

Xencor, Inc.  
1Q17 Earnings Conference Call Script  
Tuesday, May 9, 2017  
4:30pm ET (1:30pm PT)

**Operator Remarks and Welcome:**

Good afternoon and welcome to the Xencor first quarter 2017 financial results conference call. At this time, all participants are in a listen-only mode. Following the formal remarks, we will open the call up for your questions.

Please be advised that this call is being recorded at the Company's request. At this time, I'd like to turn it over to Josh Rappaport of Stern Investor Relations. Please proceed.

**Forward Looking Statements and Agenda:**

Thank you, operator.

Good afternoon. This is Josh Rappaport with Stern Investor Relations, and welcome to Xencor's first quarter 2017 financial results conference call.

This afternoon, we issued a press release which outlines the topics that we plan to discuss today. The release is available at [www.xencor.com](http://www.xencor.com).

Today on our call, Bassil Dahiyat, Ph.D., President and Chief Executive Officer, will discuss the Company's business highlights and provide an update on the Company's clinical programs and pipeline progress; and John Kuch, Vice President of Finance, will review the financial results from the first quarter of 2017. Then, we will open the call up for your questions.

Before we begin, I would like to remind you that during the course of this conference call, Xencor management may make forward-looking statements, including statements regarding the company's future financial and operating results, future market conditions, the plans and objectives of management for future operations, the company's partnering efforts, the company's capital requirements, the company's future product offerings and the company's research and development programs. These forward-looking statements are not historical facts but rather are based on Xencor's current expectations and beliefs and are based on information currently available to us. The outcome of the events described in these forward-looking statements is subject to known and unknown risks, uncertainties and other factors that could cause actual results to differ materially from the results anticipated by these forward-looking statements, including, but not limited to those factors contained in the "Risk Factors" sections of Xencor's most recently filed Annual Report on Form 10-K and Quarterly Report on Form 10-Q.

With that, let me turn it over to Bassil.

**Introduction and Corporate Remarks (Bassil):**

Thanks, Josh, and good afternoon everyone.

In the first quarter, we continued to work on advancing the broad pipeline of potential best-in-class antibodies for the treatment of autoimmune and allergic diseases and cancer that we've built using our XmAb platform. Throughout this year and in 2018, we expect to turn over the cards on multiple lead programs while we continue to expand our earlier pipeline with new bispecific oncology programs.

We are announcing one important 2017 milestone today, the results from our Phase 1 subcutaneous administration trial of XmAb5871. The data show that 5871 was safe and well-tolerated as a subcutaneous injection, and the pharmacokinetic and bioavailability data from the trial support an every other week dosing schedule. This is an important step for our 5871 program: subcutaneous dosing on an every other week schedule offers patients and doctors a simpler, more flexible treatment option than infusions. We plan to use subcutaneous dosing in our future clinical trials for XmAb5871.

For the remainder of 2017, we expect top line results from our Phase 2 study of 5871 in IgG4-Related Disease, aka IgG4-RD, this year, and to engage with the U.S. FDA to discuss the development plan, including future trials and potential registration requirements. We are also on track to announce top line data from our subcutaneous administration Phase 1 study of XmAb7195 and initial data from our Phase 1 study of XmAb14045, pending alignment with Novartis.

Big picture, we expect to have proof-of-concept clinical data for our four lead programs by the end of 2018. This will allow us to select the best program, or programs, to independently advance into later stages of development, as we continue to mature Xencor into a fully-integrated biotech company. And we are planning the IND filings and clinical trial starts for our next two bispecific oncology programs, XmAb18087 and XmAb20717 in late 2017 and 2018, with more programs to follow.

I will now get into specifics of our clinical and preclinical efforts.

Today, I'll start with XmAb5871, our first-in-class monoclonal antibody in Phase 2 development for IgG4-RD and Systemic Lupus Erythematosus.

XmAb5871 targets CD19 with its variable domain and uses our XmAb immune inhibitor Fc domain to target FcγRIIb, a receptor that inhibits B-cell function. As you know, B cell inhibition is a proven strategy for many autoimmune diseases. Current agents require large doses delivered by infusions and often deplete B cells for months, with the potential for irreversible immune suppression during that time. As the first drug to target FcγRIIb, 5871 offers a differentiated product profile and unique mechanism of action. It is a highly potent, reversible inhibitor of B-cell function.

Now I'll describe our Phase 1 clinical trial to study the pharmacokinetics and bioavailability of subcutaneous administration of 5871. 40 subjects were given 3 administrations of subcu 5871 ranging from 125 mg to 375 mg on an every other week or weekly schedule, and an additional 10 subjects were given 5871 IV as a comparator. First on safety. Subcutaneous 5871 was safe and well-tolerated, with only mild treatment emergent adverse events, or TEAEs, reported for subjects receiving any dose of SC 5871. There were no serious adverse events and no discontinuations due to AEs for SC 5871 subjects. The only drug related treatment emergent adverse event reported in more than 2 subjects was mild injection site bruising in 3 subjects or 8%. This bruising is typically caused by the insertion of the needle. One drug related gastrointestinal TEAE, mild diarrhea, was reported, in contrast to the GI infusion reactions seen in about 20% of patients for IV 5871. At least one anti-drug antibody positive sample was observed for 4 subjects, or 10%, with one subject possibly having had an impact on drug concentration.

On to the PK data. Bioavailability of SC 5871 was typical for a monoclonal antibody; drug exposure was dose proportional and the terminal half-life was similar to that reported for IV 5871. Drug exposure kinetics support an every other week schedule for SC 5871. We're very encouraged by the tolerability and drug exposure of the SC formulation of 5871 in this trial and are looking forward to using it in our future trials.

Now for an update on our ongoing Phase 2 studies of 5871 in IgG4-RD and lupus.

First, with regard to IgG4-RD: as you know, we reported preliminary data from our ongoing Phase 2 study at the ACR Annual Meeting in November, which showed that nine of 11 treated patients, or 82%, achieved an initial response to therapy of at least a three-point reduction in the IgG4-RD responder index within two weeks of their first dose with responses deepening over time. We are on track to report topline data from the trial later this year and also will discuss with the FDA future development plans, including clinical trial design and potential registration requirements.

As part of our ongoing Phase 2 study, we are monitoring a variety of immune cell populations of patients with IgG4-RD both before and during treatment with XmAb5871, in order to better understand its pathology and hopefully create new tools to monitor its progression. At the 3<sup>rd</sup> International Symposium on IgG4-Related Disease and Fibrosis in February, Dr. John Stone and Dr. Xin Kai, two investigators on our study, presented on IgG4-RD biomarker development and on preliminary flow cytometry data characterizing circulating immune cell levels from the initial patients enrolled in our study. As of the data cutoff, results were consistent with previous clinical experience, with a partial reduction in B cells and rapid reductions in circulating plasmablasts observed following treatment, and no significant apoptosis of B cells or CD4+ T cells.

We also continue to progress our Phase 2 study in SLE, another disease with a high unmet need and a strong rationale for B-cell inhibition. As a reminder, this is a randomized, double-blind, placebo-controlled, multiple dose study designed to evaluate XmAb5871's ability to maintain the improvement in SLE disease activity after a short course of intra-muscular steroid therapy and an in the absence of immunosuppressant medication. We have designed this study to assess 5871's effect on disease activity with a shorter time to endpoint and with fewer patients compared to standard SLE trials, and expect to report data in late 2018 or early 2019.

Turning now to XmAb7915, our first-in class monoclonal antibody that targets IgE with its variable domain and uses the same XmAb immune Inhibitor Fc domain as XmAb5871 to target FcγRIIb. This design creates three distinct mechanisms of action for reducing IgE levels: 7195 sequesters free IgE to block IgE signaling; it suppresses B-cell differentiation into IgE-secreting plasma cells; and it enables the rapid clearance of IgE from circulation. This is in contrast to existing, approved therapies for controlling IgE in severe asthma, which have only a single mechanism of action – blocking IgE receptor binding. intravenous administration of XmAb7195 induced a rapid and potent suppression of free IgE below the limit of detection, including from single doses in 75% of high IgE subjects. We believe 7195 therefore has the potential for as a treatment for allergic diseases, even in patients with very high IgE levels. We continue to evaluate a subcutaneously administered formulation of 7195 in a Phase 1b trial, and expect results later this year.

Next, a few comments on our XmAb bispecific oncology pipeline. Xencor's bispecific antibodies are built on a novel Fc domain, which provides a scaffold for two, or more, different antigen binding domains. The

result is a single molecule that can bind to multiple targets simultaneously, while preserving native antibody properties, like long circulating half-life, stability and easy of manufacture.

Our lead bispecific programs are tumor-targeted antibodies that contain both a tumor antigen binding domain to target malignant cells and a cytotoxic T-cell binding domain to activate cytotoxic T cells. We exploit the plug-and-play modularity of XmAb bispecifics to tune the potency of the T-cell killing to potentially improve the tolerability of tumor immunotherapy.

We currently have two bispecific oncology candidates in Phase 1 clinical testing. The first is XmAb14045, in development for the treatment of acute myeloid leukemia and other CD-123 expressing hematologic malignancies. 14045 engages the immune system against AML by binding to CD123, a protein that is highly expressed on AML and leukemic stem cells, and to CD3, a protein on cytotoxic T-cells, thus activating a targeted T-cell immune response against the cancer cells.

The second program is XmAb13676. Like 14045, 13676 binds to CD3 on T cells, but it targets CD20, which is highly-expressed on B-cell tumors, for example in CLL and NHL. We started the Phase 1 trial of 13676 in February.

We hope to announce initial data from our 14045 study later this year, and from our 13676 study in 2018, pending alignment with our partner Novartis on timing.

Next in our oncology bispecific pipeline are XmAb18087 and XmAb20717. XmAb18087 targets SSRT2 and CD3 for the treatment of neuroendocrine tumors. At the American Association for Cancer Research, or AACR, Annual Meeting in April, we presented preclinical data demonstrating 18087's ability to eliminate SSRT2-positive tumor cells by stimulating T-cell cytotoxicity *in vitro* and in mouse models, and to stimulate SSRT2-dependent T cell activation, T cell margination and cytokine release in cynomolgus monkeys. We plan to file an IND later this year.

20717 is our first candidate to simultaneously target two T-cell checkpoint targets – PD-1 and CTLA-4 – and is designed for potential use in multiple oncology indications. As a dual checkpoint bispecific antibody, we believe 20717 has the potential to improve the selectivity of combination checkpoint inhibitor therapy and eliminate the need for multiple checkpoint antibodies. We expect to file an IND in 2018.

We are advancing additional bispecific oncology programs and plan additional INDs in 2018. We presented preclinical data on several of our lead candidates at AACR, including a bispecific antibody targeting PD-1 and an undisclosed co-stimulatory receptor on T cells (PD1 x costim) and an IL15/IL15R $\alpha$  heterodimeric Fc-fusion for T-cell activation.

Just a quick update on our partnerships: in March 2017, CSL Limited, through its licensee Janssen, advanced CSL362, now called talacotuzumab -- to the Phase 3 portion of its ongoing Phase 2/3 study. It uses our XmAb Cytotoxic Fc domain, and is in development for AML. This triggered a milestone payment to Xencor of \$3.5 million dollars. With this advancement, we now have two partnered programs in Phase 3 testing, the other being Alexion with a program using our half-life extension Fc domain, in addition to five in earlier stages of clinical development, across nine pharmaceutical companies and the NIH.

Last, a brief comment on our team – I want to welcome Kevin Gorman to our Board of Directors. Kevin has substantial experience bringing drugs from clinical development through FDA approval and

commercial launch as the CEO of Neurocrine Biosciences, and I am confident that he will bring important insights to Xencor as we continue to mature into a fully-integrated company. I would also like to thank on behalf of the entire Xencor team Bruce Carter and Bob Baltera, who will be departing our Board following our Annual Meeting in June, for their many contributions in helping Xencor since before our IPO.

With that, I'll pass it over to John Kuch to review our financial results.

### **Financials (John):**

Thank you, Basil.

In this afternoon's press release, we reported cash, cash equivalents and marketable securities totaling 392.7 million dollars as of March 31, 2017, compared to 403.5 million dollars as of December 31, 2016. The decrease reflects net spending on operations for the first quarter of 2017.

Total revenue for the first quarter of 2017 was 4.3 million dollars, compared to 7.3 million dollars for the same period in 2016. Revenues are earned from technology licensing fees and milestone payments from Xencor's partners for the license of its drug candidates and use of its proprietary XmAb antibody engineering technologies. Decreased revenue for the first quarter of 2017 over revenue reported for the same period in 2016 is primarily the result of revenue earned from our Amgen collaboration in the first quarter of 2016 compared to milestone revenue received from CSL in the first quarter of 2017.

Research and development expenditures for the first quarter of 2017 were 15.0 million dollars, compared to 10 million dollars for the same period in 2016. The increased R&D spending in the three-months ended March 31, 2017 over amounts reported in the first quarter of 2016 reflects increased spending on our bispecific pipeline of candidates including our first two clinical candidates, XmAb14045 and XmAb13676 and development spending on the next two candidates, XmAb18087 and XmAb20717.

General and administrative expenses in the first quarter of 2017 were 4.8 million dollars, compared to 4.0 million dollars for the same period in 2016. The increased spending on G&A for the first quarter 2017 over the comparable period in 2016 reflects increases in stock based compensation charges in 2017.

Non-cash, share-based compensation expense for the first quarter ended March 31, 2017 was 3.2 million, compared to 2.0 million for the same period in 2016.

The net loss for the first quarter of 2017 was 14.6 million dollars, or 0.31 cents on a fully diluted per share basis, compared to a net loss of 6.4 million dollars, or 16 cents on a fully diluted per share basis, for the same period in 2016. The increased loss for the first quarter 2017 over the loss reported in 2016 is primarily due to lower revenue of \$2.9 million and increased spending of \$5.9 million in the first quarter of 2017 compared to the first quarter of 2016.

The weighted-average shares outstanding used to compute net loss per share was 46,598,797 shares for the quarter ended March 31, 2017, compared to 40,626,729 shares for the quarter ended March 31, 2016. The increase in shares outstanding reflects the additional shares issued in our December 2016 follow-on financing.

Based on our current operating plans, we expect to have cash to fund research and development programs and operations beyond the end of 2020. We expect to end 2017 with approximately \$340 million dollars in cash, cash equivalents, and marketable securities.

With that, we would now like to open the call up for your questions. Operator?

**Question & Answer Session:**

**Closing:**

Thank you, operator.

2017 will continue to be a very busy year, as we advance our XmAb pipeline and announce new clinical data from across our wholly-owned programs. Thank you all for your time today, and I look forward to updating everybody again soon.