UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, DC 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d) of the
Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): **January 11, 2016**

Idera Pharmaceuticals, Inc.
(Exact Name of Registrant as Specified in Charter)

Delaware	**001-31918**	**04-3072298**
(State or Other Jurisdiction of Incorporation)	(Commission File Number)	(IRS Employer Identification No.)

167 Sidney Street
Cambridge, Massachusetts 02139
(Address of principal executive offices) (Zip Code)

Registrant's telephone number, including area code: **(617) 679-5500**

(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (see General Instruction A.2. below):

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Item 7.01 Regulation FD Disclosure.

On February January 11, 2016, we uploaded a presentation to our website, www.iderapharma.com, discussing the state of the Company. We may rely on all or part of this presentation any time we are discussing the current state of the Company in communications with investors or at conferences. A copy of the presentation is attached to this Current Report on Form 8-K as Exhibit 99.1 (the "Slides").

By filing this Current Report on Form 8-K and furnishing the information contained herein, the Company makes no admission as to the materiality of any information in this report that is required to be disclosed solely by reason of Regulation FD.

The information contained in the Slides is summary information that is intended to be considered in the context of the Company's Securities and Exchange Commission ("SEC") filings and other public announcements that the Company may make, by press release or otherwise, from time to time. The Company undertakes no duty or obligation to publicly update or revise the information contained in this report, although it may do so from time to time as its management believes is warranted. Any such updating may be made through the filing of other reports or documents with the SEC, through press releases or through other public disclosure.

In accordance with General Instruction B.2 of this Current Report on Form 8-K, the information presented in Item 7.01 of this Current Report on Form 8-K and Exhibit 99.1 shall not be deemed to be "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section, unless the Company specifically states that the information is to be considered "filed" under the Exchange Act or incorporates it by reference into a filing under the Securities Act of 1933, as amended, or the Exchange Act.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits.

See Exhibit Index attached hereto.

SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, as amended, the Registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Idera Pharmaceuticals, Inc.

Date: January 11, 2016

By: /s/ Mark J. Casey
Mark J. Casey
Senior Vice President,
General Counsel and Secretary

EXHIBIT INDEX

Exhibit No.	Description
99.1	Investor presentation uploaded to Idera Pharmaceuticals, Inc. website on January 11, 2016

Exhibit 99.1

Forward Looking Statements

- This presentation contains forward-looking statements that involve substantial risks and uncertainties. Any statements in or made orally during this presentation about future expectations, plans and prospects for the company, including statements regarding our strategy, future operations, future financial position, future revenues, projected costs, prospects, plans and objectives of management, are forward-looking statements. The words “anticipate,” “believe,” “estimate,” “expect,” “intend,” “may,” “plan,” “predict,” “project,” “target,” “potential,” “would,” “could,” “should,” “continue,” and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. We may not actually achieve the plans, intentions or expectations disclosed in our forward-looking statements, and you should not place undue reliance on our forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in the forward-looking statements we make. Important factors that may cause or contribute to such differences include the factors set forth under the captions “Risk Factors” in our most recent quarterly report on Form 10-Q that we filed with the U.S. Securities and Exchange Commission for the period ended September 30, 2015. The forward-looking statements contained in this presentation reflect our current views with respect to future events, and we assume no obligation to update any forward-looking statements except as required by applicable law.

Targeting Medicines for Patients

Committed to advancing patient care

Focused on serious unmet needs in Cancers & Rare Diseases

Leading scientific discovery in TLR Immune Modulation and Antisense

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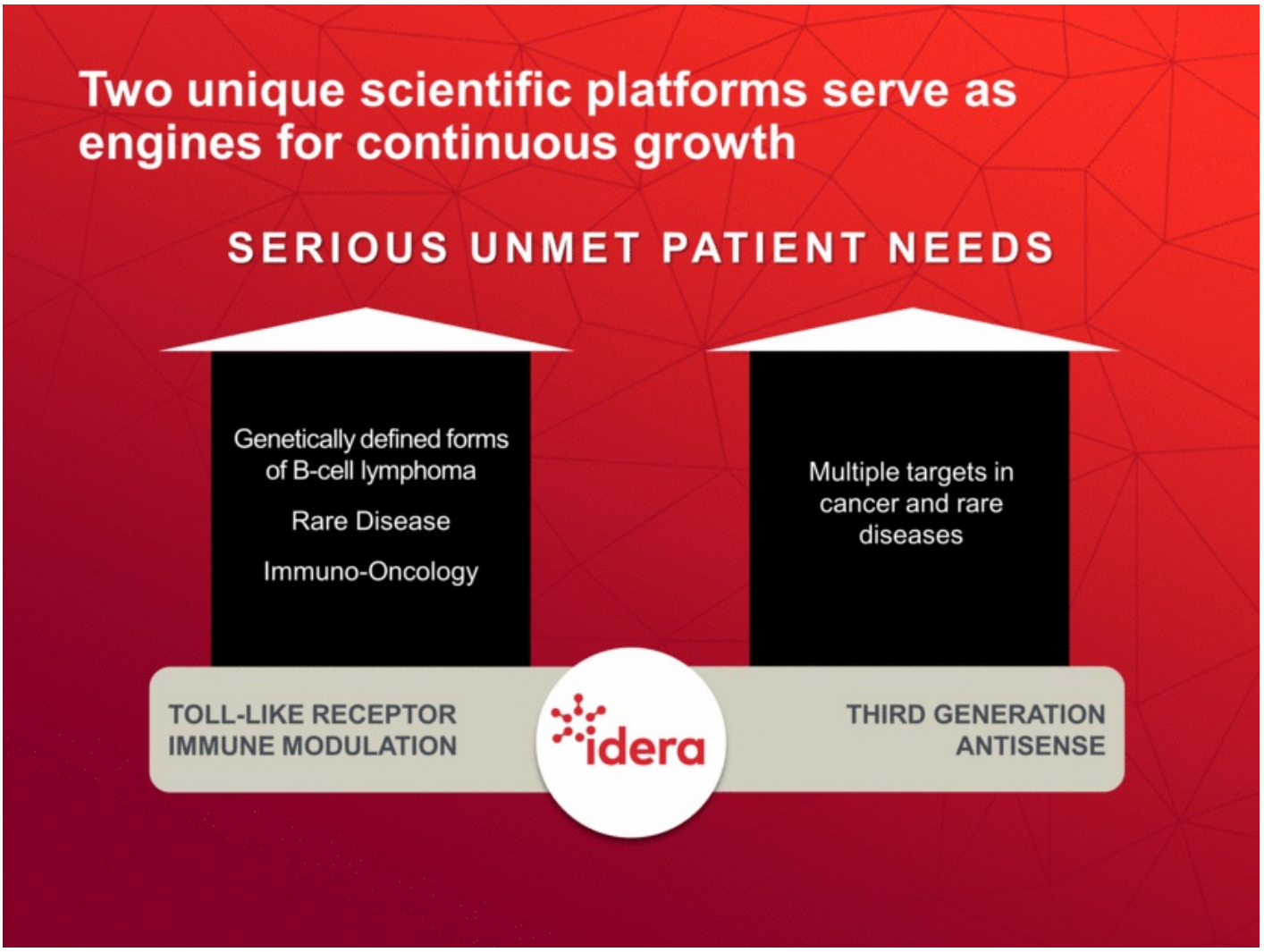
Two unique scientific platforms serve as engines for continuous growth



2015 Accomplishments

Delivering on the Promises

- Presented first positive IMO-8400 safety and efficacy data in B-cell lymphoma target
- Initiated clinical studies in Melanoma and Dermatomyositis
- Named first gene targets for 3rd Generation Antisense development platform (3GA)
- Announced first collaboration for 3GA platform
- Instituted new corporate culture
- Re-built leadership team and strengthened employee base

Experienced Leadership

SUDHIR AGRAWAL, D.Phil. ~ *President, Research*

LOUIS ARCUDI ~ *Chief Financial Officer*

MARK CASEY ~ *General Counsel*

JILL CONWELL ~ *Human Resources*

ROBERT DOODY ~ *IR & Comms*

CLAYTON FLETCHER ~ *Strategy & BD*

JOANNA HOROBIN, MB, ChB ~ *Chief Medical Officer*

VIN MILANO ~ *Chief Executive Officer*

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Toll Like Receptor (TLR) Antagonism Clinical Programs

Genetically-defined B-cell Lymphomas

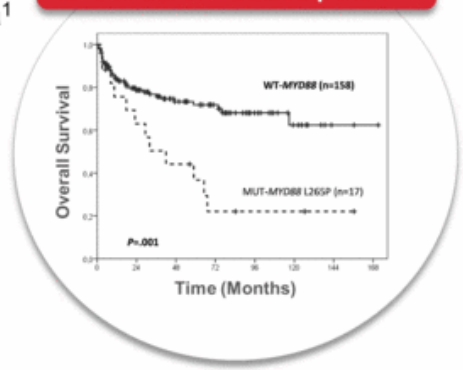
Waldenström's Macroglobulinemia (WM)

- Rare and slow-growing form B-cell lymphoma[1]
- ~1,000-1,500 new cases diagnosed annually in US[1]
- 90% carry MYD88 L265P mutation[2]
- Serious complications include anemia, retinopathy and peripheral neuropathy[1]

Diffuse Large B-Cell Lymphoma (DLBCL)

- Fast growing and potentially lethal form of B-cell lymphoma[1]
- ~20,000 new cases diagnosed annually in US[3]
- ~10% carry MYD88 L265P mutation[4,5]
- Data show poor prognosis in MYD88 L265P+ population[6]

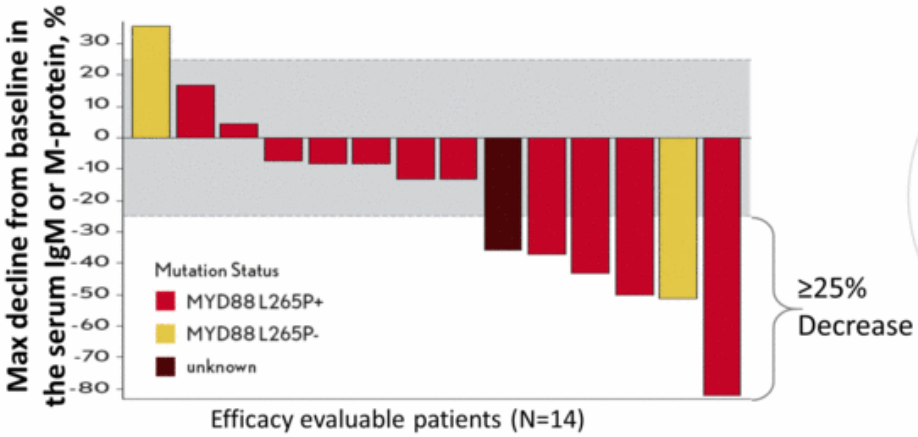
Survival is impaired in MYD88 L265P+ DLBCL patients[6]

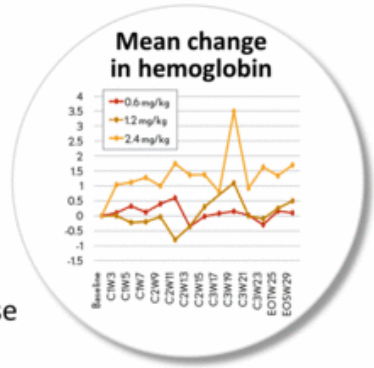


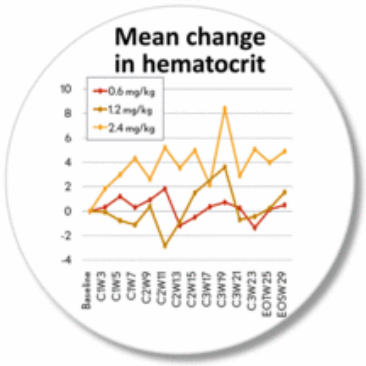
MYD88 L265P mutation also present in chronic lymphocytic lymphoma (5-10%)[7], splenic marginal zone lymphoma (13%)[8], primary CNS lymphoma (36%)[9], and other cancers

[1] American Cancer Society; [2] Treon SP, et al. N Engl J Med. 2012.; [3] Cultrera JL, et al. Cancer Control. 2012.; [4] Wang, et al. Blood Lymph Canc 2013. [5] Rosenwald A, et al. N Engl J Med. 2002. [6] Fernandez- Rodriguez C, et al. Leukemia. 2014. [7] Puente, et al. Nature. 2011. [8] Yan, et al. Haematologica. 2011. [9] Montesinos-Rongen, et al. Acta Neuropathol. 2011.

Demonstration of Clinical Activity with IMO-8400







- In the highest dose cohort studied to date (1.2 mg/kg twice a week):
  - Among 6 evaluable patients, 3 had responses and 2 had stable disease
  - Median time to first response was ~10.5 weeks
  - Improvements in symptoms, hemoglobin and bone marrow were seen
  - One of these responders was refractory to ibrutinib

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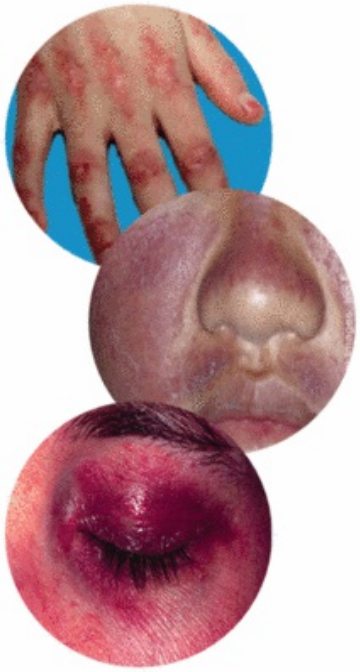
We have not yet reached IMO-8400's MTD

- IMO-8400 was generally well tolerated at all dose levels tested
- Most reported adverse events (AEs) were mild or moderate (grade 1 or 2)
- The most common AEs observed were fatigue, injection site erythema, headache, injection site pain, nausea and pain in extremity
- Grade 3 AEs reported as possibly or probably related to study drug included neutropenia, anemia and arthritis
  - 1 of 8 patients treated with 2.4 mg/kg in the safety population had a dose-limiting toxicity (DLT) deemed possibly related to study drug. This patient experienced a grade 3 probable flare of pre-existing arthritis

Our Path Forward

- Amending study protocols in both ongoing WM and DLBCL studies to advance to higher doses
- Will continue to analyze emerging data from these ongoing studies

Applying TLR Platform to Rare Diseases



Dermatomyositis

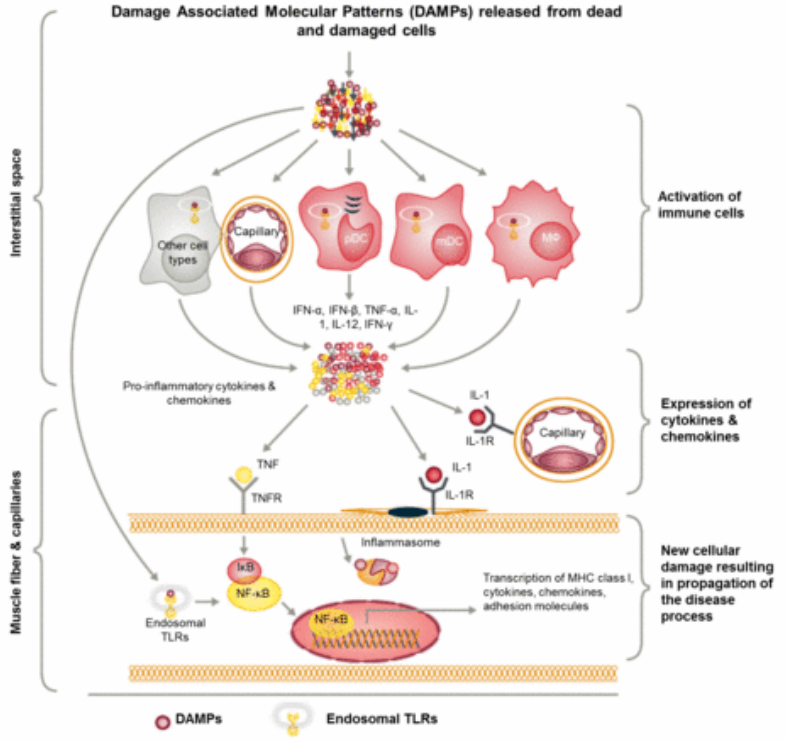
Therapeutic Rationale

- Cell damage may be caused by stress, injury or infection
- Onset typically occurs between ages 40-60 years
- Symptoms can be severely disabling, and include:
  - Muscle weakness, skin rash and/or calcinosis, joint pain, and difficulty swallowing
- Corticosteroids and immunosuppressive drugs have limited efficacy and serious side effects
- ~25k patients in U.S.

Opportunity

- TLR antagonism may disrupt the autoimmune cycle of tissue damage to improve disease symptoms

TLRs Play Role in Pathogenesis of DM



- Damaged skeletal muscle and skin tissue release Damage Associated Molecular Patterns (DAMPs)
- DAMPs bind to and initiate immune signaling through TLRs 7, 8 and 9 in skeletal muscle fibers and immune cells
- TLR signaling induces pro-inflammatory cytokines, driving downstream effects including damage to capillaries and hypoxia in affected tissue, inhibition of new muscle fiber formation, and cell death

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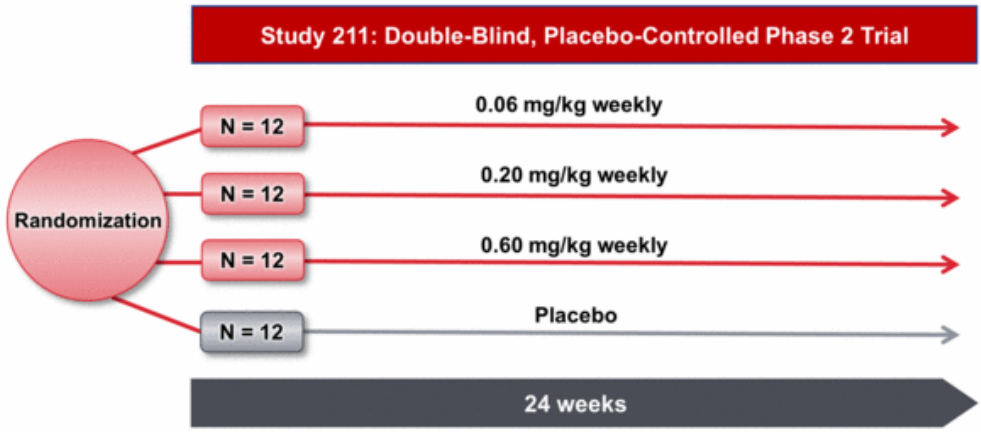
Rayavarapu, et al. Skeletal Muscle, 2013.

DM Phase 2 Study Underway



Study Design

- 24-week randomized, double-blinded placebo-controlled assessment

Major Eligibility Criteria

- DM diagnosis, aged 18-75 years, active skin and muscle disease, stable regimen of con-meds

Primary endpoint

- CDASI activity score

Exploratory endpoints

- MMT-8, IMACS core set measures, 10-meter run walk, 5D itch scale, SF-36 health survey

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Toll Like Receptor Agonist Clinical Programs

TLR9 Agonist to Induce the Immune System

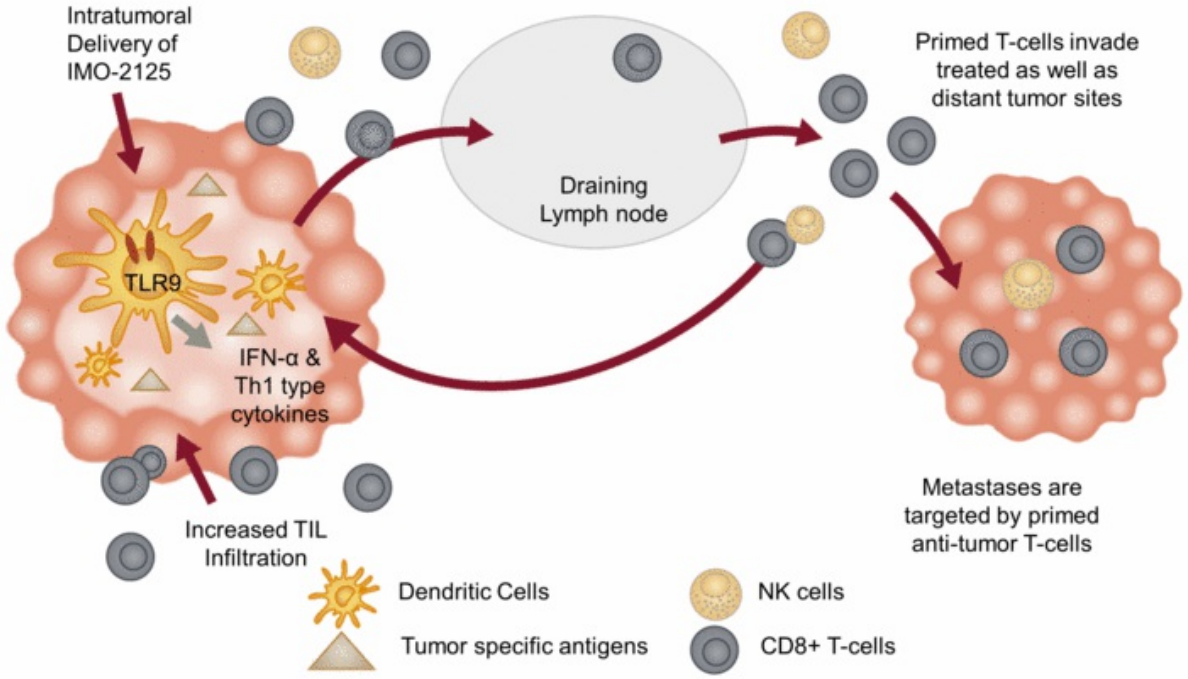
Therapeutic Rationale

- Emerging class of checkpoint inhibitors (CPIs)
  - Designed to block pathways that inhibit anti-tumor immune responses
  - PD-1 and CTLA-4 inhibitors are FDA approved for the treatment of certain cancers
- Intratumoral administration of IMO-2125 in preclinical models has stimulated dendritic cell maturation and T-cell activation in the tumor microenvironment, leading to increased local and systemic antitumor immune responses and tumor regression both alone and as well as in CPI combinations

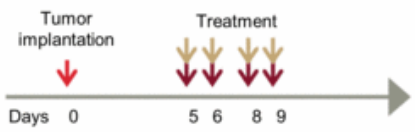
Opportunity

- Advance clinical development of IMO-2125 and CPI combinations

Intratumoral IMO-2125 Mechanism of Action



Intratumoral IMO-2125 and CTLA-4 induce systemic anti-tumor response in multiple preclinical studies

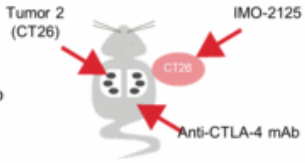


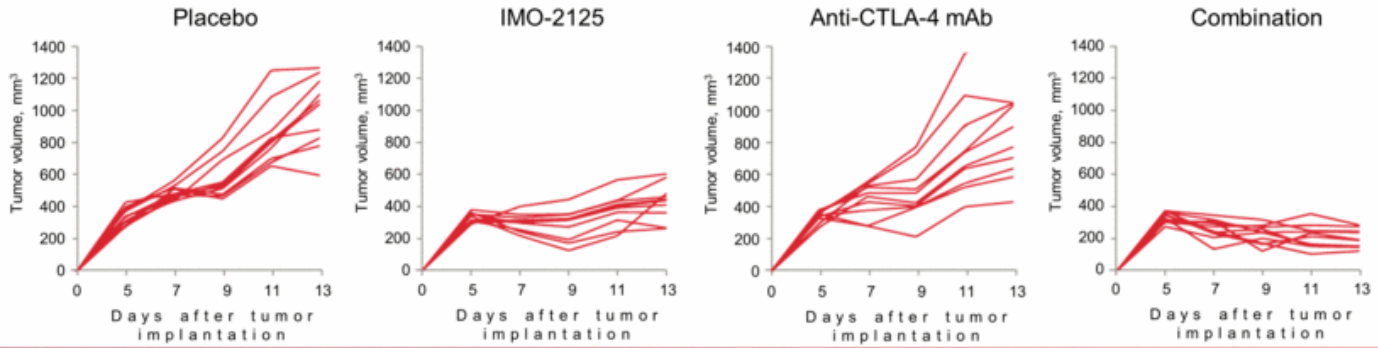
Tumor implantations:
1. Solid tumor (2 x 10^7 CT26, s.c., right flank)
2. Lung metastases (3 x 10^6 CT26, i.v.)

IMO-2125:
2.5 mg/kg i.t. injection (Tumor 1)

Anti-CTLA-4 mAb:
10 mg/kg i.p. injection

Groups:
1. Placebo
2. IMO-2125
3. Anti-CTLA-4 mAb
4. IMO-2125 + anti-CTLA-4 mAb



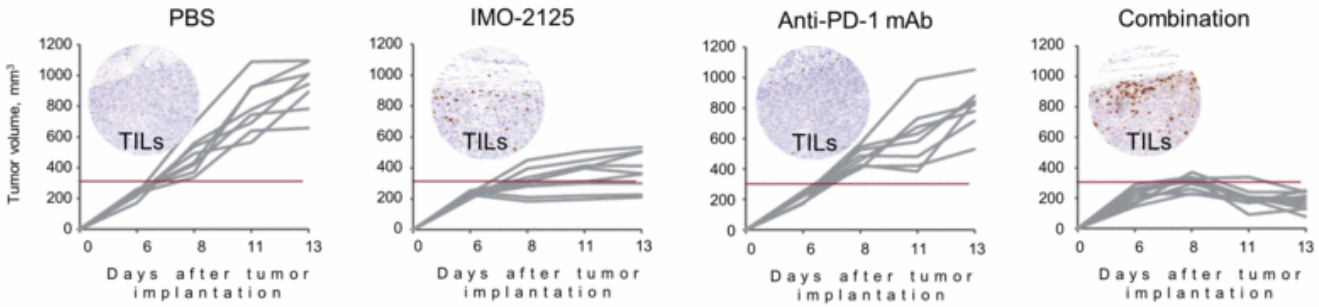


IMO-2125/ipilimumab in melanoma underway

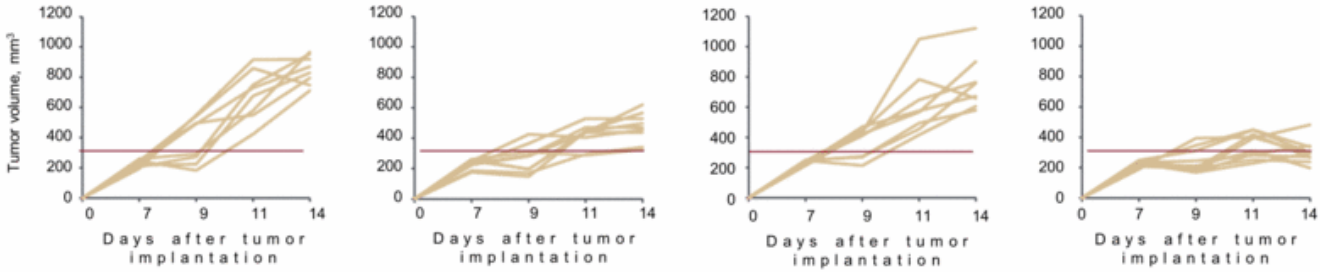
- Phase I Dose Escalation:
  - Recruit patients to cohorts (3+3 design with Bayesian component) of increasing IMO-2125 dose levels in combination with ipilimumab at its approved dose/schedule
  - IMO-2125 intratumoral injection to be administered weekly X 3 in first 4 week cycle, then once every 3 weeks in subsequent 3 week cycles for 9 weeks
  - Approximately 24 melanoma patients who have relapsed after prior therapy
- Phase 2:
  - Approximately 30 pts with metastatic melanoma
  - Preliminary assessment of efficacy in addition to biomarker analyses

IMO-2125 and anti-PD-1 mAb preclinical combo demonstrated improved tumor growth inhibition in both treated and distant tumors vs. monotherapy

Treated tumors



Distant tumors



Our Path Forward

- Execution of open-label Phase 1/2 clinical trial with MDACC
- Will continue to analyze data from this ongoing study
- Formulate and execute broader immuno-oncology development program
  - Identify next studies to execute
    - Additional treatable tumor types
    - Other CPI combinations based on extensive pre-clinical modeling

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Third Generation Antisense (3GA) Platform

Why is third generation antisense (3GA) needed?

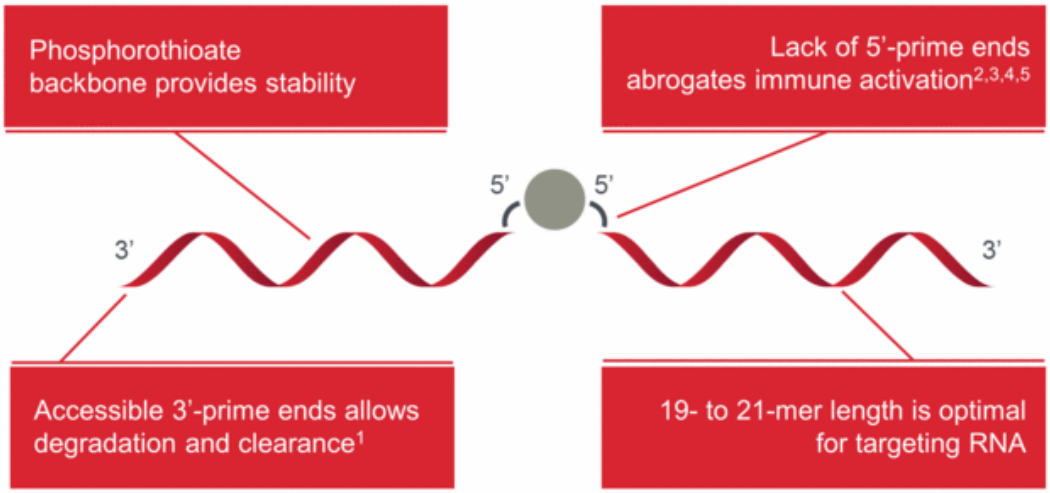
- To realize the full potential of antisense technology for the treatment of diverse diseases
- To overcome the limitations of the first and second generation antisense technology
  - Immunotoxicity
  - Therapeutic Index

Applied learnings to create third generation antisense

Development of a thorough understanding of interaction of nucleic acids and innate immune receptors

Utilization of the insights gained to create 3GA to mitigate immunotoxicity and off-target effects

3GA is a patented construct

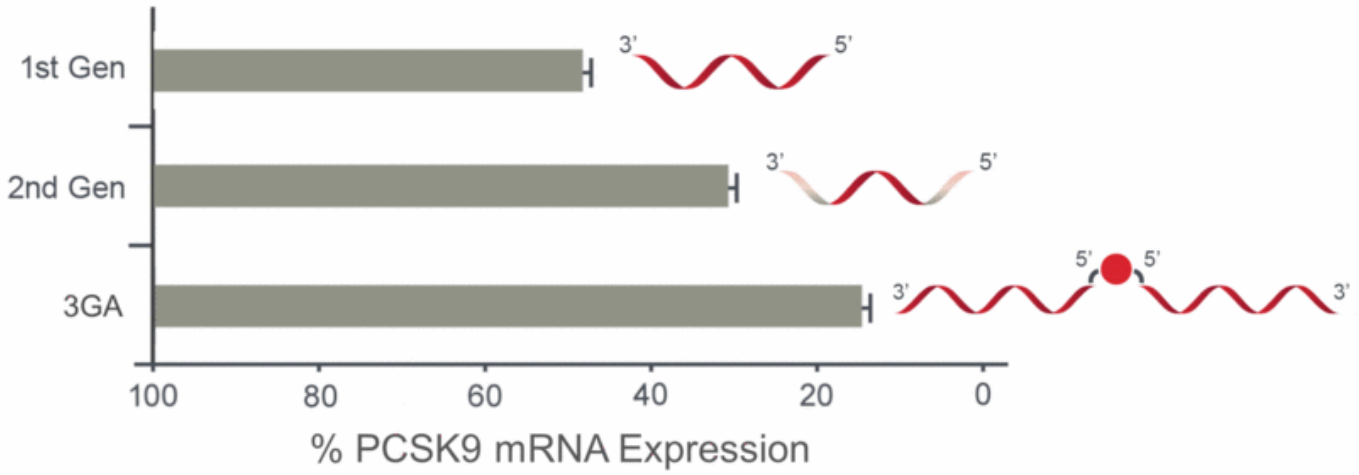


U.S. patent #8,431,544 issued to Idera in 2013.

[1] J Med Chem 2011;54:3027-36. [2] Bioorg Med Chem Lett 2000;10:2585-8. [3] Bioconjugate Chem. 2002;13:966-74. [4] Nucleic Acid Res 2002;30:4460-9. [5] Bioconjugate Chem. 2010;21:39-48 .

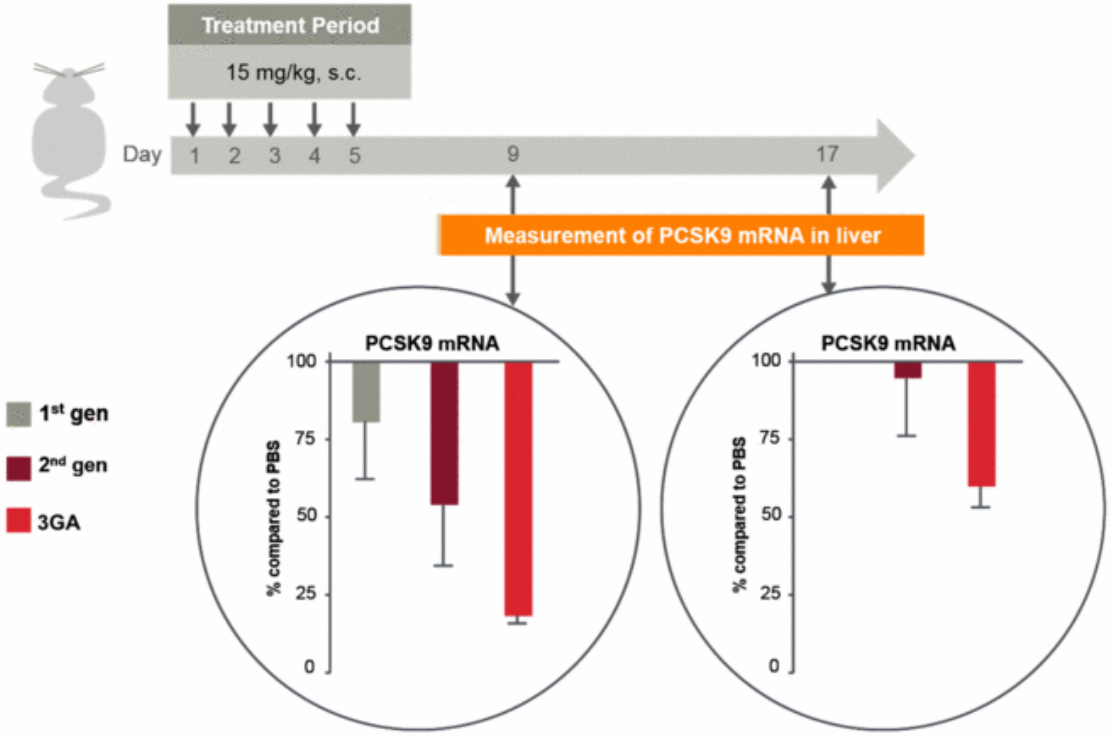
3GA is potentially more potent than first and second generation ASO

Potency of 3GA has been validated for multiple RNA targets



Assays were conducted with antisense constructs in Hepa 1-6 cells; RNA levels were quantified by qPCR

3GA has the potential for more potency and a more sustained effect than 1st and 2nd generation ASO



15 mg/kg of compounds were administered subcutaneously daily for 5 days. On day 9 and 17, PCSK9 mRNA was measured in liver lysates and compared to untreated animals.

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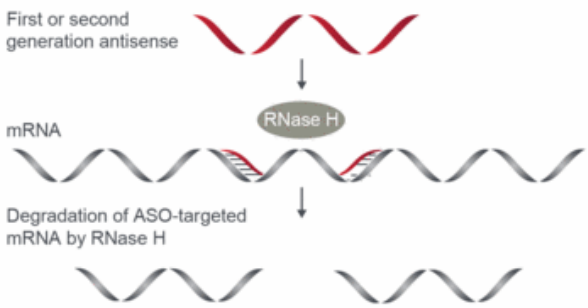
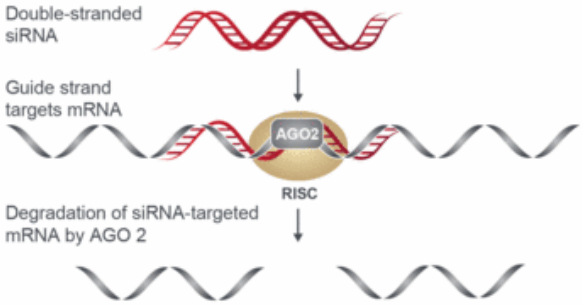
3GA introduces a new and novel mechanism

Two major mechanisms of RNA targeting





1st Gen. ASO:

mRNA target 5' –GT**GGTGCTGATGGAGGAGACC**CA – 3'

3' - CCACGACTACCTCCTCTCTGG - 5

2nd Gen. ASO:

mRNA target 5' –GT**GGTGCTGATGGAGGAGACC**CA – 3'

3' - CCACGACTACCTCCTCUGG - 5

siRNA

mRNA target 5' –GT**GGTGCTGATGGAGGAGACC**CA – 3'

3' - TTCCACGACTACCTCCTCTGG - 5

3GA:

mRNA target 5' –GT**GGTGCTGATGGAGGAGACC**CA – 3'

3' - CCACGACTACCTCCTCTCTGG - 5

Site of cleavage

PCSK9 targeted antisense/siRNA constructs were evaluated in Hepa 1-6 cells; RNA cleavage products were analyzed by 5' RLM RACE. These are major cleavage products and could vary based on experiment conditions.

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Systemic delivery of 3GA allows us to target disease-associated RNA in multiple organs

Next step: Building the pipeline

Our 3GA disease prioritization process

Key Considerations

- Gene target associated with the disease
- Over expression of the gene correlates with disease
- Gene target/pathway proof-of-concept established
- Gene target/pathway not “druggable” with small molecules or antibodies
- Rare disease and oncology indications with commercial viability
- Possibility of local delivery to the site of gene expression
  - Bladder, Ocular, Intratumoral, Lungs, GI/Colon

Rational Selection of 3GA target RNA

More efficient process to identify potent 3GA

Target mRNA

Targeting the entire transcript

↓

Screening of hundreds of ASOs

↓

Identification of ASO candidates

Our proprietary insights allow us to target specific regions of the transcript

↓

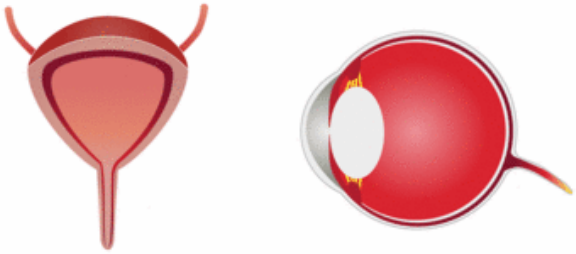
Screening of selected ASOs

↓

Identification of 3GA candidates

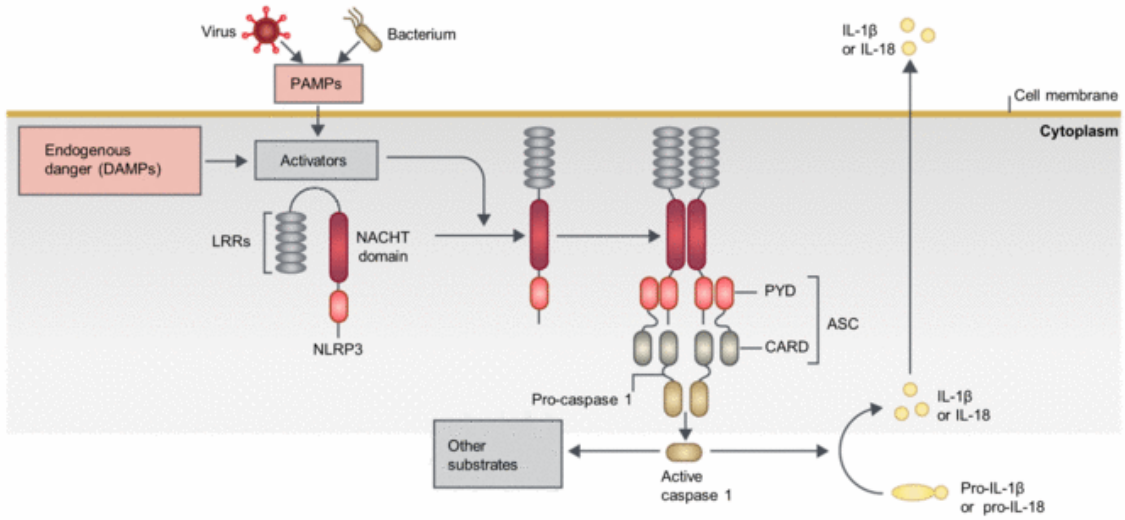
Example of selected gene target

Targeting NOD-like receptor protein 3 (NLRP3) with 3GA for the treatment of interstitial cystitis and uveitis



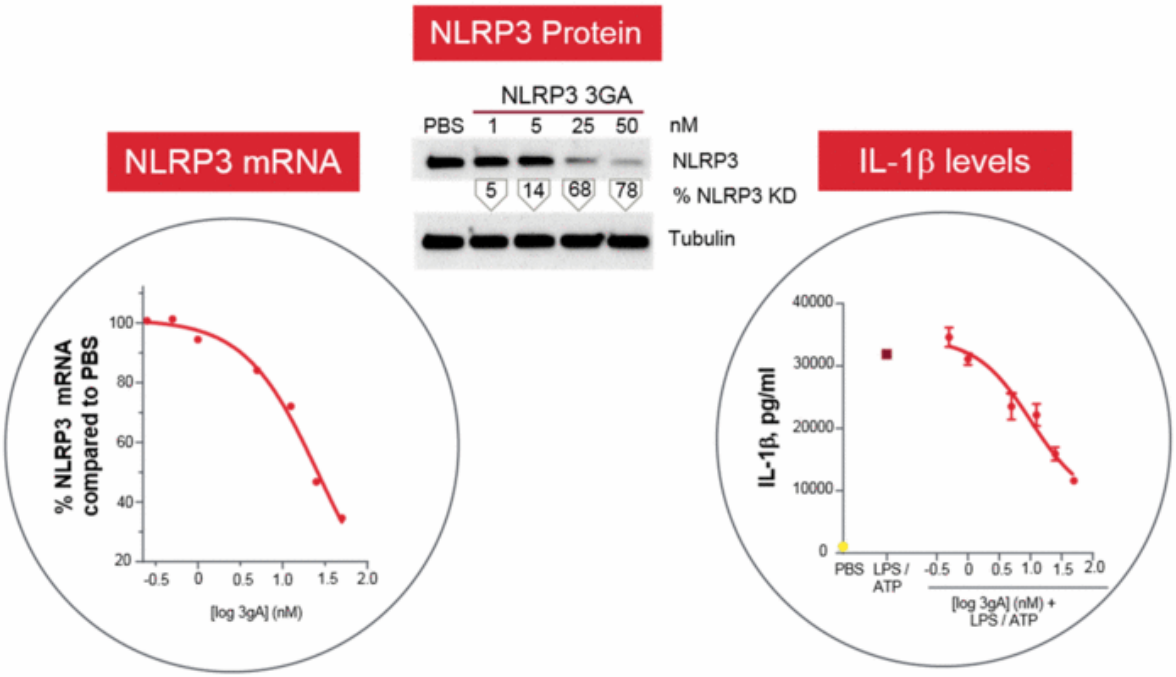
NLRP3, a key component of Inflammasomes

- Inflammasomes are complexes that control the activation of caspase-1, which leads to production of IL-1β and IL-18
- NLRP3 inflammasome is implicated in multiple diseases
- Targeting NLRP3 by 3GA offers the benefit of blocking the release of both IL-1β and IL-18



Adapted from Nature Reviews Nature Reviews Immunology 10, 210-215

3GA targeting NLRP3 downregulates NLRP3 and levels of IL-1β

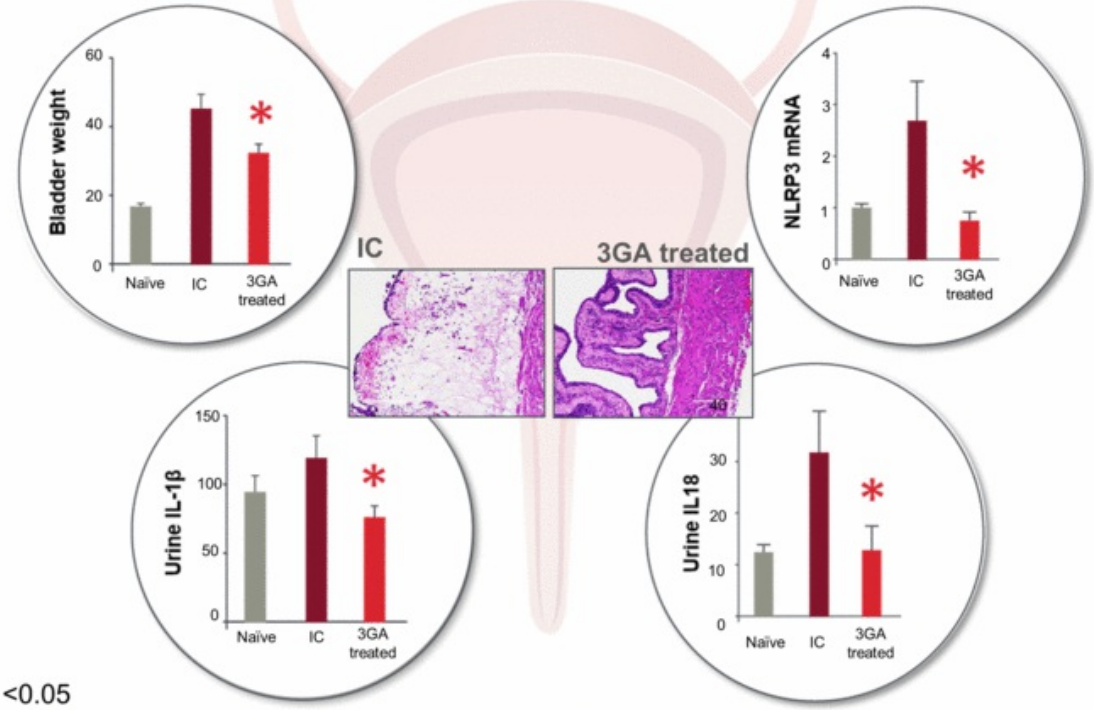


Differentiated THP-1 cells were incubated with 3GA for 24 hr. NLRP3 RNA levels were determined by qPCR and NLRP3 protein levels were analyzed by Western blotting. For IL-1β secretion, differentiated THP-1 cells were incubated with 3gA for 24 hr followed by stimulation with 100 ng/ml LPS for 4hr and 5mM ATP for 1 hr. Supernatants were collected and analyzed for IL-1b levels by ELISA

Treatment of Interstitial Cystitis by 3GA targeting NLRP3

- Interstitial cystitis (IC) is a chronic inflammation of the bladder that affects quality of life
- An estimated 4-12 million in the US may suffer from IC. Approximately 5-10% of these patients suffer severe symptoms, including ulcerations, intense pain, and frequent urination, that are in need of effective treatment
  (Interstitial Cystitis Association)
- NLRP3 pathway has been implicated in IC
  - Human bladder epithelium expresses high levels of NLRP3
    (*J Histochem Cytochem.* 2007; 443-52)
  - Positive IL-1β staining in bladder epithelial cell lining in IC patients
    (*J Urol.* 1998; 2185-92)
  - Increased serum IL-1β level in IC patients (*PLoS One*. 2013; e76779)
  - IL-18 is upregulated in human IC patient biopsy samples (*World J Urol*. 2013; 241-6)

Systemic delivery of 3GA targeting NLRP3 is believed to improve IC-associated parameters



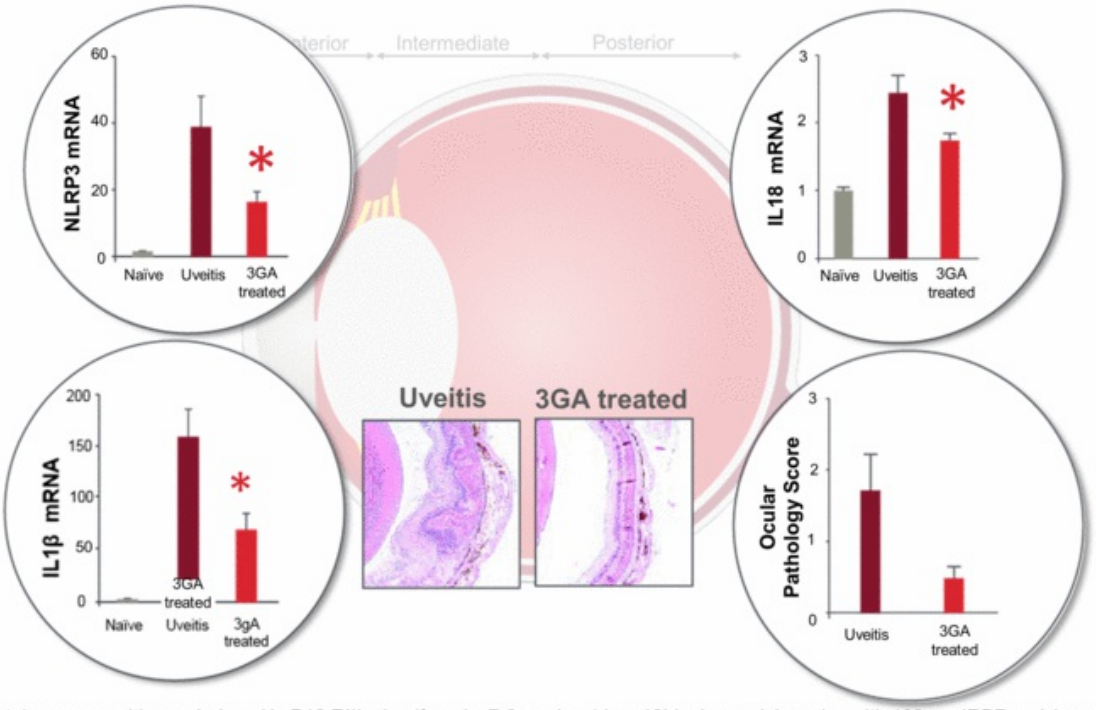
* p <0.05

Cystitis was induced in CD1 mice (female, age of 7-8 weeks, n=10) by intraperitoneally administered 200 mg/kg cyclophosphamide. 3GA was administered subcutaneously at 25 mg/kg 1 hour post cyclophosphamide. Bladder and urine samples were collected 24 hours post treatment, analyzed for bladder weight, NLRP3 mRNA, and levels of IL-1β and IL-18 in urine

Treatment of Experimental Autoimmune Uveitis by 3GA targeting NLRP3

- Uveitis is an inflammation of the uvea
- Approximately 10% of blindness for those under 65 is caused by uveitis and its complications (*Am Fam Physician.* 2000; 434.)
- NLRP3 pathway has been implicated in Uveitis
  - Circulating IL-1β level is elevated in uveitis (*Curr Eye Res.* 2000; 211-4)
  - Intraocular IL-1β level is elevated in uveitis (*Mol Vis.* 2011; 2003-10)
  - Interfering with the NLRP3 pathway reduced ocular IL-1β in uveitis model (*Hum Gene Ther.* 2015; 59-68)

Systemic delivery of 3GA targeting NLRP3 is believed to improve uveitis associated parameters



* p <0.05

Experimental autoimmune uveitis was induced in B10.RIII mice (female, 7-8 weeks old, n=10) by immunizing mice with 100 mg IRBP and 1 mg bovine eye homogenate emulsified with complete freund's adjuvant and 0.5 mg pertussis toxin on day 0 and day 7. 3GA was administered subcutaneously at 15 mg/kg on day 8, 11 and 13. Mouse eyes were enucleated on day 14, expression of NLRP3, IL-18, and IL-1βwas evaluated. In addition, pathology scores were also evaluated.

3GA platform is ready to realize the potential of antisense technology

- 3GA is designed to address the shortcomings/limitations of 1st and 2nd generation ASO
- Distinct mechanism with potent gene knockdown
- Rapid process from target selection to potential drug candidate
- We expect 1 to 2 targets per year to push into IND enabling studies for certain cancers and rare diseases
- We believe we can further exploit the 3GA technology through partnerships with companies whose interests lie outside of oncology and rare diseases

Financial Overview

Cash Runway through Second Quarter of 2017

Cash & Investments	~$95M as of 9/30/15
Recent Closing Price	$2.91 as of 1/4/16
Trading Volume	~1.3M shares daily (90 day average)
Market Capitalization	~$344M as of 1/4/16
Shares Outstanding	121.3M as of 12/31/15

Growing Development Pipeline Advancing

DEVELOPMENT PROGRAM	PRECLINICAL	PHASE 1	PHASE 2	PIVOTAL
TLR Antagonism – IMO-8400				
Waldenström's macroglobulemia				
Diffuse large B-cell lymphoma (MYD88 L265P+)				
Dermatomyositis				
Duchenne muscular dystrophy	*Planning underway*			
TLR Agonism – IMO-2125				
Refractory/Relapsed Melanoma w/ CTLA4				
Additional Tumor Types / CPI Combos	*Planning underway*			

RESEARCH PROGRAM	PRECLINICAL	PHASE 1	PHASE 2	PIVOTAL
THIRD GENERATION ANTISENSE				
NLRP				
DUX4				

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Thank You

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