IDERA PHARMACEUTICALS, INC.
Form 424B5
February 13, 2015
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Filed Pursuant to Rule 424(b)(5)
Registration No. 333-195896

Prospectus Supplement to Prospectus dated May 22, 2014.

20,000,000 Shares

Idera Pharmaceuticals, Inc.

Common Stock

$3.75 Per Share

We are offering 20,000,000 shares of our common stock.

Our common stock is listed on The Nasdaq Capital Market under the symbol IDRA. The last sale price of our common stock on February 12, 2015, as reported by The Nasdaq Capital Market, was $4.24 per share.

Entities affiliated with two of our directors, Julian C. Baker and Dr. Kelvin M. Neu, have agreed to purchase an aggregate of 5,333,333 shares of the common stock offered in this offering at the price offered to the public.

Investing in our securities involves a high degree of risk. See Risk Factors, beginning on page S-12 of this prospectus supplement, as well as in the documents incorporated or deemed to be incorporated by reference into this prospectus supplement and the accompanying prospectus, for a discussion of the factors you should carefully consider before deciding to purchase our securities.

Neither the Securities and Exchange Commission nor any state securities commission has approved or disapproved of these securities or determined if this prospectus supplement or the accompanying prospectus is truthful or complete. Any representation to the contrary is a criminal offense.

	Per Share	Total
Public offering price	$ 3.75	$ 75,000,000
Underwriting discount(1)	$ 0.225	$ 4,500,000
Proceeds, before expenses, to Idera	$ 3.525	$ 70,500,000

(1) See Underwriting beginning on page S-52 for additional information regarding underwriting compensation. We have granted the underwriters a 30-day option to purchase up to an additional 3,000,000 shares of our common stock at the public offering price less the underwriting discount.

The underwriters expect to deliver the shares of common stock against payment on or about February 19, 2015.

Goldman, Sachs & Co. **J.P. Morgan**

Piper Jaffray

The date of this prospectus supplement February 12, 2015.

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Prospectus Supplement

Accompanying Prospectus

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ABOUT THIS PROSPECTUS SUPPLEMENT

This document is in two parts. The first part is the prospectus supplement, including the documents incorporated or deemed to be incorporated herein by reference, which describes the specific terms of this offering. The second part, the accompanying prospectus, including the documents incorporated or deemed to be incorporated therein by reference, provides more general information about us and our securities. Generally, when we refer to this prospectus, we are referring to both parts of this document combined together with all documents incorporated or deemed incorporated by reference. Before you invest, you should carefully read this prospectus supplement, the accompanying prospectus, all information incorporated or deemed to be incorporated by reference herein and therein, as well as the additional information described under Where You Can Find More Information on page S-57 of this prospectus supplement. These documents contain information you should consider when making your investment decision. This prospectus supplement may add, update or change information contained in the accompanying prospectus. To the extent there is a conflict between the information contained in this prospectus supplement, on the one hand, and the information contained in the accompanying prospectus or any document incorporated or deemed to be incorporated by reference therein filed prior to the date of this prospectus supplement, on the other hand, you should rely on the information in this prospectus supplement. If any statement in one of these documents is inconsistent with a statement in another document having a later date for example, a document filed after the date of this prospectus supplement and deemed to be incorporated by reference in this prospectus supplement and the accompanying prospectus the statement in the document having the later date modifies or supersedes the earlier statement.

You should rely only on the information contained in or incorporated or deemed to be incorporated by reference in this prospectus supplement, the accompanying prospectus and in any free writing prospectuses we may provide to you in connection with this offering. We have not, and the underwriters have not, authorized any other person to provide you with any information that is different. If anyone provides you with different or inconsistent information, you should not rely on it. We are offering to sell, and seeking offers to buy, shares of our common stock only in jurisdictions where offers and sales are permitted. The distribution of this prospectus supplement and the offering of the common stock in certain jurisdictions may be restricted by law. Persons outside the United States who come into possession of this prospectus supplement must inform themselves about, and observe any restrictions relating to, the offering of the common stock and the distribution of this prospectus supplement outside the United States. This prospectus supplement does not constitute, and may not be used in connection with, an offer to sell, or a solicitation of an offer to buy, any securities offered by this prospectus supplement by any person in any jurisdiction in which it is unlawful for such person to make such an offer or solicitation.

We further note that the representations, warranties and covenants made by us in any agreement that is filed as an exhibit to any filing that is incorporated or deemed to be incorporated by reference into this prospectus supplement or the accompanying prospectus were made solely for the benefit of the parties to such agreement, including, in some cases, for the purpose of allocating risk among the parties to such agreements, and should not be deemed to be a representation, warranty or covenant to you. Moreover, such representations, warranties or covenants were accurate only as of the date when made. Accordingly, such representations, warranties and covenants should not be relied on as accurately representing the current state of our affairs.

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PROSPECTUS SUPPLEMENT SUMMARY

This summary highlights selected information about us, this offering and information appearing elsewhere in this prospectus supplement, in the accompanying prospectus and in the documents we incorporate by reference. This summary is not complete and does not contain all the information you should consider before investing in our common stock pursuant to this prospectus supplement and the accompanying prospectus. Before making an investment decision, to fully understand this offering and its consequences to you, you should carefully read this entire prospectus supplement and the accompanying prospectus, including Risk Factors beginning on page S-12 of this prospectus supplement and the financial statements and related notes and the other information that we incorporated by reference herein, including our Annual Report on Form 10-K and Quarterly Reports on Form 10-Q, that we file from time to time.

Idera Pharmaceuticals, Inc.

Overview

We are a clinical-stage biopharmaceutical company focused on the discovery, development and commercialization of novel therapeutics for oncology and rare diseases. We use two distinct proprietary drug discovery technology platforms to design and develop drug candidates. We developed these platforms based on our scientific expertise and pioneering work with synthetic oligonucleotides as therapeutic agents. Using our Toll-like receptor, or TLR, targeting technology, we design synthetic oligonucleotide-based drug candidates to act by modulating the activity of specific TLRs. In addition, using our gene silencing oligonucleotide, or GSO, technology, we are developing GSOs to turn off the messenger RNA, or mRNA, associated with disease causing genes. We consider our GSO technology to be a third generation antisense technology that can potentially reduce the immunotoxicity and increase the potency of gene silencing oligonucleotides.

Our business strategy focuses on the development of drug candidates for oncology and rare diseases, as we believe we can develop and commercialize targeted therapies on our own in disease indications characterized by small, well-defined patient populations with serious unmet medical needs. To the extent we seek to develop drug candidates for broader disease indications, we plan to execute early-stage development through proof-of-concept clinical trials and explore potential collaborative alliances to support late-stage development and commercialization.

RESEARCH AND DEVELOPMENT PROGRAMS

Drug Candidate(s)	Indication / Application	Development Status
Programs for the Modulation of Specific Toll-like Receptors		
Oncology		
B-cell Lymphomas with MYD88 L265P oncogenic mutation		
IMO-8400	Waldenström s Macroglobulinemia	Phase 1/2 clinical trial Anticipated completion and data in the fourth quarter of 2015
IMO-8400	Diffuse Large B-Cell Lymphoma	Phase 1/2 clinical trial Currently screening patients

for enrollment

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Drug Candidate(s)	Indication / Application	Development Status
Immuno-oncology		
IMO-2055/IMO-2125	Intratumoral Combination with Checkpoint Inhibitors	Two Phase 1/2 Clinical Trials Planned initiation in the second half of 2015
Rare Diseases		
IMO-8400	Dermatomyositis	Phase 2 Clinical Trial Planned initiation by the end of 2015
IMO-8400	Duchenne Muscular Dystrophy	Phase 1/2 Clinical Trial Planned initiation in early 2016
Autoimmune Diseases		
IMO-9200	Selected Autoimmune Disease	Preclinical studies and Phase 1 trial in healthy subjects ongoing
Gene Silencing Oligonucleotides		
Discovery Candidates	Inhibition of Gene Expression by Targeting RNA	Research

TLR Modulation Technology Platform

TLRs play a central role in the innate immune system by regulating signaling cascades that stimulate inflammation. As a result, we believe TLRs are potential therapeutic targets for the treatment of a broad range of diseases. Using our chemistry-based platform, we have designed TLR antagonists and agonists to act by modulating the activity of targeted TLRs. A TLR antagonist is a compound that inhibits an immune response by downregulating the targeted TLR. A TLR agonist is a compound that stimulates an immune response through the targeted TLR.

Our TLR antagonist lead drug candidates are IMO-8400 and IMO-9200, which are both antagonists of TLR7, TLR8 and TLR9. We also have created compounds that are agonists of TLR3, TLR7, TLR8 and TLR9. Our TLR agonist lead drug candidates are IMO-2055 and IMO-2125, which are both agonists of TLR9.

Our lead drug candidate is IMO-8400, a novel synthetic oligonucleotide antagonist of TLR7, TLR8, and TLR9. Currently, we are developing IMO-8400 for the treatment of certain genetically defined forms of B-cell lymphoma and for the treatment of rare diseases. We also are conducting a Phase 1 clinical trial of IMO-9200 in healthy subjects, as well as additional preclinical studies of IMO-9200 for a selected autoimmune disease. In addition, we are planning to advance at least one of our TLR9 agonists, IMO-2055 or IMO-2125, into clinical development for intratumoral injection in combination with checkpoint inhibitors for selected oncology targets.

IMO-8400 Development Program in Genetically Defined Forms of B-cell Lymphoma

We are developing IMO-8400 for the treatment of certain B-cell lymphomas in which the MYD88 L265P oncogenic mutation is present. Oncogenic mutations are changes in the DNA of tumor cells that promote the survival and proliferation of tumor cells. MYD88 is an adaptor protein in the TLR signaling pathway that mediates TLR signaling. The MYD88 L265P oncogenic mutation has been reported to lead to increased TLR signaling and malignant proliferation in certain B-cell lymphomas, including

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Waldenström s macroglobulinemia, diffuse large B-cell lymphoma, or DLBCL, and other forms of B-cell malignancies, including Burkitt s lymphoma, cutaneous diffuse large B-cell lymphoma (leg type), chronic lymphocytic leukemia, gastric mucosa-associated lymphoid tissue lymphoma, marginal zone lymphoma, and splenic marginal zone lymphoma.

We believe, based on independent research and our own preclinical research, that the inhibition of specific TLRs may be a useful approach in the treatment of certain B-cell lymphomas in which the MYD88 L265P oncogenic mutation is present. In independent research reported by investigators from the National Cancer Institute at the American Association for Cancer Research Annual Meeting in 2013, it was shown that the MYD88 L265P oncogenic mutation over-activated TLR7 and TLR9-mediated signaling and that inhibition of TLR7 and TLR9 promoted tumor cell death in preclinical models.

In addition, in preclinical studies of IMO-8400 that we presented in April 2014 at the American Association for Cancer Research Annual Meeting, and in August 2014 at both the 18th International Workshop on Waldenström s Macroglobulinemia and at the American Society of Hematology Meeting on Lymphoma Biology, IMO-8400 induced cell death in human Waldenström s macroglobulinemia tumor cells and in DLBCL tumor cells harboring the MYD88 L265P oncogenic mutation. These results were observed in preclinical studies evaluating IMO-8400 as a monotherapy and in combination with rituximab. Consistent with its proposed mechanism of action, IMO-8400 treatment in these studies inhibited cell signaling pathways that promote tumor cell survival and proliferation including those referred to scientifically as IRAK1/4, NF- KB, STAT3, p38, and BTK. Further, in these studies, IMO-8400 suppressed tumor cell production of cytokines, such as interleukin-10, or IL-10, that create a favorable microenvironment for tumor cell survival and proliferation. In addition, in preclinical studies in xenograft models, IMO-8400 decreased tumor burden in mice, even where treatment was initiated after tumors had become well established. In these same studies, tumor cells that did not harbor the MYD88 L265P oncogenic mutation were not affected by IMO-8400 treatment, demonstrating the specificity of the treatment effect in these cells.

Based on independent research, we believe that approximately 90% of patients with Waldenström s macroglobulinemia and approximately 10% of patients with DLBCL have the MYD88 L265P oncogenic mutation. We believe that this prevalence data, together with preclinical data generated by us with IMO-8400, supports our plans to develop IMO-8400 in Waldenström s macroglobulinemia and in DLBCL.

In December 2014, we announced that the FDA had granted orphan drug designation for IMO-8400 for the treatment of Waldenström s macroglobulinemia. Orphan drug designation is granted by the FDA Office of Orphan Products Development to drugs intended to treat a rare disease or condition that affects fewer than 200,000 people in the United States. This designation provides certain incentives, including eligibility for federal grants, research and development tax credits, a waiver of PDUFA filing fees and a seven-year marketing exclusivity period, once the product is approved and as long as orphan drug designation is maintained.

Prior to commencing our ongoing clinical trials of IMO-8400, we conducted a Phase 1 clinical trial of IMO-8400 in healthy subjects and a Phase 2 clinical trial of IMO-8400 in patients with moderate to severe psoriasis. To date, we have administered more than 550 doses of IMO-8400 to more than 85 healthy subjects and patients.

Phase 1/2 Clinical Trial of IMO-8400 in Waldenström s Macroglobulinemia. In 2014, we initiated patient treatment in our ongoing open-label, dose-escalation Phase 1/2 clinical trial of IMO-8400 in patients with Waldenström s macroglobulinemia who have relapsed or were refractory to prior therapy.

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Objectives of the trial include evaluation of safety and tolerability of escalating IMO-8400 dose levels and assessment of IMO-8400 clinical activity using disease-specific international guidelines for classifying clinical response. In this trial, we are evaluating doses of 0.6, 1.2 and 2.4 mg/kg per week administered as subcutaneous injections for 24 weeks. For the 2.4 mg/kg dose level, we are administering IMO-8400 in two doses of 1.2 mg/kg per week. We expect to enroll up to approximately 30 patients in this trial.

As of January 31, 2015, we had enrolled patients at each of the three dose levels. In each case, we advanced dosing to the higher dose level upon the recommendation of an independent committee following its review of safety data from the trial. We plan to complete this trial and have the full data available during the fourth quarter of 2015.

Phase 1/2 Trial of IMO-8400 in Diffuse Large B-cell Lymphoma. We are also conducting an open-label, dose-escalation Phase 1/2 clinical trial of IMO-8400 in patients with DLBCL who have relapsed or were refractory to prior therapy. With the concurrence of the FDA Center for Devices and Radiological Health, or CDRH, we plan to enroll in this trial only patients who are positive for the presence of the MYD88 L265P oncogenic mutation. Objectives of the trial include evaluation of safety and tolerability of escalating IMO-8400 dose levels and assessment of IMO-8400 clinical activity using disease-specific international guidelines for classifying clinical response. In this trial, we plan to evaluate escalating doses of 0.6, 1.2 and 2.4 mg/kg per week, administered as subcutaneous injections for 24 weeks. For each dose level, we are administering IMO-8400 subcutaneously in equally divided doses given twice per week. We expect to enroll up to approximately 30 patients in this trial. As of January 31, 2015, we had activated multiple clinical sites and initiated screening of potential study participants for the MYD88 L265P oncogenic mutation. We plan to complete this trial and have the full data available during 2016.

We believe that Waldenström s macroglobulinemia and DLBCL in patients with the MYD88 L265P oncogenic mutation are rare diseases with serious unmet medical needs, based on prevalence of the indications and our understanding of the current treatment paradigms. If we observe sufficient tolerability and a therapeutic effect in either or both of our Phase 1/2 clinical trials, we plan to meet with regulatory authorities to discuss the possibility of an accelerated clinical development and regulatory path for the applicable program. We cannot predict whether or when any of our drug candidates will prove effective or safe in humans, if we will be able to participate in FDA expedited review and approval programs, including breakthrough and fast track designation, or if they will receive regulatory approval.

Companion Diagnostic for MYD88 L265P. In May 2014, we entered into a collaboration with Abbott Molecular, Inc., or Abbott Molecular, for the development of a companion diagnostic that can be used to identify patients with the MYD88 L265P oncogenic mutation. Under the agreement, Abbott Molecular is primarily responsible for developing and obtaining regulatory approvals for the companion diagnostic test in accordance with an agreed development plan and regulatory plan and for making the companion diagnostic test commercially available in accordance with an agreed commercialization plan.

In November 2014, Abbott Molecular completed initial development of the prototype companion diagnostic for the MYD88 L265P oncogenic mutation. We have incorporated the prototype companion diagnostic into our ongoing Phase 1/2 clinical trial of IMO-8400 in patients with DLBCL.

Application of TLR Agonists in Immuno-Oncology

Our pipeline of drug candidates includes IMO-2055 and IMO-2125, two TLR9 agonists that may have potential applications as immune therapies for the treatment of cancer. Recent advancements in cancer immunotherapy have included the approval and late-stage development of multiple checkpoint inhibitors, which are therapies that target mechanisms by which tumor cells evade detection by the

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immune system. Because TLR9 agonists stimulate the immune system, we believe that there is a scientific rationale to evaluate the combination of our TRL9 agonists with checkpoint inhibitors. In independent research in preclinical cancer models, intratumoral injection of TLR9 agonists has potentiated the anti-tumor activity of checkpoint inhibitors. We believe that intratumoral injection of our TLR9 agonists activates a local immune response at the tumor which complements the systemic effect of the checkpoint inhibitors. We believe that, these data support evaluation of combination regimens including a TLR9 agonist and a checkpoint inhibitor for the treatment of cancer.

We and our collaborators have previously conducted clinical trials of IMO-2055 and IMO-2125. In these clinical trials, IMO-2055 was evaluated as a monotherapy and in combination with other oncology therapeutics in more than 300 patients with various types of cancers, and IMO-2125 was evaluated in more than 95 patients with hepatitis C. To support future potential development in cancer, we have conducted preclinical studies in which our TLR9 agonists have demonstrated anti-tumor activity in combination with the checkpoint inhibitor ipilimumab, an anti-CTLA4 antibody marketed as Yervoy® by Bristol-Myers Squibb Company. In December 2014, we presented data at the American Association for Cancer Research (AACR) Tumor Immunology and Immunotherapy Meeting from a preclinical study in which IMO-2055 delivered intratumorally in combination with ipilimumab demonstrated potent and systemic anti-tumor activity in multiple preclinical cancer models, including increased and sustained inhibition of treated and distant tumor growth in preclinical models of lung, colon and bladder cancer compared to treatment with either agent alone. We are conducting preclinical studies to characterize potential combination regiments with various checkpoint inhibitors. We intend to initiate clinical development of either IMO-2055 or IMO-2125 in combination with these checkpoint inhibitors by submitting an IND for, and initiating, two Phase 1/2 clinical trials in the second half of 2015.

Program in Rare Diseases

We are planning to initiate clinical development of IMO-8400 for the treatment of rare diseases. We have selected dermatomyositis and Duchenne muscular dystrophy, or DMD as the first non-cancer rare diseases for which we plan to develop IMO-8400. We selected these indications for development based on the reported increase in TLR expression in these disease states, expression of cytokines indicative of key TLR-mediated pathways, the identification of prospective biomarkers for evaluation in early clinical trials and with respect to dermatomyositis, the presence of auto-antibodies that can induce TLR-mediated immune responses. We anticipate commencing clinical development in these two indications by initiating a Phase 2 clinical trial in dermatomyositis by the end of 2015 and a Phase 1/2 clinical trial in DMD in early 2016.

In determining whether to proceed in these two rare diseases, we considered that multiple independent research studies across a broad range of autoimmune diseases, including both dermatomyositis and psoriasis, have demonstrated that the over-activation of TLRs plays a critical role in disease maintenance and progression. In autoimmune diseases, endogenous nucleic acids released from damaged or dying cells initiate signaling cascades through TLRs, leading to the induction of multiple pro-inflammatory cytokines. This inflammation causes further damage to the body s own tissues and organs and the release of more self-nucleic acids, creating a self-sustaining autoinflammatory cycle that contributes to chronic inflammation in the affected tissue, promoting disease progression. Research studies have shown a similar pathological amplification cycle in DMD, where endogenous nucleic acids are released from leaky dystrophin-deficient skeletal muscle cells. We believe that TLR antagonism has the potential to improve patient outcomes by disrupting these disease processes.

We believe that we demonstrated proof of concept for our approach of using TLRs to inhibit the over-activation of specific TLRs for the treatment of psoriasis and potentially other autoimmune

diseases in a randomized, double-blind, placebo-controlled Phase 2 clinical trial of IMO-8400 that we conducted in patients with moderate to severe plaque psoriasis, a well-characterized autoimmune disease. In this study, we evaluated IMO-8400 at four subcutaneous dose levels of 0.075, 0.15, 0.3, and 0.6 mg/kg, versus placebo, administered once weekly for 12 weeks in 44 patients. The trial met its primary objective as IMO-8400 was well tolerated at all dose levels with no treatment-related discontinuations, treatment-related serious adverse events or dose reductions. The trial also met its secondary objective of demonstrating clinical activity in psoriasis patients, as assessed by the Psoriasis Area Severity Index. We plan to present additional results from this Phase 2 clinical trial at a future medical congress. With our focus on rare diseases, like dermatomyositis and DMD, we do not currently plan to conduct further clinical development of IMO-8400 for the treatment of psoriasis.

IMO-8400 Development Program for Dermatomyositis. Myositis is a group of rare chronic inflammatory muscle disorders that cause muscle destruction, and includes dermatomyositis. Major symptoms of dermatomyositis include muscle tissue loss, muscle weakness, joint pain and difficulty swallowing, with skin involvement resulting in rash and/or calcinosis. Potential complications of dermatomyositis include severe disability, interstitial lung disease and cancer. In this form of myositis, over-activated TLRs stimulate a pro-inflammatory response leading to further damage of muscle, skin and other tissue. Current treatments, including corticosteroids and immunosuppressive agents, often provide limited benefit or have unfavorable safety profiles, and there is a significant unmet medical need for new therapies to treat dermatomyositis.

In August 2014, we initiated a collaboration with The Myositis Association, or TMA, a leading U.S. patient advocacy organization focused on myositis, to advance the clinical development of IMO-8400 for the treatment of myositis. Under the collaboration, we and TMA agreed to develop educational programs for patients and healthcare providers on TLR antagonism and opportunities to participate in clinical research. In addition, we formed an advisory committee of leading independent experts in the treatment of myositis to advise us on the development of IMO-8400 in myositis. Based on these ongoing efforts, we have focused our development strategy on dermatomyositis, a form of the disease in which there is muscle and skin involvement. We are finalizing our clinical trial plan for a Phase 2 clinical trial of IMO-8400 in dermatomyositis and anticipate initiating this trial by the end of 2015. If this clinical trial is successful, we may evaluate the potential of IMO-8400 to treat additional forms of myositis.

IMO-8400 Development Program for Duchenne Muscular Dystrophy. DMD is an X-linked genetic disorder characterized by progressive muscle weakness leading to severe disability, pulmonary and cardiac dysfunction and death in affected males, typically before age 30. Patients with DMD lack dystrophin, a critical muscle protein, resulting in excessive muscle damage following normal exercise. Damaged muscle cells release endogenous nucleic acids that stimulate TLRs, thereby activating a pro-inflammatory response that propagates a cycle of further muscle cell damage and destruction. In a research article published in Human Molecular Genetics in January 2014, we and scientists from Children s National Medical Center, Washington, DC, reported that, in preclinical studies, over-expression of TLR7 exacerbated inflammation and caused muscle degeneration in an *mdx* mouse model of DMD. In addition, in studies with the *mdx* mouse model of DMD, an antagonist of TLR7 and TLR9 significantly reduced muscle inflammation and increased muscle force, providing support for TLR antagonism as a potential treatment approach for DMD.

Current pharmacologic treatment of DMD is generally limited to corticosteroids, which have been shown to have side effects in children including behavioral changes, short stature from slow growth rate, weight gain, facial puffiness known as Cushingoid appearance, and cataracts. The most advanced investigational therapies in development are designed to correct for certain genetic

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mutations, representing small percentages of the total affected DMD population, enabling production of new dystrophin protein. We believe TLR antagonism is a potential non-steroid-based anti-inflammatory treatment approach for all DMD patients regardless of their genetic mutation.

We are conducting additional preclinical studies of TLR antagonist drug candidates in DMD models and are working in collaboration with Parent Project Muscular Dystrophy, or PPMD, a leading U.S. patient advocacy organization, on the design of a clinical development program for IMO-8400 in DMD. We anticipate initiating a Phase 1/2 clinical trial of IMO-8400 in DMD in early 2016.

Program in Auto-Immune Diseases

IMO-9200 for Autoimmune Disease. We have developed a second novel synthetic oligonucleotide antagonist of TLR7, TLR8, and TLR9, IMO-9200, as a drug candidate in clinical development for potential use in selected autoimmune disease indications. In October 2014, we initiated subcutaneous dosing in a Phase 1 clinical trial of IMO-9200 in healthy subjects. We have also initiated additional preclinical studies of IMO-9200 for a selected autoimmune disease.

Gene Silencing Oligonucleotide Technology to Target RNA

We are developing our GSOs to turn off the mRNA associated with disease causing genes. We have designed our GSOs to specifically address challenges associated with earlier generation antisense and RNA interference, or RNAi, technologies. Although currently used technologies to silence RNA have demonstrated the ability to inhibit the expression of disease-associated proteins, we believe that to reach their full therapeutic potential, gene silencing technologies need to achieve an improved therapeutic index with efficient systemic delivery without using a delivery technology, reduced immunotoxicity and increased potency. We have designed our GSOs to provide these attributes. For example, in preclinical studies, our GSOs have exerted gene-silencing activity in animals following systemic administration. Preclinical data also have shown that systemic delivery of GSOs targeted to the mRNA of apolipoprotein B and proprotein convertase subtilisin/kexin type 9 (PCSK9), which are proteins associated with cardiovascular diseases, resulted in reduced serum total cholesterol and low-density-lipoprotein cholesterol, in addition to reduced levels of the targeted mRNA and associated proteins. Additionally, in mouse models, systemic administration of GSOs showed significant specific gene-silencing activity with minimized induction of immune responses.

We are currently undertaking an analysis of oncology and rare disease indications for development of drug candidates from our GSO technology. Our key considerations in identifying disease indications in our GSO program include: strong evidence that the disease is caused by a specific protein; clear criteria to identify a target patient population; biomarkers for early assessment of clinical proof-of-concept; a targeted therapeutic mechanism for action; and unmet medical need to allow for a rapid development path to approval. We are planning to conduct disease model studies and begin IND-enabling development programs in each of the first two disease indications selected for further development in our GSO program in the second half of 2015.

Cash Position and Funding Requirements

We had cash and cash equivalents of approximately $58.3 million as of September 30, 2014. We estimate that we had cash, cash equivalents and investments of approximately $48.6 million as of December 31, 2014. Our estimate of our cash, cash equivalents and investments as of December 31, 2014 is an estimate prepared by management in good faith based upon internal reporting and expectations as of and for the three months ended December 31, 2014. This estimate is preliminary, and unaudited, and may be revised as a result of management s further review of our results. We

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and our auditors have not completed the normal annual audit procedures as of and for the year ended December 31, 2014, and there can be no assurance that our final results for this annual period will not differ from this estimate.

We believe that the net proceeds of this offering, together with our existing cash, cash equivalents and investments, will enable us to fund our operations into the first quarter of 2017. Specifically, we believe that our available funds following this offering will be sufficient to enable us to:

- complete our ongoing Phase 1/2 clinical trial of IMO-8400 in patients with Waldenström s macroglobulinemia and our ongoing Phase 1/2 clinical trial of IMO-8400 in patients with DLBCL harboring the MYD88 L265P oncogenic mutation;
- initiate two Phase 1/2 clinical trials involving either IMO-2055 or IMO-2125 in combination with a checkpoint inhibitor for selected oncology targets and complete at least one of these trials;
- initiate a Phase 2 clinical trial of IMO-8400 in patients with dermatomyositis and a Phase 1/2 clinical trial of IMO-8400 in patients with DMD;
- complete our ongoing Phase 1 clinical trial of IMO-9200 in healthy subjects; and
- conduct disease model studies and begin IND-enabling development programs in each of the first two disease indications selected for further development in our GSO program.

We expect that we will need to raise additional funds in order to conduct any other clinical development of our TLR drug candidates or to conduct any other development of our GSO technology.

Corporate Information

Our offices are located at 167 Sidney Street, Cambridge, Massachusetts 02139 and 760 Constitution Drive, Suite 14, Exton, Pennsylvania 19341, our telephone number is (617) 679-5500 and our Internet address is www.iderapharma.com. The information on our Internet website is not incorporated by reference in this prospectus and should not be considered to be part of this prospectus. Our website address is included in this prospectus as an inactive technical reference only. Unless the context otherwise requires, references in this prospectus to Idera Pharmaceuticals, we, us, and our refer to Idera Pharmaceuticals, Inc.

Idera® and IMO® are our trademarks. All other trademarks and service marks appearing in this prospectus are the property of their respective owners.

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THE OFFERING

Common stock offered by us	20,000,000 shares.
Common stock to be outstanding after this offering	114,829,040 shares.
Underwriters Option	The underwriters have a 30-day option to purchase up to an additional 3,000,000 shares of our common stock from us.
Use of proceeds	We estimate that the net proceeds to us from this offering, after deducting underwriting discounts and commissions and estimated offering expenses payable by us, will be approximately $70,060,000, or approximately $80,635,000 if the underwriters exercise their option to purchase additional shares from us in full. We plan to use the net proceeds from this offering, together with our existing cash, cash equivalents and investments, to advance the clinical development of our TLR antagonists in our genetically defined B-cell lymphoma program and our rare disease program, the development of our TLR agonists in our immuno-oncology program, and the development of our GSOs under our GSO program; and for working capital and other general corporate purposes. Please see Use of Proceeds on page S-42.
Risk factors	See Risk Factors beginning on page S-12 of this prospectus supplement, as well as the other information included in or incorporated by reference in this prospectus supplement and the accompanying prospectus, for a discussion of risks you should carefully consider before investing in our securities.
Nasdaq Capital Market listing	IDRA

The number of shares of our common stock to be outstanding after this offering set forth above is based on 94,829,040 shares of our common stock outstanding as of December 31, 2014.

Unless otherwise indicated, all information in this prospectus, including the number of shares of our common stock to be outstanding after this offering set forth above, excludes the following:

16,950,988 shares of common stock issuable upon exercise of stock options outstanding as of December 31, 2014, at a weighted-average exercise price of $3.56 per share;

2,804,945 shares of common stock reserved as of December 31, 2014 for future issuance under our equity incentive plans;

1,926 shares of common stock reserved as of December 31, 2014 for issuance upon any conversion of our outstanding Series A convertible preferred stock, or Series A preferred stock;

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35,536,417 shares of common stock issuable upon exercise of warrants outstanding as of December 31, 2014, at a weighted average exercise price of $0.63 per share; and

22,151,052 shares of common stock issuable upon exercise of pre-funded warrants outstanding as of December 31, 2014, at an exercise price of $0.01 per share.

In addition, this prospectus reflects and assumes no exercise of outstanding options or warrants since December 31, 2014. Unless we specifically state otherwise, all information in this prospectus supplement assumes that the underwriters do not exercise the option to purchase up to 3,000,000 additional shares of our common stock.

Entities affiliated with two of our directors, Julian C. Baker and Dr. Kelvin M. Neu, have agreed to purchase an aggregate of 5,333,333 shares of the common stock offered in this offering at the price offered to the public.

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RISK FACTORS

Investing in our securities involves a high degree of risk. You should carefully consider the risks described below, together with all of the other information included or incorporated by reference in this prospectus supplement, the accompanying prospectus and the documents we incorporate by reference, before making an investment decision. Our business, financial condition and results of operations could be materially and adversely affected by any of these risks or uncertainties. In that case, the market price of our common stock could decline, and you may lose all or part of your investment in our securities.

Risks Relating to Our Financial Results and Need for Financing

We will need additional financing, which may be difficult to obtain. Our failure to obtain necessary financing or doing so on unattractive terms could result in the termination of our operations and the sale and license of our assets or otherwise adversely affect our research and development programs and other operations.

We had cash and cash equivalents of approximately $58.3 million as of September 30, 2014. We estimate that we had cash, cash equivalents and investments of approximately $48.6 million as of December 31, 2014. Our estimate of our cash, cash equivalents and investments as of December 31, 2014 is an estimate prepared by management in good faith based upon internal reporting and expectations as of and for the three months ended December 31, 2014. This estimate is preliminary, unaudited and may be revised as a result of management s further review of our results. We and our auditors have not completed the normal annual audit procedures as of and for the year ended December 31, 2014, and there can be no assurance that our final results for this annual period will not differ from this estimate.

We believe that the net proceeds of this offering, together with our existing cash, cash equivalents and investments, will enable us to fund our operations into the first quarter of 2017. Specifically, we believe that our available funds following this offering will be sufficient to enable us to:

- complete our ongoing Phase 1/2 clinical trial of IMO-8400 in patients with Waldenström s macroglobulinemia and our ongoing Phase 1/2 clinical trial of IMO-8400 in patients with DLBCL harboring the MYD88 L265P oncogenic mutation;
- initiate two Phase 1/2 clinical trials involving either IMO-2055 or IMO-2125 in combination with a checkpoint inhibitor for selected oncology targets and complete at least one of these trials;
- initiate a Phase 2 clinical trial of IMO-8400 in patients with dermatomyositis and a Phase 1/2 clinical trial of IMO-8400 in patients with DMD;
- complete our ongoing Phase 1 clinical trial of IMO-9200 in healthy subjects; and
- conduct disease model studies and begin IND-enabling development programs in each of the first two disease indications selected for further development in our GSO program.

We expect that we will require substantial additional funds beyond the proceeds of this offering to conduct any additional research and development of our TLR drug candidates or GSO technology, including preclinical testing and clinical trials of our drug candidates, and to fund our operations. We are seeking and expect to continue to seek additional funding through collaborations, the sale or license of assets or financings of equity or debt securities. We believe that the key factors that will affect our ability to obtain funding are:

- the results of our clinical and preclinical development activities in our genetically defined forms of B-cell lymphoma and rare disease programs, our immuno-oncology program, and our GSO program and our ability to advance our drug candidates and GSO technology on the timelines anticipated;

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- the cost, timing, and outcome of regulatory reviews;
- competitive and potentially competitive products and technologies and investors receptivity to our drug candidates and the technology underlying them in light of competitive products and technologies;
- the receptivity of the capital markets to financings by biotechnology companies generally and companies with drug candidates and technologies such as ours specifically; and
- our ability to enter into additional collaborations with biotechnology and pharmaceutical companies and the success of such collaborations.

In addition, increases in expenses or delays in clinical development may adversely impact our cash position and require additional funds or further cost reductions.

Additional financing may not be available to us when we need it or may not be available to us on favorable or acceptable terms or at all. We could be required to seek funds through collaborative alliances or through other means that may require us to relinquish rights to some of our technologies, drug candidates or drugs that we would otherwise pursue on our own. In addition, if we raise additional funds by issuing equity securities, our then existing stockholders will experience dilution. The terms of any financing may adversely affect the holdings or the rights of existing stockholders. An equity financing that involves existing stockholders may cause a concentration of ownership. Debt financing, if available, may involve agreements that include covenants limiting or restricting our ability to take specific actions, such as incurring additional debt, making capital expenditures or declaring dividends, and are likely to include rights that are senior to the holders of our common stock. Any additional debt financing or equity that we raise may contain terms, such as liquidation and other preferences, or liens or other restrictions on our assets, which are not favorable to us or our stockholders.

If we are unable to obtain adequate funding on a timely basis or at all, we will be required to terminate, modify or delay preclinical or clinical trials of one or more of our drug candidates, significantly curtail or terminate discovery or development programs for new drug candidates or relinquish rights to portions of our technology, drug candidates and/or products.

We have incurred substantial losses and expect to continue to incur losses. We will not be successful unless we reverse this trend.

We have incurred losses in every year since our inception, except for 2002, 2008, and 2009 when our recognition of revenues under license and collaboration agreements resulted in our reporting net income for those years. As of September 30, 2014, we had an accumulated deficit of $439.6 million. Since January 1, 2001, we have primarily been involved in the development of our TLR pipeline. From January 1, 2001 to September 30, 2014, we incurred losses of $179.4 million. We incurred losses of $260.2 million prior to December 31, 2000, during which time we were primarily involved in the development of non-TLR-targeted antisense technology. These losses, among other things, have had and will continue to have an adverse effect on our stockholders equity, total assets, and working capital.

We have never had any products of our own available for commercial sale and have received no revenues from the sale of drugs. As of September 30, 2014, almost all of our revenues have been from collaborative and license agreements. We have devoted substantially all of our efforts to research and development, including clinical trials, and have not completed development of any drug candidates. Because of the numerous risks and uncertainties associated

with developing drugs, we are unable to predict the extent of any future losses, whether or when any of our drug candidates will become commercially available, or when we will become profitable, if at all. We expect to incur substantial operating losses in future periods.

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Risks Relating to Our Business, Strategy and Industry

We are depending heavily on the development of TLR-targeted drug candidates for the treatment of certain genetically defined forms of B-cell lymphoma and rare diseases and in our immuno-oncology program and on the development of our GSO technology. If we terminate the development of any of our programs or any of our drug candidates in such programs, are unable to successfully develop and commercialize any of our drug candidates, or experience significant delays in doing so, our business may be materially harmed.

We have invested a significant portion of our time and financial resources in the development of TLR-targeted clinical stage lead drug candidates as part of our rare disease program. In the future, we intend to invest a significant portion of our time and financial resources in the development of our TLR-targeted candidates for the treatment of certain genetically defined forms of B-cell lymphoma and rare diseases and in our immuno-oncology program. We also plan to invest substantial time and resources to further advance the development of our GSOs under our GSO program. For instance:

- we initiated a Phase 1/2 clinical trial of IMO-8400 in patients with Waldenström s macroglobulinemia and a Phase 1/2 clinical trial of IMO-8400 in patients with DLBCL harboring the MYD88 L265P oncogenic mutation;
- we are planning to conduct two Phase 1/2 clinical trials involving either IMO-2055 or IMO-2125 in combination with a checkpoint inhibitor for selected oncology targets;
- we are planning to conduct a Phase 2 clinical trial of IMO-8400 in patients with dermatomyositis and a Phase 1/2 clinical trial of IMO-8400 in patients with DMD;
- we initiated a Phase 1 clinical trial of IMO-9200 in healthy subjects; and
- we are planning to conduct disease model studies and begin IND-enabling development programs in each of the first two disease indications selected for further development in our GSO program.

We anticipate that our ability to generate product revenues will depend heavily on the successful development and commercialization of our TLR drug candidates in our genetically defined forms of B-cell lymphoma, rare disease and immuno-oncology programs, and the successful identification, development and commercialization of drug candidates in our GSO program.

Our ability to generate product revenues under our collaboration with Merck Sharp & Dohme Corp. (formerly Merck & Co., Inc.), or Merck & Co., and under any other collaboration that we enter into with respect to our other programs, will depend on the development and commercialization of the drug candidates being developed.

Our efforts, and the efforts of Merck & Co., to develop and commercialize these compounds are at an early stage and are subject to many challenges. We have experienced setbacks with respect to our programs for IMO-3100, a TLR7 and TLR9 antagonist, IMO-2125, and IMO-2055, including:

In July 2011, the FDA placed a clinical hold on the protocol that we had submitted for a phase 2 clinical trial of IMO-3100 that we planned to conduct in patients with psoriasis in light of some reversible immune responses that were observed in 13-week nonclinical toxicology studies of IMO 3100 that were inconsistent with observations made in our other nonclinical studies of IMO-3100.

In April 2011, we chose to delay initiation of our planned 12-week Phase 2 randomized clinical trial of IMO-2125 plus ribavirin in treatment-naïve, genotype 1 hepatitis C virus, or HCV, patients based on observations of lymphoproliferative malignancies in an ongoing 26-week chronic nonclinical toxicology study of IMO-2125 in rodents. We subsequently completed a 39-week chronic nonclinical toxicology study of IMO-2125 in non-human primates in which there were no similar observations.

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In July 2011, Merck KGaA, Darmstadt, Germany, or Merck KGaA, informed us that, based on increased incidence of neutropenia and electrolyte imbalances reported in its Phase 1 clinical trial of IMO-2055 in combination with cisplatin/5-FU and cetuximab in patients with first-line squamous cell carcinoma of the head and neck, or SCCHN, and subsequent re-evaluation of its clinical development program, Merck KGaA had determined that it would not conduct further clinical development of IMO-2055. In May 2012, we announced that in the Phase 2 clinical trial of IMO-2055 in combination with cetuximab in patients with second-line SCCHN, the combination of IMO-2055 and cetuximab did not meet the primary endpoint of the trial.

We are conducting multiple clinical trials of IMO-8400 in different indications. If patients in any of these trials experience adverse safety events, we may be required to delay, discontinue or modify all of our clinical trials of IMO-8400.

We may seek to enter into collaborative alliances with pharmaceutical companies to advance our TLR antagonist candidates in broader autoimmune disease indications and with respect to applications of our GSO technology program. Our previous setbacks with respect to our programs for IMO-3100, IMO-2125, and IMO-2055 could negatively impact our ability to license any of such compounds to a third party.

Our ability to successfully develop and commercialize these drug candidates, or other potential candidates, will depend on our ability to overcome these recent challenges and on several factors, including the following:

- the drug candidates demonstrating activity in clinical trials;
- the drug candidates demonstrating an acceptable safety profile in nonclinical toxicology studies and during clinical trials;
- timely enrollment in clinical trials of IMO-8400 and other drug candidates, which may be slower than anticipated, potentially resulting in significant delays;
- satisfying conditions imposed on us and/or our collaborators by the FDA or equivalent foreign regulatory authorities regarding the scope or design of clinical trials;
- the ability to demonstrate to the satisfaction of the FDA, or equivalent foreign regulatory authorities, the safety and efficacy of the drug candidates through current and future clinical trials;
- timely receipt of necessary marketing approvals from the FDA and equivalent foreign regulatory authorities;
- the ability to combine our drug candidates and the drug candidates being developed by Merck & Co. and any other collaborators safely and successfully with other therapeutic agents;

achieving and maintaining compliance with all regulatory requirements applicable to the products;

establishment of commercial manufacturing arrangements with third-party manufacturers;

the successful commercial launch of the drug candidates, assuming FDA approval is obtained, whether alone or in combination with other products;

acceptance of the products as safe and effective by patients, the medical community, and third-party payors;

competition from other companies and their therapies;

changes in treatment regimens;

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the strength of our intellectual property portfolio in the United States and abroad; and

a continued acceptable safety and efficacy profile of the drug candidates following marketing approval.

We have recently begun to focus our efforts on the research and development of drug candidates for use in the treatment of certain genetically defined forms of B-cell lymphoma, and our approach for the treatment of these genetically defined B-cell lymphomas is novel and may not result in any approved and marketable products.

We are in the early stages of developing our program in genetically defined forms of B-cell lymphoma, an area in which we have little experience. In connection with this program, we are focusing our efforts on the research and development of TLR antagonist drug candidates for use in the treatment of certain genetically defined forms of B-cell lymphoma. The scientific evidence to support the feasibility of developing drug candidates for this use is both preliminary and limited. We have conducted preclinical studies in human lymphoma cell lines that carry the MYD88 L265P oncogenic mutation to evaluate our TLR antagonists as a potential approach to the treatment of certain genetically defined forms of B-cell lymphoma. Although the preliminary results of our preclinical studies have been promising, it is unknown whether these results are indicative of results that may be obtained in our clinical trials. Therefore, we do not know if our approach of inhibiting TLRs to treat patients with genetically defined forms of B-cell lymphoma will be successful or if we will ever succeed in obtaining regulatory approval to market any product for this purpose. In addition, in the event that our development efforts for such a drug candidate progress towards commercialization, we likely will need to develop companion diagnostics for such drug candidate. We have no experience in developing companion diagnostics and will be dependent on the efforts of third-party collaborators to successfully develop and commercialize these companion diagnostics on our behalf. In May 2014, we entered into an agreement with Abbott Molecular to develop a companion diagnostic for identification of patients with B-cell lymphomas harboring the MYD88 L265P oncogenic mutation. We cannot assume that the program under this agreement will be successful.

We are in the early stages of developing our GSO program, which is a novel technology, and our efforts may not be successful or result in any approved and marketable products.

We are in the early stages of developing our GSO technology program, and the scientific evidence to support the feasibility of developing drugs based on this technology is preliminary. Further, neither we nor any other company has received regulatory approval to market therapeutics utilizing GSOs.

The future success of our GSO technology program depends on our success in identifying and developing marketable products based on such technology. Although the results of our preclinical studies to date have been supportive of the viability of this technology, it is unknown whether these results are indicative of results that may be obtained in any future clinical trials that we may conduct. We are currently undertaking an analysis of priority oncology and rare disease indications and development strategies to determine next steps in developing our GSO technology, and are planning to conduct disease model studies and begin IND-enabling development programs in each of the first two disease indications selected for further development in our GSO program in the second half of 2015. However, many steps must be successfully achieved prior to the declaration of a GSO-based drug candidate and the initiation of clinical development. Given the level of uncertainty of our ability to successfully achieve these many steps and the uncertainty of the drug discovery and clinical development processes in general, there can be no assurance that we will succeed in developing any marketable product as a result of our efforts with respect to our GSO technology program.

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If we experience delays or difficulties in the enrollment of patients in clinical trials, our receipt of necessary regulatory approvals could be delayed or prevented.

We may not be able to initiate or continue clinical trials for our drug candidates if we are unable to locate and enroll a sufficient number of eligible patients to participate in these trials as required by the FDA or similar regulatory authorities outside the United States. In particular, because there are a limited number of patients with Waldenström s macroglobulinemia or patients with DLBCL harboring the MYD88 L265P oncogenic mutation, and a limited number of patients with dermatomyositis, DMD, or other rare diseases having indications for which we may determine to develop our TLR antagonists, our ability to enroll eligible patients in any clinical trials for these indications may be limited or may result in slower enrollment than we anticipate. In addition, the relapsed or refractory DLBCL patients that we are seeking to enroll in our Phase 1/2 clinical trial of IMO-8400, typically have progressed disease with a severe prognosis. As a result, some patients for which we have initiated screening may not survive to complete screening for the MYD88 L265P oncogenic mutation. If enrolled, the disease in these patients may be too progressed for them to receive any benefit from treatment or for their treatment to contribute meaningful data to the clinical trial. In addition, some of our competitors have ongoing clinical trials for drug candidates that treat the same indications as our drug candidates, and patients who would otherwise be eligible for our clinical trials may instead enroll in clinical trials of our competitors drug candidates.

Patient enrollment is affected by other factors including:

- the severity of the disease under investigation;
- the eligibility criteria for the study in question;
- the perceived risks and benefits of the TLR antagonist drug candidates under study;
- the efforts to facilitate timely enrollment in clinical trials;
- the availability of competing clinical trials or other therapies;
- the patient referral practices of physicians;
- the ability to monitor patients adequately during and after treatment; and
- the proximity and availability of clinical trial sites for prospective patients.

Our inability to enroll a sufficient number of patients for our clinical trials would result in significant delays and could require us to abandon one or more clinical trials altogether. Enrollment delays in our clinical trials may result in increased development costs for our drug candidates, which would cause the value of our company to decline and limit our ability to obtain additional financing.

If our clinical trials are unsuccessful, or if they are delayed or terminated, we may not be able to develop and commercialize our drug candidates.

In order to obtain regulatory approvals for the commercial sale of our drug candidates, we are required to complete extensive clinical trials in humans to demonstrate the safety and efficacy of our drug candidates. Clinical trials are lengthy, complex, and expensive processes with uncertain results. We may not be able to complete any clinical trial of a potential product within any specified time period. Moreover, clinical trials may not show our potential products to be both safe and efficacious. The FDA or other equivalent foreign regulatory agencies may not allow us to complete these trials or commence and complete any other clinical trials. For example, in July 2011, the FDA placed a clinical hold on a protocol we had submitted for a proposed Phase 2 clinical trial of IMO-3100 in patients with psoriasis.

The results from preclinical testing of a drug candidate that is under development may not be predictive of results that will be obtained in human clinical trials. In addition, the results of early human clinical trials may not be predictive of results that will be obtained in larger scale, advanced stage

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clinical trials. Furthermore, interim results of a clinical trial do not necessarily predict final results, and failure of any of our clinical trials can occur at any stage of testing. Companies in the biotechnology and pharmaceutical industries, including companies with greater experience in preclinical testing and clinical trials than we have, have suffered significant setbacks in clinical trials, even after demonstrating promising results in earlier trials. Moreover, effects seen in nonclinical studies, even if not observed in clinical trials, may result in limitations or restrictions on clinical trials. Numerous unforeseen events may occur during, or as a result of, preclinical testing, nonclinical testing or the clinical trial process that could delay or inhibit the ability to receive regulatory approval or to commercialize drug products.

Only one TLR-targeted drug, imiquimod, which is marketed as Aldara® and Zyclara® by Meda AB, Graceway Pharmaceuticals LLC, and iNova Pharmaceuticals (Australia) Pty Limited has been approved by the FDA. Other companies developing drugs targeted to TLRs have experienced setbacks in clinical trials. For example in 2007, Coley Pharmaceutical Group, which since has been acquired by Pfizer, Inc., discontinued four clinical trials for PF-3512676, its investigational TLR9 agonist compound, in combination with cytotoxic chemotherapy in cancer, and suspended its development of Actilon®, a TLR9 agonist, for HCV infection. In July 2007, Anadys Pharmaceuticals, Inc. and its partner Novartis AG, or Novartis, discontinued the development of ANA975, the investigational TLR7 agonist compound for HCV infection. Dynavax Technologies Corporation, or Dynavax, announced in May 2008 discontinuation of the clinical development program for TOLAMBA®, an investigational vaccine which contained a TLR9 agonist adjuvant, and in February 2013 Dynavax announced receipt of a Complete Response Letter from FDA regarding its Biological License Application for HEPLISAV®, which is an investigational hepatitis B vaccine that contains a TLR9 agonist adjuvant. These setbacks may result in enhanced scrutiny by regulators or institutional review boards, or IRBs, of clinical trials of our drug candidates, including our TLR-targeted drug candidates, which could result in regulators or IRBs prohibiting the commencement of clinical trials, requiring additional nonclinical studies as a precondition to commencing clinical trials or imposing restrictions on the design or scope of clinical trials that could slow enrollment of trials, increase the costs of trials or limit the significance of the results of trials. Such setbacks could also adversely impact the desire of investigators to enroll patients in, and the desire of patients to enroll in, clinical trials of our drug candidates.