Bill, living with transthyretin amyloidosis, and his wife, Maura

NTLA-2001 for ATTR Amyloidosis: Interim Clinical Results from Ongoing Phase 1 Trial

June 28, 2021

Intellia THERAPEUTICS

Agenda

Welcome

Introduction

John Leonard, M.D.
Chief Executive Officer, Intellia Therapeutics

Review of NTLA-2001 Interim Phase 1 Clinical Trial Data

Dr. Julian Gillmore, M.D, Ph.D., FRCP, FRCPath,
Professor of Medicine, National Amyloidosis Centre, UCL Division of Medicine, Royal Free Hospital, U.K.; National Coordinator for Intellia's Phase 1 Study of NTLA-2001 in the U.K.

NTLA-2001 Clinical Development Plans

David Lebwohl, M.D.
Chief Medical Officer, Intellia Therapeutics

Platform Outlook

Laura Sepp-Lorenzino, Ph.D.
Chief Scientific Officer, Intellia Therapeutics

Closing Remarks and Q&A Session

Intellia THERAPEUTICS

Intellia Therapeutics' Legal Disclaimer

This presentation contains "forward-looking statements" of Intellia Therapeutics, Inc. ("Intellia", "we" or "our") within the meaning of the Private Securities Litigation Reform Act of 1995. These forward-looking statements include, but are not limited to, express or implied statements regarding Intellia's beliefs and expectations regarding our: ability to enroll and dose the necessary subjects in the clinical studies for NTLA-2001 for the treatment of transthyretin amyloidosis ("ATTR"), provide timing on data readouts from the clinical studies, and successfully secure additional clinical studies authorizations, such as investigational new drug applications ("IND") and clinical trial applications ("CTA"), in other countries; ability to evaluate NTLA-2001 in a broader ATTR population; expectation that clinical results will support NTLA-2001's safety and activity profile; belief that NTLA-2001 can be approved as a single-dose therapy or that it can halt and reverse ATTR progression; plans to present data at upcoming scientific conferences; advancement, expansion and acceleration of our CRISPR/Cas9 technology and in vivo pipeline to develop breakthrough genome editing treatments for people living with severe diseases; ability to demonstrate our platform's modularity and replicate or apply results achieved in preclinical studies, including those in our ATTR program, in any future studies, including human clinical trials; ability to optimize the impact of our collaborations on our development programs, including but not limited to our collaboration with Regeneron Pharmaceuticals, Inc. ("Regeneron"); statements regarding the timing of regulatory filings and clinical trial execution, including dosing of patients, regarding our other development programs, including NTLA-2002, NTLA-5001 and our other research programs; and potential commercial opportunities, including value and market, for our product candidates

Any forward-looking statements in this presentation are based on management's current expectations and beliefs of future events, and are subject to a number of risks and uncertainties that could cause actual results to differ materially and adversely from those set forth in or implied by such forward-looking statements. These risks and uncertainties include, but are not limited to: risks related to our ability to protect and maintain our intellectual property position; risks related to our relationship with third parties, including our licensors and licensees; risks related to the ability of our licensors to protect and maintain their intellectual property position; uncertainties related to regulatory agencies' evaluation of regulatory filings and other information related to our product candidates; uncertainties related to the authorization, initiation and conduct of studies and other development requirements for our product candidates; the risk that any one or more of our product candidates, including those that are co-developed, will not be successfully developed and commercialized; the risk that the results of preclinical studies or clinical studies will not be predictive of future results in connection with future studies; and the risk that our collaborations with Regeneron or our other ex vivo collaborations will not continue or will not be successful. For a discussion of these and other risks and uncertainties, and other important factors, any of which could cause Intellia's actual results to differ from those contained in the forward-looking statements, see the section entitled "Risk Factors" in Intellia's most recent annual report on Form 10-K as well as discussions of potential risks, uncertainties, and other important factors in Intellia's other filings with the Securities and Exchange Commission ("SEC"). All information in this presentation is as of the date of the release, and Intellia undertakes no duty to update this information unless required by law.

Intellia THERAPEUTICS

Agenda

Welcome

Introduction

John Leonard, M.D.
Chief Executive Officer, Intellia Therapeutics

Review of NTLA-2001 Interim Phase 1 Clinical Trial Data

Dr. Julian Gillmore, M.D, Ph.D., FRCP, FRCPath,
Professor of Medicine, National Amyloidosis Centre, UCL Division of Medicine, Royal Free Hospital, U.K.; National Coordinator for Intellia's Phase 1 Study of NTLA-2001 in the U.K.

NTLA-2001 Clinical Development Plans

David Lebwohl, M.D.
Chief Medical Officer, Intellia Therapeutics

Platform Outlook

Laura Sepp-Lorenzino, Ph.D.
Chief Scientific Officer, Intellia Therapeutics

Closing Remarks and Q&A Session

Intellia THERAPEUTICS

NTLA-2001 for Transthyretin (ATTR) Amyloidosis

First systemically delivered CRISPR-based therapy to enter clinical development

Impact of NTLA-2001
Interim Clinical Data Readout

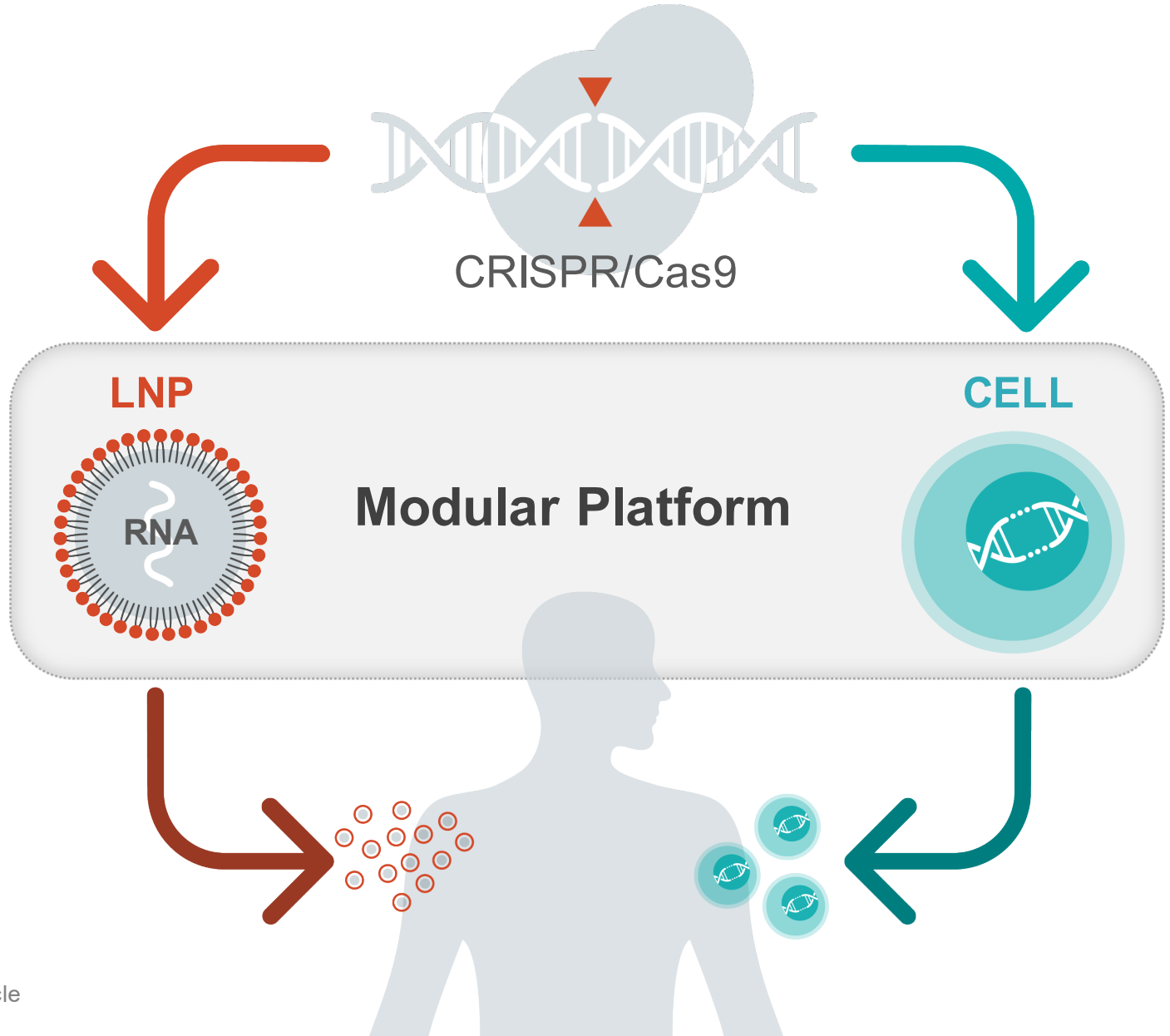
Intellia THERAPEUTICS

Building a Full-Spectrum Genome Editing Company

In Vivo

CRISPR is the therapy

Genetic diseases



Ex Vivo

CRISPR creates the therapy

Immuno-oncology

Autoimmune diseases

LNP: Lipid Nanoparticle

Intellia THERAPEUTICS

Diverse Therapeutic Pipeline of *In Vivo* and *Ex Vivo* Assets

PROGRAM	APPROACH	Research	Candidate Selection	IND-Enabling	Early-stage Clinical	Late-stage Clinical	PARTNER
***In Vivo:* CRISPR is the therapy**							
NTLA-2001: Transthyretin Amyloidosis	Knockout						LEAD Intellia* THERAPEUTICS REGENERON
NTLA-2002: Hereditary Angioedema	Knockout						Intellia THERAPEUTICS
Hemophilia A and B	Insertion						LEAD REGENERON* Intellia THERAPEUTICS
Research Programs	Knockout, Insertion, Consecutive Edits						Intellia THERAPEUTICS
Research Programs	Various						Intellia THERAPEUTICS REGENERON**
***Ex Vivo:* CRISPR creates the therapy**							
OTQ923 / HIX763: Sickle Cell Disease	HSC						NOVARTIS Intellia*** THERAPEUTICS
NTLA-5001: Acute Myeloid Leukemia	WT1-TCR						Intellia THERAPEUTICS
Solid Tumors	WT1-TCR						Intellia THERAPEUTICS
Allo Undisclosed	Undisclosed						Intellia THERAPEUTICS
Other Novartis Programs	CAR-T, HSC, OSC	Undisclosed					NOVARTIS Intellia*** THERAPEUTICS

* Lead development and commercial party ** Rights to certain *in vivo* targets *** Milestones & royalties only
CAR-T: Chimeric Antigen Receptor T cells **HSC:** Hematopoietic Stem Cells **OSC:** Ocular Stem Cells **TCR:** T Cell Receptor

Landmark Clinical Data Show Deep, Dose-Dependent TTR Reduction After Single Dose of NTLA-2001

Key Takeaways

- **87% mean reduction in serum TTR** at second dose level (0.3 mg/kg) in three patients by day 28
  - **Maximum 96% serum TTR reduction**
- **Dose-dependent response**
- **Encouraging safety profile** and no serious adverse events in first six patients by day 28

Support NTLA-2001
as potential single-dose treatment for ATTR amyloidosis

Validate LNP Platform
for systemic delivery of CRISPR

Accelerate *In Vivo* Pipeline
by unlocking the liver

REGENERON

Intellia THERAPEUTICS

Agenda

Welcome

Introduction

John Leonard, M.D.
Chief Executive Officer, Intellia Therapeutics

Review of NTLA-2001 Interim Phase 1 Clinical Trial Data

Dr. Julian Gillmore, M.D, Ph.D., FRCP, FRCPath,
Professor of Medicine, National Amyloidosis Centre, UCL Division of Medicine, Royal Free Hospital, U.K.; National Coordinator for Intellia's Phase 1 Study of NTLA-2001 in the U.K.

NTLA-2001 Clinical Development Plans

David Lebwohl, M.D.
Chief Medical Officer, Intellia Therapeutics

Platform Outlook

Laura Sepp-Lorenzino, Ph.D.
Chief Scientific Officer, Intellia Therapeutics

Closing Remarks and Q&A Session

Intellia THERAPEUTICS

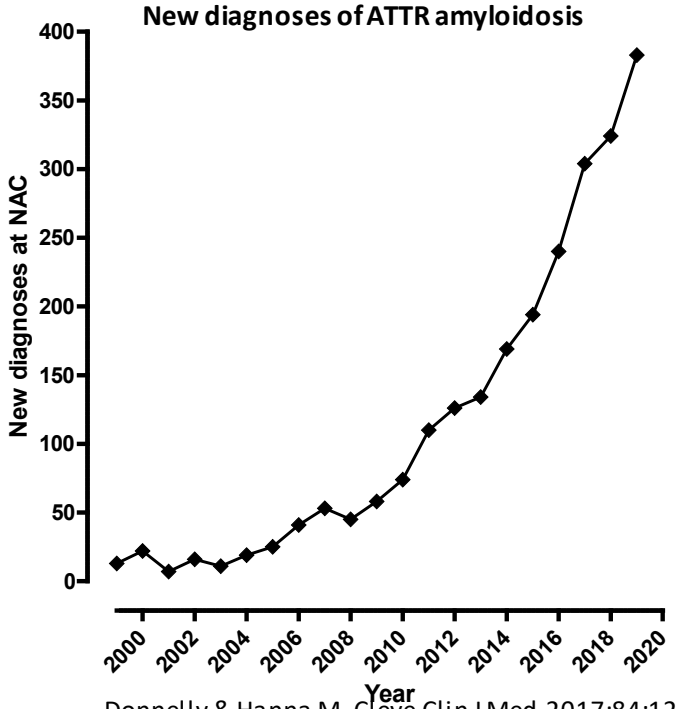
Transthyretin (ATTR) amyloidosis

- **ATTR amyloidosis is a rare, progressive, and fatal disease caused by accumulation of amyloid deposits composed of misfolded transthyretin (TTR) protein**

- **Hereditary ATTR amyloidosis (hATTR/ATTRv) causes a spectrum of clinical disease**
  - More than 130 amyloidogenic mutations of *TTR* gene
  - Autosomal dominant pattern of inheritance
  - Estimated 50,000 individuals worldwide
  - Variable phenotype dominated by:
    - **peripheral & autonomic neuropathy (ATTRv-PN)**
    - **amyloid cardiomyopathy (ATTRv-CM)**

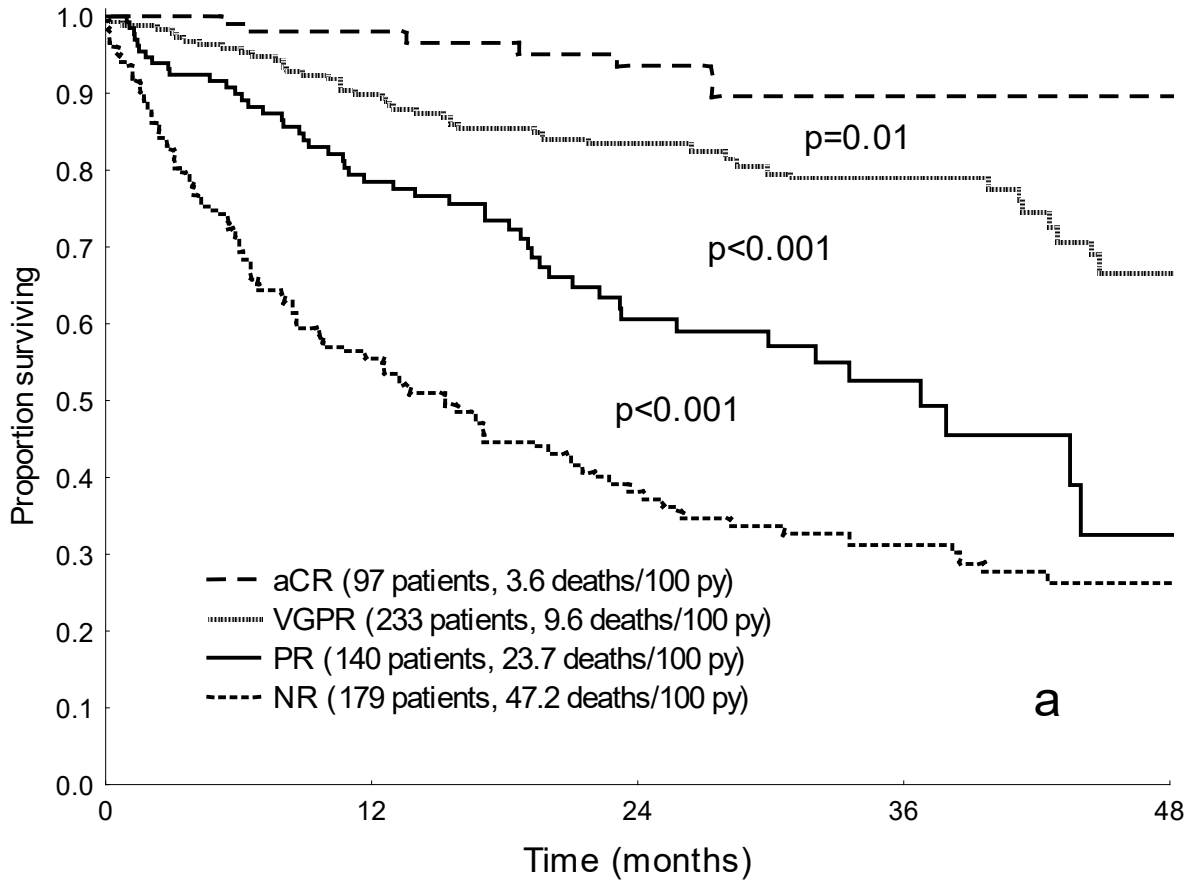
    } **ATTRv-Mixed**

- **Wild-type ATTR amyloidosis (ATTRwt) is a cardiomyopathy**
  - Increasingly recognized cause of heart failure in individuals over 50s
  - Progressive and fatal within 3-10 years
  - Majority never diagnosed



Donnelly & Hanna M. Cleve Clin J Med 2017;84:12–26
Lane T *et al,* Circulation 2019;140:16–26
Pinney *et al,* J Am Heart Assoc 2013;2: e000098
Rowczenio *et al,* Orphanet J Rare Dis. 2017;12(Suppl 1):165

AL Amyloidosis: Greater fibril precursor protein suppression associated with improved patient survival



Palladini G *et al*, JCO 2012;30:4541-4549

AA Amyloidosis: Greater fibril precursor protein suppression associated with improved patient survival

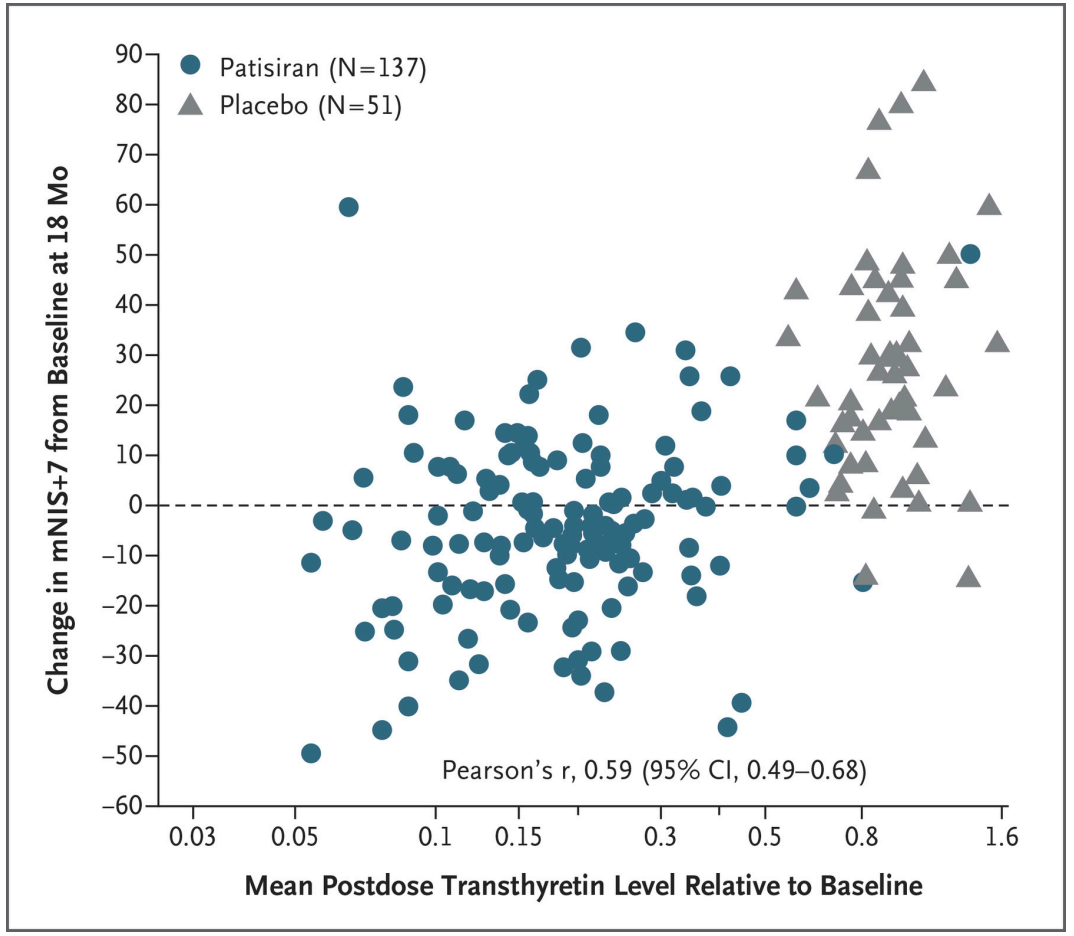
Table 3. Unadjusted Relative Risk of Death Associated with the Most Recent Median Annual SAA Concentration during Follow-up.*

SAA Octile (mg/liter)	Relative Risk (95% CI)	P Value
<4	1.0	
≥4 to <9	3.9 (1.5–10.4)	0.007
≥9 to <16.7	5.1 (2.7–9.4)	0.003
≥16.7 to <28	7.0 (3.7–13.4)	0.07
≥28 to <45.6	9.1 (4.8–17.2)	0.008
≥45.6 to <87	12.1 (6.9–21.4)	<0.001
≥87 to <155	17.0 (8.6–33.8)	<0.001
≥155	17.7 (8.7–36.0)	<0.001

* The SAA value is the median concentration within each 12-month period and was incorporated into the Cox regression model as a time-dependent covariate.

Lachmann HJ *et al,* NEJM 2007:356;2361-71

ATTR Amyloidosis: Greater TTR knockdown in amyloidosis associated with improved neuropathy score



Adams D *et al*, NEJM 2018;379:11–21

2021 PNS ANNUAL MEETING

Virtually Anywhere

12-13 JUNE / 25-27 JUNE 2021

PNS
PERIPHERAL NERVE SOCIETY

In vivo CRISPR/Cas9 Editing of the *TTR* Gene by NTLA-2001 in Patients with Transthyretin Amyloidosis

Julian D. Gillmore[1], Jorg Taubel[2], Justin Kao[3], Marianna Fontana[1], Michael L. Maitland[4], Jessica Seitzer[4], Daniel O'Connell[4], Jonathan Phillips[4], Kristy Wood[4], Yuanxin Xu[4], Adam Amaral[4], Adam P. Boyd[4], Jeffrey E. Cehelsky[4], David E. Gutstein[5], Laura Sepp-Lorenzino[4], David Lebwohl[4], Ed Gane[6,7]

1. National Amyloidosis Centre, Division of Medicine, University College London, Royal Free Hospital, London, UK
2. Richmond Pharmacology Limited, St George's University of London, London, UK
3. Department of Neurology, Auckland City Hospital, Auckland, New Zealand
4. Intellia Therapeutics, Inc., Cambridge, MA, USA
5. Regeneron Pharmaceuticals, Inc., Tarrytown, NY, USA
6. New Zealand Clinical Research, Auckland, New Zealand
7. University of Auckland, Auckland, New Zealand.

2021 PNS ANNUAL MEETING
Virtually Anywhere

Disclosures

- JDG: Expert adviser for Alnylam Pharmaceuticals, Eidos Therapeutics, Ionis Pharmaceuticals Inc., and Intellia Therapeutics Inc.

Potential for gene editing to address unmet need for hATTR/ATTRv amyloidosis

- Therapy in amyloidosis is intended to reduce or stabilize precursor protein, in ATTR amyloidosis = transthyretin (TTR)
  - Gene silencing therapy (patisiran) knocks serum TTR down by ~80% (mean) and benefits neuropathy in ATTRv[1]
  - Patients on standard treatment experience debilitating effects, disease progression and ultimately fatal complications
  - Greater TTR knockdown is expected to achieve better clinical outcomes, and can potentially reverse the disease

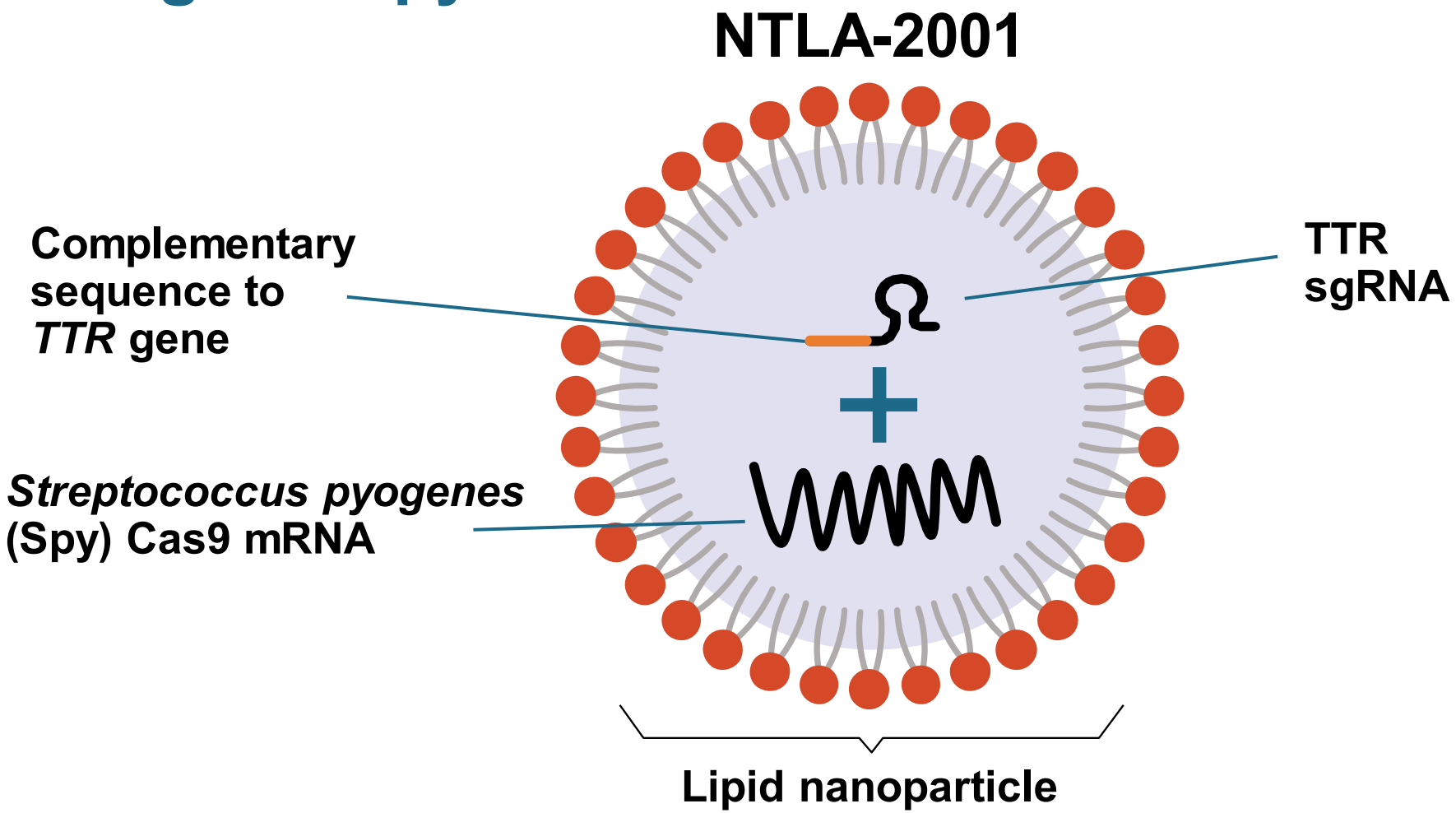
- Editing of the *TTR* gene is an attractive alternative therapeutic strategy

➢ **Potentially providing permanent, profound TTR knockdown, without the need for chronic therapy**

TTR, transthyretin

1. Adams D, et al. N Engl J Med 2018;379:11–21.

NTLA-2001 is a novel CRISPR/Cas9-based *in vivo* gene editing therapy



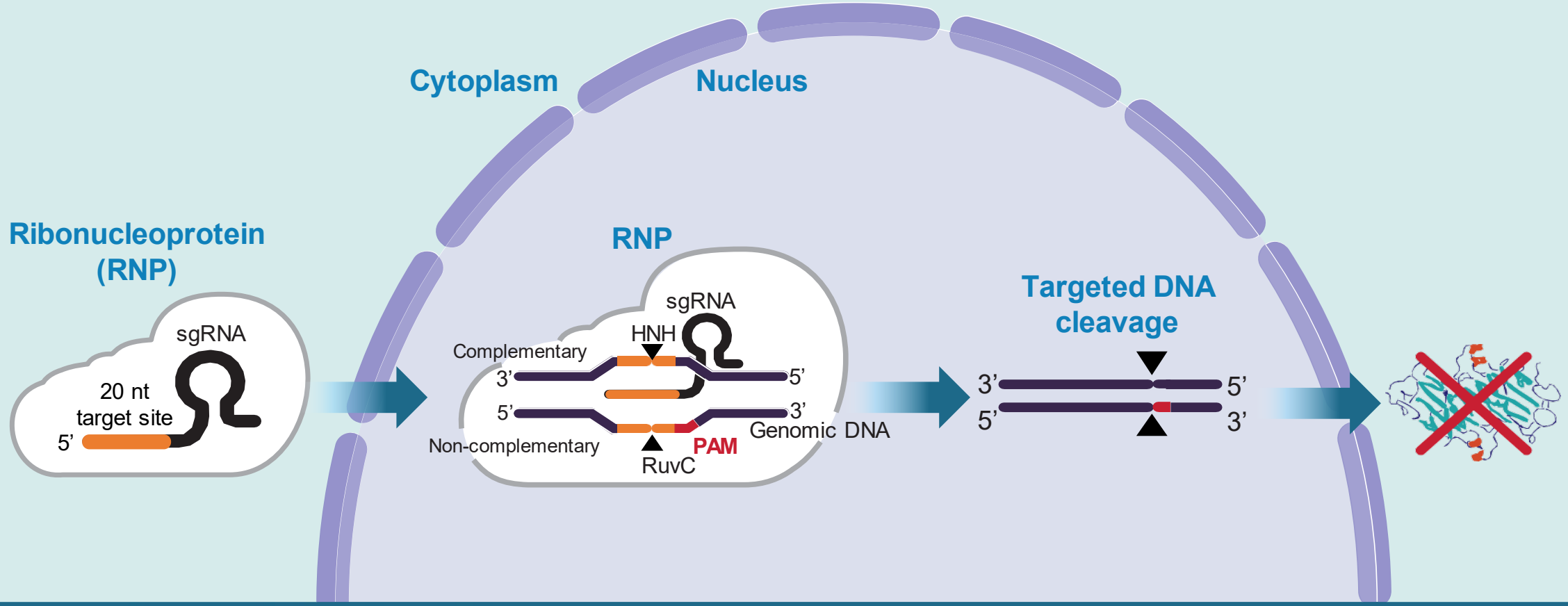
sgRNA; single guide RNA; TTR, transthyretin

NTLA-2001 delivers sgRNA and Cas9 into the nucleus, which precisely edit and inactivate the *TTR* gene

Cas9/sgRNA ribonucleoprotein enters nucleus

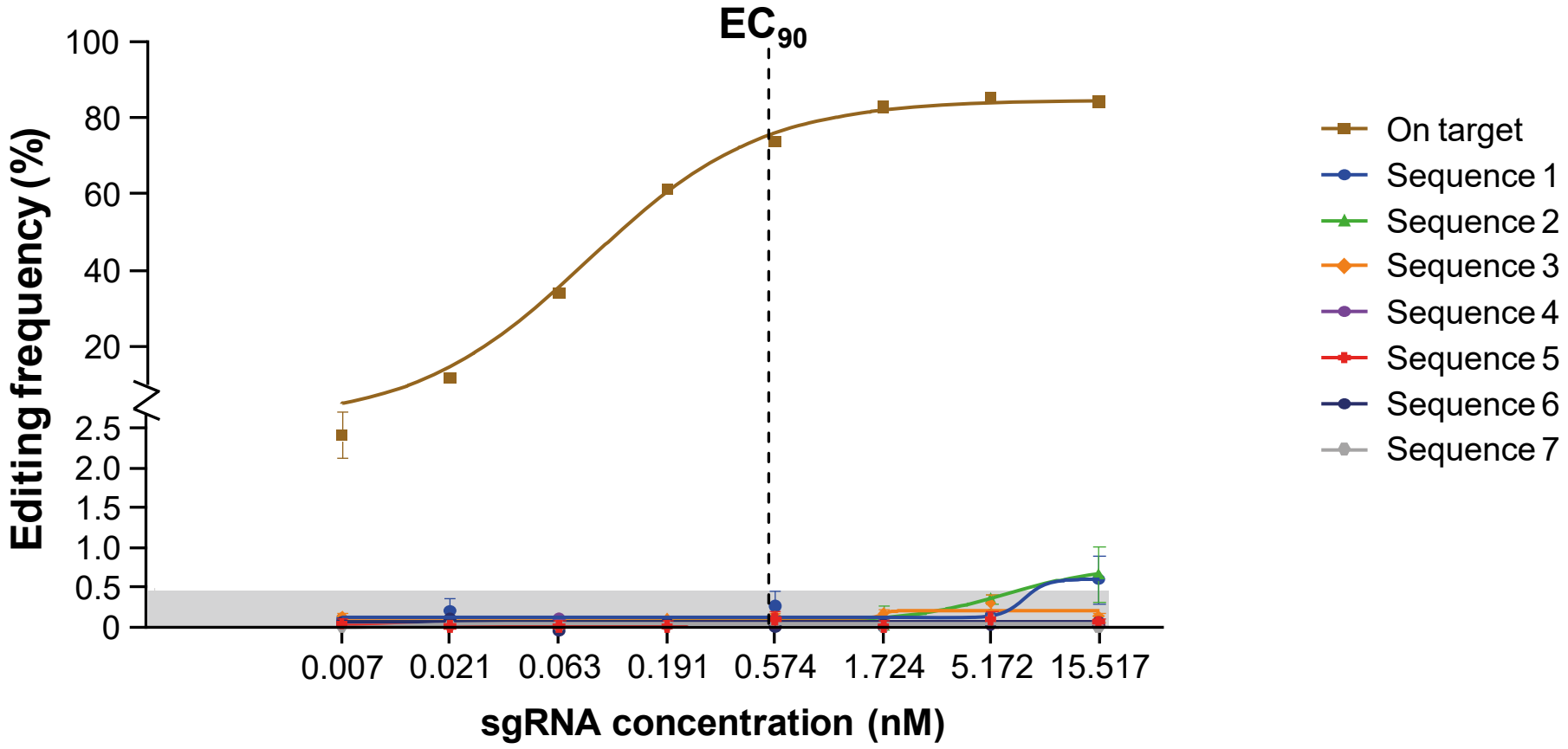
Ribonucleoprotein induces unwinding of chromosomal DNA, then binds to DNA at *TTR* gene, inducing targeted DNA cleavage

Endogenous DNA repair via nonhomologous end joining results in introduction of indels into *TTR* gene, leading to frameshift mutations that prevent production of functional TTR protein



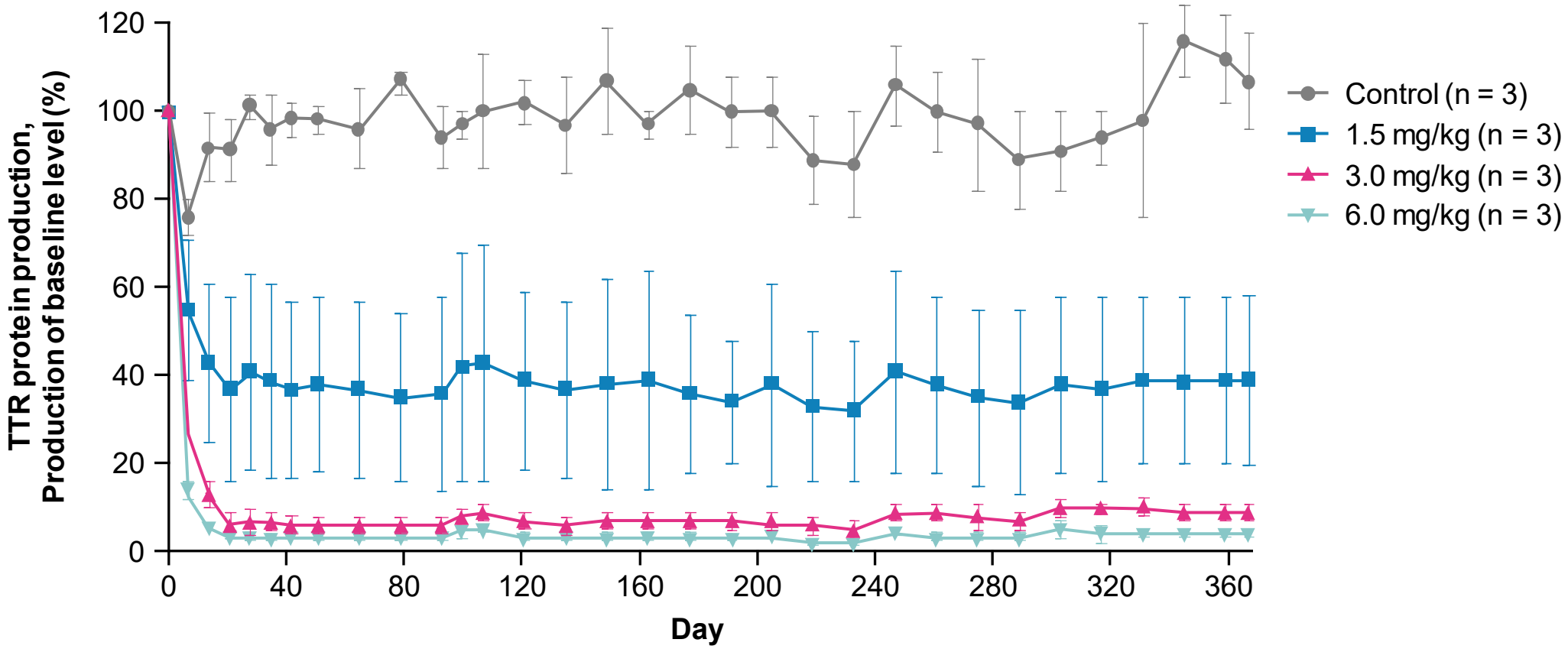
HNH, an endonuclease domain named for characteristic histidine and asparagine residues; nt, nucleotide; RNP, ribonucleoprotein; RuvC, an endonuclease domain; sgRNA, single guide RNA; TTR, transthyretin

In vitro: No detectable off-target editing with pharmacologic concentration of sgRNA



EC_{90}, concentration inducing 90% of maximal effect; sgRNA, single guide RNA

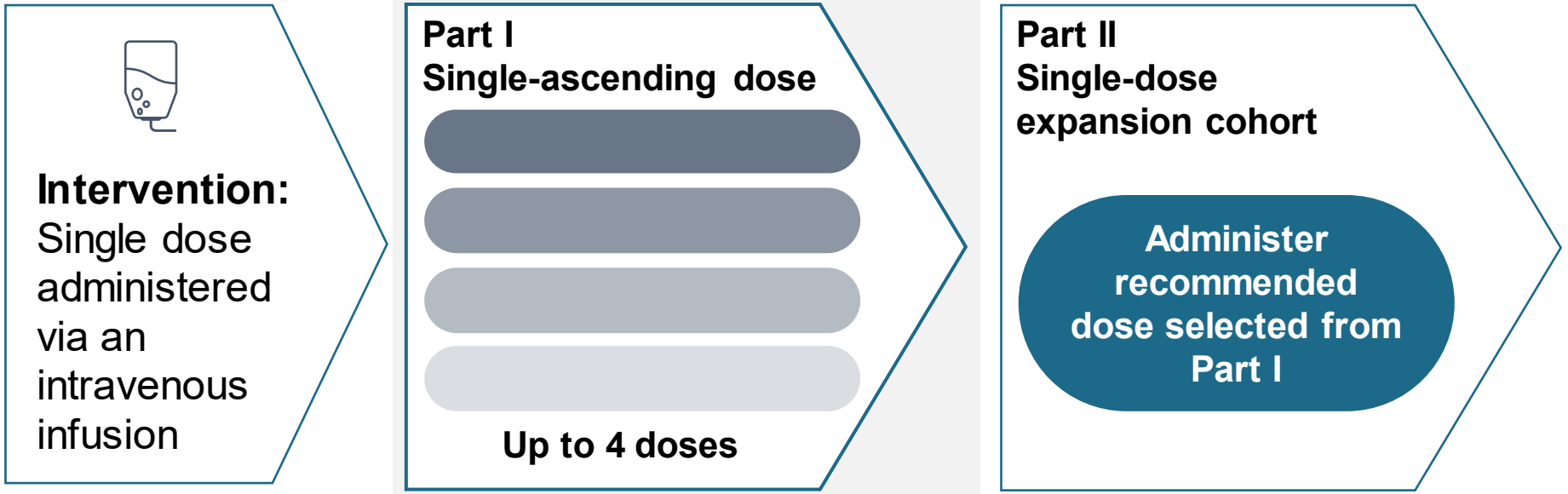
NHP: Durable, >95% TTR reduction after a single dose



NHP; non-human primate, TTR, transthyretin

First-in-human: Two-part phase 1 study of NTLA-2001

Population: Adults with ATTRv with polyneuropathy



Today's interim data cover the first six patients dosed across two cohorts through Day 28

Primary objectives

- Evaluate safety, tolerability, PK and PD
- Measure TTR levels

PD, pharmacodynamics; PK, pharmacokinetics; TTR, transthyretin.
ClinicalTrials.gov identifier: NCT04601051

NTLA-2001 first-in-human study: Demographics

Parameter	0.1 mg/kg dose (n = 3)	0.3 mg/kg dose (n = 3)
Age, years		
Median (min, max)	54 (50, 63)	53 (46, 64)
Sex, n		
Male	1	3
Female	2	0
Weight, kg		
Median (min, max)	82 (70, 89)	84 (82, 90)
Mutation status, n		
p.H110D	0	1
p.S97Y	1	1
p.T80A	2	1
Prior therapy, n		
None	1	2
Diflunisal	2	1

Parameter	0.1 mg/kg dose (n = 3)	0.3 mg/kg dose (n = 3)
Clinical scores, n		
Polyneuropathy disability score 1	3	3
NYHA Functional Classification I	3	3
NT-proBNP (ng/L), median (min, max)	127 (89, 596)	119 (50, 359)
Years since diagnosis (min, max)	2 (2, 9)	3 (1, 11)

NT-proBNP, N-terminal pro-brain natriuretic peptide; NYHA, New York Heart Association

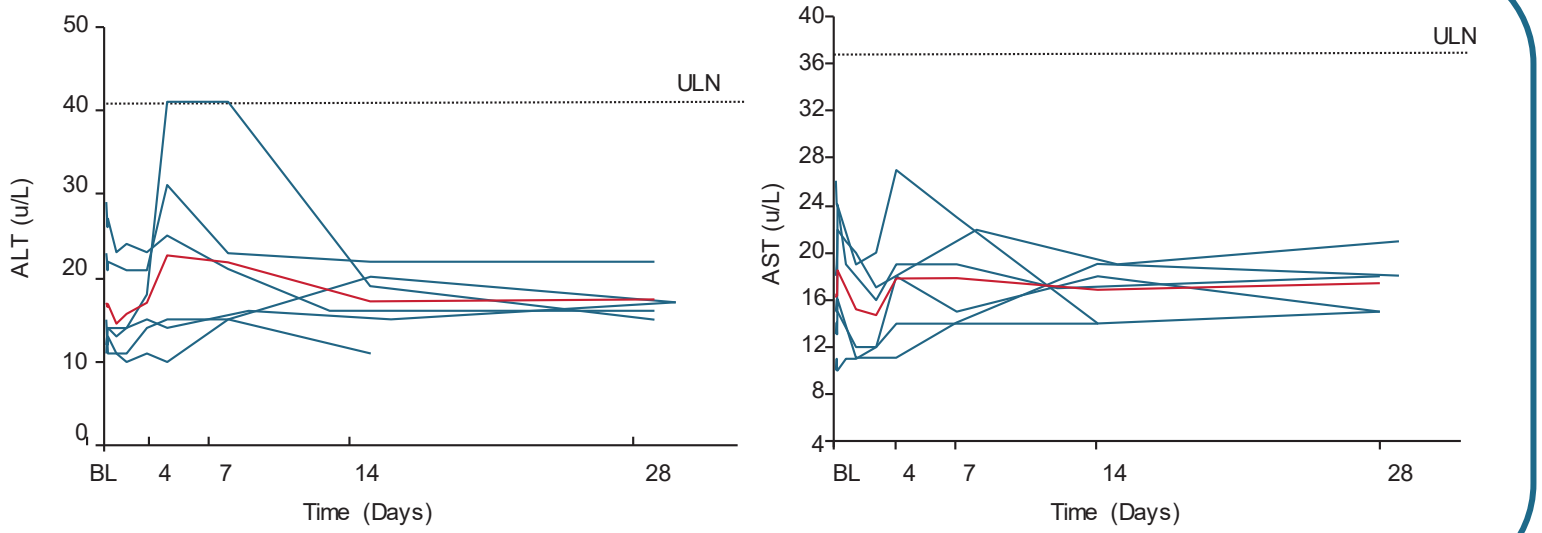
NTLA-2001 generally well tolerated in acute phase (N=6): all AEs Grade 1 with no serious AEs

Preferred Term	0.1 mg/kg (n = 3)	0.3 mg/kg (n = 3)
Subjects with at least one TEAE	**2**	**1**
Headache	2	
Diarrhea	1	
Nausea	1	
Infusion-related reaction	1	
Skin abrasion		1
Vertigo positional	1	
Foreign body sensation in eyes	1	
Catheter site swelling	1	
Acute sinusitis	1	
Thyroxine decreased	1	
Rhinorrhea	1	
Pruritis	1	
Rash	1	

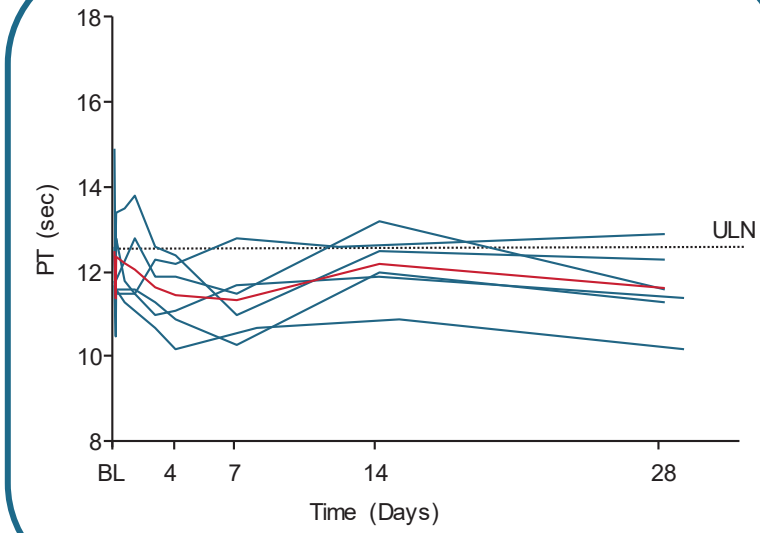
AE, adverse event; TEAE, treatment-emergent adverse event

No liver findings or coagulopathy based on laboratory testing

Liver function



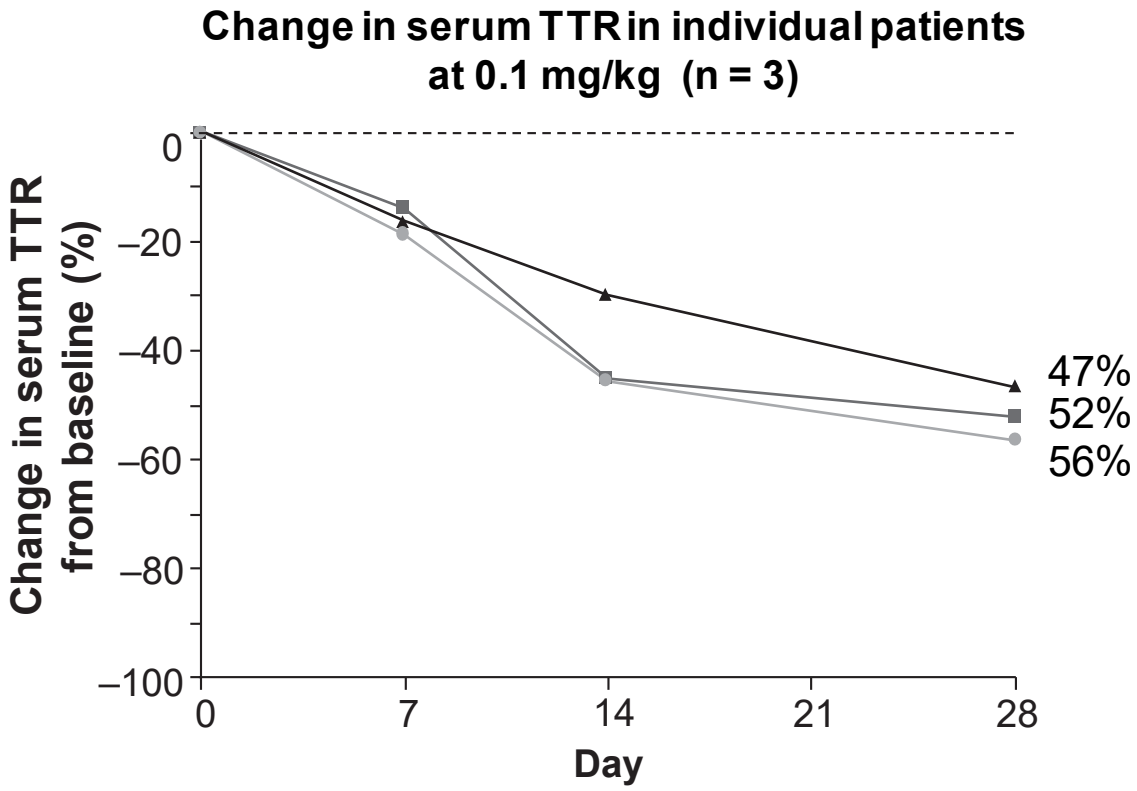
Coagulation



Individual patients — Mean ---- Reference value

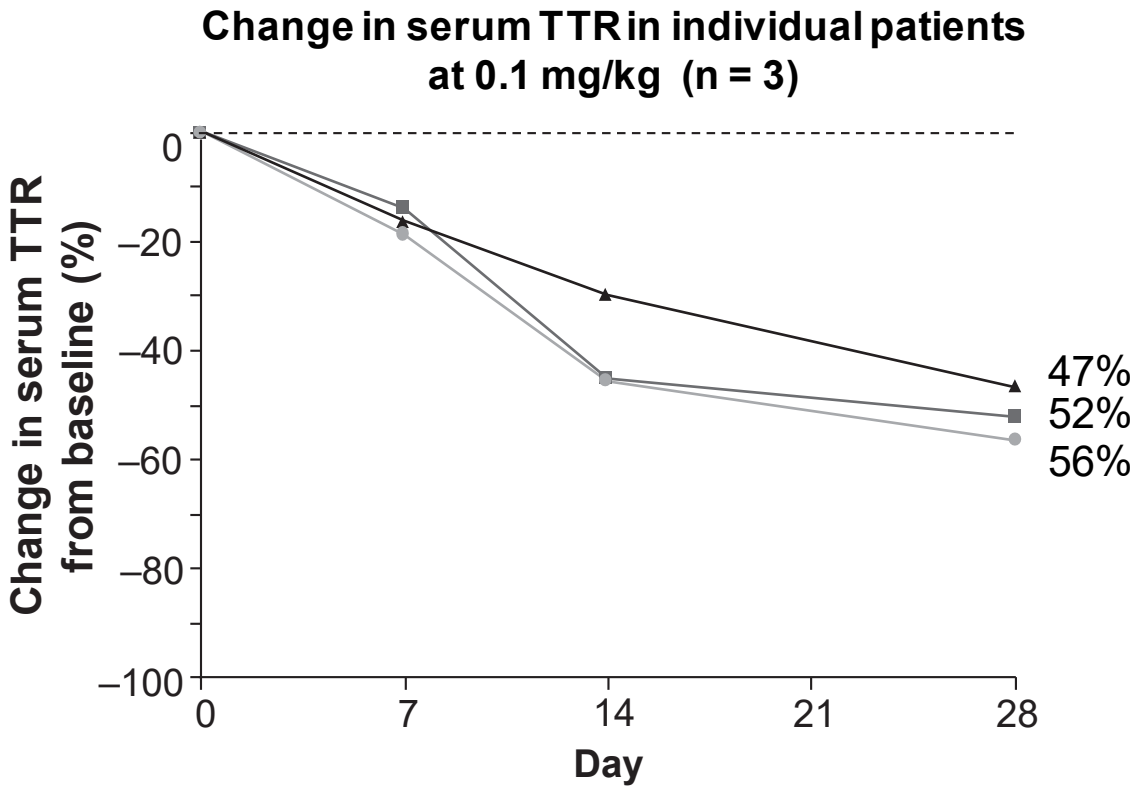
ALT, alanine aminotransferase; AST, aspartate aminotransferase; PT, prothrombin, ULN, upper limit of normal.

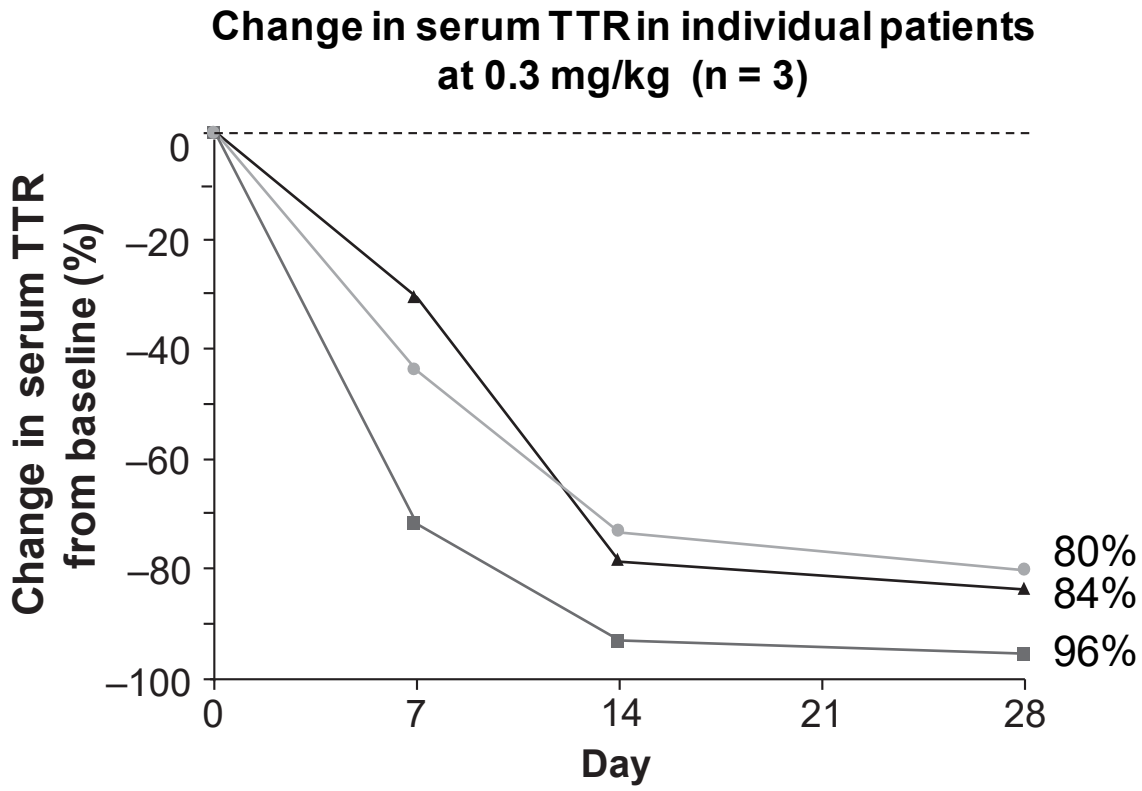
Dose-dependent serum TTR reduction after NTLA-2001



TTR, transthyretin

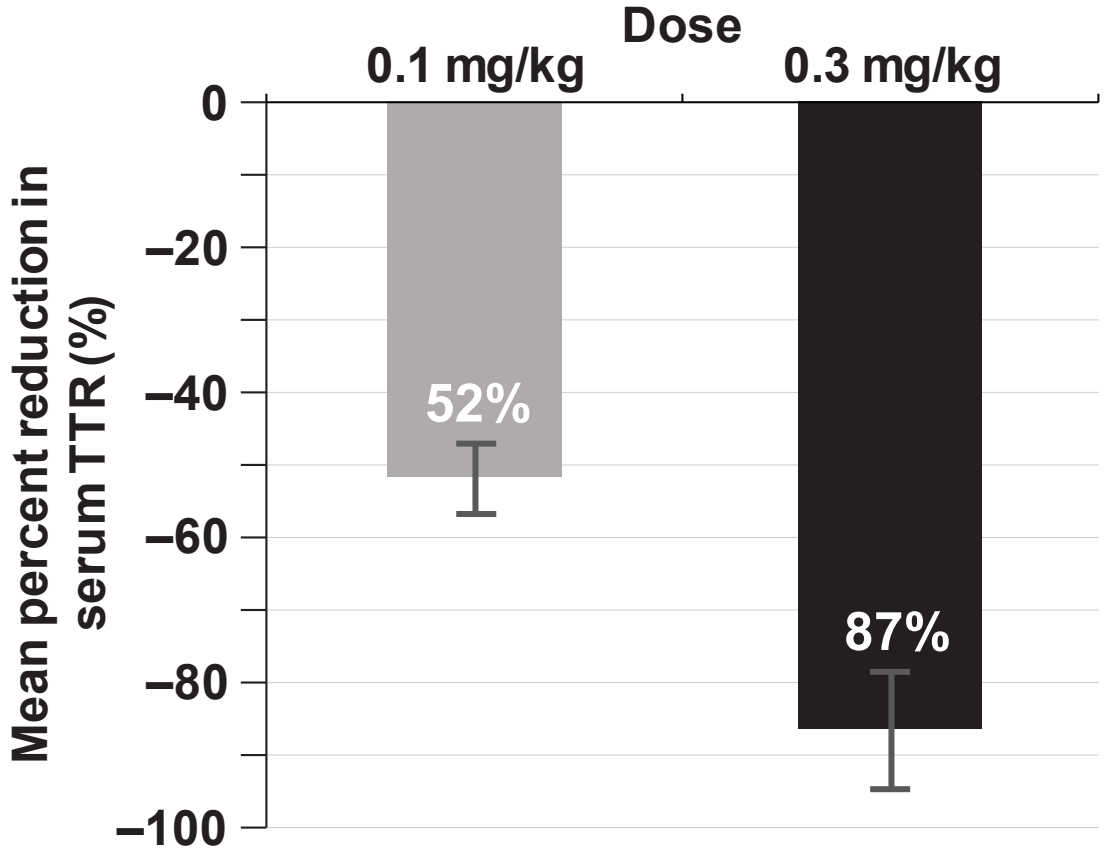
Dose-dependent serum TTR reduction after NTLA-2001





TTR, transthyretin

Average TTR reduction of 87% for 0.3 mg/kg: Predicted to result in clinical benefit for patients



Bars represent standard deviation.
TTR, transthyretin

Conclusions:
In vivo CRISPR/Cas9 editing of the *TTR* gene by NTLA-2001

- A single systemic administration of NTLA-2001 in patients with ATTRv amyloidosis-PN caused a profound reduction in serum TTR protein concentrations
  - Effect of NTLA-2001 was dose-dependent
  - 0.1 mg/kg: 52% mean reduction in TTR (56% maximum)
  - 0.3 mg/kg: 87% mean reduction in TTR (96% maximum)
- NTLA-2001 treatment was generally well tolerated: all acute AEs were of mild severity
- Further dose escalation is ongoing in this First-In-Human study
  - Greater reduction in TTR than provided by currently available agents may be achieved
  - Those greater reductions in TTR are expected to result in improved clinical benefit

- This is the first demonstration of CRISPR-based *in vivo* gene editing in humans
  - ➢ Provides proof-of-concept for a promising new therapeutic strategy

AE, adverse event; ATTRv, hereditary transthyretin amyloidosis; PN, polyneuropathy; TTR, transthyretin

Acknowledgements

- We thank the patients who participated in this trial, and their families
- We thank New Zealand Clinical Research and Richmond Pharmacology for contract research assistance, and Charles River Laboratory, Altasciences, Precision for Medicine, PPD, and QPS for serum TTR ELISA measurements and PK and biomarker tests
- We acknowledge valuable input in the development of NTLA-2001 from:
  - Intellia Therapeutics: Carri Boiselle, James Butler, David Cooke, Tracy DiMezzo, Richard Duncan, Eva Essig, Noah Gardner, Bo Han, Denise Hernandez, Kellie Kolb, John Leonard, Rebecca Lescarbeau, Reynald Lescarbeau, Mark McKee, Nishit Patel, Austin Ricker, Joseph Rissman, Matthew Roy, Andrew Schiermeier, Philipp Schneggenburger, Palak Sharma, Samantha Soukamneuth, and Kathryn Walsh
  - Regeneron Pharmaceuticals: Olivier Harari, Christos Kyratsous, Andrew Murphy, Randy Soltys, and Brian Zambrowicz
- Medical writing support was provided by Ben Caldwell, BSc, ISMPP CMPP™ of Arc, a division of Spirit Medical Communications Group Limited, and funded by Intellia Therapeutics and Regeneron Pharmaceuticals, in accordance with Good Publication Practice 3 (GPP3) guidelines (www.ismpp.org/gpp3)

Agenda

Welcome

Introduction

John Leonard, M.D.
Chief Executive Officer, Intellia Therapeutics

Review of NTLA-2001 Interim Phase 1 Clinical Trial Data

Dr. Julian Gillmore, M.D, Ph.D., FRCP, FRCPath,
Professor of Medicine, National Amyloidosis Centre, UCL Division of Medicine, Royal Free Hospital, U.K.; National Coordinator for Intellia's Phase 1 Study of NTLA-2001 in the U.K.

NTLA-2001 Clinical Development Plans

David Lebwohl, M.D.
Chief Medical Officer, Intellia Therapeutics

Platform Outlook

Laura Sepp-Lorenzino, Ph.D.
Chief Scientific Officer, Intellia Therapeutics

Closing Remarks and Q&A Session

Intellia THERAPEUTICS

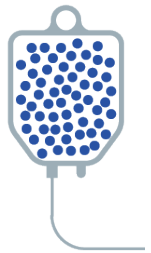
Ongoing Two-Part First-in-Human Study of NTLA-2001

NEXT STEPS IN 2021:

- Complete Part I and initiate Part II
- Share additional clinical data at scientific or medical meeting

Population: Adults with ATTRv with polyneuropathy

Intervention:
Single dose administered via an intravenous (IV) infusion



PART I
Single-Ascending Dose

Up to 4 dose-escalation cohorts

PART II
Single Dose Expansion Cohort

N = 8 subjects

Administer recommended dose selected from Part I

PRIMARY OBJECTIVES

Evaluate safety, tolerability, PK and PD

SECONDARY OBJECTIVES

Evaluate efficacy on clinical measures of neurologic function

*Minimum of 3 subjects per cohort
Clinicaltrials.gov ID: NCT04601051
NIS: Neuropathy Impairment Score **mNIS+7:** modified NIS+7 **PK:** Pharmacokinetics **PD:** Pharmacodynamics

REGENERON

Intellia THERAPEUTICS

Agenda

Welcome

Introduction

John Leonard, M.D.
Chief Executive Officer, Intellia Therapeutics

Review of NTLA-2001 Interim Phase 1 Clinical Trial Data

Dr. Julian Gillmore, M.D, Ph.D., FRCP, FRCPath,
Professor of Medicine, National Amyloidosis Centre, UCL Division of Medicine, Royal Free Hospital, U.K.; National Coordinator for Intellia's Phase 1 Study of NTLA-2001 in the U.K.

NTLA-2001 Clinical Development Plans

David Lebwohl, M.D.
Chief Medical Officer, Intellia Therapeutics

Platform Outlook

Laura Sepp-Lorenzino, Ph.D.
Chief Scientific Officer, Intellia Therapeutics

Closing Remarks and Q&A Session

Intellia THERAPEUTICS

Clinical Validation of LNP Delivery Platform Supports *In Vivo* Pipeline Acceleration

Address diseases with genetically defined targets in the liver

- ☑ Remove a toxic protein via knockout
- ❑ Restore a functional protein via insertion

ATTR, HAE
AATD, Hem A and B, PH, Undisclosed Indications

Enable access to treat diseases across multiple tissue types

Bone Marrow, CNS, Other Tissues

AATD: Alpha-1 Antitrypsin Deficiency **CNS:** Central Nervous System **HAE:** Hereditary Angioedema
Hem A and B: Hemophilia A and B **PH:** Primary Hyperoxaluria`

Intellia THERAPEUTICS

Agenda

Welcome

Introduction

John Leonard, M.D.
Chief Executive Officer, Intellia Therapeutics

Review of NTLA-2001 Interim Phase 1 Clinical Trial Data

Dr. Julian Gillmore, M.D, Ph.D., FRCP, FRCPath,
Professor of Medicine, National Amyloidosis Centre, UCL Division of Medicine, Royal Free Hospital, U.K.; National Coordinator for Intellia's Phase 1 Study of NTLA-2001 in the U.K.

NTLA-2001 Clinical Development Plans

David Lebwohl, M.D.
Chief Medical Officer, Intellia Therapeutics

Platform Outlook

Laura Sepp-Lorenzino, Ph.D.
Chief Scientific Officer, Intellia Therapeutics

Closing Remarks and Q&A Session

Intellia THERAPEUTICS

NTLA-2001 for Transthyretin (ATTR) Amyloidosis

First systemically delivered CRISPR-based therapy to enter clinical development

Impact of NTLA-2001
Interim Clinical Data Readout

Intellia THERAPEUTICS

Executing Against Strategic Priorities and R&D Goals

Clinical Validation

NTLA-2001 for Transthyretin Amyloidosis (ATTR Amyloidosis):

- ☑ Reported positive interim clinical data from ongoing Phase 1 study
- ○ Initiate Part II, a single-dose expansion cohort, in 2021
- ○ Share additional data at medical or scientific meeting in 2021

Pipeline Advancement

NTLA-2002 for Hereditary Angioedema (HAE):

- ☑ Submitted first CTA to initiate Phase 1 study
- ○ Enroll first patient in the Phase 1 study in 2021

NTLA-5001 for Acute Myeloid Leukemia (AML):

- ○ Submit IND in mid-2021

Research Programs:

- ○ Nominate at least 1 new development candidate in 2021
- ○ Nominate first allogeneic development candidate by 1H 2022

Platform Innovation

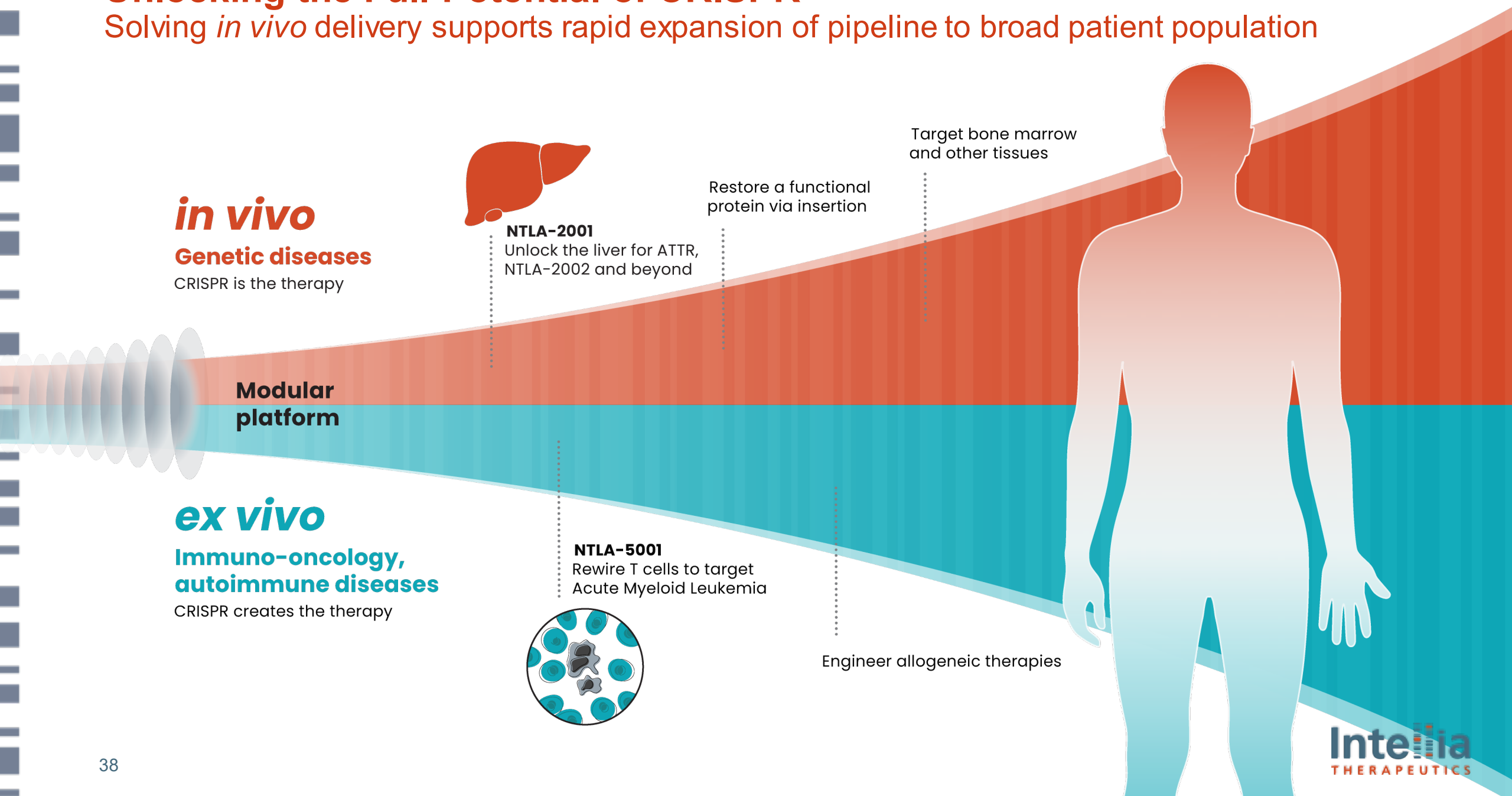
- ☑ Demonstrated preclinical proof-of-concept for *in vivo* editing of bone marrow
- ☑ Presented first preclinical data on Intellia's proprietary base editor

IND: Investigational New Drug or IND-equivalent

Intellia THERAPEUTICS

Unlocking the Full Potential of CRISPR

Solving *in vivo* delivery supports rapid expansion of pipeline to broad patient population



Q&A

NTLA-2001 Interim Phase 1 Clinical Data

Intellia

THERAPEUTICS