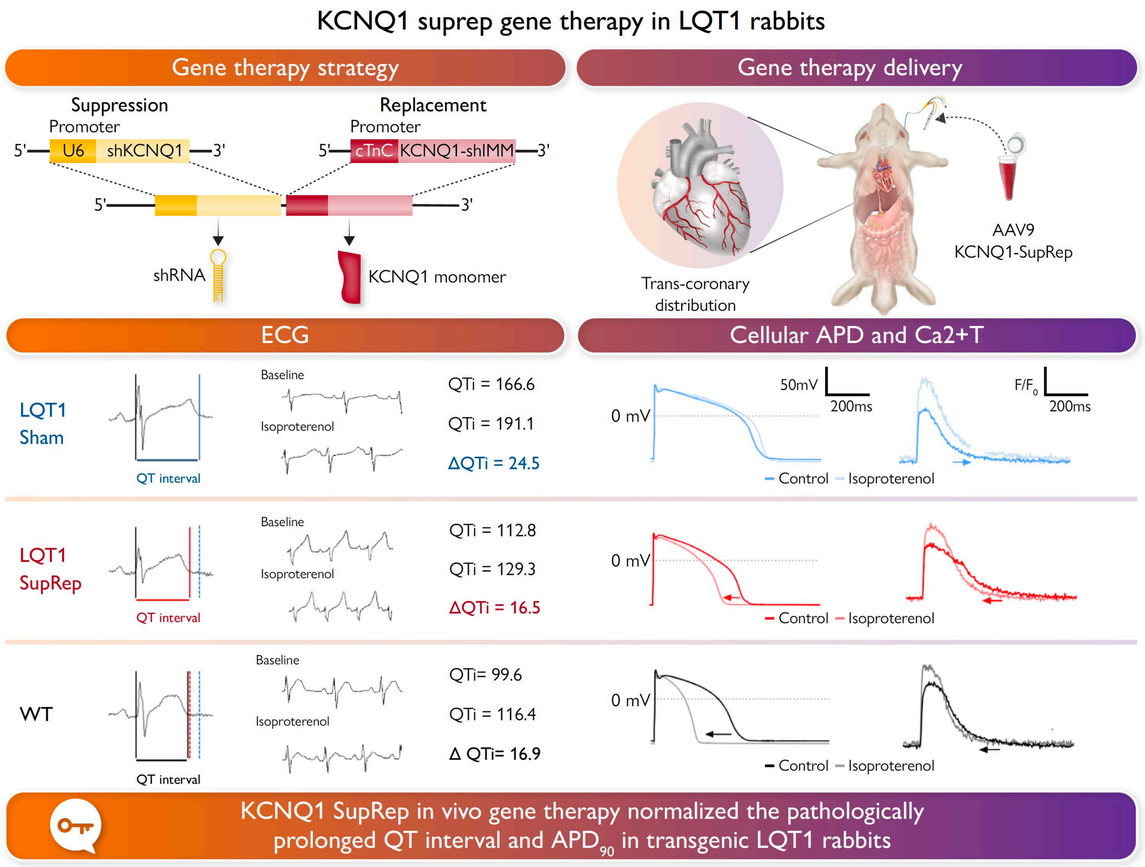
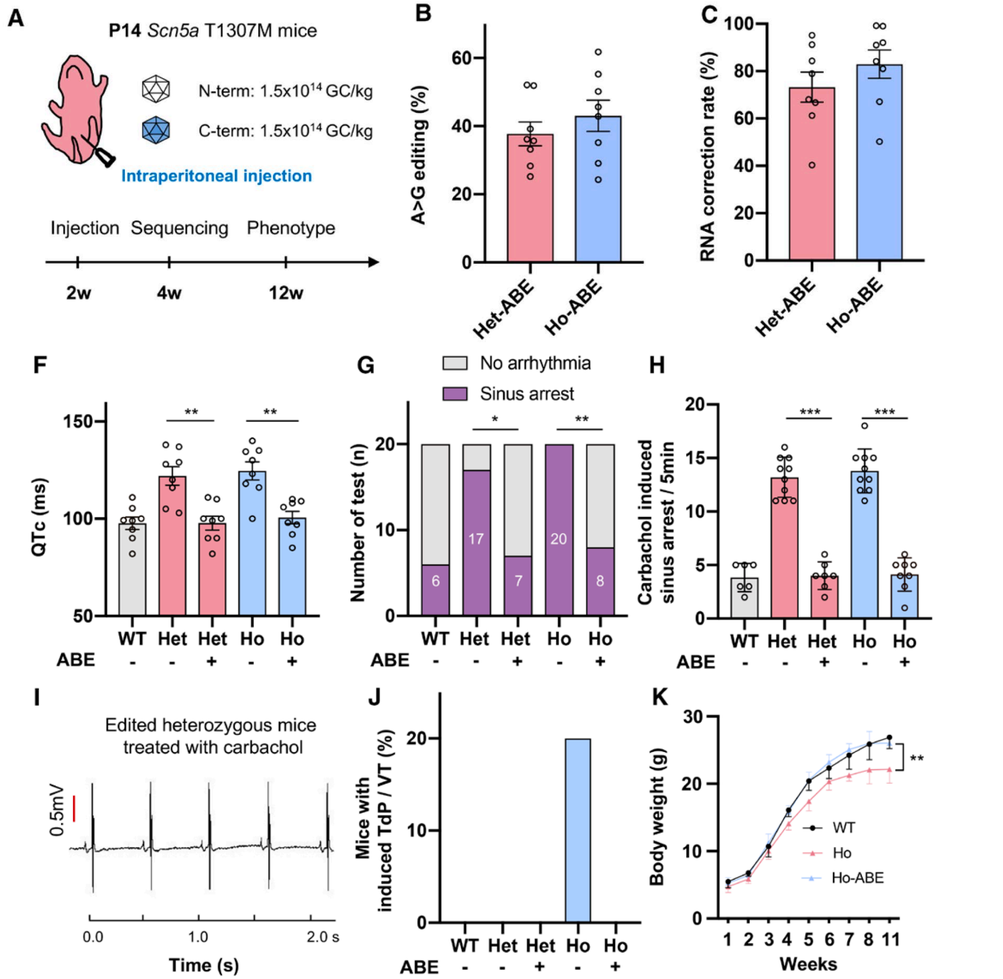


Familial Dyslipidemia

Emerging Therapies for Long QT Syndrome: Gene Therapy



"Suppression-Replacement" for LQTS 1



In Vivo Base-Editing for LQTS 3

Bains S, et al, Eur Heart J, 2024
Qi M, et al, *Circulation*, 2023

Cardiovascular Gene Therapy Trials

- **24** — **Trials for Cardiac Disease**
- **12** — **Metabolic Diseases with Cardiomyopathy** *(e.g., Pompe, Danon, Fabry)*
- **6** — **Familial Hypercholesterolemia**
- **6** — **Genetic or Acquired Cardiomyopathy**
- **11** — **Trials for Muscular Dystrophy***

ClinicalTrials.gov (as of 6/1/2025)

Challenges and The Way Forward

Scientific Challenges

- Target specificity (tissue, cell-type, and DNA sequence)
- Improved delivery and expanded delivery mechanisms
- Minimizing the immune response
- Natural history studies
- Voice of patients and families

Implementation Challenges

- Cost
- Precise knowledge of the genetic basis of disease
- Equity in trials and access in the marketplace

Terralyn Schmidt
MS, CGC

Leigh Pedersen
BSN, RN

Brandi Umstead

National Heart, Lung, and Blood Institute

R01-HL149870
R01-EB032726
R01-HL160654
R01-HL166217

CDC Centers for Disease Control and Prevention

5NU50-DD000054

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colin's kids, inc. est. 2011

Andrew Landstrom, MD, PhD
andrew.landstrom@duke.edu

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Amyloidosis 101 & ATTR-CM Therapeutics

Joshua Hare, MD, FACC, FAHA

Louis Lemberg Professor of Medicine/Director,
Interdisciplinary Stem Cell Institute
University of Miami Miller School of Medicine

CARDIAC AMYLOIDOSIS

Devastating cause of restrictive cardiomyopathy

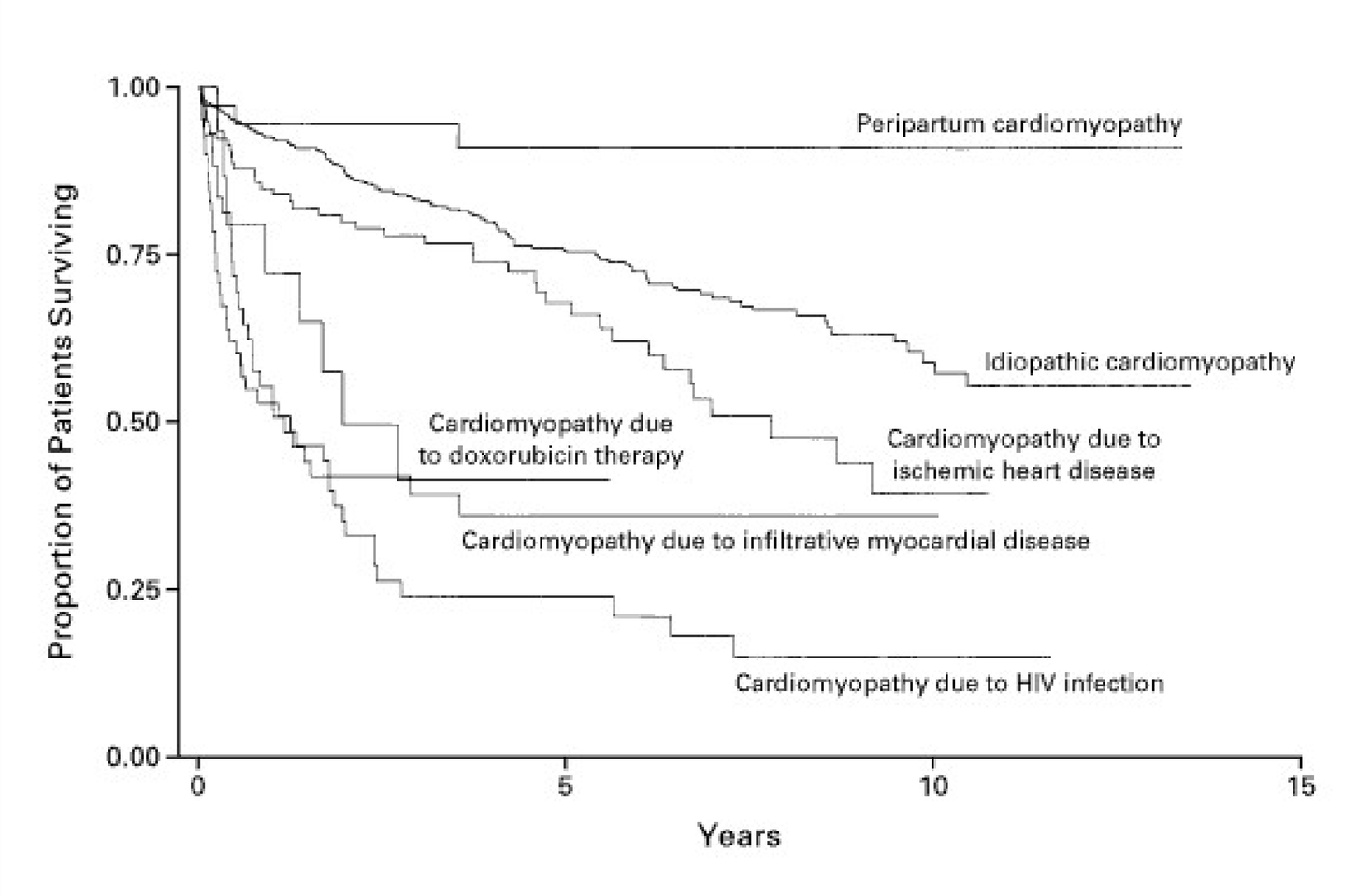
- Heart failure
- Conduction system disease
- Atrial fibrillation

Peripheral neuropathy

- Small fiber peripheral
- Autonomic polyneuropathy

Amyloid deposition

- Transthyretin
- Light chains



Refs: Ruberg et al. JAMA 2024; Felker et al. NEJM 2000

ATTR AMYLOID

TRANSTHYRETIN

Liver synthesized tetramer. Misfolding leads to tissue deposition of amyloid fibrils in heart and peripheral nerves

CARDIAC AMYLOIDOSIS

Restrictive cardiomyopathy with high morbidity and mortality

PREVALENCE

50 to 150,000 people

TREATMENT STRATEGIES:

- Inhibit hepatic TTR synthesis
- Stabilize tetramer
- Disrupt amyloid fibrils

Refs: Ruberg et al. JAMA 2024; Felker et al. NEJM 2000

ATTR
CLINICAL MANIFESTATIONS

Cardiomyopathy

- Heart failure
- Arrhythmia
- Death

Polyneuropathy

- Small fiber
- Autonomic

Ligament deposition

- Carpal Tunnel Syndrome

Table 2. Primary Clinical Manifestations of ATTR Amyloidosis[a]

Organ system	Clinical manifestation
Cardiovascular	• Atrioventricular block • Congestive heart failure • Arrhythmia (atrial fibrillation/flutter, ventricular tachycardia)
Nervous	• Peripheral neuropathy (weakness/numbness in hands and feet) • Autonomic neuropathy (orthostatic hypotension, erectile dysfunction, sweating abnormalities)
Gastrointestinal	Dysmotility (diarrhea, constipation, nausea/vomiting, early satiety)
Musculoskeletal	• Spinal stenosis • Carpal tunnel syndrome • Ligamentous rupture
Kidney	Acute and chronic kidney disease (cardiorenal syndrome, hemodynamically mediated kidney impairment)

[a] Information for this table was adapted from Kittleson et al.[6]

Refs: Ruberg et al. JAMA 2024; Felker et al. NEJM 2000

Drug Therapy for ATTR Amyloid

Small molecules that act to stabilize TTR protein in the circulation

TAFAMIDIS

- First tested for familial amyloid polyneuropathy
- Approved in the US for ATTR

ACORAMIDIS

- Approved for cardiac ATTR in 2024

Lee, A. (2025). Acoramidis: First approval. Drugs, 85(6), 833–837. https://doi.org/10.1007/s40265-025-02159-z
Cruz, M. W., & Benson, M. D. (2015). A review of tafamidis for the treatment of transthyretin-related amyloidosis. Neurology and Therapy, 4(2), 61–79. https://doi.org/10.1007/s40120-015-0031-3

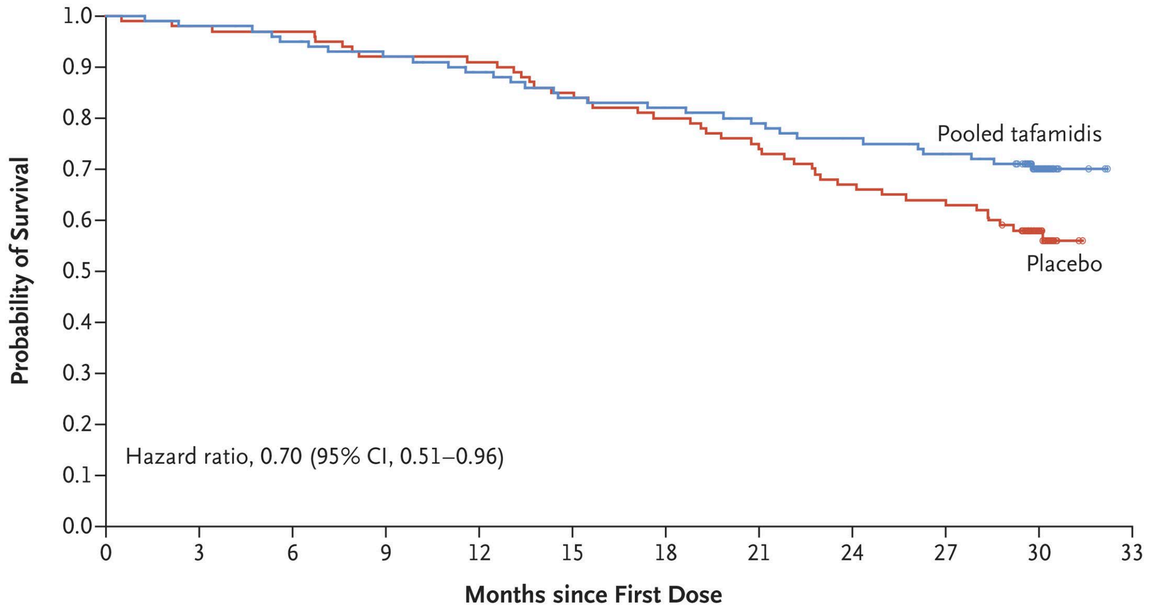
Pharmacologic Therapy:

Impact on mortality and cardiovascular hospitalization

A Primary Analysis, with Finkelstein–Schoenfeld Method

	No. of Patients	P Value from Finkelstein–Schoenfeld Method	Win Ratio (95% CI)	Patients Alive at Mo 30	Average Cardiovascular-Related Hospitalizations during 30 Mo among Those Alive at Mo 30
				no. (%)	*per patient per yr*
Pooled Tafamidis	264	<0.001	1.70 (1.26–2.29)	186 (70.5)	0.30
Placebo	177			101 (57.1)	0.46

B Analysis of All-Cause Mortality



No. at Risk (cumulative no. of events)

Pooled tafamidis	264 (0)	259 (5)	252 (12)	244 (20)	235 (29)	222 (42)	216 (48)	209 (55)	200 (64)	193 (71)	99 (78)	0 (78)
Placebo	177 (0)	173 (4)	171 (6)	163 (14)	161 (16)	150 (27)	141 (36)	131 (46)	118 (59)	113 (64)	51 (75)	0 (76)

C Frequency of Cardiovascular-Related Hospitalizations

	No. of Patients	No. of Patients with Cardiovascular- Related Hospitalizations	Cardiovascular- Related Hospitalizations	Pooled Tafamidis vs. Placebo Treatment Difference
		total no. (%)	*no. per yr*	*relative risk ratio (95% CI)*
Pooled Tafamidis	264	138 (52.3)	0.48	0.68 (0.56–0.81)
Placebo	177	107 (60.5)	0.70	

Ref: Maurer et al., 2018

JAMA Network™

From: **Cardiac Amyloidosis Due to Transthyretin Protein: A Review**

JAMA. 2024;331(9):778-791. doi:10.1001/jama.2024.0442

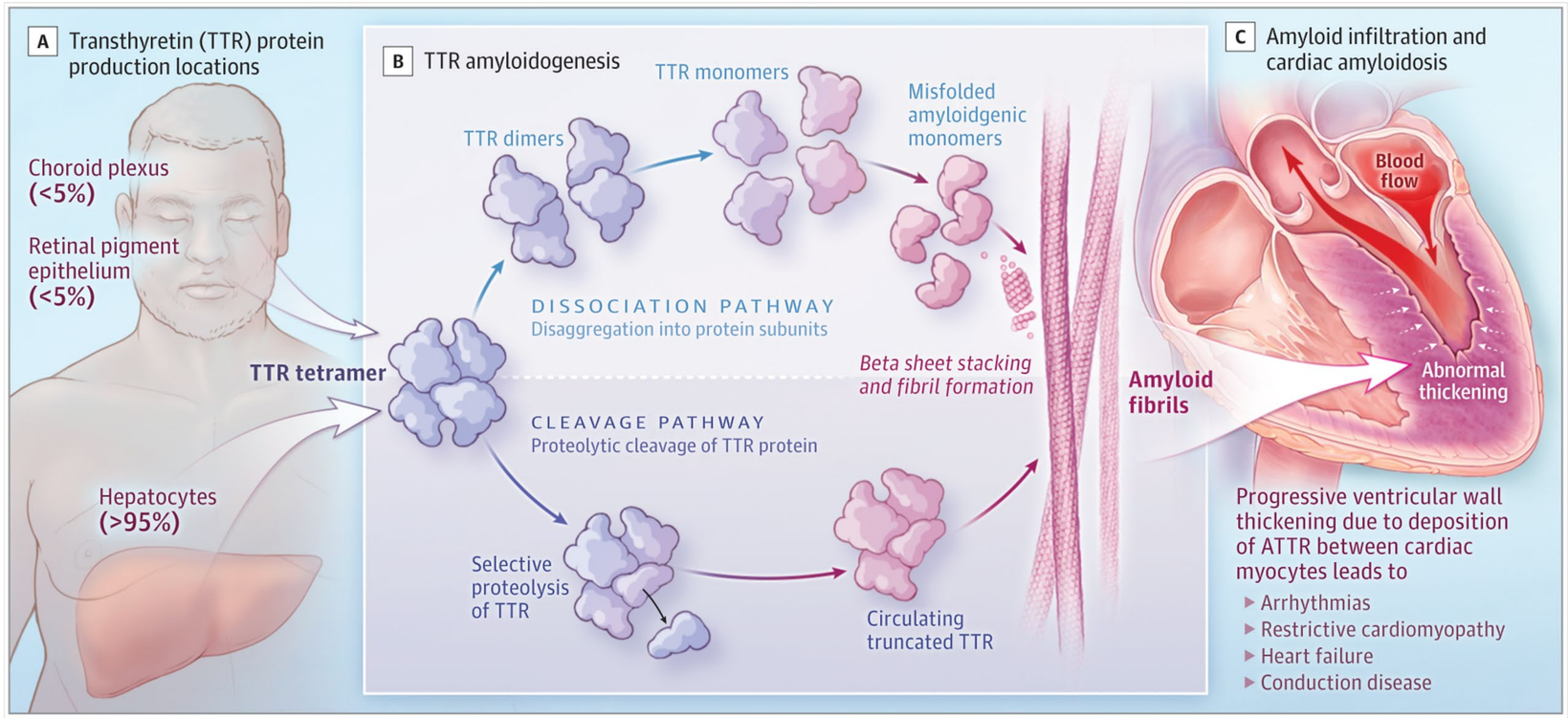


Figure Legend:

Pathobiology of ATTR Amyloidosis and Cardiac ManifestationsCirculating TTR protein is synthesized by the liver as a homotetramer that dissociates or is proteolytically cleaved into intermediates that misfold and ultimately organize into amyloid fibrils. Amyloid fibrils then deposit in the heart, resulting in arrhythmia, conduction disease often requiring placement, and heart failure.

Gene Therapy Strategies
For ATTR Cardiomyopathy

Antisense oligonucleotides (ASO)

- Single strand oligonucleotide
- Usually 16-20 nucleotides in length
- Bind to complementary mRNA and cause degradation
- ASO's can enter the cell without a transfection agent

Small Interfering RNAs (SiRNAs)

- Double strand RNA molecules
- Bind complementary RNA and cause degradation
- Must enter cytoplasm of target cells

CRISPR-Cas9 – in vivo Genome Editing

Ioannou et al. BioDrugs 2023

Gene Therapy Examples

ATTR Cardiomyopathy

Patisiran

- SiRNA
- Approved for polyneuropathy

Vutrisiran

- RNA interference;
- Approved 2025 for cardiac ATTR

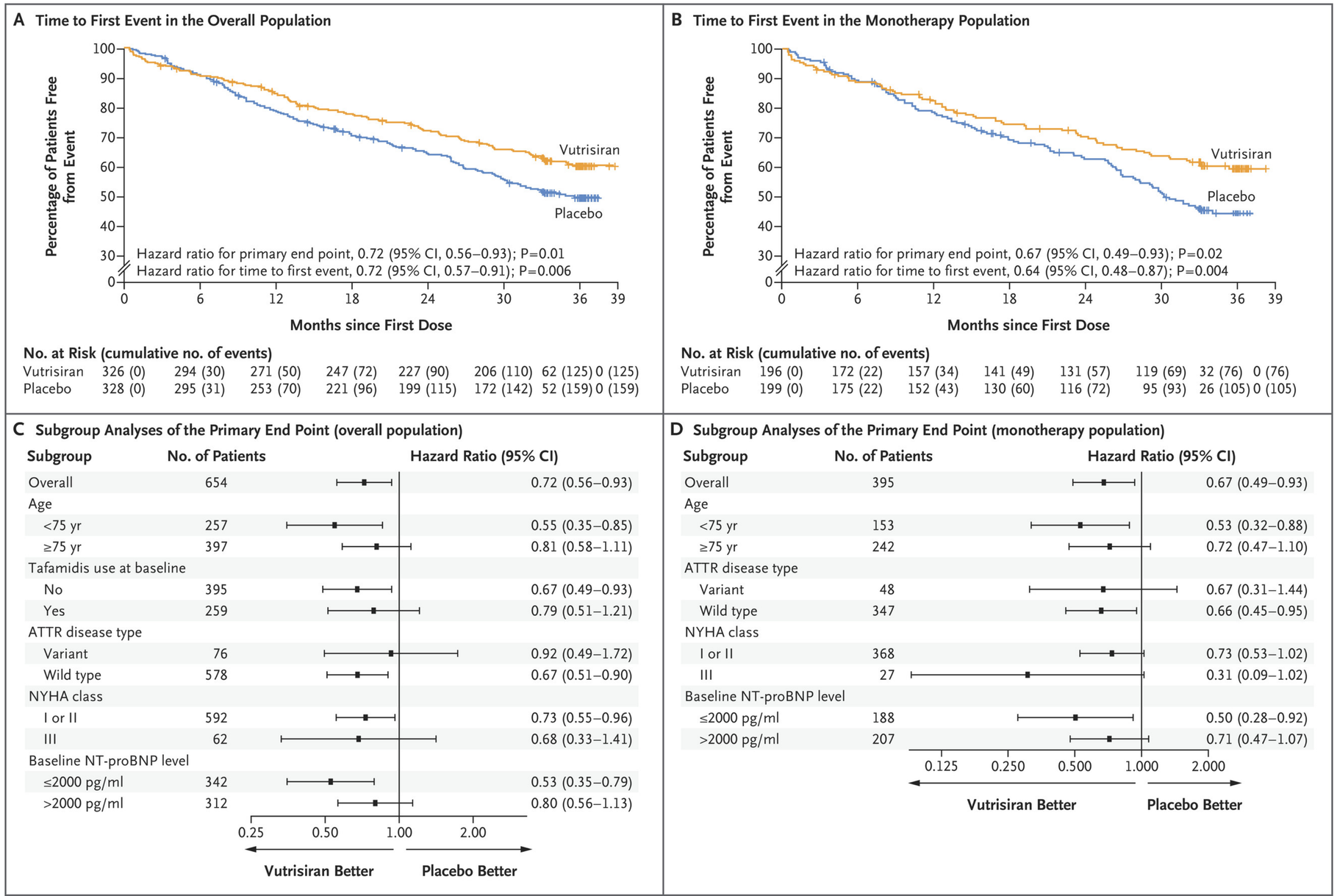
Inotersen

- Antisense oligonucleotide

Nexiguran ziclumeran

- CRISPR-Cas9
- Lipid nanoparticle mediated delivery system
- Produces a dose-dependent TTR knockdown

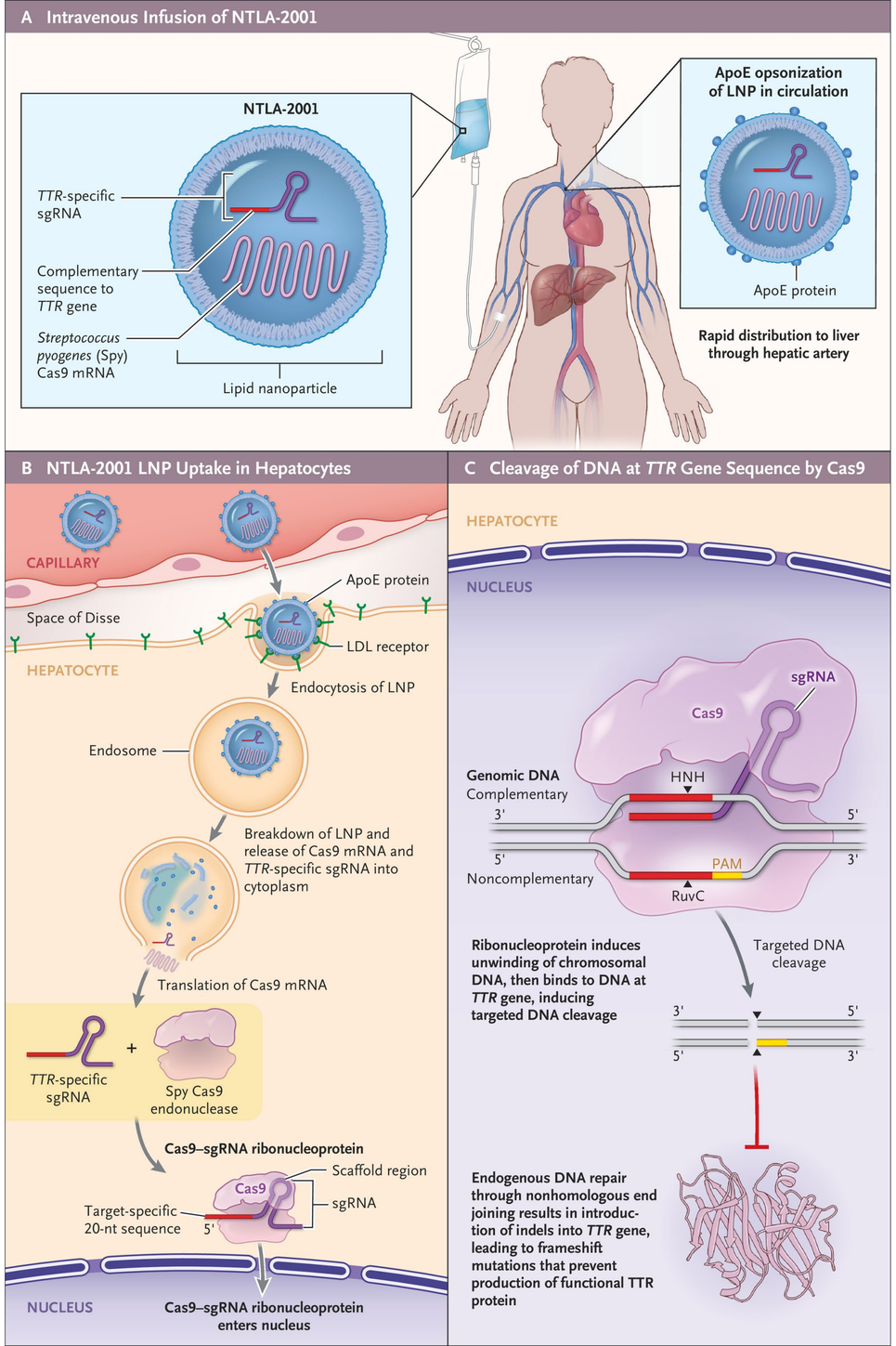
HELIOS-B Study: Vutrisiran

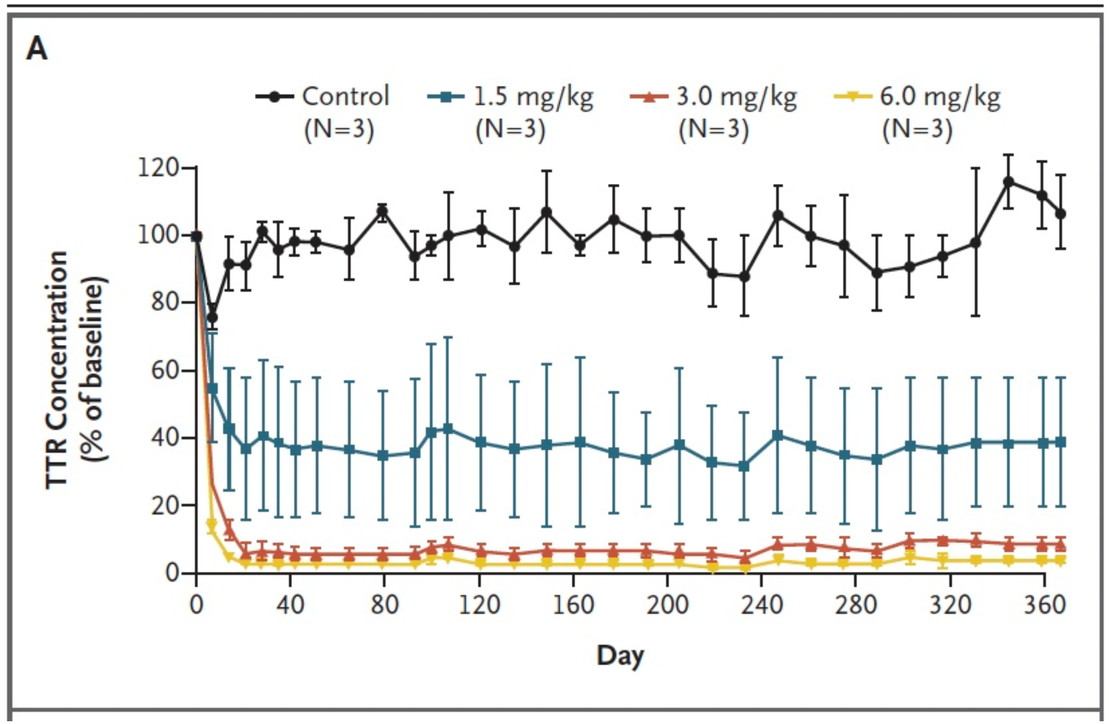


Ref: Fontana et al., 2025

Intellia Therapeutics

2021 Trial Results





Ref: Gillmore et al., 2021

****Updated 2024 data to be shared on next webinar*

Summary

AMYLOIDOSIS

Devastating cause of heart failure (50% of restrictive cardiomyopathy)

TREATMENTS

Rapidly evolving landscape leading to dramatic change in the outlook for affected patients.

There are now therapies based on TTR stabilization or using siRNA shown to reduce all-cause mortality

ATTR CARDIOMYOPATHY

Drug therapy and evolving gene therapeutic strategies deploying several state-of-the-art modalities

ATTR-CM Educational Toolkit

Coming July 2025!

Scan the QR code to Learn More!

www.heart.org/ATTR-CM

The American Heart Association is leading a national effort to improve the recognition, diagnosis, and management of Transthyretin Amyloid Cardiomyopathy (ATTR-CM). In collaboration with clinicians, scientists, and patient advocates, this initiative seeks to reduce diagnostic delays, strengthen multidisciplinary care, and expand awareness of emerging therapies, including gene-based approaches to cardiovascular disease.

This educational toolkit supports both healthcare professionals and patients/caregivers in navigating the complexities of ATTR-CM. The resources are designed to promote disease awareness and education, early recognition, informed decision-making, and meaningful conversations about clinical trial participation. An accompanying clinician guide equips providers to initiate and support discussions around gene therapy and evolving treatment pathways.

Toolkit & Resources Coming July 2025!

Educational materials for healthcare professionals and patients/caregivers will be available here in July 2025.

Stay tuned for updates and access to downloadable guides, patient education materials, and clinician-focused resources.

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Home / Health Topics / Cardiomyopathy / What is Cardiomyopathy? / Transthyretin Amyloid Cardiomyopathy (ATTR-CM)

Transthyretin Amyloid Cardiomyopathy (ATTR-CM)

What is transthyretin amyloid cardiomyopathy?

Transthyretin (trans-thy-re-tin) amyloid cardiomyopathy (ATTR-CM) is an underdiagnosed and potentially fatal disease of the heart muscle. In ATTR-CM, a protein called transthyretin that normally circulates in the bloodstream becomes misshapen and builds up in the heart, nerves and other organs

Cardiomyopathy

- What is Cardiomyopathy? −
  - Dilated Cardiomyopathy (DCM)
  - Hypertrophic Cardiomyopathy (HCM)
  - HCM in Young Adults and Student Athletes
  - Peripartum Cardiomyopathy (PPCM)
  - Restrictive Cardiomyopathy
  - Transthyretin Amyloid Cardiomyopathy (ATTR-CM)
  - Arrhythmogenic Right Ventricular Dysplasia
  - Is Broken Heart Syndrome Real?
- Understand Your Risk for Cardiomyopathy +
- Symptoms and Diagnosis of Cardiomyopathy
- Prevention and Treatment of Cardiomyopathy
- HCM Personal Stories +

Related Articles

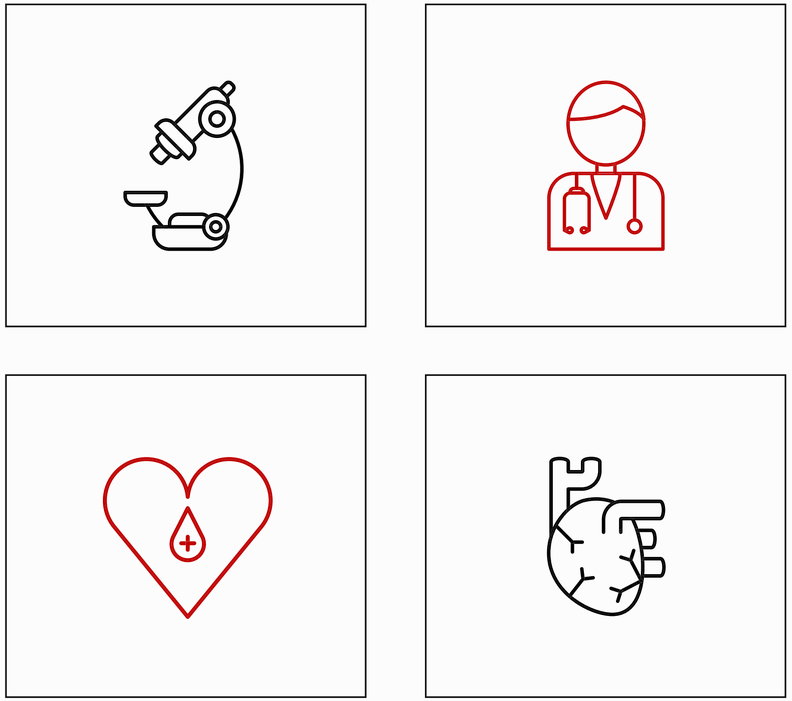
ATTR-CM Resources Available

Health Topics:
What is ATTR-CM?

Health Topics:
Genetic Testing and Counseling for hATTR

Health Topics:
A Guide to Understanding Clinical Trials

Continuing the Conversation



Webinar #2 – October 2025

Advances in Diagnosis & Treatment of Amyloidosis

Webinar #3 – February 2026

Multidisciplinary Care & Future Directions in Amyloidosis

STAY TUNED!

Invitation and registration details for upcoming webinars will be shared in the coming months!

Scan the QR code to Learn More! Or visit: www.heart.org/ATTR-CM

Q & A

American Heart Association®

Thank you for joining us today!

Recordings of today's sessions will be enduring resources in a few weeks on www.heart.org/ATTR-CM

Connect with Us! Scan to email ClinicalStudies@heart.org