# 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 12, 2015**

# 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**<br>**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.**

Idera Pharmaceuticals, Inc. (the "Company") has included on its website an updated corporate slide presentation, which it intends to present and/or distribute to the investment community and utilize in various industry and other conferences. The corporate slide presentation is accessible in the Investors and Media section of the Company's website at www.iderapharma.com and is attached hereto as Exhibit 99.1. The Company undertakes no obligation to update, supplement or amend the materials attached hereto as Exhibit 99.1.

The information furnished pursuant to Item 7.01 of this current report, including Exhibit 99.1, shall not be deemed to be "filed" for the purposes of Section 18 of the Securities Exchange Act of 1934 or otherwise subject to the liabilities of that Section. As such, this information shall not be incorporated by reference into any of the Company's reports or other filings made with the Securities and Exchange Commission. The furnishing of the information in this current report is not intended to, and does not, constitute a determination or admission by the Company that the information in this current report is material or complete, or that investors should consider this information before making an investment decision with respect to any security of the Company.

**Item 9.01 Financial Statements and Exhibits.**

(d) Exhibit

The following exhibit is furnished as part of this current report on Form 8-K:

Exhibit 99.1 — Solving Unmet Patient Needs with Strong Scientific Foundations – Idera Pharmaceuticals – January 2015

**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 12, 2015

By: /s/ Louis J. Arcudi, III
Louis J. Arcudi, III
*Senior Vice President of Operations,*
*Chief Financial Officer, Treasurer and Secretary*

**Exhibit Index**

| Exhibit No. | Description |
| --- | --- |
| 99.1 | Solving Unmet Patient Needs with Strong Scientific Foundations – Idera Pharmaceuticals – January 2015 |

Exhibit 99.1

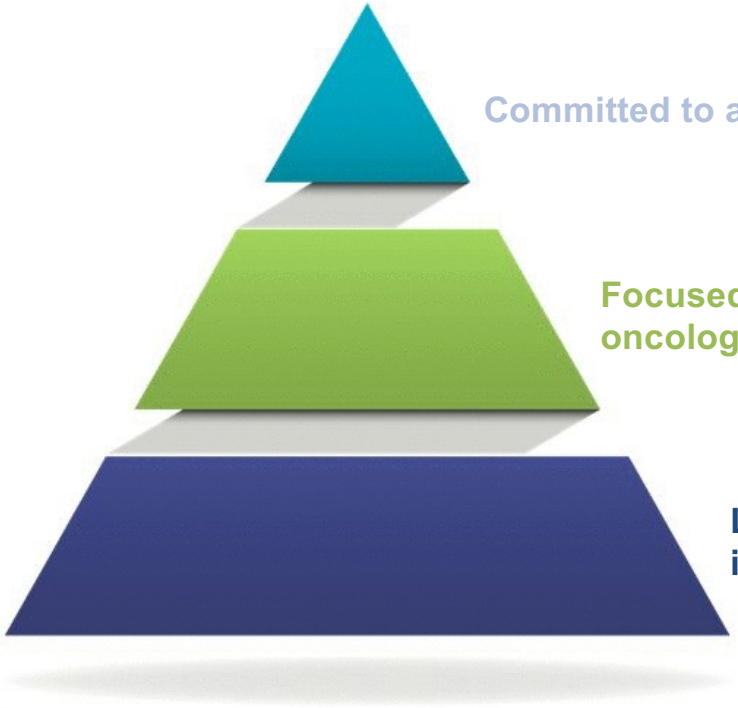

# Solving Unmet Patient Needs with Strong Scientific Foundations

IDERA PHARMACEUTICALS
JANUARY 2015

# Forward looking statements

Any statements that we may make in this presentation about future expectations, plans and prospects for the Company constitute forward-looking statements for purposes of the safe harbor provisions under The Private Securities Litigation Reform Act of 1995. Actual results may differ materially from those indicated by these forward-looking statements as a result of various important factors, including whether Idera's cash resources will be sufficient to fund the Company's continuing operations and the further development of the Company's programs; whether results obtained in preclinical studies and clinical trials such as the results described in this presentation will be indicative of the results that will be generated in future clinical trials; whether products based on Idera's technology will advance into or through the clinical trial process when anticipated or at all or warrant submission for regulatory approval; whether such products will receive approval from the U.S. Food and Drug Administration or equivalent foreign regulatory agencies; whether, if the Company's products receive approval, they will be successfully distributed and marketed; whether the Company's collaborations will be successful; and such other important factors as are set forth under the caption "Risk Factors" in the Company's Quarterly Report on Form 10-Q for the quarter ended September 30, 2014. Idera disclaims any intention or obligation to update any forward-looking statements.

# Our goal is to translate scientific breakthroughs into important medicines for patients



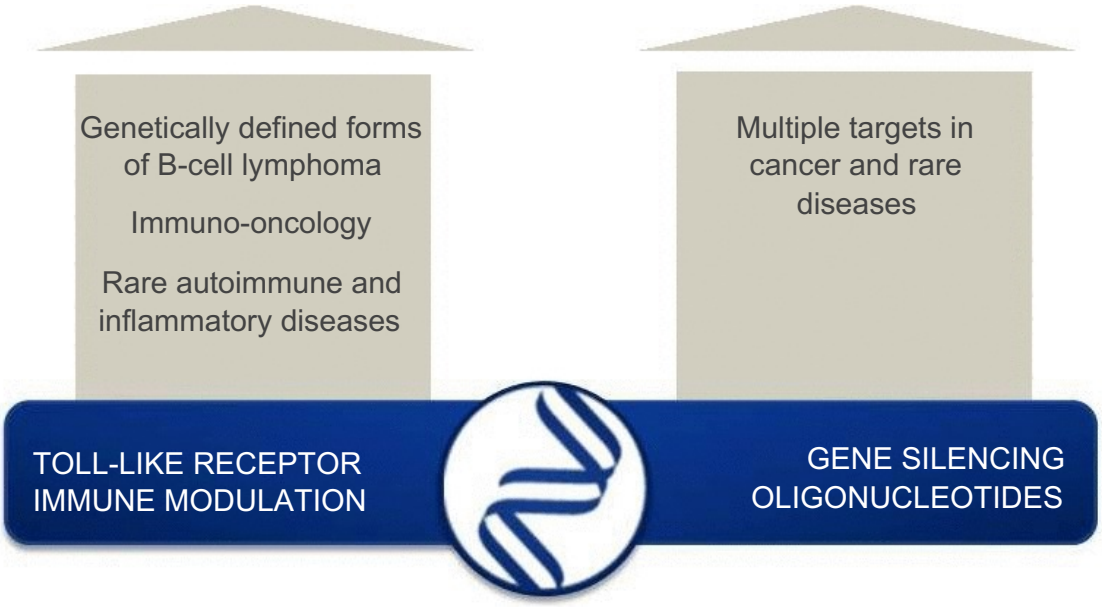
Committed to advancing patient care

Focused on serious unmet needs in oncology & rare diseases

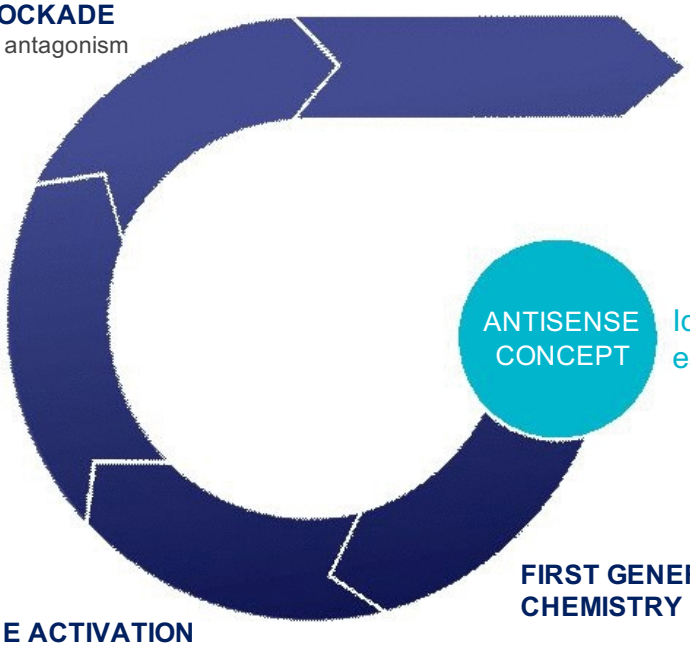
Leading scientific discovery in immunotherapy & gene silencing

# Scientific platforms support broad pipeline opportunities

SERIOUS UNMET PATIENT NEEDS



# Idera is a pioneer of immunotherapy and gene silencing technologies



**THIRD GENERATION CHEMISTRY**
Gene silencing oligonucleotides

**IMMUNE BLOCKADE**
Toll-like receptor antagonism

**SECOND GENERATION CHEMISTRY**
Modified RNA-DNA hybrid structure;
Most successful to date,
but limitations persist

ANTISENSE CONCEPT

Idera scientists were early pioneers

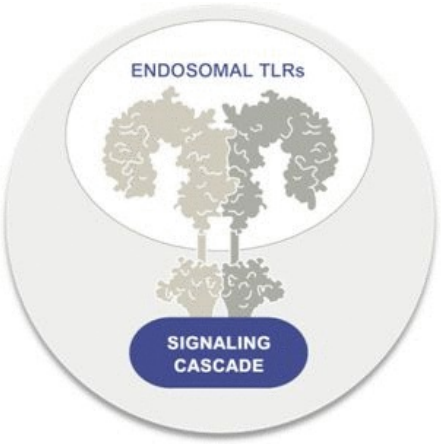
**FIRST GENERATION CHEMISTRY**

**IMMUNE ACTIVATION**
Toll-like receptor agonism

# Understanding of TLR biology has informed development of immunotherapy and gene silencing technologies

**Toll-like Receptors (TLRs)**

- Discovered in the 1990s; subject of Nobel Prize in 2011
- Play central role in the innate immune system
- Initiate signaling cascades that activate inflammation and adaptive immune responses
- Nucleic acids are ligands for endosomal TLRs 7, 8 and 9




## Therapeutic Implications

**TLR immune modulation** exploits TLR-nucleic acid interaction to stimulate or block TLR-mediated signaling

**Gene silencing oligonucleotides** are specifically designed to avoid activation of TLRs, thereby enabling enhanced gene silencing activity

# Idera has developed two leading nucleic acid chemistry-based platforms

| | TOLL-LIKE RECEPTOR IMMUNE MODULATION | GENE SILENCING OLIGONUCLEOTIDES |
|---|---|---|
| **TECHNOLOGY** | Oligonucleotide-based | Third generation oligonucleotide chemistry |
| **BIOLOGICAL TARGET** | Toll-like receptors | RNA |
| **BIOLOGICAL IMPACT** | Activate or inhibit immune activity | Silence disease-related genes |

# Idera's growing pipeline

| DEVELOPMENT PROGRAM | PRECLINICAL | PHASE 1 | PHASE 2 | PIVOTAL |
|---|---|---|---|---|
| **B-CELL LYMPHOMA – IMO-8400** | | | | |
| Waldenström's macroglobulemia | | | | |
| Diffuse large B-cell lymphoma (MYD88 L265P+) | | | | |
| **IMMUNO-ONCOLOGY – IMO-2125 / IMO-2055** | | | | |
| Intratumoral combination with CPI | *Planning underway* | | | |
| **RARE DISEASES – IMO-8400** | | | | |
| Dermatomyositis | *Planning underway* | | | |
| Duchenne muscular dystrophy | *Planning underway* | | | |
| **GENE SILENCING OLIGONUCLEOTIDES** | | | | |
| Undisclosed targets | | | | |
| **AUTOIMMUNE DISEASES** | | | | |
| Undisclosed targets | | | | |

**Major Anticipated 2015 Milestones:**

- Results from Phase 1/2 Waldenström's trial in 4Q
- Immuno-oncology, Dermatomyositis and Duchenne programs advance into or toward clinical trials
- Selection of first GSO development candidates for development

# Idera's future is bright: long-term value drivers

- **Immunotherapy and gene silencing platforms** are the foundation for potentially transformational therapies for cancer and rare diseases
- **Genetically defined B-cell lymphoma programs** are progressing in the clinic
- **New immuno-oncology program** offers opportunity to enhance emerging class of checkpoint inhibitors and improve outcomes for cancer patients
- **Rare disease programs** focus on serious unmet medical needs in collaboration with strong advocacy communities
- **Enhanced executive management team** has track record of success and expertise to build the business

# B-cell lymphoma programs

Committed to advancing patient care

**FOCUSED ON SERIOUS UNMET NEEDS IN ONCOLOGY & RARE DISEASES**

Leading scientific discovery in immunotherapy & gene silencing

# B-cell lymphomas characterized by the MYD88 L265P mutation are rare and have limited treatment options

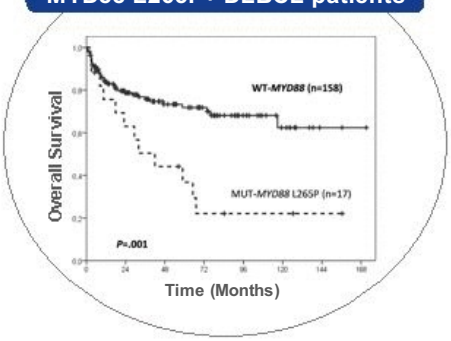
## Waldenström's Macroglobulinemia (WM)

- Rare and slow-growing form B-cell lymphoma [1]
- ~1,000-1,500 new cases diagnosed annually in US [1]
- Currently incurable with no FDA approved therapies [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]

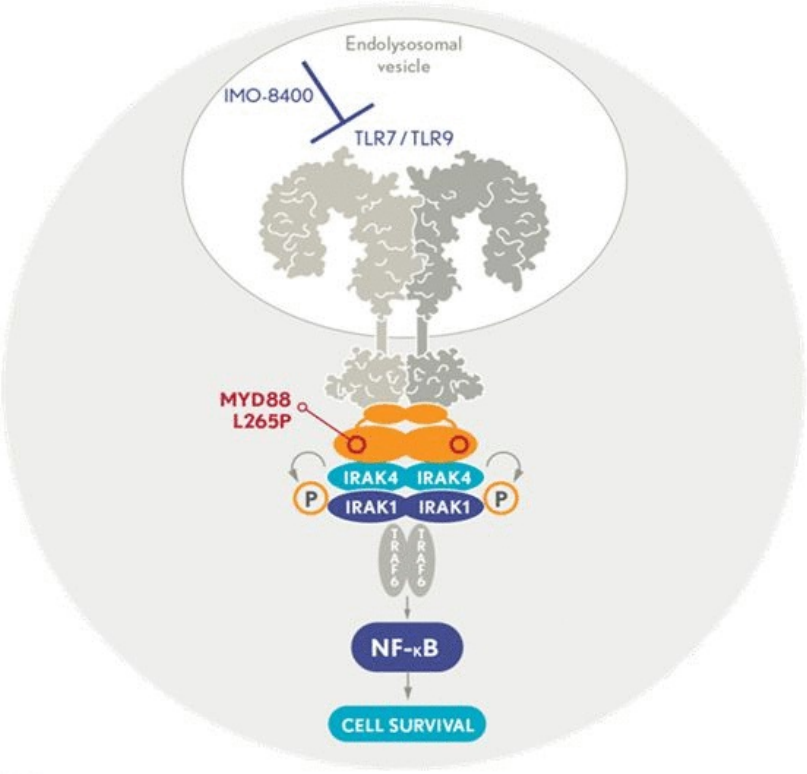
**Survival is impaired in MYD88 L265P+ DLBCL patients [6]**



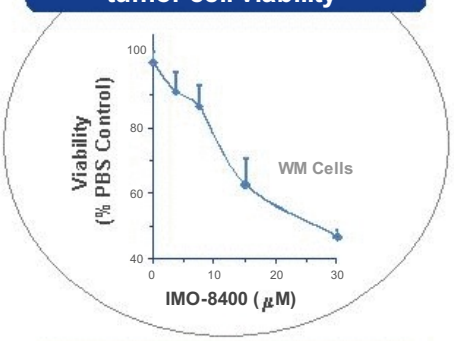

*MYD88 L265P mutation also present in chronic lymphocytic lymphoma (5-10%)[7], splenic marginal zone lymphoma (13%)[8], primary CNS lymphoma (36%)[9], and other cancers*

[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.

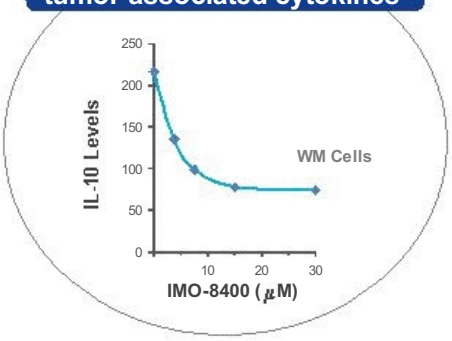
# Targeted therapy for B-cell lymphoma patients carrying oncogenic mutation in TLR signaling pathway




**IMO-8400 inhibits tumor cell viability**



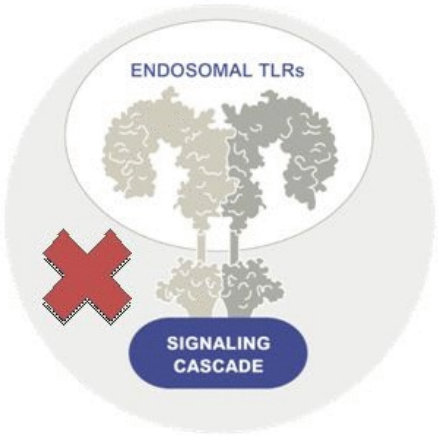

**IMO-8400 suppresses tumor-associated cytokines**




*Graphic adapted from Cancer Res. 2013,73 Suppl. 1.2332. IMO-8400 data presented at AACR 2014.*

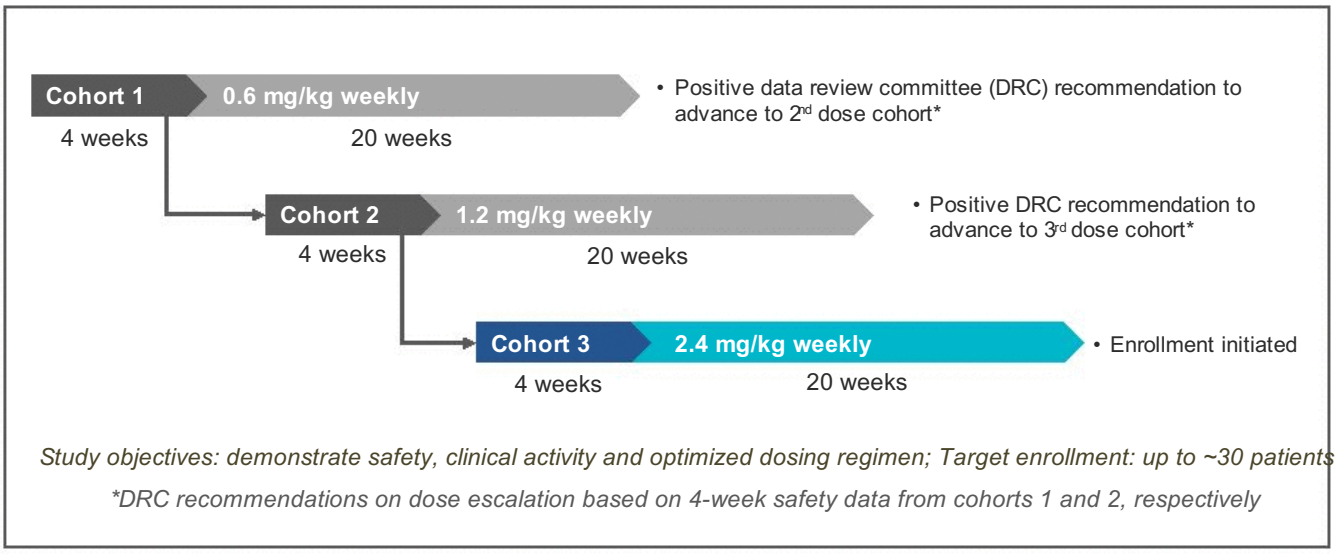
# IMO-8400 has demonstrated tolerability and evidence of clinical activity

***IMO-8400 blocks TLR signaling and immune system activation***




- IMO-8400 is a synthetic oligonucleotide-based antagonist of TLRs 7, 8 and 9
- Activity observed in preclinical models of autoimmune diseases and lymphoma
- Evidence of TLR antagonism established in human clinical trials in psoriasis
- IMO-8400 generally well tolerated in ~85 patients and healthy subjects with >550 doses administered to date

# Following safety review, enrollment initiated in third dose cohort of Phase 1/2 Waldenström's trial

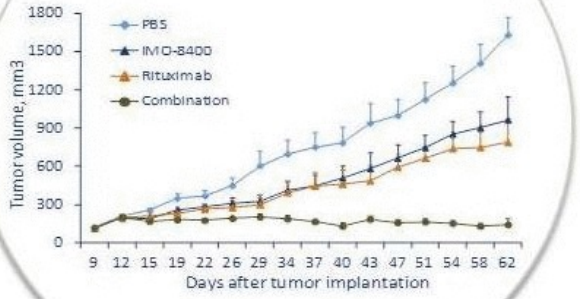



- Received FDA orphan drug designation for IMO-8400 in Waldenström's in December 2014
- Trial results expected in 4Q 2015

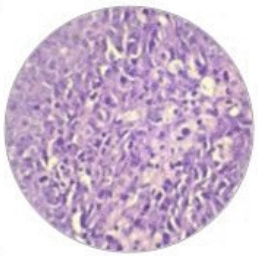
# Additional B-cell lymphoma program updates

- Completed initial development of prototype CDx under collaboration with Abbott Molecular
- Activated multiple clinical sites and expanded patient screening in Phase 1/2 DLBCL trial
- Presented abstracts at ASH 2014 included:
  - Safety of IMO-8400 in Phase 1 and 2 clinical trials in healthy volunteers and psoriasis patients
  - Preclinical activity of IMO-8400 in combination with rituximab

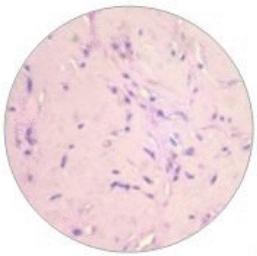
**IMO-8400 + rituximab inhibited tumor growth**




**IMO-8400 + rituximab improved tumor histology**



**Rituximab alone**



**IMO-8400 + rituximab**

Data from ABC-DLBCL MYD88 L265P+ OCI-Ly10 xenograft model. Presented at ASH 2014.

# Immuno-oncology program

Committed to advancing patient care

**FOCUSED ON SERIOUS UNMET NEEDS IN ONCOLOGY & RARE DISEASES**

Leading scientific discovery in immunotherapy & gene silencing

# Cancer immunotherapy with intratumoral TLR 9 agonist and checkpoint inhibitors

## Therapeutic Rationale

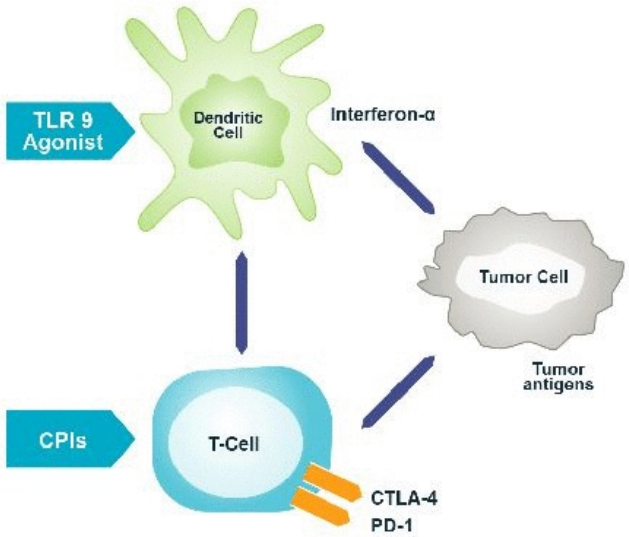
- Emerging class of checkpoint inhibitors (CPIs) represents a significant advance in cancer immunotherapy
  - Designed to block pathways that inhibit anti-tumor immune responses
  - PD-1 and CTLA-4 inhibitors are FDA approved for the treatment of melanoma; clinical development in additional cancer types is ongoing
- Intratumoral administration of TLR 9 agonists have demonstrated immunostimulatory activity in preclinical models of cancer

## Opportunity

- Combination of complementary cancer immunotherapy agents may increase the duration and durability of clinical responses

# Intratumoral TLR 9 agonist stimulates immune response while CPI inhibits tumor defense against immune attack

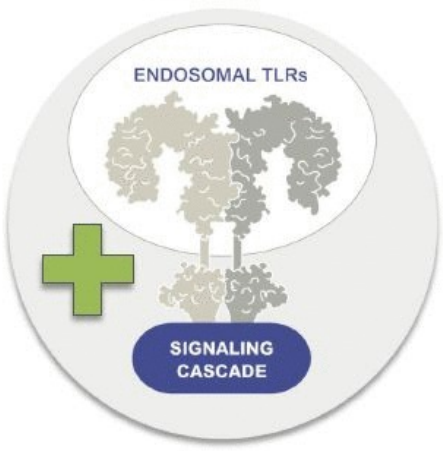
**Modulation of the Tumor Microenvironment**




- Intratumoral TLR 9 agonist stimulates dendritic cell maturation and T-cell activation; induces interferon-α
- CPIs block inhibitory pathways enabling activation of CD4+ and CD8+ T-cell responses
- Dying tumor cells release antigens enabling T-cell memory, systemic anti-tumor immune responses

# IMO-2125 & IMO-2055 are clinical-stage TLR 9 agonists

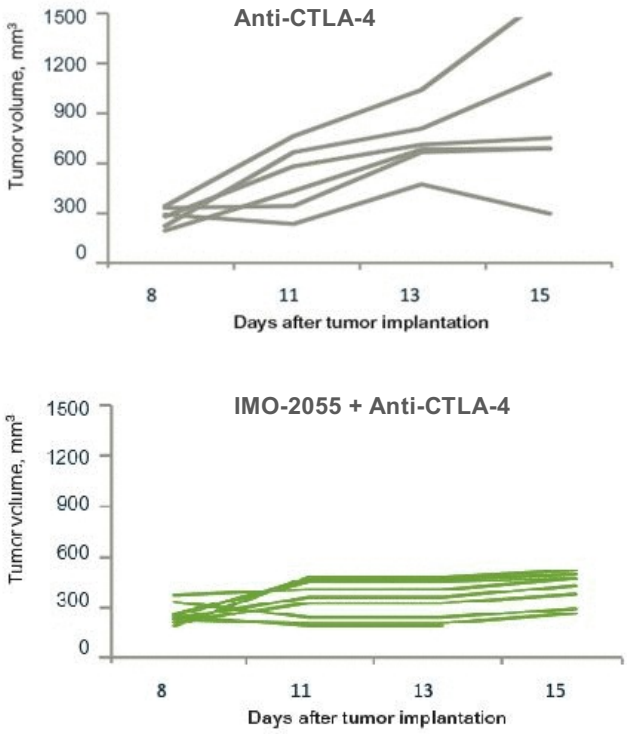
***IMO-2125 and IMO-2055 stimulate TLR signaling to induce an immune response***



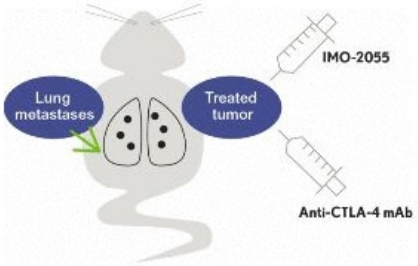

- IMO-2125 and IMO-2055 are synthetic oligonucleotide-based agonists of TLR 9
- Completed trials involving systemic administration of IMO-2125 and IMO-2055 included:
  - 80 patients with hepatitis C
  - 300 patients with various types of cancer
- Idera's TLR 9 agonists have demonstrated evidence of anti-tumor activity in multiple settings
  - Clinical trial in non-small cell lung cancer
    - Smith, et al. Cancer Immunol Immunother 2014.
  - Preclinical models
    - Damiano, et al. Clin Cancer Res 2006.
    - Damiano, et al. PNAS 2007.
    - Damiano, et al. Clin Cancer Res 2009.
    - Rosa, et al. Clin Cancer Res 2011.

# Intratumoral IMO-2055 and anti-CTLA-4 mAb demonstrated potent and systemic anti-tumor activity in preclinical models

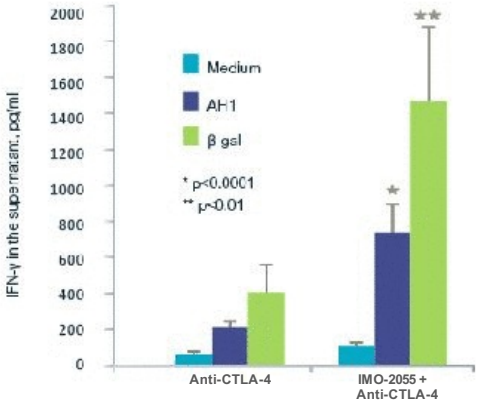
## Inhibited growth of treated tumors




## Induced systemic anti-tumor immune response




- AH1 antigen presented in locally treated tumors
- ß-gal antigen presented in disseminated lung metastases




Data from CT26 colon carcinoma model. Presented at AACR Tumor Immunology 2014.

# Priorities for immuno-oncology program

- Rapidly advance a combination regimen with an intratumoral TLR 9 agonist and a CPI into clinical development
- Complete ongoing preclinical studies to characterize potential combination regimens with various CPIs

*Intratumoral TLR 9 agonism has the potential to enhance cancer immunotherapy regimens with CPIs*

# Idera's growing pipeline

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**Major Anticipated 2015 Milestones:**

- Results from Phase 1/2 Waldenström's trial in 4Q
- Immuno-oncology, Dermatomyositis and Duchenne programs advance into or toward clinical trials
- Selection of first GSO development candidates for development

# Enhanced executive management team has a track record of success across the industry

| | |
|---|---|
| VIN MILANO | *Chief Executive Officer* |
| SUDHIR AGRAWAL, D.Phil. | *President, Research* |
| LOU ARCUDI | *SVP, Chief Financial Officer* |
| LOU BRENNER, M.D. | *SVP, Chief Medical Officer* |
| PETE WOLF | *SVP, General Counsel* |
| BOB DOODY | *VP, Investor Relations and Corporate Communications* |
| LIZ EBERHARDT | *VP, Oncology Team* |
| KATE HAVILAND | *VP, Rare Diseases* |

# Financial overview

| | |
|---|---|
| COMMON SHARES OUTSTANDING | 85.8M as of 9/30/14 |
| RECENT CLOSING PRICE | $4.96 as of 1/8/15 |
| TRADING VOLUME | ~1.7M shares daily (90 day average) |
| MARKET CAPITALIZATION | ~$425.6M as of 1/8/15 |
| CASH & INVESTMENTS | ~$58M as of 9/30/14 |

# Idera's future is bright: long-term value drivers

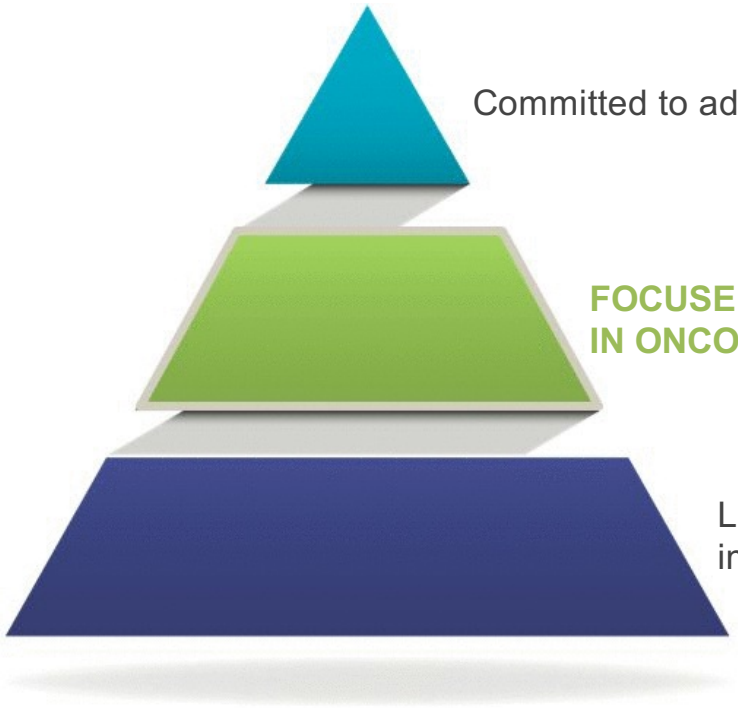
- **Immunotherapy and gene silencing platforms** are the foundation for potentially transformational therapies for cancer and rare diseases
- **Genetically defined B-cell lymphoma programs** are progressing in the clinic
- **New immuno-oncology program** offers opportunity to enhance emerging class of checkpoint inhibitors and improve outcomes for cancer patients
- **Rare disease programs** focus on serious unmet medical needs in collaboration with strong advocacy communities
- **Enhanced executive management team** has track record of success and expertise to build the business

idera PHARMACEUTICALS

# Solving Unmet Patient Needs with Strong Scientific Foundations

APPENDIX

# B-cell lymphoma programs

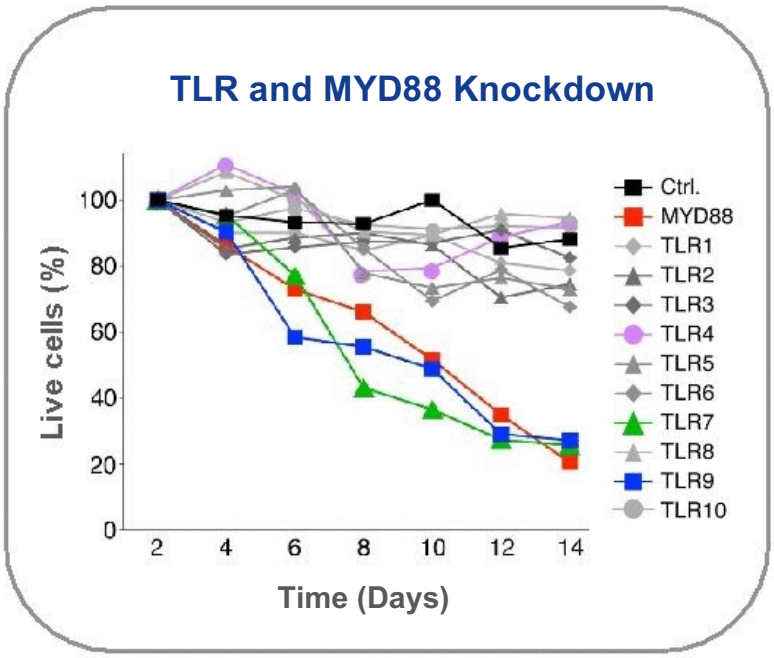


Committed to advancing patient care

**FOCUSED ON SERIOUS UNMET NEEDS IN ONCOLOGY & RARE DISEASES**

Leading scientific discovery in immunotherapy & gene silencing

# Oncogenic MYD88 L265P signaling requires TLRs 7 and 9




## Lim, Barton, Staudt. AACR 2013.

*"…Here we show that the MYD88 L265P oncoprotein binds constitutively to two members of the Toll-like receptor family, TLR7 and TLR9, thereby amplifying signals that emanate from these receptors.*

*Knockdown of TLR7 or TLR9 potently suppressed NF-κB activity in ABC-DLBCL cell lines and promoted apoptosis…*

***These new insights into the oncogenic mechanisms of MYD88 mutants provide a rationale for targeting of TLR7 and TLR9 signaling for the therapy of ABC-DLBCL."***

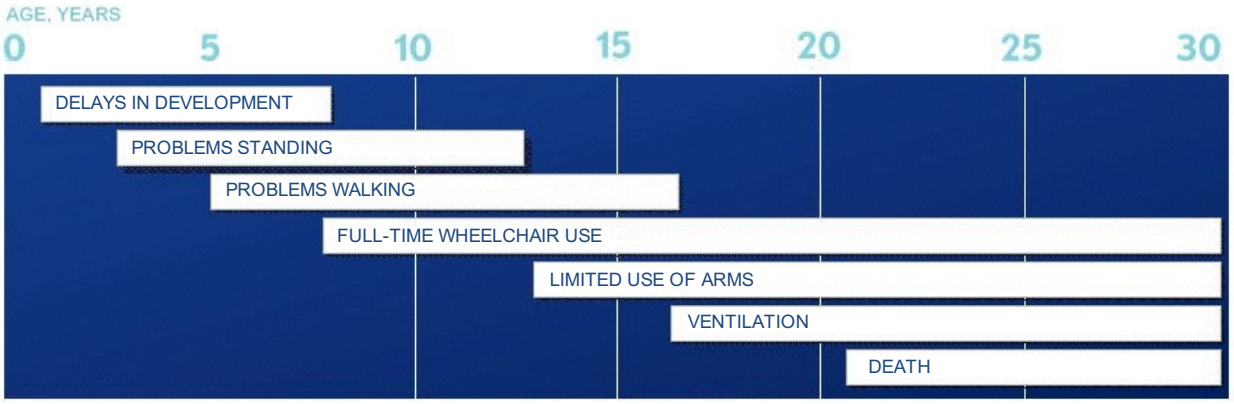
# Rare disease programs

Committed to advancing patient care

**FOCUSED ON SERIOUS UNMET NEEDS IN ONCOLOGY & RARE DISEASES**

Leading scientific discovery in immunotherapy & gene silencing

# Duchenne muscular dystrophy is a rare and universally fatal neuromuscular disorder




Graphic adapted from McDonald C, et al. Muscle & Nerve, 2013; Bushby K, et al. Lancet, 2010

## Therapeutic Rationale

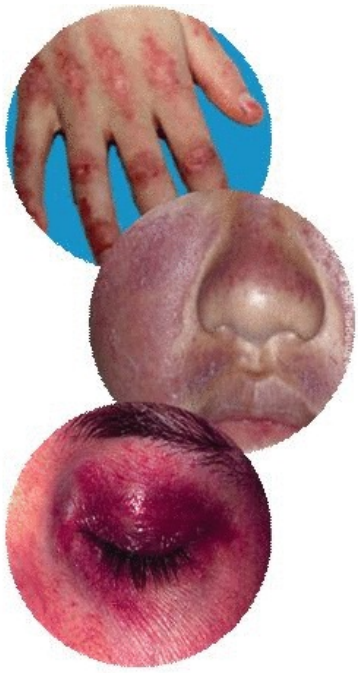
- Muscle cell damage caused by lack of functional dystrophin protein
- TLR 7 significantly over-expressed, even in pre-symptomatic infants
- Corticosteroids are standard of care, but have limited efficacy and serious side effects
- ~15-20k patients in U.S.

## Opportunity

- TLR antagonism has the potential to reduce muscle inflammation and slow disease progression
- Immune modulation may potentiate dystrophin restoration therapies such as exon skipping by inducing immunologic tolerance to dystrophin

MMWR 2009; Chen, et al. Neurology 2005; Henriques-Pons, et al. Hum Mol Gen 2014.

# Dermatomyositis is a rare and disabling inflammatory muscle disease with skin involvement



## Therapeutic Rationale

- Cell damage may be caused by stress, injury or infection
- TLR 9 and mRNA for MYD88 significantly over-expressed
- 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.

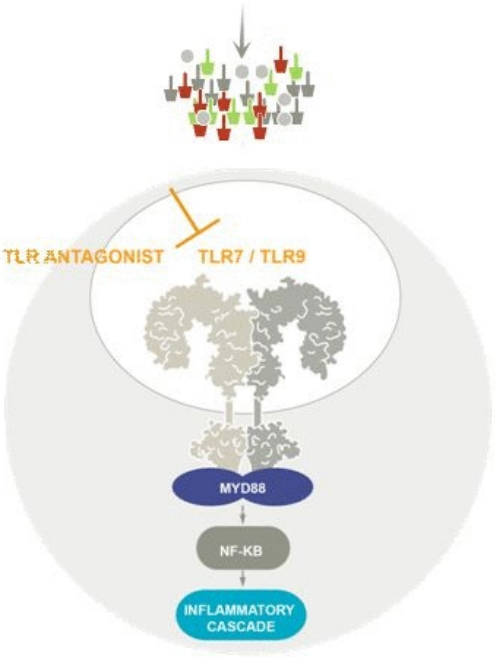
## Opportunity

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

Data provided by The Myositis Association. Rayavarapu, et al. Skeletal Muscle 2013.

# Scientific rationale for TLR antagonism in dermatomyositis and Duchenne muscular dystrophy

**Damaged or Dying Cells**




**TLR ANTAGONIST INHIBITS SIGNALING CASCADE**

Damaged cells release self DNA, self RNA and other molecules to form Damage Associated Molecular Patterns (DAMPs).

DAMPs stimulate TLR signaling to promote inflammation.

Inflammation causes additional cellular damage, thereby propagating the diseases process.

Adapted from Rayavarapu, et al. Skeletal Muscle 2013.

# Rare disease programs are advancing toward clinical development

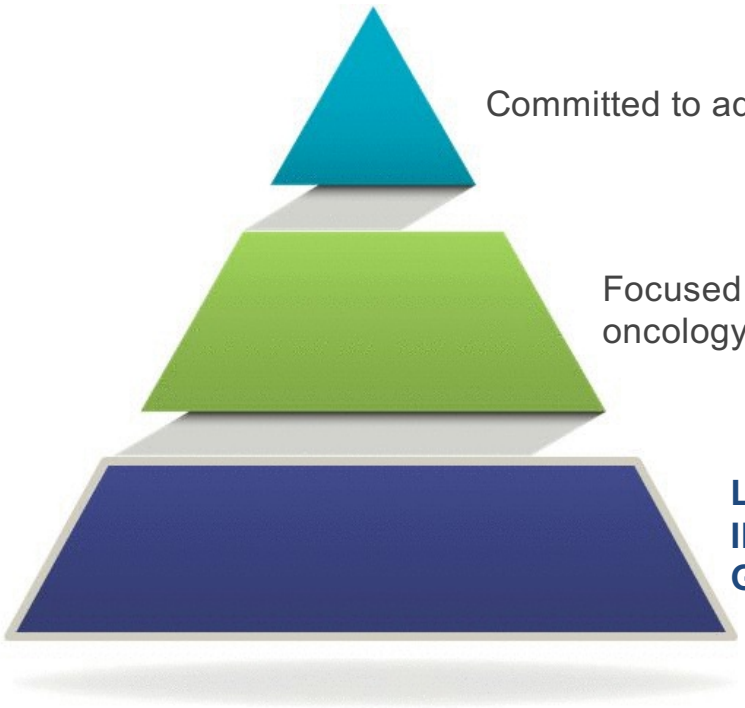
### Duchenne Muscular Dystrophy

- Collaborating with Parent Project Muscular Dystrophy
- Conducting additional preclinical studies in well-established *mdx* mouse model
- Evaluating potential clinical trial design options with disease experts, including population and endpoint selection

### Dermatomyositis

- Collaborating with The Myositis Association
- Organized advisory board of expert clinicians to inform design of Phase 2 clinical trial
- Finalizing clinical trial plan with IMO-8400; anticipate pre-IND meeting with FDA in 1H 2015

# Gene silencing oligonucleotide program



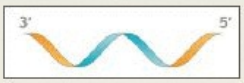
Committed to advancing patient care

Focused on serious unmet needs in oncology & rare diseases

**LEADING SCIENTIFIC DISCOVERY IN IMMUNOTHERAPY & GENE SILENCING**

# Novel Gene Silencing Oligonucleotides (GSOs) overcome limitations of current antisense technologies

## FIRST GENERATION




### Chemistry

- Single-stranded structure with phosphorothioate backbone

### Clinical Activity

- Severe off-target immune activation limited clinical utility

## SECOND GENERATION

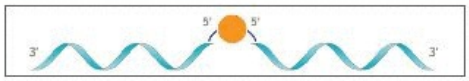

### Chemistry

- Modified RNA-DNA hybrid structure pioneered by Idera
- Most successful antisense chemistry to date

### Clinical Activity

- Immune activation, immunotoxicity, injection site reactions
- Hepatic toxicity with chronic dosing
- Limited therapeutic index

## THIRD GENERATION




### Chemistry

- Novel structure discovered by Idera
- 19- to 21-mer length is optimal for gene silencing
- No 5'-prime ends abrogates immune activation
- Accessible 3'-prime ends improves degradation and clearance
- Issued U.S. patent

### Activity in Preclinical Models

- Reduced immunotoxocity
- Decreased hepatic toxicity
- Improved potency
- Increased therapeutic index

***In vivo proof-of-concept established against multiple gene targets***

Bhagat, et al. J Med Chem 2011.

# Assessment of potential GSO targets in oncology and rare diseases is underway