Intellia is Leading the Genome Editing Revolution

Bill, living with transthyretin amyloidosis, and his wife, Maura
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: the safety, efficacy and advancement of our clinical programs for NTLA-2001 for the treatment of transthyretin amyloidosis, NTLA-2002 for the treatment of hereditary angioedema, and NTLA-5001 for the treatment of acute myeloid leukemia pursuant to its clinical trial applications (“CTA”) and IND submissions, including the expected timing of data releases, regulatory filings, and the initiation and completion of clinical trials; the advancement of development candidates including NTLA-3001 for the treatment of alpha-1 antitrypsin deficiency (AATD)-associated lung disease; the ability to generate data to initiate clinical trials and the timing of CTA and IND submissions; the expansion of its CRISPR/Cas9 technology and related technologies, including manufacturing and delivery technologies, to advance additional development candidates; the ability to maintain and expand our related intellectual property portfolio, and avoid or acquire rights to valid intellectual property of third parties; the ability to demonstrate our platform’s modularity and replicate or apply results achieved in preclinical studies, including those in our NTLA-2001, NTLA-5001, and NTLA-2002 programs, in any future studies, including human clinical trials; the ability to optimize the impact of our collaborations on our development programs, including, but not limited to, our collaboration with Regeneron Pharmaceuticals, Inc., including our co-development programs for hemophilia A and hemophilia B, our collaboration with Avevcell Therapeutics, Inc., and our other announced collaborations; Regeneron’s ability to successfully co-develop products in the hemophilia A and B programs, and the potential timing and receipt of future milestones and royalties, or profits, as applicable, based on our license, collaboration and, if applicable, co-development agreements with Regeneron, Novartis Institutes for Biomedical Research, Inc., and other collaborators; the timing of regulatory filings and clinical trial execution, including dosing of patients, regarding our development programs; the potential commercial opportunities, including value and market, for our product candidates; our use of capital and other financial results during 2022; and our ability to fund operations beyond the next 24 months.

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 valid third party intellectual property; 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 clinical study results will not be positive; the risk that the results of preclinical studies or clinical studies will not be predictive of future results; and the risk that our collaborations with Regeneron or our other 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 quarterly report on Form 10-Q 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.
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Intellia is Leading the Genome Editing Revolution

Transforming lives of people with severe diseases by developing curative genome editing treatments

Leaders of the Field
First company to demonstrate initial safety and efficacy of in vivo genome editing in a clinical study

Setting the Standard
Extensive characterization for potent and highly specific editing

Unsurpassed Genome Editing Pipeline

Full-Spectrum Strategy
Robust R&D engine to develop in vivo and ex vivo therapies for diseases with high unmet need

Modular Solutions
Focused on building differentiated technology with broad applicability that can be applied to future candidates

Applying Novel Tools
Building an array of editing tools and delivery modalities for therapeutic application

World-class Genome Editing Toolbox
Therapeutic Strategies to Treat Life-Threatening Diseases Have Advanced Over Time

INNOVATION TIMELINE

- Biologics
- RNAi
- Gene Therapy
- Genome Editing
- Small Molecule Drugs

PROTEINS RNA DNA
Power of CRISPR: Nobel-Prize Winning Genome Editing Technology

- Precise and modular approach for editing the genome
- Locates a genetic sequence to make a permanent change
- High level of specificity to make one or multiple edits
- Potential for life-long effect following one-time treatment
- Overcomes key limitations of gene and RNAi therapies
- Provides foundational capabilities for derivative tools
In Vivo Leader: First to Demonstrate Systemic CRISPR Gene Editing in Humans

CRISPR-Cas9 In Vivo Gene Editing for Transthyretin Amyloidosis

Julian D. Gillmore, M.D., Ph.D., Ed Gane, M.B., Ch.B., Jorg Taubel, M.D., Justin Kao, M.B., Ch.B., Marianna Fontana, M.D., Ph.D., Michael L. Maitland, M.D., Ph.D., Jessica Seitzer, B.S., Daniel O’Connell, Ph.D., Kathryn R. Walsh, Ph.D., Kristy Wood, Ph.D., Jonathan Phillips, Ph.D., Yuanxin Xu, M.D., Ph.D., Adam Amaral, B.A., Adam P. Boyd, Ph.D., Jeffrey E. Cehelsky, M.B.A., Mark D. McKee, M.D., Andrew Schiermeier, Ph.D., Olivier Harari, M.B., B.Chir., Ph.D., Andrew Murphy, Ph.D., Christos A. Kyratsous, Ph.D., Brian Zambrowicz, Ph.D., Randy Soltys, Ph.D., David E. Gutstein, M.D., John Leonard, M.D., Laura Sepp-Lorenzino, Ph.D., and David Lebwohl, M.D.
Building a Full-Spectrum Genome Editing Company

CRISPR-based Modular Platform

**EMPLOY NOVEL EDITING AND DELIVERY TOOLS**

- **In Vivo**
  - CRISPR *is* the therapy
  - Fix the target gene
  - Genetic diseases

- **Ex Vivo**
  - CRISPR *creates* the therapy
  - Rewire & redirect cells
  - Immuno-oncology
  - Autoimmune diseases
**2021: Groundbreaking Year for Intellia**

<table>
<thead>
<tr>
<th>In Vivo</th>
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<tbody>
<tr>
<td><strong>NTLA-2001 ATTR</strong></td>
<td>✓ First-ever clinical data supporting initial safety and efficacy of <em>in vivo</em> CRISPR genome editing in humans</td>
</tr>
<tr>
<td><strong>NTLA-2002 HAE</strong></td>
<td>✓ Dosed first patient with NTLA-2002 in first-in-human study</td>
</tr>
</tbody>
</table>
| New Development Candidates | ✓ Nominated NTLA-3001 for alpha-1 antitrypsin deficiency (AATD)  
✓ Nominated candidate for *Factor 9* insertion program for hemophilia B in collaboration with Regeneron |

<table>
<thead>
<tr>
<th>Ex Vivo</th>
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<tbody>
<tr>
<td><strong>NTLA-5001 AML</strong></td>
<td>✓ Initiated patient screening in first-in-human study</td>
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<thead>
<tr>
<th>Platform Innovation</th>
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</table>
| Research and Platform Advancements | ✓ Demonstrated preclinical proof-of-concept for *in vivo* editing of bone marrow  
✓ Unveiled proprietary base editor with first preclinical data  
✓ Highlighted Intellia’s differentiated allogeneic platform compared to current approaches |
## 2022 and Beyond: Key Expected Milestones

### In Vivo

| NTLA-2001 ATTR | Present additional clinical data from Phase 1 study in ATTRv-PN patients in Q1 2022 |
| NTLA-2002 HAE | Present interim data from Phase 1/2 study in 2H 2022 |
| NTLA-3001 AATD | Plan to file an IND or IND-equivalent in 2023 |

### Ex Vivo

| NTLA-5001 AML | Enroll patients in Phase 1/2a study in 2022 |

### Platform Innovation

| Research and Platform Advancements | Advance at least 2 new *in vivo* development candidates by end of 2022 |
| | Nominate first wholly owned allogeneic *ex vivo* development candidate by 1H 2022 |
| | Advance additional novel platform capabilities in 2022 |
# Development Pipeline Fueled by Robust Research Engine

## In Vivo: CRISPR is the therapy

<table>
<thead>
<tr>
<th>PROGRAM</th>
<th>APPROACH</th>
<th>Research</th>
<th>IND-Enabling</th>
<th>Early-Stage Clinical</th>
<th>Late-Stage Clinical</th>
<th>PARTNER</th>
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</thead>
<tbody>
<tr>
<td>NTLA-2001: Transthyretin Amyloidosis</td>
<td>Knockout</td>
<td></td>
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<td>REGENERON</td>
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<tr>
<td>NTLA-2002: Hereditary Angioedema</td>
<td>Knockout</td>
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<td>Intellia Therapeutics</td>
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<tr>
<td>NTLA-3001: AATD-Lung Disease</td>
<td>Insertion</td>
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<td>Intellia Therapeutics</td>
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<td>Hemophilia B</td>
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<td>Hemophilia A</td>
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<td>Research Programs</td>
<td>Knockout, Insertion, Consecutive Edits</td>
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<td>Intellia Therapeutics</td>
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<td>Research Programs</td>
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<td>Intellia Therapeutics</td>
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## Ex Vivo: CRISPR creates the therapy

<table>
<thead>
<tr>
<th>PROGRAM</th>
<th>APPROACH</th>
<th>Research</th>
<th>IND-Enabling</th>
<th>Early-Stage Clinical</th>
<th>Late-Stage Clinical</th>
<th>PARTNER</th>
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<tbody>
<tr>
<td>OTQ923 / HIX763: Sickle Cell Disease</td>
<td>HSC</td>
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<td></td>
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<td>Intellia Therapeutics &amp; NOVARTIS</td>
</tr>
<tr>
<td>NTLA-5001: Acute Myeloid Leukemia</td>
<td>WT1-TCR</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Intellia Therapeutics</td>
</tr>
<tr>
<td>Solid Tumors</td>
<td>WT1-TCR</td>
<td></td>
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<td>Intellia Therapeutics</td>
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<tr>
<td>Allo Undisclosed</td>
<td>Undisclosed</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Intellia Therapeutics</td>
</tr>
<tr>
<td>Other Novartis Programs</td>
<td>CAR-T, HSC, OSC</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>NOVARTIS</td>
</tr>
</tbody>
</table>

*Lead development and commercial party  **Rights to certain in vivo targets  ***Milestones & royalties only

AATD: Alpha-1 Antitrypsin Deficiency  CAR-T: Chimeric Antigen Receptor T Cells  HSC: Hematopoietic Stem Cells  OSC: Ocular Stem Cells  TCR: T Cell Receptor
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Intellia Investment Overview

In Vivo Portfolio

Ex Vivo Portfolio

Appendix
In Vivo

CRISPR is the therapy

GENETIC DISEASES

Strategic Advantages:

Potential curative therapy from single dose

Systemic non-viral delivery of CRISPR/Cas9 provides transient expression and potential safety advantages

Permanent gain of function with targeted gene insertion

Capable of delivering to multiple tissue types for various therapeutic applications

LNP: Lipid Nanoparticle
Modular Delivery Platform Enables Rapid and Reproducible Path to Clinical Development

LNP Delivery System:

- gRNA identifies genetic target
- TTR gRNA
- KLKB1 gRNA
- Target-specific gRNA
- Cas9 mRNA
- AAAA

Key Advantages of LNP Delivery

- Clinically-proven delivery to liver
- Large cargo capacity
- Transient expression
- Biodegradable
- Low immunogenicity
- Well-tolerated
- Redosing capability
- Scalable synthetic manufacturing
- Tunable to other tissues
NTLA-2001 for Transthyretin (ATTR) Amyloidosis

- Caused by accumulation of misfolded transthyretin (TTR) protein, which affects nerves, heart, kidneys and eyes
- Chronic dosing is required with current treatments

**OUR APPROACH**

Knock out TTR gene with a single dose
- Reduce wild-type and mutant TTR protein
- Aims to address polyneuropathy and cardiomyopathy

**KEY ADVANTAGES**

- Potential to halt and reverse disease
- Potential “one-and-done” treatment
- Expect lifelong, stable TTR reduction

---

50K
ATTRv patients worldwide¹

≈200-500K
ATTRwt patients worldwide²

¹Ann Med. 2015; 47(8): 625–638. ²Compiled from various sources  
ATTRv: Hereditary ATTR  ATTRwt: Wild-Type ATTR
Sustained >95% Serum TTR Protein Reduction After a Single Dose in NHPs

Therapeutically relevant serum TTR knockdown
NTLA-2001 Expanded Phase 1 Study

Two-part, open-label, multi-center study in adults with hereditary ATTR with polyneuropathy (ATTRv-PN) or ATTR amyloidosis with cardiomyopathy (ATTR-CM)

**PRIMARY OBJECTIVES**
- Evaluate safety, tolerability, PK and PD
  - Measure serum TTR levels

**SECONDARY OBJECTIVES**
- Evaluate efficacy on clinical measures of:
  - Neurologic function in subjects with ATTRv-PN
  - Cardiac disease in subjects with ATTR-CM

Potential to advance toward a pivotal trial for NTLA-2001 based on Phase 1 safety and efficacy data

**PART I**
- Total Enrollment: Up to 74 patients
- Intervention: Single dose administered via an intravenous (IV) infusion
- Up to 38 ATTRv-PN patients
- Single-Dosing Dose Escalation Cohorts (Up to 4)
- Up to 36 ATTR-CM patients
- Single-Dosing Dose Escalation Cohorts (Up to 2)

**PART II**
- Single Dose Expansion Cohort
- Administer selected dose from Part I
- Single Dose Expansion Cohort

Clinicaltrials.gov ID: NCT04601051
- PK: Pharmacokinetics
- PD: Pharmacodynamics
NTLA-2001 Phase 1 Study: Polyneuropathy Arm

Hereditary transthyretin amyloidosis with polyneuropathy (ATTRv-PN)

**Primary Objectives**
Evaluate safety, tolerability, PK and PD

- Measure serum TTR levels

**Secondary Objectives**
Evaluate efficacy on clinical measures of neurologic function

- Neuropathic impairment endpoints include NIS (Part 1 and 2) and mNIS+7 (Part 2 only)

**Total Enrollment:**
Up to 38 patients, age 18 to 80 years

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

**PART I**
Single-Ascending Dose

- N = Up to 30 subjects*
- Up to 4 dose-escalation cohorts

**PART II**
Single Dose Expansion Cohort

- N = 8 subjects
- Administer optimal dose selected from Part I

*Minimum of 3 subjects per cohort

NIS: Neuropathy Impairment Score
mNIS+7: modified NIS+7
PK: Pharmacokinetics
PD: Pharmacodynamics

Clinicaltrials.gov ID: NCT04601051
NTLA-2001 Phase 1 Study: Cardiomyopathy Arm

Hereditary transthyretin amyloidosis with cardiomyopathy (ATTRv-CM) or wild-type cardiomyopathy (ATTRwt-CM), NYHA Class I - III

**Total Enrollment:**
Up to 36 patients, age 18 to 90 years

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

---

**PART I**
Single-Ascending Dose

N = Up to 24 subjects*

Up to 2 dose-escalation cohorts

**PART II**
Single Dose Expansion Cohort

N = 12 subjects

Administer selected dose from Part I

---

**PRIMARY OBJECTIVES**
Evaluate safety, tolerability, PK and PD
- Measure serum TTR levels

---

**SECONDARY OBJECTIVES**
Evaluate efficacy on clinical measures of cardiac disease
- Cardiac imaging, biomarkers, cardiopulmonary exercise test, 6MWT

---

*Minimum of 3 subjects per cohort
NYHA: New York Heart Association
PK: Pharmacokinetics
PD: Pharmacodynamics
6MWT: 6 Minute Walk Test
NTLA-2001 Generally Well Tolerated in Acute Phase (N=6) by Day 28: All AEs Grade 1 with No Serious AEs

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

No liver findings or coagulopathy based on laboratory testing.

Data disclosed on June 26, 2021 at 2021 Peripheral Nerve Society (PNS) Annual Meeting
AE: Adverse Event  
TEAE: Treatment-Emergent Adverse Event
This slide includes data for investigational products not yet approved by regulatory authorities
Landmark Clinical Data Show Deep, Dose-Dependent Serum TTR Reduction After Single Dose of NTLA-2001

Data disclosed on June 26, 2021 at 2021 PNS Annual Meeting
This slide includes data for investigational products not yet approved by regulatory authorities
NTLA-2002 for Hereditary Angioedema (HAE)

• Genetic disease characterized by recurring, severe and unpredictable swelling in various parts of the body
• Chronic dosing is required with current treatments

OUR APPROACH

Knock out KLKB1 gene with a single dose
• Reduce kallikrein activity to prevent attacks

KEY ADVANTAGES

• Potential “one-and-done” treatment
• Expect extensive and continuous reduction in kallikrein activity
  o Intended to minimize the risk of breakthrough attacks
• Potential to eliminate significant treatment burden

~7-14 days
Average frequency of attacks for untreated patients¹

~1 in 50,000
HAE patients worldwide¹

Achieved Sustained Therapeutically Relevant Kallikrein Activity Reduction After a Single Dose in NHPs

Kallikrein Activity Reduction

- Control
- Dose Level 1 (n=3)
- Dose Level 2 (n=3)
- Dose Level 3 (n=3)

Therapeutically relevant impact on attack rate*

Single Dose

*Banerji et al., NEJM, 2017
NTLA-2002 Phase 1/2 Trial Design

International, multi-center study to assess safety, tolerability, PK, PD and effect of NTLA-2002 on attacks in adults with Type I or Type II HAE

**KEY ENDPOINTS**
- Evaluate safety and tolerability
- Change in plasma kallikrein protein and activity levels
- Change in attack rates (Phase 2)

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

**PHASE 1**
Open-Label, Single-Ascending Dose
- 3 dose-escalation cohorts*

**PHASE 2**
Expansion study to confirm recommended dose
- Dose 1 (N=10)
- Dose 2** (N=10)
- Placebo Arm (N=5)

**Total Enrollment:**
Up to 55 patients, age 18 and older

Clinicaltrials.gov ID: NCT05120830
PK: Pharmacokinetics  PD: Pharmacodynamics
*3 to 6 subjects per cohort; up to 2 additional cohorts, if necessary  **Optional cohort
Beyond Gene Inactivation, Intellia is Also Advancing Targeted Insertion Programs

CRISPR-Enabled Targeted Insertion Approach Offers Significant Advantages Over Alternate Gene Therapy Approaches

High Levels of Protein Expression

Potential to Revolutionize Gene Replacement

Durable Protein Expression
Insertion Technology Enables Production of High Levels of Therapeutic Protein

Precisely Create Insertion Site

Deliver Insertion Template

Targeted, stable gene insertion in the albumin locus
**NTLA-3001 for Alpha-1 Antitrypsin Deficiency (AATD)**

Genetic disorder leading to progressive lung disease; with liver manifestation in 10-15% of patients

---

**OUR APPROACH**

- **Targeted insertion of a functional SERPINA1 gene**
  - Continuous expression of A1AT protein at normal levels
  - Address AATD-associated lung disease

**KEY ADVANTAGES**

- Designed as a single-dose treatment
- Aims to achieve normal human levels of A1AT protein
- Potential to eliminate weekly intravenous infusions of augmentation therapy

---

AATD patients*

> 60K in the U.S.*

~250K globally*  

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1[https://www.genome.gov/Genetic-Disorders/Alpha-1-Antitrypsin-Deficiency](https://www.genome.gov/Genetic-Disorders/Alpha-1-Antitrypsin-Deficiency)

2Brantly M. *Clin Chem*. 2006; 52:2180-2181


*In severe AATD patients defined as individuals with Pi*ZZ* genotype
Durable Physiologic Levels of hA1AT Maintained Through One Year in NHP

Human A1AT (hA1AT) Expression

Circulating hA1AT (µg/mL)

Weeks Post Insertion

Normal range

Insertion (n=3)

Buffer Control (n=3)

Therapeutically relevant

1Stoller & Aboussouan The Lancet, 2005
Clinical Validation of LNP Delivery Platform Supports *In Vivo* Pipeline Acceleration

**First Wave of Programs**

Unlock Liver Targets

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 B**
**Hem A, PH, Undisclosed Indications**

Unlock Full Potential

Targets Across Multiple Tissues

Enable access to treat diseases across multiple tissue types

- Bone Marrow, CNS, Other Tissues
Ex Vivo

CRISPR creates the therapy

IMMUNO-ONCOLOGY / AUTOIMMUNE DISEASES

Strategic Advantages:

Utilizing proprietary CRISPR engineering platform to create differentiated cell therapies for IO and AI diseases

Targeting modalities, such as TCR, with broad potential in multiple indications

Focused on reproducing natural cell physiology for potential improvements to safety and efficacy in immuno-oncology
Proprietary Engineering Platform to Power Next-Generation Engineered Cell Therapies

**LNP-BASED CELL ENGINEERING PLATFORM**

- Highly efficient sequential editing
- Optimal cell performance
- Scalable manufacturing process

**ENABLES VERSATILE SOLUTIONS BY “MIXING AND MATCHING” INCLUDING:**

**Cell Type**
- HSCs, T cells
- NK cells, Macrophages

**Targeting Modality**
- TCRs
  - CAR-Ts, Universal CARs

**Rewiring Instructions**
- Immune-enhancing edits
  - Novel targets

**Abbreviations:**
- **NK**: Natural Killer
- **TCR**: T Cell Receptor
### Differentiated Approach to Cell Therapy Genome Engineering

**Gene Editing Approach**

<table>
<thead>
<tr>
<th>Delivery</th>
<th>Lipid Nanoparticle</th>
<th>Electroporation</th>
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<tr>
<td>Editing Mode</td>
<td>Sequential</td>
<td>Simultaneous</td>
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<td>Knockout (KO)</td>
<td>Cleavase or Base Editor</td>
<td>Cleavase</td>
<td>Base Editor</td>
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<tr>
<td>Insertion</td>
<td>CRISPR insertion</td>
<td>Lenti/Retroviruses</td>
<td>Lenti/Retroviruses</td>
</tr>
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</table>

#### Key Questions From Preclinical Data

- Minimize random DSB? ✓
- Minimize random insertion? ✓
- Minimize genotoxicity risk? ✓

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"Slide based on preclinical data disclosed by Intellia. Intellia’s cell product to be further explored in additional preclinical and clinical studies."
Sequential Editing with LNP Approach Minimizes Translocations While Retaining Robust Cell Viability and Expansion

ddPCR assay to detect TRAC-TRBC translocations

% TRAC-TRBC translocated cells

LNP Approach: Cell Expansion at D10

Fold Expansion

No Edit, Electroporation, LNP
NTLA-5001 for Acute Myeloid Leukemia (AML)

Most common acute leukemia in adults\(^1\)

**OUR APPROACH**

Engineer TCR-T cells directed against Wilms’ Tumor Type 1 (WT1) to specifically kill AML blasts

**KEY ADVANTAGES**

- Potential to address all mutational subtypes of AML
- Low WT1 expression in normal tissues for improved safety
- TCR sourced from healthy donor T cells intended to minimize immune toxicity

\(~20K\) New cases in the U.S. in 2021\(^1\)

\(> 40K\) New cases in the 7 Major Markets in 2020\(^2\)

\(< 30\%\) 5-year overall survival\(^1\)

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\(^1\)NIH SEER Cancer Stat Facts: Leukemia – Acute Myeloid Leukemia (AML)

\(^2\)GlobalData EpiCast Report: Acute Myeloid Leukemia June 2021, 7MM: Seven Major Markets (includes U.S.)
NTLA-5001: Potential Best-in-Class Engineered T Cell Therapy For AML

Inserts a **natural, high-avidity TCR** to replace native TCR for upgraded safety profile
- Activates both cytotoxic and helper T cells

Specifically **targets Wilms’ Tumor 1 (WT1)**, an antigen overexpressed in >90% of AML blasts$^1$
- Recognizes an epitope (VLD$^2$) presented broadly by AML blasts with the HLA-A*02:01 allele$^3$

Modified by **proprietary cell engineering technology** for optimized cell health and function

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$^1$Cilloni et al., *J Clin Oncol*, 2009
$^2$VLD is the WT1$_{137-45}$ epitope VLDFAPPGA
$^3$Refer to http://www.allelefrequencies.net for HLA frequency data
In collaboration with IRCCS Ospedale San Raffaele
NTLA-5001: Robust Anti-Tumor Efficacy Observed Against Patient-Derived AML Blasts in Mouse Model

![Graph showing pAML cells/µL of blood over days post AML infusion for different treatment groups.](image)

- **pAML Alone**
- **MART1-TCR (Control, Electroporation Process)**
- **WT1-TCR (Electroporation Process)**
- **WT1-TCR (LNP Process)**

Intellia’s lead TCR-T cells
NTLA-5001: Uniform Expression of Therapeutic TCR for Potent Tumor Targeting

Rapid Cell Engineering: 10 Days

- **THAW** T cells
- **ACTIVATE** T cells
- **REMOVE** endogenous TCR
- **INSERT** WT1 TCR in locus
- **EXPAND** rapidly
- **HARVEST** and **FREEZE**

Sequential KO of TRAC and TRBC

Apheresis & T Cell Cryopreservation

Thaw and Re-infusion
CRISPR Engineering Overcomes Key Challenges of Traditional TCR Approaches

**Traditional tgTCR Addition**
- Mixed TCRs
- Heterogenous Cell Product

**CRISPR/Cas9 tgTCR Replacement**
- tgTCRs Only
- Homogenous Cell Product

**Removal of Endogenous TCR Prevents Mispairing**

<table>
<thead>
<tr>
<th>TCR</th>
<th>% Cells with Mispaired TCRs</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>60</td>
</tr>
<tr>
<td>B</td>
<td>40</td>
</tr>
<tr>
<td>C</td>
<td>20</td>
</tr>
<tr>
<td>D</td>
<td>0</td>
</tr>
</tbody>
</table>

- TRAC KO Only + Insertion
- Intellia’s Approach (TRAC and TRBC KO + Insertion)

tgTCR: Transgenic Therapeutic TCR
NTLA-5001 Phase 1/2a Trial Design

Open-label, multi-center study of NTLA-5001, a WT1-directed TCR immunotherapy, in adults with AML

**Total Enrollment:**
Up to 54 patients, age ≥18 years

**Key Inclusion Criteria:**
- Relapsed/refractory AML after one or more therapies
- Post transplant patients are eligible
- HLA-A*02:01 positive

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

**PHASE 1**
Dose Escalation
Two-ascending arms: Up to 3 cohorts*

- **ARM 1:** Lower Disease Burden
- **ARM 2:** Higher Disease Burden

**PHASE 2**
Expansion Cohorts
To confirm recommended dose from each arm of Phase 1

- Dose 1 (N=9)
- Dose 2 (N=9)

**KEY ENDPOINTS**
- Evaluate safety and tolerability
- Characterize cell kinetics of NTLA-5001
- Determine anti-tumor activity

*3-6 subjects per cohort

Clinicaltrials.gov ID: NCT05066165

**Lower disease burden:** Patients with less than 5% AML blasts in bone marrow

**Higher disease burden:** Patients with relapsed/refractory disease with greater than or equal to 5% AML blasts in bone marrow
Ex Vivo Pipeline Expansion Strategy

First Wave of Programs

**Address a variety of cancers**
- Target new antigens with TCR identification and cell engineering platform
- Allogeneic solution

**AML, Undisclosed Indications**

Unlock Full Potential

**Advance cell therapy for cancer and autoimmune diseases**
- Novel immune-enhancing edits

**Prioritize diseases with significant unmet need**

Hematological and Solid Tumors

Immuno-oncology
Unlocking the Full Potential of CRISPR
Solving in vivo delivery supports rapid expansion of pipeline to broad patient population

**in vivo**
Genetic diseases
CRISPR is the therapy

- **NTLA-2001**
  Unlock the liver for ATTR, NTLA-2002 for HAE and beyond

- **NTLA-3001 and Factor IX**
  Restore a functional protein via insertion for AATD and Hem B

Target bone marrow and other tissues

**ex vivo**
Immuno-oncology, autoimmune diseases
CRISPR creates the therapy

- **NTLA-5001**
  Rewire T cells to target Acute Myeloid Leukemia

Engineer allogeneic therapies
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In Vivo Portfolio

Ex Vivo Portfolio

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Intellia’s Genome Editing Toolbox

Persistence of In Vivo Edits

In Vivo Editing of Hematopoietic Stem Cells

LNP-Based Editing of T Cells

Intellia’s Allogeneic Solution

Intellia’s Proprietary Base Editor

Platform: Identifying Potent and Highly Specific Guide RNAs

Strategic Collaborations
Intellia’s Genome Editing Toolbox
World-Class Genome Editing Platform Allows for Unsurpassed Capabilities

<table>
<thead>
<tr>
<th>Proprietary CRISPR-based Modular Platform</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Editing Tools</strong></td>
</tr>
<tr>
<td>CRISPR/Cas9</td>
</tr>
<tr>
<td>Base editor</td>
</tr>
<tr>
<td>Additional enzymes</td>
</tr>
<tr>
<td><strong>Delivery Tools</strong></td>
</tr>
<tr>
<td>LNP</td>
</tr>
<tr>
<td>AAV</td>
</tr>
<tr>
<td>Additional modalities</td>
</tr>
</tbody>
</table>

ENABLES SELECTING THE BEST TOOLS FOR EACH THERAPEUTIC APPLICATION:

- Applies to *in vivo or ex vivo* application
- Capable of achieving any editing strategy
  - Precise knockout and targeted insertions
  - Multiplicity of edits
  - Single nucleotide modifications
Persistence of *In Vivo* Edits
Partial Hepatectomy Model for Investigating Persistence of Knockout Genome Editing

NHP studies demonstrate sustained KO editing and target TTR protein reduction carried through regular cell turnover for 12 months

Key Question: Can editing be carried through tissue regeneration following partial hepatectomy and accelerated cell division?
Protein Reduction Remains Unchanged Following Murine Liver Regeneration

*Similar results obtained for control and LNP when sham surgery was performed

1Next generation sequencing (NGS) analysis to evaluate the frequency of insertion and deletion events (edits).

TTR gene editing rate similarly remains unchanged post-PHx by NGS analysis

PHx: Partial Hepatectomy

*Similar results obtained for control and LNP when sham surgery was performed

1Next generation sequencing (NGS) analysis to evaluate the frequency of insertion and deletion events (edits).
Partial Hepatectomy Model for Investigating Persistence of Insertion Genome Editing

Rodent studies show sustained FIX insertion editing through 12 months, demonstrating that editing is carried through normal cell turnover.

**Key Question:** Can insertion editing be carried through tissue regeneration following partial hepatectomy?
Persistent Protein Levels Post-PHx from Targeted Gene Insertion in Murine Model, in Comparison to Significant Loss of Protein Expression with Gene Therapy

Correlating editing rate similarly remains unchanged post-PHx by NGS analysis

Next generation sequencing (NGS) analysis to evaluate the frequency of insertion and deletion events (edits).

Episomal AAV Expression
Targeted Insertion via CRISPR/Cas9 Gene Editing

~95% Loss with Episomal Expression
~85% Loss with Episomal Expression

Circulating hFIX levels (normalized to pre-PHx levels)

<table>
<thead>
<tr>
<th>Event</th>
<th>Circulating hFIX levels</th>
</tr>
</thead>
<tbody>
<tr>
<td>LNP + AAV dose (Day 0)</td>
<td></td>
</tr>
<tr>
<td>Post-Edit Pre-PHx (Day 36)</td>
<td>100</td>
</tr>
<tr>
<td>PHx (Day 40)</td>
<td></td>
</tr>
<tr>
<td>Post-Edit, Post-PHx (Day 54)</td>
<td>150</td>
</tr>
<tr>
<td>Post-Edit, Necropsy (Day 85)</td>
<td>50</td>
</tr>
</tbody>
</table>

1Next generation sequencing (NGS) analysis to evaluate the frequency of insertion and deletion events (edits).
In Vivo Editing of 
Hematopoietic Stem Cells
Ex vivo SCD gene editing still has significant limitations

Complex cell manufacturing process

- Mobilize and harvest cells
- Myeloablate the patient
- Infuse edited cells & support patient until cells engraft

Conditioning regimen toxicity

- Immunosuppression for > 1 month, predisposing to infection
- Risk of malignancy from chemotherapy drugs, especially leukemia
- Risk of infertility

Implications

- Ex vivo gene editing will be limited to highly selected SCD patients with severe disease
- Treatment complexity will limit access for patients in resource-poor settings
**In vivo non-viral SCD gene editing could overcome these limitations**

### Desired features of *in vivo* approach

- Provides clinically meaningful, durable HSC editing
- Allows for multidosing to reach therapeutic target
- Preserves regenerative potential of edited cells
- Translatable to human HSC population

### Potential improved safety and accessibility

- Avoids myeloablation and associated risks of immunosuppression, malignancy and infertility
  - Approach could become mainstream therapy for SCD
- Avoids need for complex cell manufacturing or extensive supportive care post-treatment
  - Treatment simplicity could expand access to patients in resource-poor settings

---

**Simplified process**

Lipid Nanoparticle (LNP) → Infusion of LNP → Infusion
Editing HSCs in vivo requires LNPs with bone marrow tropism

- LNPs designed, formulated and tested in vivo to identify compositions with enhanced delivery to bone marrow and HSCs

Liver-tropic LNP

Bone marrow-tropic LNP
Editing of mouse bone marrow and HSCs is durable through at least one year

- Editing was similar across all time points assessed, in both whole bone marrow and HSCs populations
- Results highlight the potential for a single-course, long-lasting therapy

Note: Guide “B” used in this experiment
Editing of mouse bone marrow and HSCs increases with multidosing

- Non-immunogenic LNP delivery platform may enable stepwise “treat-to-target” approach

![Bar chart showing editing levels with different doses](chart.png)

- **Lin-Sca-1+c-Kit+CD34-Flk2-** cell population


*Level predicted to be curative for SCD*

Note: Guide “B” used in this experiment
LNP-Based Editing of T Cells
LNP-Based Cell Engineering Technology Optimizes Cell Health and Function

DNA Damage
γ-H2AX marker

Untreated | Electroporation (EP) | Intellia’s Process

Cell Expansion

Re-stimulation Stress Test

LNP approach to editing T cells
- Enables sequential editing
- Reduces safety risks from unwanted breaks caused by EP
- Produces cells with high expansion and performance
Multiplex CRISPR/Cas9 T Cell Editing: 5 Sequential Edits with 2 Insertions

Dual site-specific insertion strategy enables co-expression of CAR/TCR construct and immune enhancing transgene

% Editing Across Loci

- >80% of cells have insertion of both the TCR and GFP transgene
- Cells retained high viability and complete editing of 3 other KO targets
- Modular platform for insertion of T cell supporting transgenes
Intellia’s Allogeneic Solution
Three Immune Concerns Must Be Addressed by Allogeneic Cell Therapies

1. **Graft versus host disease (GvHD)**
   - T cell receptor (TCR) from allogeneic T cells recognizes and kills recipient (host) cells.
   - Largely solved with knockout (KO) of endogenous TCR

2. **Rejection via host T cells**
   - Human Leukocyte Antigen (HLA) molecules must match between donor and recipient to prevent rejection from:
     - Host CD8 (HLA class I) T cells
     - Host CD4 (HLA class II) T cells

3. **Rejection via host natural killer (NK) cells**
   - NK cells will attack cells that lack HLA-I expression or have low HLA-I
   - No validated solution yet

**Patient NK cell activation**

**Patient T cell**

**TCR**

**HLA class I**

**HLA class II**

**Low or no HLA class I**

**T cell Therapy**
### Immune Concerns Unaddressed by Current Allogeneic Solutions

<table>
<thead>
<tr>
<th>Approach</th>
<th>Employ intense lymphodepletion regimen</th>
<th>Knockout (KO) HLA-I (B2M)</th>
<th>KO HLA-I &amp; express NK inhibitor (HLA-E)</th>
<th>KO HLA-II &amp; Receptor X*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avoid rejection of cell therapy by host CD8 T cells</td>
<td>✔</td>
<td>✔</td>
<td>✔</td>
<td>✔</td>
</tr>
<tr>
<td>Avoid rejection of cell therapy by host CD4 T cells</td>
<td>✔</td>
<td>✗</td>
<td>✗</td>
<td>✔</td>
</tr>
<tr>
<td>Avoid rejection of cell therapy by host NK cells</td>
<td>✔</td>
<td>✗</td>
<td>✗</td>
<td>✔</td>
</tr>
<tr>
<td>Avoid profound immunosuppression</td>
<td>✗</td>
<td>✔</td>
<td>✔</td>
<td>✔</td>
</tr>
</tbody>
</table>

*Receptor X: Undisclosed target

**B2M:** Beta-2-microglobulin  
**HLA-E:** Human leukocyte antigen class E  
Slide based on preclinical data disclosed by Intellia. Intellia’s cell product to be further explored in additional preclinical and clinical studies.
Intellia’s Differentiated Allogeneic Approach Aims to Address All Three Immune Concerns

**Potential Key Advantages**

- Approach is applicable to CAR and TCR
- Solve for host NK and T cell rejection
- Avoid long-term immunosuppression

---

1. **GvHD**
   - KO endogenous TCR and insert a target moiety (CAR or TCR)

2. **T cell-mediated rejection**
   - KO HLA class II

3. **Host NKs**
   - KO Receptor X*
Intellia’s Differentiated Allogeneic Approach Aims to Address Immune Requirements

1. **GvHD**
   - KO endogenous TCR and insert a target moiety (CAR or TCR)

2. **Prevent CD8+ and CD4+ T cell-mediated rejection**
   - KO HLA class II

3. **Prevent rejection by host NK cells**
   - KO Receptor X*

Applicable to CAR and TCR  ●  Solve for host NK and T cell rejection  ●  Avoid long-term immunosuppression

---

*Receptor X: Undisclosed target

---

**Allo TCR-T Cells (4 KOs + Insertion)**

- ~80% final yield
- 98% viability
- ~280X expansion (N=3)
Allo TCR-T Cells Resist NK Cell Killing for at Least 90 Days *In Vivo*

**B2M Knockout T cells**

* >90% B2M KO T cells killed by NKs within 24h

**Signal from Live B2M KO T Cells**

- Mock mice
- NK engrafted mice

**Allo TCR-T Cells**

* Minimal Allo T cell rejection in the presence of NK cells

**Signal from Live Allo TCR-T Cells**

- Mock mice
- NK engrafted mice

**Day -28**

Mice engrafted with human NK cells or mock treated

Luciferase+ T cells

Days 0-90

IVIS imaging to detect Luciferase+ T cell signal
Allo T Cells Have Comparable Tumor Cell Killing Activity to Autologous T Cells

*In vitro* T cell cytotoxicity assay

**Allo CAR-T Cells**

**Allo TCR-T Cells**

Readout: Tumor cell lysis

In vitro T cell cytotoxicity assay

T cells + Tumor cells

% Tumor Cell Lysis

Log[T Cells:Tumor Cells]

% Tumor Cell Lysis

Log[T Cells:Tumor Cells]
Intellia’s Proprietary Base Editor
Intellia’s Base Editor is Equipotent to Cas9 for Ex Vivo Editing

Intellia's base editor is highly active with similar activity to Cas9 cleavase

>700 constructs screened for potency
85% of guides gave >90% editing
36% of guides gave >95% C to T purity

Pure C>T edits
(without indels)

Cas9

Base Editor

Intellia’s Base Editor is Equipotent to Cas9 for Ex Vivo Editing

Intellia’s base editor is highly active with similar activity to Cas9 cleavase

>700 constructs screened for potency
85% of guides gave >90% editing
36% of guides gave >95% C to T purity

Pure C>T edits
(without indels)
Simultaneous Knockout with Base Editing **Does Not** Lead to Translocations

1. **Isolate** primary T cells

2. **Deliver** base editor + 4 sgRNAs

3. **Evaluate** editing, receptor KO and translocations

### On-target editing (amplicon sequencing)

- **TRAC**: 98.1%
- **TRBC1**: 95.9%
- **TRBC2**: 96.0%
- **Gene X**: 98.1%
- **Gene Y**: 92.9%

### Receptor KO (flow cytometry)

- **CD3**: 97.3%
- **Gene X**: 97.3%
- **Gene Y**: 93.5%

### Interchromosomal Translocations

- **Untreated**
- **Base Editor**

<table>
<thead>
<tr>
<th>Locus tested</th>
<th>Translocation (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TRAC</td>
<td>ns</td>
</tr>
<tr>
<td>Gene X</td>
<td>ns</td>
</tr>
<tr>
<td>Gene Y</td>
<td>ns</td>
</tr>
</tbody>
</table>

**Intellia Therapeutics**
Platform: Identifying Potent and Highly Specific Guide RNAs
Comprehensive gRNA Specificity Assessment: Off-Target Workflow

1: Discovery of Potential Off-Target Edits

- Computational prediction
- SITE-Seq
- Genomic DNA digest
- GUIDE-seq
- Cell-based DNA repair

Aggregate ALL potential off-target genomic loci

2: Validation of Off-Target Edits in Cells

- Multiplex panel for NGS
- Targeted Amp-Seq
- NGS follow-up

Ex Vivo
- Cell drug product

In Vivo
- Edit cells in vitro using drug product formulation
- Therapeutically relevant human primary cell type(s) (2 donors)
- Dose range to exceed projected therapeutic exposure (>10X)
Limited Overlap in Discovered Off-Target Loci by Three Leading Methods

1: Discovery of Potential Off-Target Edits

- Computational prediction
- SITE-Seq Genomic DNA digest
- GUIDE-seq Cell-based DNA repair

Aggregate ALL potential off-target genomic loci

658 non-overlapping potential off-target loci

SITE-Seq
GUIDE-Seq
Cas-OFFinder

431
37
178
17
4
7

Data above is a representative example
Off-Target Workflow In Practice: Representative Example

1: Discovery of Potential Off-Target Edits

- **658** non-overlapping potential off-target loci
- SITE-Seq
- 431
- GUIDE-Seq
- 7
- 14
- 37
- 178
- Cas-OFFinder

2: Validation of Off-Target Edits in Cells

- **In Vivo Programs**
  - Dose responses using drug product formulation
  - Therapeutically relevant human primary cell type(s) (2 donors)
  - Dose range to exceed projected therapeutic exposure (>10X)
  - Validation: off-target indels detected in edited cells
Validation of Off-Target Editing in Primary Human Hepatocytes at Supersaturating LNP CRISPR Concentrations to Maximize Sensitivity

658 potential off-target loci

7 validated off-target (OT) loci
  2 in introns and 5 in intergenic regions

- SITE-Seq discovered 100%
- GUIDE-Seq and Cas-OFFinder discovered the same 3 out of 7 validated off-target loci 43%
- Eliminate gRNA with validated off-target indels in regions of the genome associated with cancer

Data above is a representative example
**In Vitro**: No detectable off-target editing with pharmacologic concentration of sgRNA

![Graph showing editing frequency vs sgRNA concentration]

EC$_{90}$, concentration inducing 90% of maximal effect; sgRNA, single guide RNA
Strategic Collaborations
Growing Intellia’s Impact on Patients Through Strategic Collaborations

Increasing shareholder value:

- Leveraging our technology while retaining rights to key areas of focus
- Accelerate development of programs outside key areas of focus
- Expand our pipeline with valuable rights in future commercial success
- Access external expertise to enhance our platform

Genetic diseases

- REGENERON

Ophthalmology

- SPARINGVISION

Immuno-oncology (IO)

- VENCELL

Autoimmune diseases (CD19)

- kyverna.

Sickle cell disease and IO

- NOVARTIS
Foundational Partnerships Provided Access to R&D Capabilities

**REGENERON**

- Up to 15 *in vivo* targets with a mix of co-developed and licensed programs
  - Liver-centric product development
- **ATTR**: First selected Co/Co program
  - Intellia is lead party; Regeneron will share 25% of costs and profits
- **Hemophilia A and B**: Co/Co programs based on targeted insertion capabilities
  - Regeneron is lead party; Regeneron will share 65% of costs and profits
- *In vivo* targets exclusively developed by Regeneron:
  - Up to $320M in milestones per target
  - High single-to-low-double-digit royalties
- Non-exclusive license to certain platform IP on up to 10 *ex vivo* CRISPR products in defined cell types

**NOVARTIS**

- Advancing Phase 1/2 study for sickle cell disease based on CRISPR/Cas9-edited HSCs
- Research collaboration term concluded in December 2019
- Novartis selected various CAR-T, HSC and OSC targets for development
  - Up to $230M in milestone payments per product
  - Mid single-digit royalties
  - All non-selected targets revert to Intellia

Intellia THERAPEUTICS
Intellia, Cellex and Blackstone Launch AvenCell to Develop Allogeneic Universal CAR-T Cell Therapies, With $250M Committed Funding

Concurrent Cellex deal enables expansion and acceleration of Intellia's ex vivo pipeline with expanded manufacturing capabilities

- Rights to **two Co/Co options** in U.S. and key European countries on allogeneic universal CAR-T products
  - Intellia leads U.S. commercialization
- Additional **validation** of Intellia's proprietary allogeneic platform
- Hold substantial **equity stake** in NewCo
- **Access to Cellex cell therapy manufacturing site and allogeneic cell donations** via a preferred relationship
  - Supports Intellia's wholly owned *ex vivo* pipeline
  - Expanded capacity to handle additional pipeline growth

- **Expansion of existing Intellia-GEMoaB collaboration**
- Combines GEMoaB’s switchable universal CAR-T cell technology with Intellia's allogeneic platform enabled by advanced CRISPR engineering
- Addition of **validating partner** Blackstone and **infusion of $250M capital** to prosecute pipeline
- Clinical-stage autologous products from GEMoaB with **near-term milestones**
- **Seasoned management team**
- Access to Cellex cell therapy **manufacturing site**