

Xencor Inc
Form 10-K/A
February 23, 2015
Table of Contents

UNITED STATES

SECURITIES AND EXCHANGE COMMISSION

WASHINGTON, DC 20549

FORM 10 K/A

(Amendment No. 1)

(Mark One)

ANNUAL REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE
ACT OF 1934

For the fiscal year ended December 31, 2014

or

TRANSITION REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE
ACT OF 1934

For the transition period from to

Commission file number: 001 36182

Xencor, Inc.

(Exact Name of Registrant as Specified in its Charter)

Delaware	20 1622502
(State or Other Jurisdiction of Incorporation or Organization)	(I.R.S. Employer Identification No.)
111 West Lemon Avenue, Monrovia, CA	91016
(Address of Principal Executive Offices)	(Zip Code)

(626) 305 5900

(Registrant's Telephone Number, Including Area Code)

Securities registered pursuant to Section 12(b) of the Act:

Title of Each Class

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	Name of Each Exchange on Which Registered
Common Stock, par value \$0.01 per share	The NASDAQ Stock Market LLC

Securities registered pursuant to Section 12(g) of the Act: None

Indicate by check mark if the registrant is a well known seasoned issuer, as defined in Rule 405 of the Securities Act. Yes No

Indicate by check mark if the registrant is not required to file reports pursuant to Section 13 or 15(d) of the Act. Yes No

Indicate by check mark whether the registrant: (1) has filed all reports required to be filed by Section 13 or 15(d) of the Securities Exchange Act of 1934 during the preceding 12 months (or for such shorter period that the registrant was required to file such reports), and (2) has been subject to such filing requirements for the past 90 days. Yes No

Indicate by check mark whether the registrant has submitted electronically and posted on its corporate Web site, if any, every Interactive Data File required to be submitted and posted pursuant to Rule 405 of Regulation S-T (§232.405 of this chapter) during the preceding 12 months (or for such shorter period that the registrant was required to submit and post such files). Yes No

Indicate by check mark if disclosure of delinquent filers pursuant to Item 405 of Regulation S-K is not contained herein, and will not be contained, to the best of the registrant's knowledge, in definitive proxy or information statements incorporated by reference in Part III of this Form 10-K or any amendment to this Form 10-K.

Indicate by check mark whether the registrant is a large accelerated filer, an accelerated filer, a non-accelerated filer or a smaller reporting company. See definitions of "large accelerated filer", "accelerated filer" and "smaller reporting company" in Rule 12b-2 of the Exchange Act.

Large accelerated filer Accelerated filer Non-accelerated filer Smaller reporting company
(Do not check if a smaller reporting company)

Indicate by check mark whether the registrant is a shell company (as defined in Rule 12b-2 of the Securities Exchange Act of 1934). Yes No

The aggregate market value of the voting and non-voting common equity held by non-affiliates computed by reference to the price at which the common equity was last sold as of June 30, 2014 was \$151,512,499

The number of outstanding shares of the registrant's common stock, par value \$0.01 per share, as of February 6, 2015 was 31,472,763.

DOCUMENTS INCORPORATED BY REFERENCE

Portions of the registrant's proxy statement to be filed with the Securities and Exchange Commission pursuant to Regulation 14A in connection with the registrant's 2015 Annual Meeting of Stockholders, which will be filed subsequent to the date hereof, are incorporated by reference into Part III of this Form 10-K. Such proxy statement will be filed with the Securities and Exchange Commission not later than 120 days following the end of the registrant's

fiscal year ended December 31, 2014.

Table of Contents

EXPLANATORY NOTE

The sole purpose of this Amendment No. 1 to Xencor, Inc.'s Annual Report on Form 10-K (the "Form 10-K") for the period ended December 31, 2014, as filed with the Securities and Exchange Commission on February 20, 2015, is to correct clerical errors in Exhibits 23.1, 31.1 and 31.2 attached to the Form 10-K.

In connection with the filing of this Amendment and pursuant to the rules of the Securities and Exchange Commission, we are including with this Amendment new certifications by our principal executive and principal financial officer.

Except as described above, no other changes have been made to the Original Filing. The Original Filing continues to speak as of the date of the Original Filing, and we have not updated the disclosures contained therein to reflect any events which occurred at a date subsequent to the filing of the Original Filing other than as expressly indicated in this Amendment.

Table of Contents

Xencor, Inc.

FORM 10 K

For the Fiscal Year Ended December 31, 2014

Table of Contents

	Page
<u>PART I</u>	
<u>Item 1</u>	3
<u>Item 1A</u>	36
<u>Item 1B</u>	61
<u>Item 2</u>	61
<u>Item 3</u>	61
<u>Item 4</u>	62
<u>PART II</u>	
<u>Item 5</u>	62
<u>Item 6</u>	64
<u>Item 7</u>	65
<u>Item 7A</u>	82
<u>Item 8</u>	84
<u>Item 9</u>	116
<u>Item 9A</u>	116
<u>Item 9B</u>	117
<u>PART III</u>	
<u>Item 10</u>	117
<u>Item 11</u>	118
<u>Item 12</u>	118
<u>Item 13</u>	118
<u>Item 14</u>	118
<u>PART IV</u>	
<u>Item 15</u>	119
<u>Signatures</u>	123

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Table of Contents

PART I

Forward Looking Statements

This Annual Report on Form 10-K or this Annual Report, may contain “forward looking statements” within the meaning of the federal securities laws made pursuant to the safe harbor provisions of the Private Securities Litigation Reform Act of 1995. We have based these forward looking statements largely on our current expectations and projections about future events and financial trends affecting the financial condition of our business. Forward looking statements should not be read as a guarantee of future performance or results, and will not necessarily be accurate indications of the times at, or by, which such performance or results will be achieved. Forward looking statements are based on information available at the time those statements are made and/or management’s good faith belief as of that time with respect to future events, and are subject to risks and uncertainties that could cause actual performance or results to differ materially from those expressed in or suggested by the forward looking statements. Our actual results could differ materially from those anticipated in these forward looking statements as a result of various factors, including those set forth below under Part I, Item 1A, “Risk Factors” in this Annual Report. These statements, which represent our current expectations or beliefs concerning various future events, may contain words such as “may,” “will,” “expect,” “anticipate,” “intend,” “plan,” “believe,” “estimate” or other words indicating future results. Such statements may include, but are not limited to, statements concerning the following:

- the initiation, cost, timing, progress and results of our research and development activities, preclinical studies and future clinical trials;
- our ability to obtain and maintain regulatory approval of our future product candidates, and any related restrictions, limitations, and/or warnings in the label of an approved product candidate;
- our ability to obtain funding for our operations;
 - our plans to research, develop and commercialize our future product candidates;
- our strategic alliance partners’ election to pursue development and commercialization;
- our ability to attract collaborators with development, regulatory and commercialization expertise;
- our ability to obtain and maintain intellectual property protection for our future product candidates;
- the size and growth potential of the markets for our future product candidates, and our ability to serve those markets;
- our ability to successfully commercialize our future product candidates;
- the rate and degree of market acceptance of our future product candidates;
- our ability to develop sales and marketing capabilities, whether alone or with potential future collaborators;
- regulatory developments in the United States and foreign countries;
- the performance of our third party suppliers and manufacturers;
- the success of competing therapies that are or become available;
- the loss of key scientific or management personnel;
- our expectations regarding the time during which we will be an emerging growth company under the JOBS Act;
- our use of the proceeds from our recently completed initial public offering and private placement; and
- the accuracy of our estimates regarding expenses, future revenues, capital requirements and need for additional financing.

Table of Contents

Given these uncertainties, you should not place undue reliance on these forward looking statements. These forward looking statements represent our estimates and assumptions only as of the date of this Annual Report on Form 10 K and, except as required by law, we undertake no obligation to update or revise publicly any forward looking statements, whether as a result of new information, future events or otherwise after the date of this Annual Report on Form 10 K. We qualify all of our forward looking statements by these cautionary statements.

Item 1. Business.

Our Business

We are a clinical stage biopharmaceutical company focused on discovering and developing engineered monoclonal antibodies to treat severe and life threatening diseases with unmet medical needs. We use our proprietary XmAb technology platform to create next generation antibody product candidates designed to treat autoimmune and allergic diseases, cancer and other conditions. In contrast to conventional approaches to antibody design, which focus on the portion of antibodies that interact with target antigens, we focus on the portion of the antibody that interacts with multiple segments of the immune system. This portion, referred to as the Fc domain, is constant and interchangeable among antibodies. Our engineered Fc domains, the XmAb technology, can be readily substituted for natural Fc domains. We believe our Fc domains enhance antibody performance by, for example, increasing immune inhibitory activity, improving cytotoxicity, extending circulating half life or stabilizing novel antibody structures, while maintaining 99.5% identity in structure and sequence to natural antibodies. By improving over natural antibody function, we believe that our XmAb engineered antibodies offer innovative approaches to treating disease and potential clinical advantages over other treatments.

Our business strategy is based on the plug and play nature of the XmAb technology platform to modify features of natural antibodies and create numerous differentiated antibody product candidates. We have internally generated a pipeline that has allowed us to selectively partner certain development programs while maintaining full ownership of other programs. We also have a number of technology licenses under which we have licensed the XmAb technology platform to pharmaceutical and biotechnology companies for use in a limited number of programs, providing multiple revenue streams that require no further resources from Xencor. There are currently eight antibody product candidates in clinical trials that have been engineered with XmAb technology, including six candidates being advanced by licensees and development partners.

Our internally generated pipeline includes the following three lead XmAb engineered antibodies that are currently in development:

- XmAb5871, our most recently advanced wholly-owned program recently completed a Phase 1b/2a clinical trial in rheumatoid arthritis (RA) and we are planning to initiate an open-label pilot clinical trial in IgG4-related disease (IgG4-RD) to assess disease control activity measured by the IgG4-RD Responder Index (Carruthers, et al., 2012, Int J Rheum) in 2015. XmAb5871 uses our XmAb Immune Inhibitor Fc Domain and targets B cells, an important component of the immune system. In January 2015, we announced that in the Phase 2a part of the trial 15 XmAb5871-treated patients and eight placebo-treated patients were evaluable for RA disease activity at the protocol specified disease activity assessment time point of two weeks following the sixth biweekly infusion. 33% of patients (5 of 15) that received all six biweekly doses of XmAb5871 achieved DAS28-CRP remission or low disease activity compared to zero patients that were treated with the placebo achieving DAS28-CRP remission or low disease activity. Three ACR70 responses (20%) and six ACR50 responses (40%) occurred in the XmAb5871 group compared to zero and one (13%) respectively in the placebo group. ACR70 and ACR50 responses refer, respectively to 70% and 50% reductions in the American College of Rheumatology rheumatoid arthritis symptom scale, a common measure of RA disease activity. Across the entire Phase 1b/2a clinical trial, biweekly administration of XmAb5871 for 12 weeks was generally well tolerated. The most common XmAb5871 treatment

related adverse related events (AEs) observed were predominantly mild to moderate gastrointestinal toxicities (nausea, vomiting, diarrhea) occurring during the first infusion of XmAb5871. These gastrointestinal AEs did not typically recur on subsequent infusions and no infusions were discontinued due to these AEs. Other treatment related AEs experienced in more than two XmAb5871 treated patients were pyrexia (fever) and headache. Treatment related serious adverse events (SAEs) occurred in two patients that received

Table of Contents

XmAb5871: infusion related reaction and venous thrombosis (blood clot). Two patients in the placebo treated group also reported SAEs.

We believe XmAb5871 has the potential to address a key unmet need in autoimmune therapies due to its combination of potent B-cell inhibition without B-cell depletion and will be exploring other indications in 2015. In October 2014, we announced that we sought and regained all rights to XmAb5871 from our partner, Amgen Inc. (Amgen), who had an option to acquire an exclusive worldwide license. In return, we granted Amgen a first right to negotiate a proposed license for XmAb5871 prior to seeking future partners. This right expires upon the earlier of the initiation of Phase 3 clinical testing of XmAb5871, a change of control of Xencor, or October 2019.

- XmAb7195 is our wholly-owned program being developed for the treatment of severe asthma and allergic diseases. It uses our XmAb Immune Inhibitor Fc Domain and is designed to reduce blood serum levels of IgE, which mediates allergic responses and allergic disease. Its three specific mechanisms of action give it potential advantages over current therapies: (i) increased IgE binding, (ii) inhibition of IgE production and (iii) rapid clearance of IgE from circulation. In 2014 we filed an IND for XmAb7195 for allergic asthma with the FDA and in May initiated a Phase 1a single ascending dose clinical trial in healthy volunteers and in allergen-sensitive subjects with high IgE levels. In January 2015, we reported that interim data from the trial show rapid reduction of circulating free IgE levels to below the limit of detection at the end of the XmAb7195 infusion in 90% of XmAb7195 treated subjects that had detectable free IgE pre-dose, including those at the lowest dose evaluated of 0.3 mg/kg. Total IgE levels were also reduced in a parallel fashion. Two subjects with high pre-dose IgE levels (above 400 IU/mL) were treated with XmAb7195, one each at 0.75 mg/kg and 3.0 mg/kg doses, and both had reduction of free IgE levels to below the limit of detection lasting for at least one week. A dose limiting toxicity of transient, asymptomatic thrombocytopenia (low blood platelet count) was observed at the 3.0 mg/kg dose. The decrease in platelet count was transient with a minimum by 24 hours post-dose, recovery starting by 48 hours post-dose and near full platelet count recovery by study Day 8 in all cases, at which time serum drug concentrations still exceeded levels that eliminate detectable IgE. No evidence of thrombocytopenia has been observed in any of the clinical trials of XmAb5871, an anti-CD19 antibody with the identical XmAb Immune Inhibitor Fc domain as that of XmAb7195. Moderate urticaria (hives) was reported in a total of seven XmAb7195 treated subjects with an apparent correlation of dose with frequency of occurrence. In all cases regardless of dose, the signs/symptoms of urticaria were mild, non-diffuse and easily treated with oral antihistamine, and the study drug infusions were continued to completion without worsening of symptoms. Otherwise, there were no other adverse events that occurred in more than two XmAb7195 treated subjects. There were no serious adverse events reported and no subject discontinued Part 1 of trial early.
- XmAb5574/MOR208 is being developed by our partner MorphoSys AG (MorphoSys) for the treatment of blood based cancers and uses our XmAb Cytotoxic Fc Domain. In a Phase 1 clinical trial of XmAb5574/MOR208 completed by Xencor in patients with high-risk, heavily-pretreated chronic lymphocytic leukemia (CLL), in which the antibody showed encouraging signs of preliminary anti-tumor activity and an acceptable safety profile and was well tolerated. The trial protocol was amended to include a period of extended dosing for a total of eight patients at the 12 mg/kg dose to study the effect of longer duration of exposure on safety and response rate. Overall response rate by IWCLL 2008 criteria was 29.6% (eight partial responses in 27 evaluable patients). Using NCI-WG CLL 1996, response criteria resulted in a response rate of 66.7% (18 partial responses). At the highest dose studied, 12 mg/kg, 12 of 16 patients (75%) had a partial response by NCI-WG CLL 1996 and six patients (37.5%) had a partial response using additional CT criteria (IWCLL 2008). Median progression free survival for all patients was 199 days and for the extended treatment arm (at 12 mg/kg) was 420 days. Blood disease cleared in most patients, with median reduction in absolute lymphocyte count from baseline of 90.8%.

XmAb5574/MOR208 was generally well tolerated with no maximum-tolerated dose identified. Clinically-significant, treatment-related adverse events (AEs) classified as Grade 3 or higher occurred in 5 out of twenty-seven patients. The most frequent treatment-related AEs were infusion-related reactions, which were reported for 66.7% of patients, all of which were grade 1 or 2, and no reactions were seen following the first infusion. Treatment-related AEs each reported for 18.5% of patients were ALT increased, AST

Table of Contents

increased, neutropenia and thrombocytopenia; all other treatment-related AEs were reported for $\leq 15\%$ of patients. These events resolved, generally without requiring treatment, and did not lead to discontinuation.

MorphoSys is currently conducting two Phase 2 clinical trials of MOR208 in patients with B-cell acute lymphoblastic leukemia (B-ALL) and non-Hodgkin lymphomas (NHL).

In 2014 our development efforts for XmAb bispecific antibodies expanded. Bispecific antibodies are designed with two different variable domains to elicit biological effects that require simultaneous binding to two targets. Previously, industry efforts at bispecific antibody design have generally been frustrated by poor molecular stability, difficulties in production and short in vivo half-life. Xencor's XmAb® bispecific Fc domain technology is designed to maintain full-length antibody properties in a bispecific antibody, potentially enabling stable molecules with favorable in vivo half-life and allowing for the use of standard antibody production methods. These bispecific Fc domains are used to generate a broad array of novel drug candidates. In November 2014, we announced preclinical data from three programs using our XmAb bispecific Fc technology showing that bispecific antibodies targeting CD123, CD20 and CD38 antigens each activated T-cells to rapidly kill target cells from a single dose IV bolus in cynomolgus monkeys and demonstrated prolonged half-life of approximately one week in mice. We also announced that we had selected our lead anti-CD123xCD3 bispecific antibody, XmAb14045, for IND-enabling studies and cGMP process development and manufacturing.

In addition, we have licensed our XmAb technology to pharmaceutical and biotechnology companies for use in a limited number of their programs. These licensees include Boehringer Ingelheim International GmbH (Boehringer Ingelheim), CSL Limited (CSL), Janssen R&D, LLC (Janssen), Merck, Sharp and Dohme, a subsidiary of Merck & Co., Inc. (Merck), Alexion Pharmaceuticals, Inc. (Alexion), and collectively these licensees have five Phase 1 clinical development-stage programs and two pre-clinical development-stage programs. In December 2014, we announced a discovery-collaboration with Novo Nordisk A/S (Novo Nordisk) to jointly discover novel biologic drug candidates for an undisclosed target by combining multiple Xencor XmAb technologies, including our bispecific and immune inhibitor technologies.

A summary of all our licensed programs is shown below:

Licensee	Year	Xencor Technology	Indication	Milestones	Royalties	Current Development Stage
Boehringer Ingelheim	2007	Cytotoxic	Oncology	Yes	Yes	Phase 1 trials (two candidates)
Janssen	2009	Xtend	Autoimmune disease	Yes	Yes	Preclinical
CSL	2009	Cytotoxic	Oncology	Yes	Yes	Phase 1
Morphosys	2010	Cytotoxic	Oncology	Yes	Yes	Phase 2
CSL	2013	Xtend	Hematological diseases	Yes	Yes	Preclinical

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Merck	2013	Fc optimization	Autoimmune disease	Yes	Yes	Phase 1
Alexion	2013	Xtend	Undisclosed	Yes	Yes	Phase 1
Novo Nordisk	2014	Various, including Bi-specifics	Undisclosed	Yes	Yes	Preclinical

We were founded in 1997 based on protein engineering technology developed by our co-founders Bassil Dahiyat, Ph.D. and Stephen Mayo, Ph.D. at the California Institute of Technology. We began our first therapeutic monoclonal antibody engineering and discovery programs in 2002 and entered into our first XmAb technology license in 2004.

Our Strategy

Our goal is to become a leading biopharmaceutical company focused on developing and commercializing engineered monoclonal antibodies to treat severe and life-threatening diseases with unmet medical needs. Key elements of our strategy are to:

Table of Contents

- " Advance the clinical development of our lead Immune Inhibitor Fc Domain product candidates. We are developing XmAb5871 for the treatment of autoimmune diseases, including IgG4-related disease, and are developing XmAb7195 for the treatment of asthma and allergic diseases.
- " Build a large and diversified portfolio of product candidates. We aim to create new XmAb-engineered antibody product candidates that exploit the novel properties of our XmAb technology platform for preclinical and clinical development by us.
- " Continue to monetize and expand the use of our XmAb technology platform. We are seeking additional licensing and partnering opportunities, similar to our collaborations with MorphoSys and Novo Nordisk with other leading pharmaceutical and biotechnology companies.
- " Broaden the functionality of our XmAb technology platform. We are conducting further research into the function and application of antibody Fc domains in order to expand the scope of our XmAb technology platform. Our bispecific technology, which uses our heterodimeric Fc domain enabling molecules with dual target binding, is an example of the expanding functionality of our XmAb technology platform.
- " Continue to expand our patent portfolio protecting our XmAb technology platform. We seek to expand and protect our development programs and product candidates by filing and prosecuting patents in the United States and other countries.

Our Business

Antibodies as Therapeutic Agents

Antibodies are Y-shaped proteins that are produced by B cells and used by the immune system to target and neutralize foreign objects known as antigens. These objects may include tumor cells, bacteria and viruses. Antibodies are composed of two structurally independent parts, the variable domain (the Fv domain) and the constant domain (the Fc domain and the CH1 domain). The Fv domain is responsible for targeting a specific antibody to a specific antigen and is different for every type of antibody. The Fc domain interacts with various receptors on immune cells and other cells and, rather than binding antibodies to target antigens, it endows antibodies with properties beyond simple binding, such as immune response regulation and cytotoxicity. Importantly, Fc domains are the same and interchangeable from antibody to antibody.

Most antibody research to date has been based on the ability to discover and improve antigen-selective antibody Fv domains. Many pharmaceutical and biotechnology companies have efforts to discover, develop and commercialize antibody drugs using such Fv-based tools. A number of successful products have resulted from these efforts and the

Table of Contents

global market for antibody therapeutics was estimated to be approximately \$45 billion in 2011, of which the U.S. market was estimated to be \$20 billion.

Intense competition drives companies to develop differentiated antibody drugs, often because of the common pursuit of the same antigen Fv targets across the industry. Industry efforts have focused on engineering Fv domains since the mid-1980s to enhance performance. More recently, many efforts at differentiation have attempted to improve upon antibody performance by drastically changing the antibody structure or substituting new molecules altogether, for example, new antibody-like scaffolds, bi-specific antibodies and antibody-drug conjugates. A challenge to these efforts has been making these new drug molecules replicate the beneficial features of natural antibodies, including ease of production, safety, efficacy and simplicity. These efforts, however, have largely ignored the Fc domain.

In contrast, in the last decade Xencor has focused on Fc engineering. Fc engineering involves additional complexities, particularly consideration of simultaneous interactions with multiple Fc receptors and immune cell types and requires significant expertise in structural biology and immunology. We developed the XmAb technology to create significantly enhanced antibody performance while preserving over 99.5% of the natural antibody structure because we believe that maintaining native antibody structure could retain these beneficial features in our highly differentiated antibody candidates.

Our XmAb Technology Platform

Our XmAb Fc domain technology is a platform of antibody components that enable the creation of therapeutic antibody candidates that have novel interactions with the human immune and antibody regulation systems. We developed the XmAb technology platform from a systematic effort to engineer the Fc domain of antibodies to manipulate its interactions with a variety of its natural receptors. We used our patented screening technology, consisting of algorithms and computer models of the three-dimensional structure of the Fc domain, to focus on, from the vast number of possibilities, manageable sets of possible amino acid changes that result in small modifications to the Fc domain structure which effect significant changes in antibody function and performance.

We have identified a set of Fc domains, each of which is engineered to have a specific function based on its Fc receptor binding profile, including:

- Immune Inhibitor Fc Domain—selective immune inhibition and rapid target clearance, targeting the receptor Fc RIIB
- Cytotoxic Fc Domain—increased cytotoxicity, targeting the receptors Fc RIIIa on natural killer (NK) cells and Fc RIIa on other immune system cells
- Xtend Fc Domain—extended antibody half life, targeting the receptor FcRn on endothelial cells
- Bispecific Domain – heterodimeric Fc domains enabling molecules with dual target binding

In addition, we have engineered XmAb Fc domains with other properties, including rapid antigen clearance, antibody stability and multiple antigen specificity (heterodimer). Each XmAb Fc domain consists of a naturally occurring Fc domain with a small number of amino acid changes, usually two that we found to be critical for modulating interactions with the desired Fc receptors or manipulating Fc structural organization. With such limited modifications of the natural Fc domain, XmAb engineered antibodies are typically over 99.5% identical in structure and sequence to natural antibodies, simplifying product development yet enhancing function. In contrast to other engineering approaches for next-generation antibodies, we believe this conservative design allows our engineered antibodies to retain the beneficial stability, pharmacokinetics, and ease of discovery of natural antibodies, as well as to allow well-validated methods for antibody manufacturing. We believe we can thereby avoid the problems many new antibody platforms have had in production and drug stability.

Table of Contents

XmAb Immune Inhibitor Fc Domain technology

Fc RIIb is an inhibitory receptor that is expressed on B cells and other cells. Fc RIIb, when engaged by Fc domains, signals inside the cell to block immune response activation pathways, for example the B cell receptor pathway that activates in response to antigen recognition and ultimately results in the production of antibodies to antigen. We have focused on this role as an important negative feedback regulator of the B cell response, where its biology is well validated. Its expression and signaling characteristics have made it a difficult target for monoclonal antibodies, as targeting it by itself does not trigger its inhibitory properties. Fc RIIb must be associated with other specific partner proteins on the cell surface to activate its inhibitory properties. We have circumvented this problem by discovering variants of the Fc domain with enhanced binding to Fc RIIb and designed the Fv domain to target a B cell protein. This coupling of the two target proteins, in some cases, will trigger the inhibitory properties of Fc RIIb.

We have discovered a series of Fc RIIb immune inhibitor Fc variants with increased binding affinity to Fc RIIb of up to 400 fold. The high affinity variant has two amino acid substitutions in the Fc domain and has been applied to create our first immune inhibitor product development candidate XmAb5871. This antibody, described in greater detail below, targets CD19 on B cells through its variable domain and recruits Fc RIIb to induce its inhibitory properties. We have demonstrated in several preclinical studies that XmAb5871 inhibits B cell responses to a variety of stimuli. We have also applied this high affinity Immune Inhibitor Fc Domain to our anti IgE antibody XmAb7195, which as a result inhibits activation of only IgE positive B cells and hence prevents production of IgE, a key mediator of allergic response. Also, we have discovered an exciting new mechanism of action mediated by the XmAb Immune Inhibitor Fc Domains. High Fc RIIb binding causes very rapid clearance from the circulation of the complexes formed between XmAb7195 and IgE, a property that we believe is unique among IgE inhibitor antibodies. This provides another mechanism to lower the amount of circulating IgE.

Table of Contents

The rapid clearance mechanism of Immune Inhibitor Fc Domains offers a highly differentiating function for antibodies targeting soluble antigens, such as IgE, and opens opportunities for the technology beyond B cell modulation. For example, we are generating discovery candidates using XmAb Immune Inhibitor Fc Domains to clear pathologic targets from circulation.

XmAb Cytotoxic Fc Domain technology

Our XmAb Cytotoxic Fc Domain technology consists of a series of variant Fc domains that improve binding to the activating Fc receptors. This binding improvement drives increased antibody dependent cell cytotoxicity (ADCC), a primary mechanism of antibody cytotoxicity. The lead Fc variant used in nearly all of our Cytotoxic Fc Domain antibody candidates is an Fc domain with two amino acid substitutions that increase affinity for Fc RIIIa, the activating receptor expressed on natural killer (NK) cells, by approximately 40 fold. NK cells are cytotoxic lymphocytes of the innate immune system and play a major role in elimination of tumor cells and virally infected cells. Our XmAb Cytotoxic Fc Domain also increases affinity for Fc RIIa by approximately five fold, with potential for recruitment of other important effector cells such as macrophages, which play a role in both innate and adaptive immunity by engulfing and digesting foreign material. Fc RIIIa is considered an important mediator of the antitumor efficacy of antibodies such as Genentech's Herceptin (trastuzumab) and Biogen/Idc/Genentech's Rituxan (rituximab).

Numerous publications have demonstrated the importance of Fc receptors for anti tumor efficacy in mouse models and also in clinical studies of Rituxan and Herceptin. We have applied our Cytotoxic Fc Domain to a large number of validated (e.g. Rituxan, Herceptin, Bristol Myers Squibb and Eli Lilly and Company's Erbitux (cetuximab)) and unvalidated antibodies, and in all cases we have seen a marked increase of ADCC measured in vitro. We have established that the Cytotoxic Fc Domain technology increases the anti tumor efficacy of antibodies in a number of mouse models. In primate studies, we have shown that our anti CD19 antibody XmAb5574/MOR208, which incorporates our XmAb Cytotoxic Fc Domain, depletes monkey B cells whereas a similar anti CD19 antibody with an unmodified Fc domain did not successfully kill B cells.

In Phase 1 clinical studies, antibodies incorporating our XmAb Cytotoxic Fc Domain, for example XmAb2513 targeting CD30 in Hodgkin's lymphoma, have shown tumor reduction response rates comparable or superior to response rates in published reports of non Fc engineered antibodies against the same target cells. Several partners and licensees are using our Cytotoxic Fc Domain in their oncology antibodies, including four programs currently in clinical trials.

XmAb Xtend Fc Domain technology

Our XmAb Xtend Fc Domain technology consists of Fc domains designed to increase binding affinity to the receptor FcRn. FcRn is present inside lysosomes in endothelial cells lining the blood vessels and functions to rescue antibodies from the degradation that makes most proteins short lived in circulation. As a result of interactions with FcRn, all antibodies have half lives ranging from a few days to a few weeks, allowing less frequent dosing for antibody drugs than most other biologics. We have engineered a series of Fc variants that increase binding of the Fc domain to FcRn to enhance FcRn mediated rescue and thereby increase circulating half life. Our lead XmAb Xtend Fc Domain has two amino acid substitutions and has shown up to three fold increases of in vivo half life for a number of different antibodies in monkey models.

We believe extension of half life can be exploited to improve therapeutic antibody performance in several ways:

- Increased dosing interval, providing superior patient convenience and likely compliance. Such a reduced frequency of dosing also results in lower drug use in aggregate, reducing cost of goods.

- Lower drug quantities at the same dosing interval as the parent antibody. This can simplify dosage formulation and sometimes enable subcutaneous formulation. Cost of goods is reduced as well.
- Higher drug levels using the same dose and dosing interval as the parent antibody, resulting in longer drug exposure and potentially translating to better efficacy.

Table of Contents

We have licensed XmAb Xtend Fc Domain technology to several biopharmaceutical companies who are using XmAb Xtend Fc Domains to both improve existing antibody drugs and to create new drugs with long half lives. In the third quarter of 2014, our partner Alexion initiated a Phase 1 clinical trial with an undisclosed molecule to be used against an undisclosed target. It is the first human clinical trial with a molecule incorporating our XmAb Xtend Domain technology.

XmAb Bispecific Domain technology

Bispecific antibodies are designed with two different variable domains to elicit biological effects that require simultaneous binding to two targets. Previously, industry efforts at bispecific antibody design have generally been frustrated by poor molecular stability, difficulties in production and short in vivo half-life. Xencor's XmAb® bispecific Fc domain technology is designed to maintain full-length antibody properties in a bispecific antibody, potentially enabling stable molecules with favorable in vivo half-life and allowing for the use of standard antibody production methods. These bispecific Fc domains are used to generate a broad array of novel drug candidates.

We have created Fc variants that form heterodimeric Fc domains that enable the creation of bispecific antibodies that have different Fv domains on each side of the Fc domain in order to bind to a different antigen with each of their Fv domains. For example, we can readily create bispecific antibodies that bind both CD3 and a tumor antigen in order to recruit cytotoxic T cells to the tumor cell. Because of the Fc domain, these bispecific antibodies retain the long half-life and ease of production typical of standard antibodies. We have generated a number of bispecific antibody discovery programs using our XmAb heterodimer Fc domains and have demonstrated that several bispecific antibodies built on these Fc domains are highly active in primate models.

In November 2014, we announced preclinical data from three programs using our XmAb bispecific Fc technology showing that bispecific antibodies targeting CD123, CD20 and CD38 antigens each activated T-cells to rapidly kill target cells from a single dose IV bolus in cynomolgus monkeys and demonstrated prolonged half-life of approximately one week in mice. Our initial bispecific programs are tumor-targeted antibodies that contain both a tumor antigen binding domain (CD123, CD20, or CD38) and a cytotoxic T-cell binding domain (CD3).

We also announced in November 2014 that we had selected our lead anti-CD123xCD3 bispecific antibody, XmAb14045, for IND-enabling studies and cGMP process development and manufacturing. We plan to initiate clinical trials in acute myeloid leukemia with XmAb14045 in 2016. Additional development candidates against additional tumor targets are in discovery.

Additional XmAb Fc domains

We continue to design Fc domain variants and have identified improved functions in addition to those described above. Our goal is to remain at the forefront of antibody engineering by using our expertise in Fc domain engineering to create new functions for use in antibody therapeutics. We have Fc variants that improve complement dependent cytotoxicity. Other Fc variants have been engineered to eliminate binding to all Fc receptors, thereby creating Fc domains that have no cytotoxic effector function at all. Such domains have important use in therapeutics where no effector function is desired.

Antibody Fv domain engineering capabilities

We have developed tools to engineer humanized and fully human, high affinity antibody Fv domains. Usually starting from a mouse antibody Fv domain, we analyze its amino acid sequence computationally to find the best matches with human antibody sequences, which we then substitute into the murine Fv domain to create antibodies with very high

human sequence content. Our approach preserves the structural integrity of the antibody and maintains binding to antigen. We also perform antigen affinity enhancement by computationally filtering sequence changes and generating small, focused libraries of Fv variants that we screen for tighter binding. All of our internally discovered candidates, including XmAb5871, XmAb7195 and XmAb5574/MOR208, were generated using these tools.

Table of Contents

XmAb5871, a B cell Inhibitor for the Treatment of Autoimmune Diseases

Overview of XmAb5871

XmAb5871 is a monoclonal antibody for the treatment of autoimmune diseases that uses our XmAb Immune Inhibitor Fc domain to target Fc RIIb, an inhibitory receptor expressed on B cells and other immune cells, and through its Fv domain targets CD19, which is expressed on all B cells. By simultaneously targeting the B cell proteins, CD19 and Fc RIIb, XmAb5871 has an ability to engage the natural inhibitory pathway provided by Fc RIIb, preventing further activation of B cells by autoantigens and potentially also suppressing the ability of B cells to further provoke downstream autoimmune responses from T cells. CD19 and Fc RIIb are expressed broadly throughout B cell development, so we expect that XmAb5871 will confer broad suppression of B cell activation and downstream events such as antibody production. We have demonstrated that XmAb5871 inhibits B cell function in multiple animal models and in initial human clinical trials without destroying these important immune cells, in contrast to other B cell targeting therapies, such as Rituxan, that attack and destroy B cells. We believe the combination of potent inhibition without B cell depletion, which can lead to opportunistic infections, has the potential to address a key unmet need in autoimmune therapies. The coupling between CD19 and Fc RIIb, mediated by XmAb5871, promotes a strong negative signal in the B cell, preventing its activation and potentially blocking disease pathology in a variety of autoimmune and inflammatory conditions by broadly blocking all B cell populations. XmAb5871 is the first potential therapy that we are aware of that targets Fc RIIb inhibition.

Therapeutic Inhibition by XmAb5871 Mimics Natural Pathways. (A) B cell responses against antigen lead to antibody secretion, resulting in immunity and in some cases autoimmunity. (B) Excess antibodies produced in the B cell response can engage both the antigen and the inhibitory receptor Fc RIIb on the B cell surface, acting to control the immune response. (C) XmAb5871 mimics the natural feedback inhibition by targeting CD19, rather than the antigen, on the B cell surface and recruiting Fc RIIb to inhibit activation of the targeted B cell.

In December 2010, we entered into a collaboration and option agreement with Amgen for XmAb5871. During the option period, which was to expire upon completion of a data review period following our planned Phase 2b proof of concept (POC) clinical trial, we led research, development and manufacturing activities for XmAb5871 with collaborative input and development support from Amgen. Under the agreement, Amgen paid us an upfront payment and early development milestones and was obligated to pay additional milestones, both before and after payment of an

Table of Contents

option exercise fee, and royalties on sales following an exercise of the option by Amgen. In 2014 we approached Amgen to request an end to the collaboration to allow us to pursue a different clinical and commercial path from the original agreement. Amgen agreed and in October 2014 we announced that we had regained all rights to XmAb5871, subject to granting Amgen a right of first negotiation. The new agreement requires Xencor to first discuss with Amgen any proposed license prior to seeking other partners. This right expires upon the earlier of the initiation of Phase 3 clinical testing of XmAb5871, a change of control of Xencor, or October 2019. At that time we announced that we were not continuing development in RA and were planning clinical development in multiple autoimmune diseases where B-cell inhibition shows promise, including IgG4-RD.

Clinical Development Summary

The initial clinical trial application for XmAb5871 was approved by the United Kingdom Medicines and Healthcare Products regulatory agency in September 2011. To date, all clinical development for XmAb5871 has been conducted in western and central Europe. In December 2012, we completed a Phase 1a randomized, blinded, placebo-controlled, single ascending dose clinical trial to investigate the safety, tolerability and pharmacokinetics of XmAb5871 in healthy male adult volunteers. The primary objectives of this clinical trial were (1) to determine the safety and tolerability profile of single-dose intravenous administration of XmAb5871 and (2) to characterize the single-dose pharmacokinetics and immunogenicity of XmAb5871. We also included several biomarkers to evaluate the ability of XmAb5871 to suppress B-cell responses in treated subjects and we observed promising immunosuppressive activity against these biomarkers, including a tetanus antigen challenge, stimulated CD86 expression and B-cell count. XmAb5871 was well tolerated at all doses investigated. No subjects experienced a serious adverse event or a dose-limiting toxicity. The most frequently reported treatment related adverse events (AEs) were gastrointestinal AEs (including nausea, vomiting, abdominal pain, abdominal/epigastric discomfort and diarrhea) in 39% of subjects. All but one were of mild to moderate severity, with one subject experiencing severe nausea. All 48 subjects completed the clinical trial protocol. XmAb5871's half-life averaged 3.63 ± 1.24 days. Anti-drug antibodies (ADA) were detected in samples from 44% of the subjects. One subject with a positive ADA appeared to have an accelerated decline in XmAb5871 concentration. Other than this one subject there was no distinct evidence of ADA-mediated clearance of XmAb5871 in any other ADA positive subject. XmAb5871 was demonstrated to have promising immunosuppressive activity based on several biomarkers observed during the trial.

In January 2013, we initiated a Phase 1b/2a clinical trial in patients with active RA. This Phase 1b/2a clinical trial was a multi-center, randomized, placebo-controlled, double-blinded, multiple ascending dose study of the safety, tolerability, pharmacokinetics and pharmacodynamics of XmAb5871 in RA patients with active disease on stable non-biologic DMARD therapy. The primary objective of this clinical trial was to determine the safety and tolerability profile of biweekly, multiple-dose, intravenous administration of XmAb5871 in patients with RA. Secondary objectives were (1) to characterize the pharmacokinetics and immunogenicity of intravenously administered XmAb5871 in patients with RA and (2) to evaluate the effect of XmAb5871 on RA disease response as measured by changes in Disease Activity Score 28 using C-reactive protein (DAS28-CRP) at Week 13 for the Phase 2a part of this clinical trial. The clinical trial was conducted in two parts. In the Phase 1b part of this clinical trial, 29 RA patients with active disease on stable non-biologic DMARD therapy were enrolled into four consecutive dose cohorts (0.3 to 10.0 mg/kg) randomized approximately 6:2 (six XmAb5871 patients to two placebo patients), other than for the lowest dose, where it was 3:1. Each patient was administered XmAb5871 or placebo every 14 days for a total of six doses. In the Phase 2a part of this clinical trial, 27 patients were randomized 2:1 (two XmAb5871 patients to one placebo patient). The dose for this part of the trial was 10.0 mg/kg or placebo and patients were administered XmAb5871 or placebo every 14 days for a total of six doses.

Data from the Phase 2a part of the trial show promising activity in patients with RA, including multiple DAS28-CRP remissions and ACR50 and ACR70 responses. In the Phase 2a cohort of the trial 15 XmAb5871 treated patients and eight placebo treated patients were evaluable for RA disease activity at the protocol specified disease activity

assessment time point of two weeks following the sixth biweekly infusion. 33% of patients (5 of 15) that received all six biweekly doses of XmAb5871 achieved DAS28-CRP remission or low disease activity versus zero on placebo. Three ACR70 responses (20%) and six ACR50 responses (40%) occurred in the XmAb5871 group compared to zero and one (13%) respectively in the placebo group. Across the entire Phase 1b/2a clinical trial, biweekly administration of XmAb5871 for 12 weeks was generally well tolerated. The most common XmAb5871 treatment

Table of Contents

related adverse events (AEs) observed were predominantly mild to moderate gastrointestinal toxicities (nausea, vomiting, diarrhea) occurring during the first infusion of XmAb5871. These gastrointestinal AEs did not typically recur on subsequent infusions and no infusions were discontinued due to these AEs. Other treatment related AEs experienced in more than two XmAb5871 treated patients were pyrexia (fever) and headache. Treatment related serious adverse events (SAEs) occurred in two patients that received XmAb5871: infusion related reaction and venous thrombosis. Two patients in the placebo treated group also reported SAEs.

We have begun translational and mechanistic studies of XmAb5871 in the rare autoimmune disorder, immunoglobulin G4-related disease, or IgG4-RD. In 2015 we plan to file an investigational new drug application (IND) with the FDA and to initiate an open-label pilot clinical trial in patients with IgG4-RD to assess disease control activity as measured by the IgG4-RD Responder Index (Carruthers, et al., 2012, Int J Rheum).

IgG4-RD is a rare fibro-inflammatory autoimmune disorder that impacts approximately 10,000-20,000 patients in the United States, based on the incidence rate reported in Japan. IgG4-RD affects multiple organ systems and we believe is characterized by the presence of IgG4-positive plasmablast cells and the distinct histopathological appearance of diseased organs that is required for diagnosis. This objective histopathological diagnostic criterion is atypical for most autoimmune diseases and offers advantages for accurately identifying patients.

Preclinical Development

We have examined the ability of XmAb5871 to inhibit B cells in preclinical studies, including in vitro and in vivo studies. The observations in our preclinical studies include:

- " No depletion of human B cells in culture;
- " Inhibition of human B cells, including B cells donated by lupus and arthritis patients, stimulated by a variety of agents;
- " Suppression of antibody responses in humanized mouse models;
- " Suppression of disease in mouse models of arthritis and multiple sclerosis without B-cell depletion; and
- " Well tolerated at high doses in monkeys.

The lack of B-cell depletion is an important property of XmAb5871, giving it a potential safety advantage relative to B-cell depleting therapies like Rituxan. We have shown that XmAb5871 did not kill B cells in a culture of human blood cells over a wide concentration range. In contrast, Rituxan and XmAb5574, depleting antibodies for treating B-cell cancers, both significantly depleted B cells.

Table of Contents

The hallmark of B-cell activation is intracellular calcium mobilization. B cells taken from human donors can be stimulated in vitro resulting in a readily observable mobilization of calcium. In contrast, in the presence of XmAb5871, stimulation of the B cells leads to very slight calcium mobilization, barely detectable with our assays (figure below).

XmAb5871 suppresses calcium mobilization, a hallmark of B-cell activation. Upon stimulation, B cells treated with placebo showed an increase in calcium flux. In contrast, B cells treated with XmAb5871 showed a low calcium signal.

A second common measure of B-cell activation is their proliferation in response to various stimuli. In preclinical studies, we demonstrated XmAb5871 inhibits B-cell proliferation stimulated by anti-CD79b, IL-4, BLyS or lipopolysaccharide (LPS), a range of stimulants that signal through different pathways. The inhibition of the BLyS-mediated propagation is particularly notable given the recent approval of the anti-BLyS antibody Benlysta for treatment of lupus, suggesting that XmAb5871 inhibition includes the pathways blocked by Benlysta.

Because most autoimmune diseases involve contributions from T cells in addition to B cells, we examined the ability of XmAb5871 to reduce the propensity of the B cell to activate T cells. CD86 is the ligand for CD28 on T cells and their interaction is a major stimulant of T cells. For example, the blockade of CD86 by Bristol-Myers Squibb's Orencia (abatacept) is used as a treatment for rheumatoid arthritis and is also being investigated for the treatment of lupus. Upon B-cell stimulation, CD86 is increased on the B-cell surface, promoting the ability of the B cell to engage and activate the T-cell response. In the presence of XmAb5871, however, we observed that CD86 was significantly diminished. This observation led subsequently to the use of a similar assay as an activity biomarker for our Phase 1a clinical trial.

XmAb5871 was consistently immunosuppressive in mouse models of the human B-cell response. Because the antibody does not recognize mouse CD19 or mouse Fc γ RIIb, we used humanized mouse models (huSCID), in which human peripheral-blood cells, including B cells and T cells, are engrafted into an immune compromised mouse. These are well-established models and the human immune cells will normally react to immunization with antigen. Assuming that most of our human donors would have been vaccinated with tetanus toxoid, we set up humanized mouse models with a tetanus booster vaccination to see if XmAb5871 could suppress the anti-tetanus response (figure below). We ran the model numerous times and observed a robust anti-tetanus antibody response in untreated mice (the placebo control group), which we did not observe in mice treated with XmAb5871, indicating effective B-cell inhibition. Rituximab was included as a control, showing only intermediate suppression of the anti-tetanus antibody response. XmAb5871's ability to prevent antibody responses in these humanized mouse models suggests it might be capable of inhibiting antibody responses in general and thus autoantibody responses in humans with autoimmune diseases.

Table of Contents

XmAb5871 inhibited anti-tetanus antibody responses in mice engrafted with human B cells and immunized with tetanus.

We could not test XmAb5871 for activity in mouse disease models because of the lack of reactivity with the mouse CD19 and Fc γ 2b. Accordingly, we created an XmAb5871 surrogate antibody called XENP8206, which has an Fv domain that recognizes mouse CD19 and an Fc domain identical to XmAb5871. We then used mice transgenic for human Fc γ 2b as a background system for disease models. In these mice, the mouse Fc γ 2b gene has been replaced with the human Fc γ 2b gene so their Fc γ 2b receptor can be recognized by the XENP8206 Immune Inhibitor Fc Domain. In vitro experiments with B cells taken from the transgenic mice showed us that XENP8206 was capable of mimicking XmAb5871's B-cell inhibition activity, and that the activity was dependent on engagement of human Fc γ 2b. In a collagen-induced arthritis model, XENP8206-treated mice had little to no evidence of inflammation, whereas untreated mice had a 40% incidence of disease. XENP8206's ability to decrease symptoms in a mouse model of multiple sclerosis was at least as good as a Rituxan surrogate antibody, which caused complete depletion of the mouse B cells. XmAb5871's surrogate antibody XENP8206 did not cause significant B-cell depletion in our mouse studies.

We completed both 12-week and 24-week, multiple-dose, preclinical monkey toxicology studies of XmAb5871 and found no adverse events in doses up to 200 mg/kg. Additional preclinical work has also shown that XmAb5871 is capable of suppressing B cells donated by lupus and rheumatoid arthritis patients in both in vitro and in vivo models.

Market Opportunity

The indication for which we are currently pursuing XmAb5871 development is IgG4-RD, a newly designated disorder. IgG4-RD is a rare fibro-inflammatory autoimmune disorder that impacts approximately 10,000-20,000 patients in the United States, based on the incidence rate reported in Japan. IgG4-RD affects multiple organ systems and

Table of Contents

we believe is characterized by the distinct microscopic appearance of diseased organs, including the presence of IgG4-positive plasmablast cells that is required for diagnosis. There are currently no approved therapies for IgG4-RD and glucocorticoids (hormone steroids) are the current standard of care treatment.

The autoimmune disease therapeutic market generally presents an opportunity in various small and large market indications, some of which may be appropriate for XmAb14045. Currently marketed antibody-based products, include Rituxan (marketed under the trade name MabThera outside the United States), with 2012 worldwide sales for the treatment of several autoimmune indications of approximately \$1.1 billion, and GlaxoSmithKline's Benlysta (belimumab), with 2012 worldwide sales of over \$200.0 million for the treatment of lupus. We will continue to consider additional autoimmune diseases to determine whether XmAb5871 has potential as a treatment.

XmAb7195, an IgE Inhibitor for the Treatment of Asthma and Allergic Diseases

Overview

XmAb7195 is an anti IgE antibody engineered to reduce IgE levels for the treatment of asthma and other atopic diseases. Its three specific mechanisms of action give it potential advantages over current therapies: increased IgE affinity, inhibition of the transition of B cells to IgE secreting cells and rapid clearance of IgE from circulation.

- XmAb7195 is a humanized anti IgE antibody with an Fv domain that targets the same IgE epitope as Genentech and Novartis AG's Xolair (omalizumab), which is validated to block IgE. XmAb7195's affinity for IgE is approximately three times higher than that of Xolair. We believe that this contributes to the increased suppression of IgE observed in our preclinical studies.
- XmAb7195, in contrast to Xolair, has our XmAb Immune Inhibitor Fc Domain that has a 400 fold higher affinity than natural antibodies for Fc RIIb. XmAb7195 and XmAb5871 have the same Fc domain, but XmAb7195, unlike XmAb5871, inhibits only IgE positive B cells. By binding to Fc RIIb on IgE positive B cells, XmAb7195 suppresses their activation and differentiation into IgE secreting plasma cells. This binding reduces IgE production, a mechanism not seen with Xolair, and ultimately lowers IgE levels in the blood.
- XmAb7195 targets Fc RIIb, which results in rapid clearance of free and total IgE and inhibition of the function of IgE expressing B cells. Total IgE reduction differentiates XmAb7195 from other anti-IgE therapeutic antibodies, which actually increase total IgE levels. Because total IgE assays, unlike free IgE assays, are readily available to clinicians, the effect of XmAb7195 on total IgE levels could enable for the first time simple monitoring, and potentially adjustment of treatment effect.
- In our preclinical primate and other animal studies, we observed rapid reductions in IgE levels, even from the highly elevated levels found in chimpanzees, and rapid clearance of IgE from circulation. We did not observe any clearance or such magnitude of reduction with Xolair. This suggests a new mechanism of action in which high Fc RIIb binding causes very rapid clearance of the complexes formed between XmAb7195 and IgE in the liver. We believe XmAb7195 binds to Fc RIIb expressed in cells lining the blood vessels in the liver which take up and degrade the XmAb7195 IgE complex.

These three mechanisms lead to levels of serum IgE below quantifiable levels in preclinical chimpanzee studies and offer the potential for superior IgE control and superior clinical efficacy. We believe the limitations of current treatment with Xolair can be overcome with XmAb7195, and that superior IgE control means our product candidate can potentially treat a larger population with superior efficacy.

Clinical Development Plans

In 2014 we filed an IND for XmAb7195 for asthma with the FDA and in May 2014 initiated a Phase 1a single ascending dose clinical trial in healthy volunteers and in allergen-sensitive subjects with high IgE levels. This Phase 1a

Table of Contents

clinical trial is a randomized, double-blind, placebo-controlled, single ascending dose trial being conducted in two parts. In the completed Part 1, healthy subjects were enrolled into five consecutive dose cohorts of eight subjects each, randomized to receive a single intravenous (IV) administration of XmAb7195 or matching placebo 6:2 (six XmAb7195 patients to two placebo patients). In the ongoing Part 2, otherwise healthy subjects with a history of allergic rhinitis and/or allergic conjunctivitis and/or atopic dermatitis with elevated serum IgE (> 300 IU/mL), will be enrolled into three consecutive dose cohorts of eight subjects each, randomized to a single intravenous (IV) administration of XmAb7195 or matching placebo 6:2 (six XmAb7195 patients to two placebo patients). The primary and secondary objectives of the clinical trial are to determine the safety and tolerability profile of single-dose IV administration of XmAb7195 and to characterize the pharmacokinetics (PK) and immunogenicity of single-dose IV administration of XmAb7195 respectively. Exploratory objectives include the determination of the effect of XmAb7195 on serum free and total IgE and the effect on basophil surface IgE and basophil Fc RI expression levels.

In January 2015, we reported interim data from this Phase 1a clinical trial. XmAb7195 was administered to 30 subjects in Part 1 of the trial in single doses ranging from 0.3 to 3.0 mg/kg. 29 of 30 (97%) subjects had detectable free IgE levels pre-dose. Of these, 26 subjects (90%) had reduction of free IgE levels to below the detectable limit of the assay (<10 ng/ml) at the end of the XmAb7195 infusion with reduction lasting for at least one week following a single infusion, including those at the lowest dose evaluated of 0.3 mg/kg. Two subjects with high pre-dose IgE levels (above 400 IU/mL) were treated with XmAb7195, one each at 0.75 mg/kg and 3.0 mg/kg doses, and both had reduction of free IgE levels to below the limit of detection lasting for at least one week. Total IgE was reduced to below the limit of detection (<2.0 IU/mL) in 26 of 30 (87%) subjects with detectable total IgE pre-dose. Dosing through the first three cohorts (0.3, 1.0 and 3.0 mg/kg) resulted in observations of two apparent dose-related toxicities: urticaria and thrombocytopenia. There were no other AEs that occurred in more than two XmAb7195 treated subjects. There were no SAEs reported and no subject discontinued the trial early.

Asymptomatic thrombocytopenia occurred in all six subjects receiving XmAb7195 in the 3.0 mg/kg dose cohort and thrombocytopenia was deemed a dose limiting toxicity. The decrease in platelet count was transient with a low point by 24 hours post-dose, recovery starting by 48 hours post-dose and near full platelet count recovery by study Day 8 in all cases, at which time serum drug concentrations still exceeded levels that eliminate detectable IgE. There was no apparent relationship of thrombocytopenia to known polymorphisms of Fc receptors IIa or IIb.

Moderate urticaria was reported in a total of seven XmAb7195 treated subjects with an apparent correlation of dose with frequency of occurrence. In all cases regardless of dose, the signs/symptoms of urticaria were mild, non-diffuse and easily treated with oral antihistamine, and the study drug infusions were continued to completion without worsening of symptoms.

We plan to initiate a Phase 1b multiple ascending dose trial, following successful Phase 1a trial safety assessment, that will include cohorts of mild to moderate allergic patients.

Preclinical Development

We have performed a variety of in vitro and in vivo studies to explore the ability of XmAb7195 to sequester IgE and inhibit its production. These preclinical studies have shown that XmAb7195 inhibits the production of IgE in a variety of settings, with greater and/or prolonged reductions of IgE compared to Xolair. We also have observed evidence of three different mechanisms of action. The observations from our preclinical studies include:

- " Selective inhibition of IgE production in human B-cell assays;
- " Prolonged reduction of free and total IgE in humanized mice compared to Xolair;

- " Greater reduction of free and total IgE in chimpanzees compared to Xolair;
- " Well tolerated at high doses in monkeys; and
- " Well tolerated in chimpanzees.

17

Table of Contents

Important for XmAb7195's mechanisms of action is the binding of circulating IgE and our in vitro and in vivo studies reflect this activity and its three-fold tighter binding to IgE than Xolair. In a preclinical study, we treated B cells to induce their transition into IgE-secreting plasma cells and observed that XmAb7195 reduced the total amount of IgE produced. This is consistent with our prediction that the incorporation of our Immune Inhibitor Fc Domain causes the inhibition of IgE B cells. In this respect, XmAb7195 behaves similarly to XmAb5871, which we have shown to have broad capacity to inhibit the production of all classes of antibodies by B cells. In the case of XmAb7195, however, the B-cell inhibition is restricted to B cells expressing IgE on their surface, and our preclinical studies confirm this selectivity.

As with XmAb5871, XmAb7195's enhanced Fc domain does not bind well to mouse Fc γ 2b, so we used models of mice engrafted with human blood cells and examined IgE levels in response to XmAb7195. Compared to Xolair, XmAb7195 prolonged the reduction of free IgE levels, indicating an additional biological effect beyond that of simple IgE binding. Total IgE levels (which are the sum of IgE complexed with anti-IgE antibody plus any free IgE) were significantly reduced in XmAb7195-treated mice, but not reduced in the Xolair-treated mice. We interpret these data as further evidence that XmAb7195, through its Immune Inhibitor Fc Domain, engages Fc γ 2b on IgE B cells and prevents their transition into IgE-secreting plasma cells. In further studies in the humanized mice, we compared the activity of XmAb5871 to XmAb7195 and saw that the XmAb7195 suppression was restricted to IgE versus other immunoglobulins such as IgG and IgM.

We have also tested the activity of XmAb7195 in chimpanzees, which we believe is the most predictive animal model of the effects of XmAb7195 in humans. Chimpanzees, including those in our study, normally have very high levels of IgE compared to humans, and humans with these levels would be considered ineligible for Xolair because their IgE levels exceed Xolair's effective range. We treated six chimpanzees, three with XmAb7195 and three with Xolair, and observed that both antibodies caused a reduction in circulating free IgE, as shown in the figures below.

XmAb7195 reduces free IgE levels in chimpanzees to below the limits of quantification of our IgE assay, 0.004 μ g/ml. Chimpanzees treated with Xolair had transient impact, briefly reducing free IgE to approximately 0.050 μ g/ml. The plots show data from the same study at different time intervals.

Xolair only transiently reduced the free IgE, however, and never achieved the low IgE levels generally believed necessary for efficacy (0.02 μ g/ml or lower). Xolair, consistent with human clinical studies, increased total IgE three to five fold. XmAb7195, on the other hand, reduced free IgE levels to below our limit of quantification (0.004 μ g/ml), amounting to at least 10-fold lower IgE than with Xolair. XmAb7195-treated chimpanzees had marked and rapid reductions in total IgE as well, once again consistent with the added mechanisms of action contributed by the Immune Inhibitor Fc Domain. We believe that the very rapid reduction in total IgE implicates a third mechanism of action, namely the ability to rapidly clear IgE bound to XmAb7195. A second chimpanzee study confirmed these findings, and additional preclinical studies with surrogate antibodies in Fc γ 2b transgenic mice closely resemble our observations in chimpanzees, indicating that the rapid clearance mechanism is a general phenomenon and a potential new application of

Table of Contents

the Immune Inhibitor Fc Domain platform.

We have performed 12-week, multiple dose toxicology studies in cynomolgus monkeys up to 100 mg/kg and XmAb7195 is well tolerated with no adverse effects observed. Furthermore, although the chimpanzee studies were not designed as toxicology studies, XmAb7195 was well tolerated at the 5 mg/kg dose we tested at both single and multiple doses.

Market Opportunity

According to the CDC, asthma affects approximately one in 12 Americans, more than half of asthma sufferers have at least one attack each year and thousands of people die from asthma attacks each year. Disease severities cover a wide range, and the treatment landscape is multi-tiered for asthma patients. Patients with mild and moderate asthma are generally well controlled with inhaled corticosteroids and long-acting beta agonists. However, a small percentage of the estimated 25 million asthma patients in the United States have severe asthma and are refractory to high-dose combination therapy. This severe population is commonly treated with oral corticosteroids, which are associated with a host of undesirable side effects and are often insufficient to control the disease.

IgE, the target of Xolair, is the direct mediator of allergies and the allergic asthma response. When IgE binds to allergens, it triggers an allergic response, which can ultimately result in the debilitating bronchoconstriction of asthma, and other systemic pathologies such as atopic dermatitis and chronic urticaria, also known as hives. Xolair's efficacy in severe asthma through the suppression of IgE has validated IgE as a therapeutic target.

Xolair has been used to treat the severe asthma population, generating worldwide sales in 2012 of approximately \$1.3 billion. While Xolair has demonstrated efficacy in severe asthma, its modest potency has led to two key limitations:

- " Because Xolair's modest potency would require an impractically large dose to control high IgE levels, it is approved for use only in a limited number of asthma patients, leaving approximately 20% of asthma patients that have high body weight and high IgE levels ineligible; and
- " Of those patients treated with Xolair, approximately half do not reach target IgE reductions.

XmAb5574/MOR208, a Cytotoxic B cell Depleting Product Candidate for the Treatment of B cell Cancers

Overview

XmAb5574/MOR208 is a monoclonal antibody that targets CD19 and incorporates our Cytotoxic Fc Domain technology for killing of malignant B cells. XmAb5574/MOR208 was discovered by us and is now being developed by MorphoSys, pursuant to a collaboration and license agreement that we entered into in June 2010. Under this agreement, we granted MorphoSys an exclusive worldwide license to XmAb5574/MOR208 for all indications. We were responsible for completing a Phase 1 clinical trial of XmAb5574/MOR208 in chronic lymphocytic leukemia (CLL), which was completed in January 2013. MorphoSys is solely responsible, at its own cost, for all other development and commercialization activities. MorphoSys commenced Phase 2 clinical trials in patients with B ALL and Non-Hodgkin's Lymphoma (NHL), in April and May 2013, respectively.

We humanized XmAb5574/MOR208 with our proprietary technology and applied our Cytotoxic Fc Domain to enhance binding to the human Fc receptors Fc γ RIIIa and Fc γ RIIIa, thereby enhancing recruitment of natural killer (NK)

cells and other Fc R bearing effector cells. We applied further engineering to the CD19 binding Fv domain of XmAb5574/MOR208 to enhance its affinity over 10 fold for human CD19, and also increased its affinity for monkey CD19, enabling monkey toxicology and efficacy studies.

19

Table of Contents

CD19 is an alternative target to CD20 that can be used in salvage regimens for patients failing Rituxan. Further, CD19 is expressed on the B cell surface earlier in development and persists longer through B cell maturation. Therefore, XmAb5574/MOR208 may be able to target a broader spectrum of lymphoid malignancies, such as acute lymphocytic lymphoma (ALL) or CLL, where Rituxan's efficacy may be limited. Finally, we believe that combination therapy of XmAb5574/MOR208 with immunomodulatory agents, such as lenalidomide, and/or new chemotherapy agents, offers the potential for superior efficacy to existing therapies.

XmAb5574 recruits Natural Killer cells to malignant B cells to promote their destruction.

Clinical Development

In January 2013, we completed a Phase 1 clinical trial of XmAb5574/MOR208 in patients with high-risk, heavily-pretreated CLL, in which the antibody showed encouraging signs of preliminary anti-tumor activity and an acceptable safety profile and was well tolerated. The trial protocol was amended to include a period of extended dosing for a total of eight patients at the 12 mg/kg dose to study the effect of longer duration of exposure on safety and response rate. Overall response rate by IWCLL 2008 criteria was 29.6% (eight partial responses in 27 evaluable patients). Using NCI-WG CLL 1996, response criteria resulted in a response rate of 66.7% (18 partial responses). At the highest dose studied, 12 mg/kg, 12 of 16 patients (75%) had a partial response by NCI-WG CLL 1996 and six patients (37.5%) had a partial response using additional CT criteria (IWCLL 2008). Median progression free survival for all patients was 199 days and for the extended treatment arm (at 12 mg/kg) was 420 days. Blood disease cleared in most patients, with median reduction in absolute lymphocyte count from baseline of 90.8%. XmAb5574/MOR208 was generally well tolerated with no maximum-tolerated dose identified. Clinically-significant, treatment-related adverse events (AEs) classified as Grade 3 or higher occurred in 5 out of twenty-seven patients. The most frequent treatment-related AEs were infusion-related reactions, which were reported for 66.7% of patients, all of which were grade 1 or 2, and no reactions were seen following the first infusion. Treatment-related AEs each reported for 18.5% of patients were ALT increased, AST increased, neutropenia and thrombocytopenia; all other treatment-related AEs were reported for $\leq 15\%$ of patients.

Based on the Phase 1 clinical trial results, MorphoSys has continued the development of XmAb5574/MOR208 and has initiated two Phase 2 clinical trials of MOR208 in patients with ALL and NHL, respectively. The Phase 2 clinical trial in ALL began in April 2013 and is an open-label, multicenter, single-arm clinical trial designed to assess efficacy in patients suffering from relapsed or refractory B-ALL. Secondary outcome measures include response duration, safety and pharmacokinetics of XmAb5574/MOR208. In total, 30 patients are planned to be enrolled. The Phase 2 clinical trial in NHL began in May 2013 and is an open-label, multicenter, single-arm clinical trial designed to assess the efficacy of MOR208 in patients with relapsed or refractory NHL. Secondary outcome measures include response duration, safety and pharmacokinetics of MOR208. A total of up to 120 patients are planned to be enrolled in four separate sub-indications: follicular lymphoma (FL), mantle cell lymphoma (MCL), diffuse large B-cell lymphoma (DLCL), and other forms of indolent NHL (iNHL). In December 2014, MorphoSys reported interim data from the Phase 2 trial in NHL. Of 89 patients treated with the four different subtypes of relapsed or refractory NHL; four complete responses (two in DLBCL, one in FL, one in iNHL), 14 partial responses (seven in DLBCL, six in FL, one in iNHL).

Table of Contents

In addition, an investigator-sponsored Phase 2 clinical trial in CLL as a combination therapy with lenalidomide began in January 2014. Additional clinical trials in other B-cell malignancies and in combination with chemotherapy are possible and will be conducted at the discretion of and under the control of MorphoSys.

Preclinical Development Summary

Our preclinical observations include:

- " Cytotoxicity against multiple lymphoma cell lines;
- " Cytotoxicity against malignant cells from ALL and MCL patients;
- " Inhibition of tumor growth in mouse xenograft models;
- " Rapid and sustained depletion of peripheral and tissue B cells in monkeys; and
- " Well tolerated at high doses in monkeys.

In preclinical in vitro studies, we tested XmAb5574/MOR208 for ADCC activity against a large number of lymphoma-derived tumor cell lines. In these studies, XmAb5574/MOR208 was shown to mediate strong NK-mediated killing against the CD19-positive tumor cell lines tested. Similar tests were performed with tumor cells taken directly from patients with either ALL or MCL. In these studies, XmAb5574/MOR208 demonstrated substantial ADCC activity against both types of lymphomas. In all contexts examined, the control antibody, which is identical to XmAb5574/MOR208 except its Fc domain is an unmodified Fc domain (anti-CD19 IgG1), showed greatly reduced ADCC, in some cases with no detectable killing of tumor cells. This comparison highlights the impact of our Cytotoxic Fc Domain technology on the ability of anti-CD19 antibodies to recruit NK cells and attack tumor cells. In addition to NK-mediated killing, the presumed dominant mechanism of action, we also observed macrophage-mediated phagocytosis of tumor cells in vitro, and a direct anti-tumor effect (requiring no effector cells such as NK or macrophage) in which the antibody appears to slow the growth of some tumor lines.

We used mouse xenograft models to examine the in vivo activity of XmAb5574/MOR208 against subcutaneously implanted lymphoma cells. The antibody inhibits lymphoma growth in both prophylactic (tumor-prevention) models and established tumor models. Notably, anti-CD19 antibodies with unmodified Fc domains had diminished anti-tumor activity compared to XmAb5574/MOR208.

Although the precursor antibody does not react strongly with monkey CD19 or B cells, our affinity-enhanced Fv domain does react well with monkey B cells, and this enabled further POC and toxicology studies in cynomolgus monkeys. We performed an initial high-dose (10 mg/kg) study in monkeys and observed rapid depletion of peripheral B cells after a single dose of the antibody, ultimately reducing the B cells to less than five percent of their starting numbers. Significant B-cell reductions were also observed in the bone marrow, spleen and lymph nodes, notable because of Rituxan's relatively poor ability to impact tissue-resident B cells. The 10 mg/kg dose was well-tolerated by the monkeys, with no adverse effects.

In additional monkey studies, we compared the ability of different doses of XmAb5574/MOR208 to deplete monkey B cells and observed significant B-cell reductions at lower doses, 1 and 3 mg/kg. In a final study to demonstrate the impact of our Cytotoxic Fc Domain technology on in vivo tumor cell killing, we compared the ability of XmAb5574/MOR208 to an unmodified IgG1 control antibody (anti-CD19 IgG1) to deplete monkey B cells at a 3 mg/kg dose (figure below). The XmAb5574/MOR208-treated animals displayed a marked drop in peripheral B-cell counts. The unmodified control antibody anti-CD19 IgG1, on the other hand, did not noticeably affect B-cell counts and was indistinguishable from the effects of treatment with vehicle alone.

Table of Contents

A single dose of XmAb5574/MOR208 depletes peripheral B cells in cynomolgus monkeys. A control anti-CD19 antibody containing an unmodified IgG1 Fc domain and placebo, consisting of the buffer vehicle, has no effect on B cells.

Market Opportunity

B-cell cancers include lymphomas such as NHL and leukemias such as CLL and ALL. Collectively, lymphomas represent about five percent of all cancers diagnosed in the United States. NHL is the most prevalent of all lymphoproliferative diseases, with the National Cancer Institute estimating that over 69,000 new cases will be reported in the United States in 2013 and 85% of NHLs are classified as B-cell disorders. The Leukemia and Lymphoma Society estimates that over 16,000 new cases of CLL and over 6,000 new cases of ALL will be reported in 2013. CD19, the target of XmAb5871's Fv domain, is a B-cell surface protein that is highly expressed on the tumor cells in NHL and many leukemias, including ALL and CLL. We believe that targeting CD19 with XmAb5574/MOR208 offers potential advantages over the current standard of care for B-cell malignancies, which is treatment with Rituxan plus chemotherapy. Rituxan, an anti-CD20 antibody, plus chemotherapy have successfully treated many B-cell NHLs and some B-cell leukemias, demonstrating the utility of antibodies targeting B-cell diseases. Although the Rituxan-chemotherapy regimen has led to major improvements in response rates and progression-free survival, the majority of patients relapse and many lose responsiveness to Rituxan treatment. In 2014, MorphoSys gained orphan drug in the US and orphan medicinal product status in the European Union for XmAb5574/MOR208 for the treatment of both DLBCL and CLL.

Our Research and Development Pipeline

We have used our various Fc platforms and antibody optimization capabilities to produce a growing pipeline of development candidates. These include new XmAb Immune Inhibitor Fc Domain candidates designed to remove target antigens from circulation and multiple oncology candidates using our CD3 bispecifics platform. We will continue to progress these candidates as additional options for clinical development by us or as out licensing opportunities to generate additional revenue.

Bispecific Antibodies for Oncology

We have created Fc variants that form heterodimeric Fc domains that enable the creation of bispecific antibodies that have different Fv domains on each side of the Fc domain in order to bind to a different antigen with each of their Fv domains. We have focused our initial bispecific candidate discovery work on building a pipeline of bispecific antibodies that bind both CD3 and a tumor antigen in order to recruit cytotoxic T cells to the tumor cell. Because of the Fc domain, these bispecific antibodies retain the long half-life and ease of production typical of standard antibodies.

Table of Contents

Our lead bispecific programs are tumor-targeted antibodies that contain both a tumor antigen binding domain (CD123, CD20, or CD38) and a cytotoxic T-cell binding domain (CD3). In November 2014, we announced preclinical data from these three lead programs showing that each activated T-cells to rapidly kill target cells from a tolerated single dose IV bolus in cynomolgus monkeys and demonstrated prolonged half-life of approximately one week in mice.

Data released included the following:

XmAb Anti-CD123 x Anti-CD3 Bispecific Antibodies in Acute Myelogenous Leukemia

- Depletion of over 99% of circulating CD123+ cells in monkeys for over a week
- Bone marrow CD123+ cells were depleted by over 95% at all doses in monkeys
- Prolonged serum half-life in mice of 6.2 days

XmAb Anti-CD20 x Anti-CD3 Bispecific Antibodies in B-cell Lymphomas and Leukemia

Depletion of over 97% of circulating B cells in monkeys for over a week

- B cells in the more resistant lymph nodes and bone marrow were depleted by over 90% at all doses in monkeys
- Prolonged serum half-life in mice up to 6.7 days

Anti-CD38 x Anti-CD3 Bispecific Antibodies in Multiple Myeloma

- Depleted circulating CD38+ cells by greater than 95%
- Prolonged half-life in mice up to 8 days

Applying the rapid clearance property of the Immune Inhibitor Fc Domain

We are exploring multiple new candidate concepts for application of our Immune Inhibitor Fc Domain, in particular capitalizing on the newly discovered rapid clearance property, which builds off the natural scavenging role of Fc RIIb on liver sinusoidal endothelial cells, a type of cell in the blood vessels of the liver that filters antigens out of the circulation. For example, building on our lead anti IgE product candidate, XmAb7195, we are now characterizing a second generation antibody with a modified version of the IIb immune inhibitor domain. The new Fc domain has intermediate affinity enhancement for Fc RIIb, which we have discovered promotes IgE control in mouse models with a longer dosing interval than XmAb7195. We are also exploring approaches to clear pathologic immune complexes from circulation. Antibody-antigen complexes are central to the kidney pathology in lupus nephritis and a variety of other conditions and form when antigens present in the circulation are recognized by antibodies of the immune system.

Second Generation Biologics

Our Xtend Fc Domain technology can potentially improve the performance of commercially successful therapeutic antibodies by enhancing their half life and improving dosing convenience. We have produced several enhanced versions of antibodies, in some cases by applying the Xtend Fc Domain mutations, and in other cases also modifying other features. AbbVie's Humira (adalimumab) is the industry leading anti TNF antibody for the treatment of rheumatoid arthritis, reaching global sales above \$5 billion. We have produced and characterized a half life enhanced version of Humira that we call Xtend TNF (also known as XmAb 6755). It has approximately twice the in vivo half life of Humira, which is dosed on a biweekly schedule, and we believe Xtend TNF has the potential to achieve monthly dosing in rheumatoid arthritis patients without loss of efficacy. A stable cell line has been created and we have a business relationship with Boehringer Ingelheim to manufacture Xtend TNF drug supply for preclinical toxicology and clinical studies.

Collaboration Agreements and Technology Licenses

We use product development partnerships with pharmaceutical and biotechnology companies to complement our internal drug discovery and development capabilities, to assist the efficient global commercialization of our products

Table of Contents

and technology and to generate near and long term funding. To date, the revenue generated from upfront fees, license fees, option fees and milestone payments associated with these arrangements, combined with the development expenses assumed by our partners, have allowed us to better manage our operating expenses and continue to invest in building new opportunities.

Below is a table summarizing all of our technology licenses:

Licensee	Year	Xencor Technology	Indication	Milestones	Royalties	Current Development Stage
Boehringer Ingelheim	2007	Cytotoxic	Oncology	Yes	Yes	Phase 1 trials (two candidates)
Janssen	2009	Xtend	Autoimmune disease	Yes	Yes	Preclinical
CSL	2009	Cytotoxic	Oncology	Yes	Yes	Phase 1
Morphosys	2010	Cytotoxic	Oncology	Yes	Yes	Phase 2
CSL	2013	Xtend	Hematological diseases	Yes	Yes	Preclinical
Merck	2013	Fc optimization	Autoimmune disease	Yes	Yes	Phase 1
Alexion	2013	Xtend	Undisclosed	Yes	Yes	Phase 1
Novo Nordisk	2014	Various, including Bi-specifics	Undisclosed	Yes	Yes	Preclinical

Collaboration Agreement with Novo Nordisk A/S

In December 2014 we entered into a collaboration and licensing agreement with Novo Nordisk A/S (Novo) to jointly discover novel biologic drug candidates for an undisclosed target by combining multiple Xencor XmAb® technologies, including bispecific and immune inhibitor technologies. Under the terms of the agreement, we granted Novo a license to use and access these technologies over the research term that is initially a two year period. We will also provide research support in collaboration with Novo to the agreed upon research plan to discover potential drug candidates. Novo will have an exclusive right to develop and commercialize any candidates that are identified during the research discovery period. Xencor and Novo will use Xencor's XmAb® technologies, including our bispecific technology to build a variety of molecular formats that can engage the target and also modulate desired immune functions using Xencor's XmAb immune inhibitor technology. XmAb immune inhibitor Fc domains target Fc RIIb (also known as CD32b). In addition to its proprietary technologies, Xencor will also contribute expertise in studying immune functions of biologics and immune system modulation. Novo will collaborate by using its proprietary technology during the research discovery period and will have all development and commercial rights.

Under the terms of the agreement, in January 2015, we received an upfront payment of \$2.5 million and will receive funded research support of \$1.6 million per year for each year of the research term, which initially is two years. Novo has an option to extend the research term to a third year by providing written notice and payment of another year of research funding. If certain pre-established discovery milestones are achieved during the research term, we are eligible to receive \$2.0 million in milestone payments. Upon completion of the research term, Novo will have development and commercial rights to any compounds identified. If certain developmental, regulatory and sales milestones are achieved by Novo in developing the compound, we are eligible to receive up to an additional \$167.3 million in

milestone payments for a product developed. The \$167.3 million in milestone payments is comprised as follows: \$36.3 million relates to clinical development milestone events, \$51.0 million relates to the filing and completion of regulatory approvals and an additional \$80.0 million of aggregate milestone payments relate to the achievement of certain product sale goals. If licensed products are commercialized, we are also entitled to receive royalties in the mid-single digits based on net sales of products sold by Novo, its affiliates and its sublicenses in a calendar year. Novo's royalty obligations continue on a licensed product-by-product and country-by-country basis until the later to occur of the expiration of the last valid claim in a patent covering a product in such country, or 10 years after the date of the first commercial sale of such product in such country.

The term of this agreement will continue until the earlier of expiration of all royalty payment obligations under the agreement or, upon completion of the research term if Novo does not identify a compound for continued

Table of Contents

development. The agreement provides that it may be terminated by either party upon written notice to the other party in the event of the other party's bankruptcy or insolvency, or the other party's material breach of the agreement if such breach remains uncured for 90 days, or 20 days for any non-payment breach. Novo may terminate this agreement at any time following the expiration of the research term for any reason upon delivery of at least 90 days' prior written notice to us. Upon such termination, all licenses granted by us to Novo shall terminate.

Collaboration and Option Agreement with Amgen

In December 2010, we entered into a collaboration and option agreement with Amgen Inc. (Amgen) pursuant to which we agreed to collaborate with Amgen to research, develop and commercialize XmAb5871, an Fc-engineered monoclonal antibody that targets CD19 via its Fv domains and Fc RIIb via its XmAb Fc domain, and products based thereon. Under the terms of the agreement, we granted to Amgen an option to obtain an exclusive license to research, develop, manufacture and commercialize XmAb5871 and certain related products worldwide. Under the terms of the agreement, we received an initial upfront payment of \$11.0 million and a \$2.0 million milestone payment upon the initiation of our Phase 1b/2a clinical trial of XmAb5871 in January 2013 in patients with moderate to severe rheumatoid arthritis. In October 2014, we announced that we sought and regained all rights to XmAb5871 from Amgen. In return for terminating the collaboration and option agreement, we granted Amgen a first right to negotiate a proposed license for XmAb5871 prior to seeking future partners. This right expires upon the earlier of the initiation of Phase 3 clinical testing of XmAb5871, a change of control of Xencor, or October 2019.

Collaboration and License Agreement with MorphoSys AG

In June 2010, we entered into a collaboration and license agreement with MorphoSys AG (MorphoSys) which we subsequently amended in March 2012. We granted to MorphoSys an exclusive worldwide license under certain of our patents and know how to research, develop and commercialize XmAb5574/MOR208, as well as other anti CD19 antibodies that incorporate our cytotoxic Fc domain technology, with the right to sublicense under certain conditions. Under the terms of the agreement, we agreed to collaborate with MorphoSys to develop and commercialize XmAb5574/MOR208, a high potency cytotoxic monoclonal antibody developed by us for the treatment of B cell malignancies and other diseases. Under the terms of the agreement, we initiated and sponsored a Phase 1 clinical trial for XmAb5574/MOR208 in patients with chronic lymphocytic leukemia in December 2010 which was completed in January 2013. Following such completion, MorphoSys is responsible for all further clinical development and commercialization of licensed antibodies and licensed products under the agreement and is required to use commercially reasonable efforts to achieve certain developmental and regulatory milestones and other diligence obligations under the agreement. In addition, MorphoSys is responsible for all costs relating to the development and commercialization of XmAb5574/MOR208, or other antibodies covered by the agreement, including manufacturing, regulatory, clinical and registration costs.

Under the terms of the agreement, we received an upfront payment of \$13.0 million and received \$3.0 million for development milestones in 2013. If certain developmental, regulatory and sales milestones are achieved, we are also eligible to receive up to an additional \$299.0 million in milestone payments. The \$299.0 million of milestone payments is comprised as follows: \$62.0 million relates to clinical development milestone events, \$187.0 million relates to the filing and completion of regulatory approvals and an additional \$50.0 million of aggregate milestone payments relate to the achievement of certain product sale goals. If licensed products are commercialized, we are also entitled to receive tiered royalties in the high single digit to low teen percent range based upon net sales of products sold by MorphoSys, its affiliates and its sublicensees in a calendar year. MorphoSys' royalty obligations continue on a licensed product by licensed product and country by country basis until the later to occur of the expiration of the last valid claim in the licensed patent covering a licensed product in such country, or 11 years after the first sale of a licensed product following marketing authorization in such country.

The term of this agreement will continue until all of MorphoSys' royalty payment obligations have expired unless terminated earlier. The agreement provides that it may be terminated by either party upon written notice to the other party in the event of the other party's insolvency or the other party's material breach of the agreement if such breach remains uncured for 120 days, or 30 days in the case of a non payment breach. MorphoSys may terminate the agreement without cause upon 90 days' advance written notice to us. In the event that MorphoSys terminates this

25

Table of Contents

agreement for convenience or we terminate due to MorphoSys' material breach, worldwide rights to develop, manufacture and commercialize XmAb5574/MOR208, as well as any other antibodies covered by the agreement, revert back to us completely. Along with these rights, MorphoSys is obligated to transfer all regulatory documents, clinical data and know how, and we are granted a license from MorphoSys to allow us to develop, manufacture and commercialize XmAb5574/MOR208, or other antibodies covered by the agreement, worldwide, subject to reimbursing MorphoSys a portion of their development costs out of future revenue generated from the development and commercialization of XmAb5574/MOR208.

Option and License Agreement with Alexion

In January 2013, we entered into an option and license agreement with Alexion Pharmaceuticals, Inc. (Alexion). Under the terms of the agreement, we granted to Alexion an exclusive research license, with limited sublicensing rights, to make and use our Xtend technology to evaluate and advance compounds against six different target programs during a five year research term under the agreement, up to completion of the first multi dose human clinical trial for each target compound. Alexion may extend the research term for an additional three years upon written notice to us and payment of an extension fee of \$2.0 million. Alexion is responsible for conducting all research and development activities under the agreement at its own expense. In the third quarter of 2014, Alexion initiated a Phase 1 clinical trial with an undisclosed molecule to be used against an undisclosed target. It is the first human clinical trial with a molecule incorporating our Xtend Fc Domain technology.

In addition, we granted to Alexion an exclusive option, on a target by target basis, to obtain an exclusive commercial, worldwide, royalty bearing license, with sublicensing rights, under our Xtend technology to develop and commercialize products that contain the target for which the option is exercised. In order to exercise this option, Alexion must pay a \$4.0 million option fee with respect to each target for which the option is exercised. Alexion may exercise this option at any time during the research term but must exercise an option prior to the initiation of a second clinical trial.

Under the agreement, we received an upfront payment of \$3.0 million. Alexion is also required to pay an annual maintenance fee of \$0.5 million during the research term of the agreement and \$1.0 million during any extension of the research term. In addition, if certain development, regulatory and commercial milestones are achieved we are eligible to receive up to \$66.5 million for the first product to achieve such milestones on a target by target basis. If licensed products are successfully commercialized, we are also entitled to receive royalties based on a percentage of net sales of such products sold by Alexion, its affiliates or its sublicensees, which percentage is in the low single digits. Alexion's royalty obligations continue on a product by product and country by country basis until the expiration of the last to expire valid claim in a licensed patent covering the applicable product in such country.

Absent early termination, the term of the agreement will continue until the expiration of Alexion's royalty payment obligations or until the expiration of the research term if Alexion has not exercised its option for a product license under the agreement. Either party may terminate the agreement for a material breach of the agreement by the other party if such breach remains uncured for 60 days, or 30 days in the case of a non payment breach. Alexion may terminate the agreement without cause on a target by target basis upon 90 days' advance written notice to us.

Other Technology Licenses

In addition to the product development partnerships and technology license agreement described above, we also enter into non-exclusive relationships whereby we license our intellectual property around a specific XmAb technology to a pharmaceutical or biotechnology company to use in one or more of their own products. By accessing our technology, our partners hope to improve the pharmacology of their antibodies and create potential commercial differentiation for their product candidates. Under these technology licenses, we generally grant rights to our licensees that are limited to

the specific XmAb Fc domains that are required and also limited to a specific program or set of programs of the partner that are outside of our core strategic areas. This approach allows us to maintain control over the vast majority of the rights to our platform while still disseminating our technology for broad use. The plug-and-play nature of XmAb technology allows us to structure nearly all of these licenses without any work commitment on our part; hence, these licenses allow us to generate revenue to support our own internal programs with no additional obligations

Table of Contents

on our part. The revenue we generate from these licenses comes in the form of license fees, annual maintenance fees, milestone payments and royalties.

Intellectual Property

The foundation for XmAb technology and our product candidates and partnering is the generation and protection of intellectual property for novel antibody therapeutics. We combine proprietary computational methods for amino acid sequence design with laboratory generation and testing of new antibody compositions. Our design and engineering team prospectively assesses, with patent counsel, the competitive landscape with the goal of building broad patent positions and avoiding third-party intellectual property.

As a pioneer in Fc domain engineering, we systematically scanned the structure of the Fc domain to discover Fc variants. We have filed patent applications relating to thousands of specific Fc domain variants with experimental data on specific improvements of immune function, pharmacokinetics, structural stability and novel structural constructs. We have filed additional patent applications derived from these applications as we discover new properties of the Fc variants and as new business opportunities arise. We continually seek to expand the intellectual property coverage of our technology and candidates, and invest in discovering new Fc domain technologies and antibody product candidates.

As of December 31, 2014, our patent estate, on a worldwide basis, includes more than 150 issued patents (of which at least 40 are in the United States) and over 170 pending patent applications (of which at least 60 are in the United States) which we own or for which we have a fully-paid exclusive license, with claims directed to XmAb Fc domains, all of our clinical and preclinical stage antibodies and our computational protein design methods, called the PDA, protein design platform. Over 100 issued patents and 150 pending patent applications have claims directed specifically to our XmAb technology and candidates. Our XmAb Fc domain, patents and patent applications, with claims directed to their incorporation into antibodies, Fc domain engineering and compositions of matter are expected to expire in the United States between 2023 and 2031. Our three lead product candidates are covered by issued U.S. composition of matter patents relating to both the XmAb Fc domains and the individual product candidates. The composition of matter patents relating to our lead product candidates are expected to expire in the United States between 2027 and 2030, one of which relates to XmAb5574/MOR208, two relate to XmAb5871 and two relate to XmAb7195.

In addition to patent protection, we rely on trade secret protection and know-how to expand our proprietary position around our technology and other discoveries and inventions that we consider important to our business. We seek to protect this intellectual property in part by entering into confidentiality agreements with our employees, consultants, scientific advisors, clinical investigators and other contractors and also by requiring our employees, commercial contractors and certain consultants and investigators, to enter into invention assignment agreements that grant us ownership of certain discoveries or inventions made by them.

Further, we seek trademark protection in the United States and in certain other jurisdictions where available and when we deem appropriate. We have obtained registrations for the Xencor trademark, as well as certain other trademarks, which we use in connection with our pharmaceutical research and development services and our clinical-stage products, including XmAb, PDA and Protein Design Automation. We currently have registrations for Xencor and PDA in the United States, Australia, Canada, the European Community and Japan, for Protein Design Automation in the United States, Australia, Canada and the European Community, and for XmAb in the United States, Australia and the European Community.

Manufacturing

We are able to internally manufacture the quantities of our product candidates required for relatively short preclinical animal studies. We believe that this allows us to accelerate the drug development process by not having to rely on third parties for all of our manufacturing needs. We have adopted a manufacturing strategy of contracting with third parties in accordance with cGMP for the manufacture of drug substance and product, including XmAb5871, XmAb7195 and our bispecific development candidate, XmAb14045. Additional contract manufacturers are used to fill, label, package and distribute investigational drug products. This allows us to maintain a more flexible infrastructure

Table of Contents

while focusing our expertise on developing our products. XmAb5871 and XmAb7195 are produced by mammalian cell culture of a Chinese hamster ovary cell line that expresses the antibody, followed by multiple purification and filtration steps typical of those used for monoclonal antibodies. We have no long term manufacturing agreements and will ultimately depend on contract manufacturers for the manufacture of our products for commercial sale, as well as for process development. We are currently in the process of developing subcutaneous formulations for XmAb5871 and XmAb7195 with third party contract manufacturers.

Development and Manufacturing Services Agreement with Catalent

In September 2005, we entered into a development and manufacturing services agreement (the Catalent Manufacturing Agreement) with Catalent Pharma Solutions LLC (formerly Cardinal Health PTS, LLC) (Catalent). Under the terms of the agreement, Catalent may, from time to time, provide development and manufacturing services for us related to our XmAb technology. Catalent is currently performing services related to the manufacture under cGMP of drug substance of XmAb5871 and XmAb7195 under the agreement. We pay for services performed by Catalent under the agreement pursuant to statements of work entered into from time to time.

Under the terms of the agreement, if Catalent develops one or more cell lines using its proprietary GPEX product expression technology (GPEX Technology) in the course of performing services under the agreement, we have the option to license any such cell line for non cGMP research for a license fee of \$30,000 per year for a period of up to 10 years and on other terms to be agreed upon by Catalent and us. In addition, we have the option to license any cell line developed by Catalent in the course of performing services under the agreement that incorporates the GPEX Technology for use in the production of clinical and commercial supplies of gene expression products by us or any of our manufacturers for 10 years for an upfront fee that ranges between \$0 and \$0.3 million per cell line, an annual license fee of \$30,000, and development and regulatory milestones up to as much as an aggregate of \$2.9 million per cell line licensed, and on other terms to be agreed upon by Catalent and us.

This agreement will remain in effect unless either party terminates it in accordance with its terms. We may unilaterally terminate the agreement or activities under any statement of work entered into pursuant to the agreement upon 90 days written notice to Catalent. Catalent may unilaterally terminate the agreement upon 24 months written notice to us. Either party may terminate the agreement upon written notice to the other party upon the other party's insolvency or the other party's material breach of the agreement if such breach remains uncured for 30 days following notice thereof.

Cell Line Sale Agreement with Catalent

In December 2011, we entered into a GPEX derived cell line sale agreement with Catalent pursuant to which we purchased a cell line (the GPEX Cell Line) developed by Catalent under the Catalent Manufacturing Agreement for use in the manufacture of XmAb7195.

As consideration for the purchase and sale of the GPEX Cell Line under the agreement, we paid an initial upfront fee of \$125,000. In addition, we are required to pay an annual fee to Catalent and royalties based on a percentage of net sales for products that are derived from or utilize the GPEX Cell Line. Such percentage is less than 1.0%. We are also required to make payments to Catalent based upon the achievement of certain developmental and regulatory milestones totaling up to approximately \$2.9 million.

We have the unilateral right to terminate the agreement upon 30 days written notice to Catalent. In addition, either party may terminate the agreement upon written notice to the other party in the event of the other party's insolvency or the other party's material breach of the agreement if such breach remains uncured for 60 days following notice thereof. Absent early termination, the agreement will remain in effect. If we terminate the agreement without cause or if Catalent terminates the agreement for our material breach of the agreement, our ownership rights in the GPEX® Cell

Line will automatically terminate, and title thereto will revert to Catalent.

Upon acquisition of the GPEX cell line we transferred XmAb7195 manufacturing to Cook Pharmica, LLC (Cook) under which Cook has produced drug substance and drug product for use in our XmAb7195 clinical studies and

28

Table of Contents

performed related services, Cook continues to provide services under the agreement, however, we have transferred further manufacturing and development efforts for XmAb7195 back to Catalent under the Catalent Manufacturing Agreement.

Boehringer Ingelheim International GmbH

In February 2012, we entered into a collaboration agreement with Boehringer Ingelheim International GmbH (Boehringer Ingelheim) for the establishment of certain manufacturing processes and the production of our next generation monoclonal anti TNF antibody for use in our preclinical and Phase 1 clinical development. Under the terms of the agreement, we are required to use commercially reasonable efforts to complete Phase 1 clinical testing of the product and to find a licensing partner for the further development and commercialization of the antibody into a therapeutic product.

We will be required to pay for services performed and products provided by Boehringer Ingelheim under the agreement pursuant to project plans entered into from time to time. In addition, we are required to reimburse Boehringer Ingelheim for all out of pocket expenses, including the cost of raw materials, incurred in connection with the project plan. Boehringer Ingelheim has agreed to delay all payments due to them under the agreement, including an annual interest rate which is a low double digit percentage, until (A) in the case where we have entered into a license agreement with a third party, the later of (1) the effective date of such license agreement or (2) the earlier of (i) completion of the clinical summary report for a Phase 1 clinical trial of the product or (ii) February 10, 2017 or (B) in the case where we decide to continue to develop the product on our own, on or before five years from the earlier of (i) completion of the clinical summary report for a Phase 1 clinical trial of the product or (ii) February 10, 2017. Any payments due by us in the situation described in clause (A) of the preceding sentence will be made in installments each of which will be limited to a maximum percentage of any licensing revenue that we receive under the applicable third party license. We are not obligated to pay Boehringer Ingelheim any or all of the amounts owed under the agreement, including interest payments if we: (a) are not able to further develop the product for technical or scientific reasons or (b) do not decide to proceed with the further development of the product without a business partner and are unable to enter into a partnership agreement within an agreed upon period of time after Phase 1 clinical development.

Pursuant to the agreement, we have granted Boehringer Ingelheim a first right to negotiate to manufacture and supply the products for use in any future Phase 2 and Phase 3 clinical trials, and should Boehringer Ingelheim exercise such right, Boehringer Ingelheim has a first right to negotiate to manufacture and supply commercial product as our principal supplier for an agreed upon period following the first commercial launch of the products. In the event that we desire to produce the products using the process developed and performed by Boehringer Ingelheim outside the agreement or any manufacturing agreement which we may enter into with Boehringer Ingelheim, we will be required to pay Boehringer Ingelheim a one-time technology access fee of \$3.5 million in exchange for a worldwide, irrevocable, exclusive and royalty free license, with sublicensing rights, to use the process developed by Boehringer Ingelheim under the agreement to produce the products.

Absent early termination, the agreement will terminate upon completion of all projects set forth in the agreement. Either party may terminate the agreement upon 180 days prior written notice to the other party if such party will not be able to carry out the project contemplated by the agreement for scientific, technical or business reasons.

KBI Biopharma, Inc.

In July 2014, we entered into a master services agreement (KBI Agreement) with KBI Biopharma, Inc. We have engaged KBI under the KBI Agreement for process development, clinical scale-up, analytical method development, formulation development and other services related to drug substance and drug product for our first bispecific

development candidate, XmAb14045, in accordance with cGMP regulations. Under the KBI Agreement, Xencor will pay up to approximately \$3.5 million as services are incurred over the term of the Agreement. The KBI Agreement is for a three year term but is automatically extended on an annual basis until the services are completed. The KBI Agreement may be terminated by either party for a breach that is not remedied within thirty days after notice or sixty days after notice of the existence of an uncurable scientific or technical issue that renders KBI unable to render services under the KBI Agreement, by Xencor after sixty day notice, or in the event of a bankruptcy of a party. For termination

Table of Contents

other than a material breach by KBI, we must pay for all services conducted prior to the termination and to wind down the activities.

Competition

We compete in an industry that is characterized by rapidly advancing technologies, intense competition and a strong emphasis on proprietary products. Our competitors include pharmaceutical companies, biotechnology companies, academic institutions and other research organizations. We compete with these parties for promising targets for antibody based therapeutics, new technology for optimizing antibodies and in recruiting highly qualified personnel. Many competitors and potential competitors have substantially greater scientific, research and product development capabilities as well as greater financial, marketing and sales and human resources than we do. In addition, many specialized biotechnology firms have formed collaborations with large, established companies to support the research, development and commercialization of products that may be competitive with ours. Accordingly, our competitors may be more successful than we may be in developing, commercializing and achieving widespread market acceptance. In addition, our competitors' products may be more effective or more effectively marketed and sold than any treatment we or our development partners may commercialize and may render our product candidates obsolete or noncompetitive before we can recover the expenses related to developing and commercializing any of our product candidates.

Competition in autoimmune disease drug development is intense and includes multiple monoclonal antibodies, other biologics and small molecules approved for the treatment of autoimmune diseases, many of which are being developed or marketed by large multinational pharmaceutical companies such as Glaxo SmithKline plc, AbbVie Inc., Janssen Pharmaceuticals, Inc., Genentech Inc. and Amgen Inc. Benlysta is currently the only monoclonal antibody that we are aware of that is approved for the treatment of lupus, although we believe that Rituxan is prescribed, off label, for this indication. In addition, these and other pharmaceutical companies have monoclonal antibodies or other biologics in clinical development for the treatment of autoimmune diseases. There are currently no approved therapies for IgG4-related disease, a newly recognized disorder, and glucocorticoids are the current standard of care.

Many companies have approved therapies or are developing drugs for the treatment of asthma including multinational pharmaceutical companies such as GlaxoSmithKline, Novartis AG and AstraZeneca plc. Monoclonal antibody drug development has primarily focused on allergic asthma. Xolair is currently the only monoclonal antibody that we are aware of that is approved for the treatment of severe asthma. In addition, we are aware that Novartis and Genentech each have an antibody targeting IgE in Phase 1 or 2 clinical development for asthma. Other monoclonal antibodies in development target cytokines such as IL 13, IL 4, IL 5, IL 9, GM CSF or their receptors. Although these drugs function differently from our products, if successfully developed, these drugs will compete in the asthma market. We are not aware of any companies developing drugs that target Fc RIIB for the treatment of asthma.

Competition in blood cancer drug development is intense, with more than 250 compounds in clinical trials by large multinational pharmaceutical companies and Rituxan is just one of many monoclonal antibodies approved for the treatment of NHL or other blood cancers. In addition, we are aware of a number of other companies with development stage programs that may compete with XmAb5574/MOR208 in the future. We anticipate that we will face intense and increasing competition as new treatments enter the market and advanced technologies become available, such as Gavyaza for CLL and NHL marketed by Roche, Blincyto for ALL marketed by Amgen and engineered T-cell therapies targeting blood cancers from companies such as Novartis.

Regulatory Overview

Our business and operations are subject to a variety of U.S. federal, state and local and foreign supranational, national, provincial and municipal laws, regulations and trade practices. The FDA and comparable regulatory authorities in

state and local jurisdictions and in other countries impose substantial and burdensome requirements upon companies involved in the clinical development, manufacture, marketing and distribution of drugs and biologics. These agencies and other federal, state and local entities regulate research and development activities and the testing, manufacture, quality control, safety, effectiveness, labeling, storage, recordkeeping, approval, advertising and promotion and export and import of our product candidate.

30

Table of Contents

U.S. Government Regulation

U.S. Drug Development Process

In the United States, the FDA regulates drugs and biologic products under the Federal Food, Drug and Cosmetic Act (FDCA) (21 U.S.C. §301, et seq), its implementing regulations and other laws, including, in the case of biologics, the Public Health Service Act. Our antibody product candidates are subject to regulation by the FDA as a biologic. Biologics require the submission of a Biologics License Application (BLA), to the FDA and approval of the BLA by the FDA before marketing in the United States. The process of obtaining regulatory approvals for commercial sale and distribution and the subsequent compliance with applicable federal, state, local and foreign statutes and regulations require the expenditure of substantial time and financial resources. Failure to comply with the applicable U. S. requirements at any time during the product development process, approval process or after approval, may subject an applicant to administrative or judicial civil or criminal sanctions. These sanctions could include the FDA's refusal to approve pending applications, license suspension or revocation, withdrawal of an approval, imposition of a clinical hold on clinical trials, warning letters, product recalls, product seizures, total or partial suspension of production or distribution, injunctions, fines, refusals of government contracts, restitution, disgorgement or civil and/or criminal penalties. The process required by the FDA before a biologic may be marketed in the United States generally involves the following:

- completion of preclinical laboratory tests, animal studies and formulation studies performed in accordance with the FDA's current Good Laboratory Practices (GLP) regulations;
- submission to the FDA of an investigational new drug application (IND) which must become effective before human clinical trials in the United States may begin;
- performance of adequate and well controlled human clinical trials in accordance with the FDA's current good clinical practices (GCP) regulations to establish the safety and efficacy of the product candidate for its intended use;
- submission to the FDA of a BLA;
- satisfactory completion of an FDA inspection (if the FDA deems it as a requirement) of the manufacturing facility or facilities where the product is produced to assess compliance with the FDA's cGMP regulations to assure that the facilities, methods and controls are adequate to preserve the product's identity, strength, quality and purity;
- potential audits by the FDA of the nonclinical and clinical trial sites that generated the data in support of the BLA;
- review of the BLA by an external Advisory Committee to the FDA, whose recommendations are not binding on the FDA; and
- FDA review and approval of the BLA prior to any commercial marketing or sale.

Before testing any compounds with potential therapeutic value in humans, the product candidate enters the preclinical testing stage. Preclinical tests include laboratory evaluations of product chemistry, stability and formulation, as well as animal studies to assess the potential toxicity and activity of the product candidate. The conduct of the preclinical tests must comply with federal regulations and requirements including GLPs. The sponsor must submit the results of the preclinical tests, together with manufacturing information, analytical data, any available clinical data or literature and a proposed clinical protocol, to the FDA as part of the IND. The IND automatically becomes effective 30 days after receipt by the FDA, unless the FDA raises concerns or questions about the conduct of the clinical trial, including concerns that human research subjects will be exposed to unreasonable health risks. In such a case, the IND sponsor and the FDA must resolve any outstanding concerns before the clinical trial can begin. The FDA may also

Table of Contents

impose clinical holds on a product candidate at any time before or during clinical trials due to safety concerns or non-compliance, or for other reasons.

Clinical trials involve the administration of the product candidate to human patients under the supervision of qualified investigators, generally physicians not employed by or under the clinical trial sponsor's control. Clinical trials are conducted under protocols detailing, among other things, the objectives of the clinical trial, dosing procedures, subject selection and exclusion criteria and the parameters to be used to monitor subject safety and effectiveness. Each protocol must be submitted to the FDA as part of the IND. Clinical trials must be conducted in accordance with GCPs. Further, each clinical trial must be reviewed and approved by an IRB at or servicing each institution at which the clinical trial will be conducted. An IRB is charged with protecting the welfare and rights of clinical trial participants and considers such items as whether the risks to individuals participating in the clinical trials are minimized and are reasonable in relation to anticipated benefits. The IRB also approves the form and content of the informed consent that must be signed by each clinical trial subject or his or her legal representative and must monitor the clinical trial until completed. Human clinical trials are typically conducted in three sequential phases that may overlap or be combined:

- Phase 1. The product candidate is initially introduced into a limited population of healthy human subjects and tested for safety, dosage tolerance, absorption, metabolism, distribution and excretion. In the case of some products for some diseases, or when the product may be too inherently toxic to ethically administer to healthy volunteers, the initial human testing is often conducted in patients with the disease or condition for which the product candidate is intended to gain an early indication of its effectiveness.
- Phase 2. The product candidate is evaluated in a limited patient population (but larger than in Phase 1) to identify possible adverse effects and safety risks, to preliminarily evaluate the efficacy of the product for specific targeted indications and to assess dosage tolerance, optimal dosage and dosing schedule.
- Phase 3. Clinical trials are undertaken to further evaluate dosage and provide substantial evidence of clinical efficacy and safety in an expanded patient population (such as several hundred to several thousand) at geographically dispersed clinical trial sites. Phase 3 clinical trials are typically conducted when Phase 2 clinical trials demonstrate that a dose range of the product candidate is effective and has an acceptable safety profile. These trials typically have at least 2 groups of patients who, in a blinded fashion, receive either the product or a placebo. Phase 3 clinical trials are intended to establish the overall risk/benefit ratio of the product and provide an adequate basis for product labeling. Generally, two adequate and well-controlled Phase 3 clinical trials are required by the FDA for approval of a BLA.

Annual progress reports detailing the results of the clinical trials must be submitted to the FDA and written IND safety reports must be promptly submitted to the FDA and the investigators for serious and unexpected adverse events or any finding from tests in laboratory animals that suggests a significant risk for human subjects.

Concurrent with clinical trials, companies usually complete additional animal studies and must also develop additional information about the chemistry and physical characteristics of the biologic and finalize a process for manufacturing the product in commercial quantities in accordance with cGMP requirements. The manufacturing process must be capable of consistently producing quality batches of the product candidate and, among other things, must develop methods for testing the identity, strength, quality and purity of the final biologic product. Additionally, appropriate packaging must be selected and tested and stability studies must be conducted to demonstrate that the product candidate does not undergo unacceptable deterioration over its shelf life.

U.S. Review and Approval Processes

The results of product development, preclinical studies and clinical trials, along with descriptions of the manufacturing process, analytical tests, proposed labeling and other relevant information are submitted to the FDA in the form of a BLA requesting approval to market the product for one or more specified indications.

If the FDA determines that the BLA is substantially complete it will accept the BLA for filing. This process generally takes eight months to a year but in some cases may take much longer.

Table of Contents

The FDA reviews the BLA to determine, among other things, whether the proposed product is safe and effective for its intended use, and whether the product is being manufactured in accordance with cGMP to assure and preserve the product's identity, strength, quality and purity and it may inspect the manufacturing facilities to assure cGMP compliance and clinical sites used during the clinical trials to assure cGMP compliance. During the approval process, the FDA also will determine whether a risk evaluation and mitigation strategy (REMS) is necessary to assure the safe use of the product. If the FDA concludes a REMS is needed, the sponsor of the BLA must submit a proposed REMS prior to approval. A REMS can substantially increase the costs of obtaining approval.

The FDA will issue a complete response letter describing deficiencies in the BLA and recommend actions if the agency decides not to approve the BLA. The applicant will have to address all of the deficiencies which could take substantial time to address.

If the product receives regulatory approval, the approval may be significantly limited to specific diseases and dosages or the indications for use may otherwise be limited, and may require that certain contraindications, warnings or precautions be included in the product labeling. In addition, the FDA may require post marketing studies, sometimes referred to as Phase 4 testing, which involves clinical trials designed to further assess drug safety and effectiveness and may require testing and surveillance programs to monitor the safety of approved products that have been commercialized.

Post Approval Requirements

Any biologic products for which we or our collaborators receive FDA approvals are subject to continuing regulation by the FDA, including, among other things, record keeping requirements, reporting of adverse experiences with the product, providing the FDA with updated safety and efficacy information, product sampling and distribution requirements, complying with certain electronic records and signature requirements and complying with FDA promotion and advertising requirements, which include, among others, restrictions on direct to consumer advertising, promoting biologics for uses or in patient populations that are not described in the product's approved labeling (known as "off label use"), industry sponsored scientific and educational activities and promotional activities involving the internet. Failure to comply with these or other FDA requirements can subject a manufacturer to possible legal or regulatory action, such as warning letters, suspension of manufacturing, seizure of product, injunctive action, mandated corrective advertising or communications with healthcare professionals, possible civil or criminal penalties or other negative consequences, including adverse publicity.

We will rely, and expect to continue to rely, on third parties for the production of clinical and commercial quantities of our products. Our collaborators may also utilize third parties for some or all of a product we are developing with such collaborator. Manufacturers are required to comply with applicable FDA manufacturing requirements contained in the FDA's cGMP regulations and other laws. cGMP regulations require among other things, quality control and quality assurance as well as the corresponding maintenance of records and documentation. Drug manufacturers and other entities involved in the manufacture and distribution of approved biologics are subject to periodic inspections by the FDA and certain state agencies for compliance.

U.S. Patent Term Restoration and Marketing Exclusivity

Depending upon the timing, duration and specifics of the FDA approval of any of our biologic product candidates, we may apply for limited patent term extension under the Drug Price Competition and Patent Term Restoration Act of 1984, commonly referred to as the Hatch-Waxman Amendments. The Hatch Waxman Amendments permit a patent restoration term of up to five years for one patent per product as compensation for patent term lost during product development and the FDA regulatory review process of that product. The U.S. Patent and Trademark Office, in consultation with the FDA, reviews and approves the application for any patent term extension or restoration.

Market exclusivity provisions under the FDCA can also delay the submission or the approval of certain applications of other companies seeking to reference another company's BLA. Specifically, the Biologics Price Competition and Innovation Act (BPCIA) established an abbreviated pathway for the approval of biosimilar and interchangeable biological products generally not earlier than 12 years after the original BLA approval, although it can be shortened to four years if the biosimilar contains certification of patent invalidity or non-infringement to one of the

Table of Contents

patents listed with the FDA by the innovator BLA holders. The new abbreviated regulatory pathway establishes legal authority for the FDA to review and approve biosimilar biologics, including the possible designation of a biosimilar as “interchangeable” based on their similarity to existing brand product.

U.S. Foreign Corrupt Practices Act

The U.S. Foreign Corrupt Practices Act, to which we are subject, prohibits corporations and individuals from engaging in certain activities to obtain or retain business or to influence a person working in an official capacity. It is illegal to pay, offer to pay or authorize the payment of anything of value to any foreign government official, government staff member, political party or political candidate in an attempt to obtain or retain business or to otherwise influence a person working in an official capacity.

Pharmaceutical Coverage, Pricing and Reimbursement

The cost of pharmaceuticals continues to generate substantial governmental and third party payor interest. We expect that the pharmaceutical industry will experience pricing pressures due to the trend toward managed healthcare, the increasing influence of managed care organizations and additional legislative proposals. Significant uncertainty exists and will continue to exist as to the coverage and reimbursement status of any product candidate for which we obtain regulatory approval. In the United States and markets in other countries, sales of any product for which we receive regulatory approval for commercial sale will depend in part on the availability of coverage and adequate reimbursement from third party payors. Third-party payors include government payor programs at the federal and state levels, including Medicare and Medicaid, managed care providers, private health insurers and other organizations. Third party payors are increasingly challenging the price and examining the medical necessity and cost effectiveness of medical products and services, in addition to their safety and efficacy. Formulary placement by third-party payors is very competitive and can lead to lower prices and may effectively restrict patient access to our drugs. We may need to conduct expensive pharmacoeconomic studies in order to demonstrate the medical necessity and cost-effectiveness of our products, in addition to the costs required to obtain the FDA approvals. Our product candidates may not be considered medically necessary or cost-effective.

The U.S. government, state legislatures and foreign governments have shown significant interest in implementing cost containment programs to limit the growth of government-paid healthcare costs, including price controls, restrictions on reimbursement and requirements for substitution of generic products for branded prescription drugs. Different pricing and reimbursement schemes exist in other countries. In the European Community, governments influence the price of pharmaceutical products through their pricing and reimbursement rules and control of national healthcare systems that fund a large part of the cost of those products to consumers. The downward pressure on healthcare costs in general, and particularly prescription drugs, has become very intense. As a result, increasingly high barriers are being erected to the entry of new products. In addition, in some countries, cross border imports from low priced markets exert a commercial pressure on pricing within a country. There can be no assurance that any country that has price controls or reimbursement limitations for drug products will allow favorable reimbursement and pricing arrangements for any of our products.

Healthcare Reform

In the United States and foreign jurisdictions, there have been and will continue to be a number of legislative and regulatory proposals to change the healthcare system in ways that could affect our ability to sell our product candidates profitably, once they are approved for sale. Among policy makers and payors in the United States and elsewhere, there is significant interest in promoting changes in healthcare systems with the stated goals of containing healthcare costs, improving quality and/or expanding access. In the United States, the pharmaceutical industry has been a particular focus of these efforts and has been significantly affected by major legislative initiatives.

In March 2010, the Patient Protection and Affordable Health Care Act, as amended by the Health Care and Education Affordability Reconciliation Act (collectively, PPACA) was enacted, which significantly changes the way healthcare is financed by both governmental and private insurers. Among other cost containment measures, PPACA establishes: an annual, nondeductible fee on any entity that manufactures or imports certain branded prescription drugs

Table of Contents

and biologic agents; a new Medicare Part D coverage gap discount program; and a new formula that increases the rebates a manufacturer must pay under the Medicaid Drug Rebate Program. In addition, other legislative changes have been adopted and proposed since PPACA was enacted including legislation reducing Medicare payments to providers and reductions to payments under other government programs. Additional new laws may result in additional reductions in Medicare and other healthcare funding, which could have a material adverse effect on our customers and accordingly, our financial operations.

Other Healthcare Laws and Compliance Requirements

In the United States, the research, manufacturing, distribution, sale and promotion of drug products and medical devices are potentially subject to regulation by various federal, state and local authorities in addition to the FDA, including CMS, other divisions of HHS (e.g., the Office of Inspector General), the U.S. Department of Justice, state Attorneys General, and other state and local government agencies. For example, sales, marketing and scientific/educational grant programs must comply with fraud and abuse laws such as the federal Anti Kickback Statute, as amended, the federal False Claims Act, as amended, and similar state laws. Pricing and rebate programs must comply with the Medicaid Drug Rebate Program requirements of the Omnibus Budget Reconciliation Act of 1990, as amended, and the Veterans Health Care Act of 1992, as amended. If products are made available to authorized users of the Federal Supply Schedule of the General Services Administration, additional laws and requirements apply. All of these activities are also potentially subject to federal and state consumer protection and unfair competition laws.

The federal Anti Kickback Statute prohibits any person, including a prescription drug manufacturer (or a party acting on its behalf), from knowingly and willfully soliciting, receiving, offering or providing remuneration, directly or indirectly, to induce or reward either the referral of an individual, or the furnishing, recommending or arranging for a good or service, for which payment may be made under a federal healthcare program such as the Medicare and Medicaid programs. Additionally, many states have adopted laws similar to the federal Anti Kickback Statute, and some of these state prohibitions apply to referral of patients for healthcare items or services reimbursed by any third party payor, not only the Medicare and Medicaid programs. The federal False Claims Act imposes liability on any person or entity that, among other things, knowingly presents or causes to be presented, a false or fraudulent claim for payment by a federal healthcare program. The qui tam provisions of the False Claims Act allow a private individual to bring civil actions on behalf of the federal government alleging that the defendant has submitted a false claim to the federal government, and to share in any monetary recovery. In recent years, the number of suits brought by private individuals has increased dramatically. In addition, various states have enacted false claims laws analogous to the False Claims Act. Also, the Health Insurance Portability and Accountability Act of 1996 (HIPAA) created several new federal crimes, including healthcare fraud, and false statements relating to the delivery of or payments for healthcare benefits, items or services. HIPAA and its implementing regulations also established uniform federal standards for certain “covered entities” (healthcare providers, health plans and healthcare clearinghouses) governing the conduct of certain electronic healthcare transactions and protecting the security and privacy of protected health information. Many states also have similar laws. Under a federal “sunshine law” manufacturers are required to track and report to the federal government certain payments and other transfers of value made to physicians and other healthcare professionals and teaching hospitals and ownership or investment interests held by physicians and their immediate family members. There are also an increasing number of state “sunshine” laws that require manufacturers to make reports to states on pricing and marketing information. Several states have enacted legislation requiring pharmaceutical companies to, among other things, establish marketing compliance programs, file periodic reports with the state, make periodic public disclosures on sales, marketing, pricing, clinical trials and other activities and/or register their sales representatives and prohibiting or limiting certain other sales and marketing practices.

Because of the breadth of these laws and the narrowness of available statutory and regulatory exemptions, it is possible that some of our business activities could be subject to challenge under one or more of such laws. If our

operations are found to be in violation of any of the federal and state laws described above or any other governmental regulations that apply to us, we may be subject to penalties, including criminal and significant civil monetary penalties, damages, fines, imprisonment, exclusion from participation in government healthcare programs, injunctions, recall or seizure of products, total or partial suspension of production, denial or withdrawal of pre marketing product approvals, private “qui tam” actions brought by individual whistleblowers in the name of the government or refusal to allow us to

35

Table of Contents

enter into supply contracts, including government contracts, and the curtailment or restructuring of our operations, any of which could adversely affect our ability to operate our business and our results of operations.

Europe / Rest of World Government Regulation

In addition to regulations in the United States, we, and our collaborators, will be subject to a variety of regulations in other jurisdictions governing, among other things, clinical trials and any commercial sales, marketing and distribution of our products, similar or more stringent than the U.S. laws.

Whether or not we, or our collaborators, obtain FDA approval for a product, we must obtain the requisite approvals from regulatory authorities in foreign countries prior to the commencement of clinical trials or marketing of the product in those countries. The requirements and process governing the conduct of clinical trials, product licensing, pricing and reimbursement vary from country to country. In addition, we and our collaborators may be subject to foreign laws and regulations and other compliance requirements, including, without limitation, anti kickback laws, false claims laws and other fraud and abuse laws, as well as laws and regulations requiring transparency of pricing and marketing information and governing the privacy and security of health information, such as the European Union's Directive 95/46 on the Protection of Individuals with regard to the Processing of Personal Data.

If we, or our collaborators, fail to comply with applicable foreign regulatory requirements, we may be subject to, among other things, fines, suspension or withdrawal of regulatory approvals, product recalls, seizure of products, operating restrictions and criminal prosecution.

Employees

As of December 31, 2014, we had 39 employees, 37 of whom were full time, 16 of whom hold Ph.D. or M.D. degrees, 28 of whom were engaged in research and development activities and 11 of whom were engaged in business development, finance, information systems, facilities, human resources or administrative support. None of our employees are represented by any collective bargaining unit. We believe that we maintain good relations with our employees.

About Xencor

We were incorporated in California in August 1997 under the name Xencor. In September 2004, we reincorporated the state of Delaware under the name Xencor, Inc. Our principal offices are located at 111 West Lemon Avenue, Monrovia, CA 91016, and our telephone number (626) 305 5900. Our website address is www.xencor.com. Our website and the information contained on, or that can be accessed through, the website will not be deemed to be incorporated by reference in, and are not considered part of, this Annual Report on Form 10 K. Our Annual Reports on Form 10 K, Quarterly Reports on Form 10 Q, Current Reports on Form 8 K and amendments to reports filed or furnished pursuant to Section 13(a) and 15(d) of the Securities Exchange Act of 1934, as amended, are available free of charge of the Investor Relations portion of our web site at www.xencor.com as soon as reasonably practical after we electronically file such material with, or furnish it to, the Securities and Exchange Commission (SEC).

We have a single operating segment and substantially all of our operating assets are located in the United States. For information regarding our revenue and research and development expenses for the last three fiscal years, see Item 7, 'Management's Discussion and Analysis of Financial Conditions and Results of Operations.'

Item 1A. Risk Factors.

Except for the historical information contained herein or incorporated by reference, this Annual Report and the information incorporated by reference contains forward looking statements that involve risks and uncertainties. These statements include projections about our accounting and finances, plans and objectives for the future, future operating and economic performance and other statements regarding future performance. These statements are not guarantees of future performance or events. Our actual results may differ materially from those discussed here. Factors that could cause or contribute to differences in our actual results include those discussed in the following section, as well as those

Table of Contents

discussed in Part II, Item 7 entitled “Management’s Discussion and Analysis of Financial Condition and Results of Operations” and elsewhere throughout this Annual Report and in any other documents incorporated by reference into this Annual Report. You should consider carefully the following risk factors, together with all of the other information included or incorporated in this Annual Report. Each of these risk factors, either alone or taken together, could adversely affect our business, operating results and financial condition, as well as adversely affect the value of an investment in our common stock. There may be additional risks that we do not presently know of or that we currently believe are immaterial which could also impair our business and financial position.

Risks Relating to Our Business and to the Discovery, Development and Regulatory Approval of Our Product Candidates

We have incurred significant losses since our inception and anticipate that we will continue to incur significant losses for the foreseeable future.

We are a clinical stage biopharmaceutical company. To date, we have financed our operations primarily through equity and debt financings and our research and licensing agreements and have incurred significant operating losses since our inception in 1997. Our net loss for the years ended December 31, 2014 and 2013 respectively, was \$16.4 million and \$60.3 million (including a \$48.6 million loss on settlement of convertible notes). Such losses are expected to increase in the future as we execute our plan to continue our discovery, research and development activities, including the ongoing and planned clinical development of our antibody product candidates, and incur the additional costs of operating as a public company. We are unable to predict the extent of any future losses or when we will become profitable, if ever. Even if we do achieve profitability, we may not be able to sustain or increase profitability on an ongoing basis which would adversely affect our business, prospects, financial condition and results of operations.

Biopharmaceutical product development is a highly speculative undertaking and involves a substantial degree of uncertainty. We have never generated any revenue from product sales and may never be profitable.

We have devoted substantially all of our financial resources and efforts to developing our proprietary XmAb technology platform, identifying potential product candidates and conducting preclinical studies and clinical trials. We and our partners are still in the early stages of developing our product candidates, and we have not completed development of any products. Our revenue to date has been primarily revenue from the license of our proprietary XmAb technology platform for the development of product candidates by others or revenue from our partners. Our ability to generate revenue and achieve profitability depends in large part on our ability, alone or with partners, to achieve milestones and to successfully complete the development of, obtain the necessary regulatory approvals for, and commercialize, product candidates. We do not anticipate generating revenues from sales of products for the foreseeable future. Our ability to generate future revenues from product sales depends heavily on our and our partners’ success in:

- completing clinical trials through all phases of clinical development of our current product candidates, XmAb5871 and XmAb7195, as well as the product candidates that are being developed by our partners and licensees;
- seeking and obtaining marketing approvals for product candidates that successfully complete clinical trials;
- launching and commercializing product candidates for which we obtain marketing approval, with a partner or, if launched independently, successfully establishing a sales force, marketing and distribution infrastructure;
- identifying and developing new XmAb engineered therapeutic antibody candidates;
- establishing and maintaining supply and manufacturing relationships with third parties;
- obtaining additional licensing and partnering opportunities, similar to our partnership with MorphoSys for XmAb5574/MOR208, with leading pharmaceutical and biotechnology companies;
 - achieving the milestones set forth in our agreements with our partners;

Table of Contents

- conducting further research into the function and application of antibody Fc domains in order to expand the scope of our proprietary XmAb technology platform;
- maintaining, protecting, expanding and enforcing our intellectual property; and
- attracting, hiring and retaining qualified personnel.

Because of the numerous risks and uncertainties associated with biologic product development, we are unable to predict the timing or amount of increased expenses and when we will be able to achieve or maintain profitability, if ever. In addition, our expenses could increase beyond expectations if we are required by the U.S. Food and Drug Administration (FDA), or foreign regulatory agencies, to perform studies and trials in addition to those that we currently anticipate, or if there are any delays in our or our partners completing clinical trials or the development of any of our product candidates. If one or more of the product candidates that we independently develop is approved for commercial sale, we anticipate incurring significant costs associated with commercializing such product candidates. Even if we or our partners are able to generate revenues from the sale of any approved products, we may not become profitable and may need to obtain additional funding to continue operations, which may not be available to us on favorable terms, if at all. Even if we do achieve profitability, we may not be able to sustain or increase profitability on a quarterly or annual basis. Our failure to become and remain profitable would depress the value of our company and could impair our ability to raise capital, expand our business, maintain our research and development efforts, diversify our product offerings or even continue our operations. A decline in the value of our company could also cause you to lose all or part of your investment.

We will require additional financing and may be unable to raise sufficient capital, which could lead us to delay, reduce or abandon research and development programs or commercialization.

As of December 31, 2014, we had \$54.7 million in cash and cash equivalents. We expect our expenses to increase in connection with our ongoing development activities, including additional clinical trials of XmAb5871 and XmAb7195, and, continued development of our bi specific drug candidates including our initial development candidate, XmAb14045, and other research activities. Identifying potential product candidates and conducting preclinical testing and clinical trials are time consuming, expensive and uncertain processes that take years to complete, and we or our partners may never generate the necessary data or results required to obtain regulatory approval and achieve product sales. In addition, our product candidates, if approved, may not achieve commercial success.

Our commercial revenues, if any, will be derived from sales of products that we do not expect to be commercially available for many years, if at all. If we obtain regulatory approval for any of our product candidates, we expect to incur significant commercialization expenses related to product manufacturing, marketing, sales and distribution. Accordingly, we will need to obtain substantial additional funding in connection with our continuing operations. If we are unable to raise capital when needed or on attractive terms, we could be forced to delay, reduce or eliminate our research and development programs or any future commercialization efforts.

We believe our existing cash, together with interest thereon, will be sufficient to fund our operations through the end of 2016. However, changing circumstances or inaccurate estimates by us may cause us to use capital significantly faster than we currently anticipate, and we may need to spend more money than currently expected because of circumstances beyond our control. For example, our planned clinical trials for XmAb5871, XmAb7195, or clinical trials for other drug candidates may encounter technical, enrollment or other issues that could cause our development costs to increase more than we expect. We do not have sufficient cash to complete the clinical development of any of our product candidates and will require additional funding in order to complete the development activities required for regulatory approval of either XmAb5871 or XmAb7195 or XmAb14045 or any other future product candidates that we develop independently. Because successful development of our product candidates is uncertain, we are unable to estimate the actual funds we will require to complete research and development and commercialize our product candidates. Adequate additional financing may not be available to us on acceptable terms, or at all. In addition, we

may seek additional capital due to favorable market conditions or strategic considerations; even if we believe we have sufficient funds for our current or future operating plans. If we are unable to raise capital when needed or on attractive terms, we could be forced to delay, reduce or eliminate our research and development programs or any future commercialization efforts.

Table of Contents

The development and commercialization of biologic products is subject to extensive regulation, and we may not obtain regulatory approvals for any of our product candidates.

The clinical development, manufacturing, labeling, packaging, storage, recordkeeping, advertising, promotion, export, import, marketing and distribution and other possible activities relating to XmAb5871, XmAb7195, and XmAb5574/MOR208, our current lead antibody product candidates, as well as any other antibody product candidate that we may develop in the future, are subject to extensive regulation in the United States as biologics. Biologics require the submission of a Biologics License Application (BLA) to the FDA and we are not permitted to market any product candidate in the United States until we obtain approval from the FDA of a BLA for that product. A BLA must be supported by extensive clinical and preclinical data, as well as extensive information regarding chemistry, manufacturing and controls (CMC) sufficient to demonstrate the safety, purity, potency and effectiveness of the applicable product candidate to the satisfaction of the FDA.

Regulatory approval of a BLA is not guaranteed, and the approval process is an expensive and uncertain process that may take several years. The FDA and foreign regulatory entities also have substantial discretion in the approval process. The number and types of preclinical studies and clinical trials that will be required for BLA approval varies depending on the product candidate, the disease or the condition that the product candidate is designed to target and the regulations applicable to any particular product candidate. Despite the time and expense associated with preclinical studies and clinical trials, failure can occur at any stage, and we could encounter problems that require us to repeat or perform additional preclinical studies or clinical trials or generate additional CMC data. The FDA and similar foreign authorities could delay, limit or deny approval of a product candidate for many reasons, including because they:

- may not deem our product candidate to be adequately safe and effective;
- may not find the data from our preclinical studies and clinical trials or CMC data to be sufficient to support a claim of safety and efficacy;
- may not approve the manufacturing processes or facilities associated with our product candidate;
- may conclude that we have not sufficiently demonstrated long term stability of the formulation of the drug product for which we are seeking marketing approval;
- may change approval policies or adopt new regulations; or
- may not accept a submission due to, among other reasons, the content or formatting of the submission.

Generally, public concern regarding the safety of drug and biologic products could delay or limit our ability to obtain regulatory approval, result in the inclusion of unfavorable information in our labeling, or require us to undertake other activities that may entail additional costs.

We have not submitted an application for approval or obtained FDA approval for any product. This lack of experience may impede our ability to obtain FDA approval in a timely manner, if at all, for our product candidates.

To market any biologics outside of the United States, we and current or future collaborators must comply with numerous and varying regulatory and compliance related requirements of other countries. Approval procedures vary among countries and can involve additional product testing and additional administrative review periods, including obtaining reimbursement and pricing approval in select markets. The time required to obtain approval in other countries might differ from that required to obtain FDA approval. The regulatory approval process in other countries may include all of the risks associated with FDA approval as well as additional, presently unanticipated, risks. Regulatory approval in one country does not ensure regulatory approval in another, but a failure or delay in obtaining regulatory approval in one country may negatively impact the regulatory process in others, including the risk that our product candidates may not be approved for all indications requested and that such approval may be subject to limitations on the indicated uses for which the drug may be marketed. Certain countries have a very difficult reimbursement environment and we may not

Table of Contents

obtain reimbursement or pricing approval, if required, in all countries where we expect to market a product, or we may obtain reimbursement approval at a level that would make marketing a product in certain countries not viable.

If we experience delays in obtaining approval or if we fail to obtain approval of our product candidates, the commercial prospects for our product candidates may be harmed and our ability to generate revenues will be materially impaired which would adversely affect our business, prospects, financial condition and results of operations.

Even if we receive regulatory approval for any of our product candidates, we will be subject to ongoing regulatory obligations and continued regulatory review, which may result in significant additional expense. Additionally, our product candidates, if approved, could be subject to labeling and other restrictions and market withdrawal and we may be subject to penalties if we fail to comply with regulatory requirements or experience unanticipated problems with our products.

Any regulatory approvals that we or our partners receive for our product candidates may also be subject to limitations on the approved indicated uses for which the product may be marketed or to the conditions of approval, or contain requirements for potentially costly post marketing testing, including Phase 4 clinical trials, and surveillance to monitor the safety and efficacy of the product candidate. In addition, if the FDA or a comparable foreign regulatory authority approves any of our product candidates, the manufacturing processes, labeling, packaging, distribution, adverse event reporting, storage, advertising, promotion, import, export and recordkeeping for the product will be subject to extensive and ongoing regulatory requirements. These requirements include submissions of safety and other post marketing information and reports, registration, as well as continued compliance with current good manufacturing practices (cGMPs), and current good clinical practices (cGCPs), for any clinical trials that we conduct post approval. Later discovery of previously unknown problems with a product, including adverse events of unanticipated severity or frequency, undesirable side effects caused by the product, problems encountered by our third party manufacturers or manufacturing processes, or failure to comply with regulatory requirements, either before or after product approval, may result in, among other things:

- restrictions on the marketing or manufacturing of the product;
- requirements to include additional warnings on the label;
- requirements to create a medication guide outlining the risks to patients;
- withdrawal of the product from the market;
- voluntary or mandatory product recalls;
- requirements to change the way the product is administered or for us to conduct additional clinical trials;
- fines, warning letters or holds on clinical trials;
- refusal by the FDA to approve pending applications or supplements to approved applications filed by us or our strategic partners, or suspension or revocation of product license approvals;
- product seizure or detention, or refusal to permit the import or export of products;
- injunctions or the imposition of civil or criminal penalties; and
- harm to our reputation.

Additionally if any of our product candidates receives marketing approval, the FDA could require us to adopt a Risk Evaluation and Mitigation Strategy (REMS) to ensure that the benefits of the therapy outweigh its risks, which may include, among other things, a medication guide outlining the risks for distribution to patients and a communication plan to health care practitioners.

Table of Contents

Any of these events could prevent us from achieving or maintaining market acceptance of the product or the particular product candidate at issue and could significantly harm our business, prospects, financial condition and results of operations.

The FDA's policies may change and additional government regulations may be enacted that could prevent, limit or delay regulatory approval of our product candidates. We cannot predict the likelihood, nature or extent of government regulation that may arise from future legislation or administrative action, either in the United States or abroad. If we are slow or unable to adapt to changes in existing requirements or the adoption of new requirements or policies, or if we are not able to maintain regulatory compliance, we may lose any marketing approval that we may have obtained and we may not achieve or sustain profitability, which would adversely affect our business, prospects, financial condition and results of operations.

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 product 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 addition, some of our competitors have ongoing clinical trials for product candidates that treat the same indications as our product candidates, and patients who would otherwise be eligible for our clinical trials may instead enroll in clinical trials of our competitors' product candidates.

Patient enrollment is affected by other factors including:

- the severity of the disease under investigation;
- the patient eligibility criteria for the study in question;
- the perceived risks and benefits of the product candidate under study;
- our payments for conducting clinical trials;
- 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.

For example, in our Phase 1a clinical trial of XmAb5871, which we completed in December 2012, delays in patient enrollment that were outside our control caused several weeks of delay that we did not predict at the outset of that clinical trial. Our inability to enroll a sufficient number of patients for any of our clinical trials could 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 product candidates and in delays to commercially launching our product candidates, if approved, which would cause the value of our company to decline and limit our ability to obtain additional financing.

The manufacture of biopharmaceutical products, including XmAb engineered antibodies, is complex and manufacturers often encounter difficulties in production. If we or any of our third party manufacturers encounter any loss of our master cell banks or if any of our third party manufacturers encounter other difficulties, or otherwise fail to comply with their contractual obligations, our ability to provide product candidates for clinical trials or our products to patients, once approved, the development or commercialization of our product candidates could be delayed or stopped.

The manufacture of biopharmaceutical products is complex and requires significant expertise and capital investment, including the development of advanced manufacturing techniques and process controls. We and our contract

Table of Contents

manufacturers must comply with cGMP regulations and guidelines. Manufacturers of biopharmaceutical products often encounter difficulties in production, particularly in scaling up and validating initial production and contamination. These problems include difficulties with production costs and yields, quality control, including stability of the product, quality assurance testing, operator error, shortages of qualified personnel, as well as compliance with strictly enforced federal, state and foreign regulations. Furthermore, if microbial, viral or other contaminations are discovered in our products or in the manufacturing facilities in which our products are made, such manufacturing facilities may need to be closed for an extended period of time to investigate and remedy the contamination.

All of our XmAb engineered antibodies are manufactured by starting with cells which are stored in a cell bank. We have one master cell bank for each antibody manufactured in accordance with cGMP and multiple working cell banks and believe we would have adequate backup should any cell bank be lost in a catastrophic event. However, it is possible that we could lose multiple cell banks and have our manufacturing severely impacted by the need to replace the cell banks.

We cannot assure you that any stability or other issues relating to the manufacture of any of our product candidates or products will not occur in the future. Additionally, our manufacturer may experience manufacturing difficulties due to resource constraints or as a result of labor disputes or unstable political environments. If our manufacturers were to encounter any of these difficulties, or otherwise fail to comply with their contractual obligations, our ability to provide any product candidates to patients in clinical trials and products to patients, once approved, would be jeopardized. Any delay or interruption in the supply of clinical trial supplies could delay the completion of clinical trials, increase the costs associated with maintaining clinical trial programs and, depending upon the period of delay, require us to commence new clinical trials at additional expense or terminate clinical trials completely. Any adverse developments affecting clinical or commercial manufacturing of our product candidates or products may result in shipment delays, inventory shortages, lot failures, product withdrawals or recalls, or other interruptions in the supply of our product candidates or products. We may also have to take inventory write offs and incur other charges and expenses for product candidates or products that fail to meet specifications, undertake costly remediation efforts or seek more costly manufacturing alternatives. Accordingly, failures or difficulties faced at any level of our supply chain could materially adversely affect our business and delay or impede the development and commercialization of any of our product candidates or products and could have a material adverse effect on our business, prospects, financial condition and results of operations.

Adverse side effects or other safety risks associated with our product candidates could delay or preclude approval, cause us to suspend or discontinue clinical trials, abandon product candidates, limit the commercial profile of an approved label, or result in significant negative consequences following marketing approval, if any.

Undesirable side effects caused by our product candidates could result in the delay, suspension or termination of clinical trials by us, our collaborators, the FDA or other regulatory authorities for a number of reasons. If we elect or are required to delay, suspend or terminate any clinical trial of any product candidates that we develop, the commercial prospects of such product candidates will be harmed and our ability to generate product revenues from any of these product candidates will be delayed or eliminated. Serious adverse events observed in clinical trials could hinder or prevent market acceptance of the product candidate at issue. Any of these occurrences may harm our business, prospects, financial condition and results of operations significantly.

In our Phase 1b/2a clinical trial of XmAb5871, for example, some subjects reported mild to moderate gastrointestinal toxicities (nausea, vomiting and diarrhea). Other treatment related adverse events experienced in more than two XmAb5871-treated patients were pyrexia (fever) and headache. Treatment related serious adverse events occurred in two patients that received XmAb5871: infusion related reaction and venous thrombosis. Further, interim analysis in our Phase 1a clinical trial of XmAb7195 resulted in subjects having urticaria and dose limiting thrombocytopenia. If

these or other side effects cause excessive discomfort, safety risks or reduction in acceptable dosage, then the development and commercialization of XmAb5871 or XmAb7195 could suffer significant negative consequences. We cannot predict if additional types of adverse events or more serious adverse events will be observed in future clinical trials of XmAb5871, XmAb7195 or any future product candidate.

Table of Contents

In addition, we observed detectable levels of immunogenicity, or the creation by the immune system of anti XmAb5871 antibodies, in 44% of subjects receiving XmAb5871 in the Phase 1a clinical trial. While a common occurrence for antibody therapies, immunogenicity to XmAb5871 or any of our other product candidates could neutralize the therapeutic effects of XmAb5871 or such other candidates and/or alter their pharmacokinetics, which could have a material adverse effect on the effectiveness of our product candidates and on our ability to commercialize them.

We may not be successful in our efforts to use and expand our XmAb technology platform to build a pipeline of product candidates and develop marketable products.

We are using our proprietary XmAb technology platform to develop engineered antibodies, with an initial focus on four properties: immune inhibition, cytotoxicity, extended half life and most recently, heterodimeric Fc domains enabling molecules with dual target binding. This platform has led to our three lead product candidates, XmAb5871, XmAb7195 and, XmAb5574/MOR208, and our first bispecific development candidate, XmAb14045, as well as the other programs that utilize our technology and that are being developed by our partners and licensees. While we believe our preclinical and clinical data to date, together with our established partnerships, has validated our platform to a degree, we are at a very early stage of development and our platform has not yet, and may never lead to, approved or marketable therapeutic antibody products. Even if we are successful in continuing to build our pipeline, the potential product candidates that we identify may not be suitable for clinical development, including as a result of their harmful side effects, limited efficacy or other characteristics that indicate that they are unlikely to be products that will receive marketing approval and achieve market acceptance. If we do not successfully develop and commercialize product candidates based upon our technological approach, we may not be able to obtain product or partnership revenues in future periods, which would adversely affect our business, prospects, financial condition and results of operations.

We face significant competition from other biotechnology and pharmaceutical companies and our operating results will suffer if we fail to compete effectively.

The biotechnology and pharmaceutical industries are intensely competitive. We have competitors both in the United States and internationally, including major multinational pharmaceutical companies, biotechnology companies, universities and other research institutions. Many of our competitors have substantially greater financial, technical and other resources, such as larger research and development staff and experienced marketing and manufacturing organizations and well established sales forces. Competition may increase further as a result of advances in the commercial applicability of technologies and greater availability of capital for investment in these industries. Our competitors may succeed in developing, acquiring or licensing on an exclusive basis, drug products that are more effective or less costly than any product candidate that we are currently developing or that we may develop.

We face intense competition in autoimmune disease drug development from multiple monoclonal antibodies, other biologics and small molecules approved for the treatment of autoimmune diseases many of which are being developed or marketed by large multinational pharmaceutical companies such as GlaxoSmithKline plc, AbbVie Inc., Janssen Pharmaceuticals, Inc., Roche/Genentech Inc. and Amgen Inc. GlaxoSmithKline's Benlysta (belimumab) is currently the only monoclonal antibody that we are aware of that is approved for the treatment of lupus although we believe that Biogen Idec/Genentech's Rituxan (rituximab) is prescribed, off label, for this indication. In addition, these and other pharmaceutical companies have monoclonal antibodies or other biologics in clinical development for the treatment of autoimmune diseases.

Many companies have approved therapies or are developing drugs for the treatment of asthma including multinational pharmaceutical companies such as GlaxoSmithKline, Roche/Genentech, Novartis AG and AstraZeneca plc. Monoclonal antibody drug development has primarily focused on allergic asthma. Xolair is currently the only

monoclonal antibody that we are aware of that is approved for the treatment of severe asthma. In addition, Novartis and Genentech each have an antibody targeting IgE in Phase 1 or 2 clinical development for asthma.

Competition in blood cancer drug development is intense, with more than 250 compounds in clinical trials by large multinational pharmaceutical companies and Rituxan is just one of many monoclonal antibodies approved for the treatment of non-Hodgkin lymphomas or other blood cancers.

Table of Contents

Our ability to compete successfully will depend largely on our ability to leverage our experience in drug discovery and development to:

- discover and develop products that are superior to other products in the market;
- attract qualified scientific, product development and commercial personnel;
- obtain and maintain patent and/or other proprietary protection for our products and technologies;
- obtain required regulatory approvals; and
- successfully collaborate with pharmaceutical companies in the discovery, development and commercialization of new products.

The availability and price of our competitors' products could limit the demand, and the price we are able to charge, for any of our product candidates, if approved. We will not achieve our business plan if acceptance is inhibited by price competition or the reluctance of physicians to switch from existing drug products to our products, or if physicians switch to other new drug products or choose to reserve our products for use in limited circumstances.

Established biopharmaceutical companies may invest heavily to accelerate discovery and development of products that could make our product candidates less competitive. In addition, any new product that competes with an approved product must demonstrate compelling advantages in efficacy, convenience, tolerability and safety in order to overcome price competition and to be commercially successful. Accordingly, our competitors may succeed in obtaining patent protection, receiving FDA approval or discovering, developing and commercializing medicines before we do, which would have a material adverse impact on our business. We will not be able to successfully commercialize our product candidates without establishing sales and marketing capabilities internally or through collaborators.

Risks Relating to Our Dependence on Third Parties

Our existing partnerships are important to our business, and future partnerships may also be important to us. If we are unable to maintain any of these partnerships, or if these partnerships are not successful, our business could be adversely affected.

Because developing biologics products, conducting clinical trials, obtaining regulatory approval, establishing manufacturing capabilities and marketing approved products are expensive, we have entered into partnerships, and may seek to enter into additional partnerships, with companies that have more resources and experience than us, and we may become dependent upon the establishment and successful implementation of partnership agreements.

Our partnership and license agreements include those we have announced with MorphoSys AG, Novo Nordisk, Boehringer Ingelheim and others. These partnerships and license agreements also have provided us with important funding for our development programs, and we expect to receive additional funding under these partnerships in the future. Our existing partnerships, and any future partnerships we enter into, may pose a number of risks, including the following:

- collaborators have significant discretion in determining the efforts and resources that they will apply to these partnerships;
- collaborators may not perform their obligations as expected;
- collaborators may not pursue development and commercialization of any product candidates that achieve regulatory approval or may elect not to continue or renew development or commercialization programs based on clinical trial results, changes in the collaborators' strategic focus or available funding, or external factors, such as an acquisition, that divert resources or create competing priorities;

Table of Contents

- collaborators may delay clinical trials, provide insufficient funding for a clinical trial program, stop a clinical trial or abandon a product candidate, repeat or conduct new clinical trials or require a new formulation of a product candidate for clinical testing;
- collaborators could independently develop, or develop with third parties, products that compete directly or indirectly with our products or product candidates if the collaborators believe that competitive products are more likely to be successfully developed or can be commercialized under terms that are more economically attractive than ours, which may cause collaborators to cease to devote resources to the commercialization of our product candidates;
- a collaborator with marketing and distribution rights to one or more of our product candidates that achieve regulatory approval may not commit sufficient resources to the marketing and distribution of such product or products;
- disagreements with collaborators, including disagreements over proprietary rights, contract interpretation or the preferred course of development, might cause delays or termination of the research, development or commercialization of product candidates, might lead to additional responsibilities for us with respect to product candidates, or might result in litigation or arbitration, any of which would be time consuming and expensive;
- while we have generally retained the right to maintain and defend our intellectual property under our agreements with collaborators, certain collaborators may not properly maintain or defend certain of our intellectual property rights or may use our proprietary information in such a way as to invite litigation that could jeopardize or invalidate our intellectual property or proprietary information;
- collaborators may infringe the intellectual property rights of third parties, which may expose us to litigation and potential liability;
- collaborators may learn about our technology and use this knowledge to compete with us in the future;
- results of collaborators' preclinical or clinical studies could produce results that harm or impair other products using our XmAb technology platform;
- there may be conflicts between different collaborators that could negatively affect those partnerships and potentially others; and
- the number and type of our partnerships could adversely affect our attractiveness to future collaborators or acquirers.

If our partnerships and license agreements do not result in the successful development and commercialization of products or if one of our collaborators terminates its agreement with us, we may not receive any future research and development funding or milestone or royalty payments under the arrangement. If we do not receive the funding we expect under these arrangements, our continued development of our product candidates could be delayed and we may need additional resources to develop additional product candidates. All of the risks described in these risk factors relating to product development, regulatory approval and commercialization described in this Annual Report also apply to the activities of our collaborators and there can be no assurance that our partnerships and license agreements will produce positive results or successful products on a timely basis or at all.

Our partnership agreements generally grant our collaborators exclusive rights under certain of our intellectual property, and may therefore preclude us from entering into partnerships with others relating to the same or similar compounds, indications or diseases. In addition, partnership agreements may place restrictions or additional obligations on our ability to license additional compounds in different indications, diseases or geographical locations. If we fail to comply with or breach any provision of a partnership agreement, a collaborator may have the right to terminate, in whole

Table of Contents

or in part, such agreement or to seek damages. Many of our collaborators also have the right to terminate the partnership agreement for convenience. If a partnership agreement is terminated, in whole or in part, we may be unable to continue the development and commercialization of the applicable product candidates, and even if we are able to do so, such efforts may be delayed and result in additional costs.

There is no assurance that a collaborator who is acquired by a third party would not attempt to change certain contract provisions that could negatively affect our partnership. The acquiring company may also not accept the terms or assignment of our contracts and may seek to terminate the agreements. Any one of our partners could breach covenants, restrictions and/or sub license agreement provisions leading us into disputes and potential breaches of our agreements with other partners.

We may in the future determine to partner with additional pharmaceutical and biotechnology companies for development and potential commercialization of therapeutic products. We face significant competition in seeking appropriate collaborators. Our ability to reach a definitive agreement for a partnership will depend, among other things, upon our assessment of the collaborator's resources and expertise, the terms and conditions of the proposed partnership and the proposed collaborator's evaluation of a number of factors. If we elect to fund and undertake development or commercialization activities on our own, we may need to obtain additional expertise and additional capital, which may not be available to us on acceptable terms or at all. If we fail to enter into partnerships and do not have sufficient funds or expertise to undertake the necessary development and commercialization activities, we may not be able to further develop our product candidates or bring them to market or continue to develop our product platform and our business, prospects, financial condition and results of operations may be materially and adversely affected.

We rely upon third party contractors and service providers for the execution of most aspects of our development programs. Failure of these collaborators to provide services of a suitable quality and within acceptable timeframes may cause the delay or failure of our development programs.

We outsource certain functions, tests and services to contract research organizations (CROs), medical institutions and collaborators as well as outsourcing manufacturing to collaborators and/or contract manufacturers, and we rely on third parties for quality assurance, clinical monitoring, clinical data management and regulatory expertise. We also have engaged, and may in the future engage, a CRO to run all aspects of a clinical trial on our behalf. There is no assurance that such individuals or organizations will be able to provide the functions, tests, biologic supply or services as agreed upon or in a quality fashion and we could suffer significant delays in the development of our products or processes.

In some cases there may be only one or few providers of such services, including clinical data management or manufacturing services. In addition, the cost of such services could be significantly increased over time. We rely on third parties and collaborators as mentioned above to enroll qualified patients and conduct, supervise and monitor our clinical trials. Our reliance on these third parties and collaborators for clinical development activities reduces our control over these activities. Our reliance on these parties, however, does not relieve us of our regulatory responsibilities, including ensuring that our clinical trials are conducted in accordance with GCP regulations and the investigational plan and protocols contained in the regulatory agency applications. In addition, these third parties may not complete activities on schedule or may not manufacture under GMP conditions. Preclinical or clinical studies may not be performed or completed in accordance with Good Laboratory Practices (GLP) regulatory requirements or our trial design. If these third parties or collaborators do not successfully carry out their contractual duties or meet expected deadlines, obtaining regulatory approval for manufacturing and commercialization of our product candidates may be delayed or prevented. We rely substantially on third party data managers for our clinical trial data. There is no assurance that these third parties will not make errors in the design, management or retention of our data or data systems. There is no assurance these third parties will pass FDA or regulatory audits, which could delay or prohibit

regulatory approval.

46

Table of Contents

We rely on third parties to manufacture supplies of our preclinical and clinical product candidates. The development of such candidates could be stopped or delayed if any such third party fails to provide us with sufficient quantities of product or fails to do so at acceptable quality levels or prices or fails to maintain or achieve satisfactory regulatory compliance.

We do not currently have nor do we plan to acquire the infrastructure or capability internally to manufacture our clinical drug supplies for use in the conduct of our clinical trials, and we lack the resources and the capability to manufacture any clinical candidates on a clinical scale. Instead, we rely on our third party manufacturing partners, Catalent Pharma Solutions LLC (Catalent) for the production of XmAb5871 and XmAb7195 and third parties for fill and testing services. We are also working with KBI Biopharma, Inc. (KBI) to develop manufacturing processes to initiate manufacturing of our bispecific development candidate, XmAb14045. Any of our contract manufacturers may not perform as agreed, may be unable to comply with cGMP requirements and with FDA, state and foreign regulatory requirements or may terminate their respective agreements with us.

In addition, manufacturers are subject to ongoing periodic unannounced inspection by the FDA and other governmental authorities to ensure strict compliance with government regulations. We do not control the manufacturing processes of either Catalent or KBI and are currently completely dependent on each of Catalent and KBI for the production of XmAb5871, XmAb7195 and XmAb14015 in accordance with cGMP, which include, among other things, quality control, quality assurance and the maintenance of records and documentation. If we were to experience an unexpected loss of supply, we could experience delays in our planned clinical trials, as Catalent or KBI would need to manufacture additional clinical drug supply and would need sufficient lead time to schedule a manufacturing slot. While there are other potential suppliers of clinical supplies of our biologics, the long transition periods necessary to switch manufacturers for any of XmAb5871, XmAb7195 or XmAb14045 would significantly delay our clinical trials and the commercialization of such products, if approved.

We intend to rely on third parties to manufacture commercial supplies of our product candidates, if and when approved. If we are unable to obtain a license agreement from Catalent for the manufacture of XmAb5871, if we are unable to enter into commercial supply agreements with third party suppliers or if any such third party supplier fails to provide us with sufficient quantities or fails to comply with regulatory requirements, commercialization of such products could be delayed or stopped.

We do not currently have nor do we plan to acquire the infrastructure or capability internally to manufacture our products on a commercial scale. Although we have entered into agreements for the manufacture of clinical supplies of XmAb5871, XmAb7195 and XmAb14045, we have not entered into a commercial supply agreement with either Catalent or KBI and neither has demonstrated that it will be capable of manufacturing XmAb5871, XmAb7195 or XmAb14045 on a large commercial scale. We might be unable to identify manufacturers for commercial supply on acceptable terms or at all. Moreover, our existing license with Catalent to use certain technology and know how in the production of our XmAb5871 product candidate only applies for so long as manufacturing services are provided by Catalent. We expect to move manufacturing services to another contract manufacturing organization to support late stage clinical trials for XmAb5871 as well as commercial supplies which would require negotiation of a license from Catalent. We expect to be able to finalize such a license agreement with Catalent for XmAb5871 in due course. However, we can provide no assurances as to when such a license agreement will be executed or if it will be executed at all. If we are not able to secure a commercial license from Catalent, or not able to obtain a commercial license on acceptable terms, we may be required to change the manufacturing process for XmAb5871. A change to the manufacturing process for XmAb5871 or any of our product candidates would cause us to incur significant costs and to devote significant efforts to implement such a change. Additionally, the late stage clinical development and commercialization of XmAb5871 or other product candidates by us or our collaborators may be delayed as a result, which would materially and adversely affect our business.

If our third party manufacturers cannot successfully manufacture material that conforms to our specifications and the applicable regulatory authorities' strict regulatory requirements, or pass regulatory inspection, they will not be able to secure or maintain regulatory approval for the manufacturing facilities. In addition, we have no control over the ability of any third party manufacturer to maintain adequate quality control, quality assurance and qualified personnel. The facilities used by our third party manufacturers to manufacture XmAb5871, XmAb7195, XmAb14045 and any

Table of Contents

other potential product candidates that we may develop in the future must be approved by the applicable regulatory authorities, including the FDA, pursuant to inspections that will be conducted after we submit our BLA to the FDA. In addition, manufacturers are subject to ongoing periodic unannounced inspection by the FDA and other governmental authorities to ensure strict compliance with government regulations. If the FDA or any other applicable regulatory authorities do not approve these facilities for the manufacture of our products or if they withdraw any such approval in the future, or if our suppliers or third party manufacturer decide they no longer want to supply our biologics or manufacture our products, we may need to find alternative manufacturing facilities, which would significantly impact our ability to market our products and our business, prospects, financial condition and results of operations may be materially and adversely affected.

Risks Relating to Our Intellectual Property

If we are unable to obtain, maintain and enforce intellectual property protection covering our products, others may be able to make, use or sell products substantially the same as ours, which could adversely affect our ability to compete in the market.

Our commercial success depends, in part, on our ability to obtain, maintain and enforce patents, trade secrets, trademarks and other intellectual property rights and to operate without having third parties infringe, misappropriate or circumvent the rights that we own or license. If we are unable to obtain, maintain and enforce intellectual property protection covering our products, others may be able to make, use or sell products that are substantially the same as ours without incurring the sizeable development and licensing costs that we have incurred, which would adversely affect our ability to compete in the market. As of December 31, 2014, we held at least 150 issued patents and 170 pending patent applications. We file patent applications in the United States, Canada, Japan, Europe and other major markets either directly or via the Patent Cooperation Treaty. Our ability to stop third parties from making, using, selling, offering to sell or importing our product candidates is dependent upon the extent to which we have rights under valid and enforceable patents or trade secrets that cover these activities. However, the patent positions of biopharmaceutical companies, including ours, can be highly uncertain and involve complex legal and factual questions for which important legal principles remain unresolved. No consistent policy regarding the breadth of claims allowed in patents in these fields has emerged to date in the United States. The U.S. patent laws have recently changed, there have been changes regarding how patent laws are interpreted, and the U.S. Patent and Trademark Office (the PTO) has also implemented changes to the patent system. Some of these changes are currently being litigated, and we cannot accurately determine the outcome of any such proceedings or predict future changes in the interpretation of patent laws or changes to patent laws which might be enacted into law. Those changes may materially affect our patents, our ability to obtain patents or the patents and applications of our collaborators and licensors. The patent situation in the biopharmaceutical industry outside the United States is even more uncertain. Therefore, there is no assurance that our pending patent applications will result in the issuance of patents or that we will develop additional proprietary products which are patentable. Moreover, patents issued or to be issued to us may not provide us with any competitive advantage. Our patent position is subject to numerous additional risks, including the following:

- we may fail to seek patent protection for inventions that are important to our success;
- our pending patent applications may not result in issued patents;
- we cannot be certain that we are the first to invent the inventions covered by pending patent applications or that we were the first to file such applications and, if we are not, we may be subject to priority disputes;
- we may be required to disclaim part or all of the term of certain patents or all of the term of certain patent applications;
- we may file patent applications but have claims restricted or we may not be able to supply sufficient data to support our claims and, as a result, may not obtain the original claims desired or we may receive restricted claims. Alternatively, it is possible that we may not receive any patent protection from an application;

Table of Contents

- we could inadvertently abandon a patent or patent application, resulting in the loss of protection of certain intellectual property rights in a certain country. We, our collaborators or our patent counsel may take action resulting in a patent or patent application becoming abandoned which may not be able to be reinstated or if reinstated, may suffer patent term adjustments;
- the claims of our issued patents or patent applications when issued may not cover our product candidates;
- no assurance can be given that our patents would be declared by a court to be valid or enforceable or that a competitor's technology or product would be found by a court to infringe our patents. Our patents or patent applications may be challenged by third parties in patent litigation or in proceedings before the PTO or its foreign counterparts, and may ultimately be declared invalid or unenforceable, or narrowed in scope;
- there may be prior art of which we are not aware that may affect the validity or enforceability of a patent claim. There also may be prior art of which we are aware, but which we do not believe affects the validity or enforceability of a claim, which may, nonetheless, ultimately be found to affect the validity or enforceability of a claim;
- third parties may develop products which have the same or similar effect as our products without infringing our patents. Such third parties may also intentionally circumvent our patents by means of alternate designs or processes or file applications or be granted patents that would block or hurt our efforts;
- there may be dominating patents relevant to our product candidates of which we are not aware;
- our patent counsel, lawyers or advisors may have given us, or may in the future give us incorrect advice or counsel. Opinions from such patent counsel or lawyers may not be correct or may be based on incomplete facts;
- obtaining regulatory approval for biopharmaceutical products is a lengthy and complex process, and as a result, any patents covering our product candidates may expire before, or shortly after such product candidates are approved and commercialized;
- the patent and patent enforcement laws of some foreign jurisdictions do not protect intellectual property rights to the same extent as laws in the United States, and many companies have encountered significant difficulties in protecting and defending such rights in foreign jurisdictions. If we encounter such difficulties or we are otherwise precluded from effectively protecting our intellectual property rights in foreign jurisdictions, our business prospects could be substantially harmed; and
- we may not develop additional proprietary technologies that are patentable.

Any of these factors could hurt our ability to gain full patent protection for our products. Registered trademarks and trademark applications in the United States and other countries are subject to similar risks as described above for patents and patent applications, in addition to the risks described below.

Many of our product development partnership agreements are complex and may call for licensing or cross licensing of potentially blocking patents, know how or intellectual property. Due to the potential overlap of data, know how and intellectual property rights there can be no assurance that one of our collaborators will not dispute our right to use, license or distribute data, know how or other intellectual property rights, and this may potentially lead to disputes, liability or termination of a program. There are no assurances that our actions or the actions of our collaborators would not lead to disputes or cause us to default with other collaborators. For example, we may become involved in disputes with our collaborators relating to the ownership of intellectual property developed in the course of the partnership. We also cannot be certain that a collaborator will not challenge the validity or enforceability of the patents we license.

Table of Contents

We cannot be certain that any country's patent and/or trademark office will not implement new rules which could seriously affect how we draft, file, prosecute and/or maintain patents, trademarks and patent and trademark applications. We cannot be certain that increasing costs for drafting, filing, prosecuting and maintaining patents, trademarks and patent and trademark applications will not restrict our ability to file for patent protection. For example, we may elect not to seek patent protection in certain jurisdictions or for certain inventions in order to save costs. We may be forced to abandon or return the rights to specific patents due to a lack of financial resources.

We currently rely, and may in the future rely, on certain intellectual property rights licensed from third parties to protect our technology. In particular, we have licensed and sublicensed certain intellectual property relating to our Xtend technology from a third party. Under our license, we have no right to control patent prosecution of this intellectual property or to enforce the patents, and as such the licensed rights may not be adequately maintained by the licensors. The termination of this or other licenses could also prevent us from commercializing product candidates covered by the licensed intellectual property.

Furthermore, the research resulting in the in licensed patents was developed in the course of research funded by the U.S. government. As a result, the U.S. government may have certain rights ("march in rights") to intellectual property embodied in our Xtend products. Government rights in certain inventions developed under a government funded program include a non exclusive, non transferable, irrevocable worldwide license to use inventions for any governmental purpose. Circumstances that trigger march in rights include, for example, failure to take, within a reasonable time, effective steps to achieve practical application of the invention in a field of use, failure to satisfy the health and safety needs of the public and failure to meet requirements of public use specified by federal regulations. Federal law requires any licensor of an invention that was partially funded by the federal government to obtain a covenant from any exclusive licensee to manufacture products using the invention substantially in the United States. The U.S. government also has the right to use and disclose, without limitation, scientific data relating to licensed technology that was developed in whole or in part at government expense. The government funding agency can elect to exercise these march in rights on their own initiative or at the request of a third party.

We intend to file applications for trademark registrations in connection with our product candidates in various jurisdictions, including the United States. No assurance can be given that any of our trademark applications will be registered in the United States or elsewhere, or that the use of any registered or unregistered trademarks will confer a competitive advantage in the marketplace. Furthermore, even if we are successful in our trademark registrations, the FDA and regulatory authorities in other countries have their own process for drug nomenclature and their own views concerning appropriate proprietary names. No assurance can be given that the FDA or any other regulatory authority will approve of any of our trademarks or will not request reconsideration of one of our trademarks at some time in the future. The loss, abandonment, or cancellation of any of our trademarks or trademark applications could negatively affect the success of the product candidates to which they relate.

If we are not able to prevent disclosure of our trade secrets and other proprietary information, the value of our technology and products could be significantly diminished.

We rely on trade secret protection to protect our interests in proprietary know how and in processes for which patents are difficult to obtain or enforce. We may not be able to protect our trade secrets adequately. We have a policy of requiring our consultants, advisors and collaborators to enter into confidentiality agreements and our employees to enter into invention, non disclosure and non compete agreements. However, no assurance can be given that we have entered into appropriate agreements with all parties that have had access to our trade secrets, know how or other proprietary information. There is also no assurance that such agreements will provide for a meaningful protection of our trade secrets, know how or other proprietary information in the event of any unauthorized use or disclosure of information. Furthermore, we cannot provide assurance that any of our employees, consultants, contract personnel, or collaborators, either accidentally or through willful misconduct, will not cause serious damage to our programs and/or

our strategy, for example by disclosing important trade secrets, know how or proprietary information to our competitors. It is also possible that our trade secrets, know how or other proprietary information could be obtained by third parties as a result of breaches of our physical or electronic security systems. Any disclosure of confidential data into the public domain or to third parties could allow our competitors to learn our trade secrets and use the information in competition against us. In addition, others may independently discover our trade secrets and proprietary information. Any action to enforce our

Table of Contents

rights is likely to be time consuming and expensive, and may ultimately be unsuccessful, or may result in a remedy that is not commercially valuable. These risks are accentuated in foreign countries where laws or law enforcement practices may not protect proprietary rights as fully as in the United States or Europe. Any unauthorized disclosure of our trade secrets or proprietary information could harm our competitive position.

We may be required to reduce the scope of our intellectual property due to third party intellectual property claims.

Our competitors may have filed, and may in the future file, patent applications covering technology similar to ours. Any such patent application may have priority over our patent applications, which could further require us to obtain rights to issued patents covering such technologies. If another party has filed a U.S. patent application on inventions similar to ours that claims priority to an application filed prior to March 16, 2013, we may have to participate in an interference proceeding declared by the PTO to determine priority of invention in the United States. The costs of these proceedings could be substantial, and it is possible that such efforts would be unsuccessful if, unbeknownst to us, the other party had independently arrived at the same or similar invention prior to our own invention, resulting in a loss of our U.S. patent position with respect to such inventions. In addition, changes enacted on March 15, 2013 to the U.S. patent laws under the America Invents Act resulted in the United States changing from a “first to invent” country to a “first to file” country. As a result, we may lose the ability to obtain a patent if a third party files with the PTO first and could become involved in proceedings before the PTO to resolve disputes related to inventorship. We may also become involved in similar proceedings in other jurisdictions.

Furthermore, recent changes in U.S. patent law under the America Invents Act allows for post issuance challenges to U.S. patents, including ex parte reexaminations, inter parte reviews and post grant oppositions. There is significant uncertainty as to how the new laws will be applied and if our U.S. patents are challenged using such procedures, we may not prevail, possibly resulting in altered or diminished claim scope or loss of patent rights altogether. Similarly, some countries, notably members of the European Union, also have post grant opposition proceedings that can result in changes in scope and/or cancellation of patent claims.

Our products could infringe patents and other property rights of others, which may result in costly litigation and, if we are not successful, could cause us to pay substantial damages or limit our ability to commercialize our products, which could have a material adverse effect on our business.

Our commercial success depends upon our ability, and the ability of our collaborators, to develop, manufacture, market and sell our product candidates and use our proprietary technologies without infringing the patents and other proprietary rights of third parties. There is considerable intellectual property litigation in the biotechnology and pharmaceutical industries. For example, we are aware of issued U.S. patents and patent applications owned by Genentech that may relate to and claim components of certain of our product candidates, including XmAb5871, XmAb7195 and XmAb5574/MOR208 or their manufacture. We believe that these patents and patent applications will expire in the United States in 2020 and 2021, respectively, but it is possible that the terms could be extended, for example, as a result of patent term restoration to compensate for regulatory delays. While we believe that our current development of these candidates currently falls into the “safe harbor” of non infringement under 35 U.S.C. §271(e)(1), this protection terminates upon commercialization. In addition, there can be no assurance that our interpretation of this statutory exemption would be upheld. Furthermore, while we believe that claims in these patents are either invalid or not infringed, we cannot assure you that if we were sued for infringement of these patents that we would prevail. In order to successfully challenge the validity of any issued U.S. patent, we would need to overcome a presumption of validity. This burden is a high one requiring us to present clear and convincing evidence as to the invalidity of such claims. There is no assurance that a court would find these claims to be invalid or not infringed.

In addition, as the biopharmaceutical industry expands and more patents are issued, the risk increases that there may be patents issued to third parties that relate to our products and technology of which we are not aware or that we must

challenge to continue our operations as currently contemplated. Our products may infringe or may be alleged to infringe these patents. Because some patent applications in the United States may be maintained in secrecy until the patents are issued, because patent applications in the United States and many foreign jurisdictions are typically not published until eighteen months after filing and because publications in the scientific literature often lag behind actual discoveries, we cannot be certain that others have not filed patents that may cover our technologies, our product candidates or their use. Additionally, pending patent applications which have been published can, subject to certain

Table of Contents

limitations, be later amended in a manner that could cover our technologies, our products or the use of our products. We may become party to, or threatened with, future adversarial proceedings or litigation regarding intellectual property rights with respect to our products and technology. Third parties may assert infringement claims against us based on existing patents or patents that may be granted in the future.

If we are sued for patent infringement, we would need to demonstrate that our products or methods either do not infringe the patent claims of the relevant patent or that the patent claims are invalid, and we may not be able to do this. Proving invalidity is difficult. For example, in the United States, proving invalidity requires a showing of clear and convincing evidence to overcome the presumption of validity enjoyed by issued patents. Even if we are successful in these proceedings, we may incur substantial costs and divert management's time and attention in pursuing these proceedings, which could have a material adverse effect on us.

Any such claims are likely to be expensive to defend, and some of our competitors may be able to sustain the costs of complex patent litigation more effectively than we can because they have substantially greater resources.

If we are found to infringe a third party's intellectual property rights, we could be required to obtain a license from such third party to continue developing and marketing our products and technology. We may also elect to enter into such a license in order to settle litigation or in order to resolve disputes prior to litigation. However, we may not be able to obtain any required license on commercially reasonable terms or at all. Even if we were able to obtain a license, it could be non-exclusive, thereby giving our competitors access to the same technologies licensed to us, and could require us to make substantial royalty payments. We could also be forced, including by court order, to cease commercializing the infringing technology or product. In addition, we could be found liable for monetary damages, including treble damages and attorneys' fees if we are found to have willfully infringed a patent. A finding of infringement could prevent us from commercializing our product candidates or force us to cease some of our business operations, which could materially harm our business. Claims that we have misappropriated the confidential information or trade secrets of third parties could have a similar negative impact on our business.

Our intellectual property may be infringed upon by a third party.

Third parties may infringe one or more of our issued patents or trademarks. We cannot predict if, when or where a third party may infringe one or more of our issued patents or trademarks. To counter infringement, we may be required to file infringement claims, which can be expensive and time consuming. There is no assurance that we would be successful in a court of law in proving that a third party is infringing one or more of our issued patents or trademarks. Any claims we assert against perceived infringers could also provoke these parties to assert counterclaims against us, alleging that we infringe their intellectual property. In addition, in a patent infringement proceeding, a court may decide that a patent of ours is invalid or unenforceable, in whole or in part, construe the patent's claims narrowly and/or refuse to stop the other party from using the technology at issue on the grounds that our patents do not cover the technology in question, any of which may adversely affect our business. Even if we are successful in proving in a court of law that a third party is infringing one or more of our issued patents or trademarks there can be no assurance that we would be successful in halting their infringing activities, for example, through a permanent injunction, or that we would be fully or even partially financially compensated for any harm to our business. We may be forced to enter into a license or other agreement with the infringing third party at terms less profitable or otherwise commercially acceptable to us than if the license or agreement were negotiated under conditions between those of a willing licensee and a willing licensor. We may not become aware of a third party infringer within legal timeframes for compensation or at all, thereby possibly losing the ability to be compensated for any harm to our business. Such a third party may be operating in a foreign country where the infringer is difficult to locate and/or the intellectual property laws may be more difficult to enforce. Some third party infringers may be able to sustain the costs of complex infringement litigation more effectively than we can because they have substantially greater resources. Any inability to stop third party infringement could result in loss in market share of some of our products or even lead to a delay,

reduction and/or inhibition of the development, manufacture or sale of certain products by us. There is no assurance that a product produced and sold by a third party infringer would meet our or other regulatory standards or would be safe for use. Such third party infringer products could irreparably harm the reputation of our products thereby resulting in substantial loss in market share and profits.

Table of Contents

We may be subject to claims that our employees, consultants or independent contractors have wrongfully used or disclosed confidential information of third parties.

We employ individuals who were previously employed at other biotechnology or pharmaceutical companies. We may be subject to claims that we or our employees, consultants or independent contractors have inadvertently or otherwise used or disclosed confidential information of our employees' former employers or other third parties. We may also be subject to claims that former employers or other third parties have an ownership interest in our patents. Litigation may be necessary to defend against these claims. There is no guarantee of success in defending these claims, and if we do not prevail, we could be required to pay substantial damages and could lose rights to important intellectual property. Even if we are successful, litigation could result in substantial cost and be a distraction to our management and other employees.

Risks Related to Employee Matters and Managing Growth and Other Risks Related to Our Business

We are subject to competition for our skilled personnel and may experience challenges in identifying and retaining key personnel that could impair our ability to conduct and grow our operations effectively.

Our future success depends on our ability to retain our executive officers and to attract, retain and motivate qualified per