**Kymera Therapeutics Second Quarter 2026 Results Call | August 5, 2026**

Moderator:

Good day everyone. My name is Chelle and I will be your conference operator today. At this time, I would like to welcome you to the Kymera Therapeutics Second Quarter 2026 Results Call. All lines have been placed on mute to prevent any background noise. After the speaker's remarks, there will be a question-and-answer session. If you would like to ask a question during this time and if you have joined via the webinar, please use the raise hand icon, which can be found at the bottom of your webinar application. If you have joined by phone, please press star nine on your keypad to raise your hand. At this time, I would like to turn the call over to Justine Koenigsberg, Vice President, Investor Relations.

Justine Koenigsberg:

Good morning, and welcome to Kymera Therapeutics' quarterly update conference call.

Joining me today with prepared remarks are Nello Mainolfi, our Founder, President and CEO and Bruce Jacobs, our Chief Financial Officer. We'll also have brief comments from Jared Gollob and Terence Rooney, our new Chief Medical Officer, who will also join us for Q&A.

Following our prepared remarks, we will open the call for questions from our covering analysts. To ensure we have time to hear from everyone, we will limit each analyst to one question.

Before we begin, I would like to remind you that today's discussion will include forward-looking statements subject to risks and uncertainties described in our most recent Form 10-Q filed with the SEC. Please note that any forward-looking statements speak only as of today's date and we undertake no obligation to update them.

With that, I will now turn the call over to Nello.

Nello Mainolfi:

Thank you, Justine, and good morning, everyone. Before we dive into the quarterly update, as we announced last week, I'd like to take a moment to recognize Jared as he prepares to retire and thank him for his many contributions over the past eight years.

Jared has been instrumental in helping build Kymera and has made a meaningful impact across virtually every aspect of the company. His leadership, dedication, and partnership have helped shape where we are today, and we're incredibly grateful for everything he's done.

While we'll certainly miss him, he leaves the organization in a position of great strength. With Terence stepping into the role, I'm confident that we'll have a seamless transition and continue to build on the strong foundation Jared has helped create. You'll hear from Terence later in the call, but I'm very excited to have him join the team and believe that his long and deep experience across all phases of immunology development and commercialization, along with his insights and capabilities, will be critical to helping advance our next phase of growth.

On behalf of everyone at Kymera, thank you, Jared. We wish you nothing but the very best in this well-deserved next chapter. Before we get started, we wanted to give him the opportunity to share a few words. Jared?

Jared Gollob:

Thank you, Nello.

While it's certainly bittersweet to say goodbye, I have made the decision to retire after having spent 20 years in academic medicine followed by 20 years in industry, including the last 8 years at Kymera. This is obviously an exciting time for Kymera, and I could not be prouder, not only of all that we have accomplished, but also of all that the company will accomplish in the future. I am happy to note that I'll be staying on as an advisor through year-end to help with the transition, as needed. But I am ultimately excited for this next chapter and for the opportunity to spend more time with my family.

But before I go, I want to say a heartfelt thank you to all of you. It has been an incredible privilege to be part of this organization and to work alongside such talented, dedicated, and inspiring people. I have also enjoyed all the interactions I have had with all of the investors and analysts on the call today, which I will miss as well. Looking back, I'm filled with gratitude for everything we've accomplished. I have tremendous confidence in the future of Kymera and can't wait to see all that's still to come. It's been an absolute honor to be part of this journey.

Thank you.

Nello Mainolfi:

Thanks, Jared.

Now, with that, let's turn to our quarterly update. We enter today's call with strong momentum across the business, driven by progress in our lead programs, advancement of our broader pipeline, and continued execution against our long-term strategy.

At Kymera, our ambition is not only just to develop new medicines but to redefine what is possible for patients by changing treatment paradigms.

We believe we have the ability, by leveraging not only targeted protein degradation, but also our broad small molecule capabilities, to fundamentally reshape how diseases are treated. Our focus remains firmly on translating that science into transformative oral medicines that can create meaningful impact for patients.

Reflecting on our recent accomplishments, last quarter's highlight was undoubtedly the announcement that we completed enrollment in our Phase 2b AD trial approximately six months ahead of schedule. As a result, we now expect to report topline data by year-end 2026 and to initiate Phase 3 development around mid-2027, both approximately six months earlier than previously planned.

We view the rapid enrollment as a reflection of many factors, including: the compelling preclinical and Phase 1b AD data generated to date, appreciation and confidence from investigators and KOL's in a well-understood pathway, the incredible excitement from investigators and patients for the opportunity of a once-a-day oral therapy, as we have seen in other areas, outstanding execution by our clinical trial operations and medical teams.

We completed this trial well ahead of expectations as I mentioned earlier. I would add that we did that while maintaining our strict focus on quality, which included many measures that we put in place, some of which could be perceived as burdensome to patients and sites. And this approach remained consistent throughout enrollment.

In addition to the momentum around our STAT6 program, we continue to demonstrate that our platform can repeatedly generate differentiated investigational medicines against high-value targets, reinforcing our strategy of building a robust pipeline capable of driving high patient impact and, in doing so, delivering long-term value.

Our second wholly owned program is also progressing in the clinic with the ongoing Phase 1 study of KT-579, our oral IRF5 degrader. We believe IRF5 represents a highly compelling target in autoimmune diseases with the potential to address multiple high-value indications. We expect to report Phase 1 healthy volunteer results in the fourth quarter of 2026 and to advance the program into its first proof-of-concept study in lupus patients soon thereafter.

In addition, KT-485, our second generation IRAK4 degrader partnered with Sanofi, recently entered Phase 1 development, resulting in a $20 million milestone payment to Kymera. The Phase 1 is designed to evaluate KT-485 in both healthy volunteers and patients with hidradenitis suppurativa, and we look forward to its advancement under Sanofi's leadership.

And, last but not least, we look forward to providing updates as our CDK2 molecular glue, KT-200, partnered with Gilead, advances toward an expected IND and clinical start next year.

Taken together, our progress across all of these programs highlights both the power of our discovery and development capabilities and our ability to consistently translate into potentially transformative medicines for patients and meaningful value creation for shareholders.

Turning more specifically now to KT-621.

As we prepare for the upcoming readout later this year, I wanted to quickly refresh you all on the details of the trial and then touch on how we view the opportunity for KT-621.

As a reminder, the study is evaluating three doses of KT-621 compared to placebo. The primary endpoint is percent change from baseline in EASI score at Week 16. Key secondary endpoints include EASI-50, EASI-75, vIGA 0-1, and Pruritus NRS. When we report topline results later this year, these, along with safety, are among the key endpoints we expect to share.

As we think about the upcoming clinical readouts, our overarching goal is very clear. We are developing KT-621 to deliver on what we hear patients want: an active, safe, oral therapy, in diseases like AD, asthma, EOE, COPD and others that lack such an option.

This is not only true in these Type 2 indications, but also more broadly. There is an overwhelming demand for oral pills that can help manage debilitating chronic diseases. It comes down to clear preference among patients and physicians for treatments that are better suited to fit their lives.

With respect to the market opportunity, we have made this point in the past, but it bears repeating. Market data tell us that there are a significant number of patients that are not well-served with existing treatments. In fact, there are an estimated 43 million adults in the US, EU5, and Japan with atopic dermatitis and only a small percentage of patients, single digits, really, with moderate-to-severe disease are on advanced systemic therapies.

There is no doubt that this represents a significant, if not unprecedented opportunity, for KT-621. If we are successful and can deliver an oral therapy that is both clinically efficacious and well tolerated, we have the potential to meaningfully expand the use of systemic treatment and dramatically change the existing treatment landscape.

Finally, on the opportunity, I think it’s important to highlight that KT-621 has the potential to become not only the first oral therapy with a favorable safety profile for individual Type 2 inflammatory diseases, such as atopic dermatitis and asthma, but also the first and only oral therapy capable of addressing the full spectrum of Type 2 diseases and their associated comorbidities. This would represent a powerful tool for physicians and position KT-621 as a potential first and best option for all patients with Type 2 diseases, given more than 50% of this population presents with additional Type 2 comorbidities.

Now, while most of my comments have focused on the AD trial, we're similarly excited about the progress we're making in asthma. We continue to hear strong enthusiasm for the potential of an effective, oral therapy in Type 2 asthma, based on our discussions with KOLs, including most recently at the ATS conference. Importantly, we hear consistently from KOLs that a well-tolerated oral therapy delivering clinically meaningful benefit could address a significant unmet need and expand treatment options for a broader population of moderate-to-severe patients. Enrollment is underway in the BREADTH Phase 2b trial, and we remain on track to report topline data by late 2027.

As we continue to advance KT-621 in the Phase 2b trial, we're also focused on generating the long-term data that will be important both for regulatory purposes and ultimately for commercialization.

We are pleased to share that we have initiated an open-label extension asthma study, which will allow patients who complete the BREADTH study to continue receiving KT-621 for up to an additional 52 weeks.

Taken together, we believe our development strategy across both AD and asthma positions us, once these two studies are completed, to fully explore the broad potential of KT-621 across other dermatology and respiratory diseases in later stage trials.

Now turning to KT-579, our oral IRF5 degrader, we remain on track to report topline data from the ongoing healthy volunteer study in the fourth quarter of 2026.

As a reminder, IRF5 is a genetically validated driver of innate immune dysfunction and inflammation and is implicated across multiple autoimmune diseases, such as lupus and IBD.

KT-579 seeks to confront a challenge that has been elusive among traditional drug development approaches in this space. By selectively degrading IRF5, KT-579 is designed to modulate multiple disease-driving pathways with a single mechanism and therefore has the potential to be the first novel mechanism with broad utility in diseases where patients are desperately seeking more efficacious and well-tolerated oral therapies.

The Phase 1 healthy volunteer study is designed to evaluate single- and multiple-ascending doses of KT-579, with the objective of achieving more than 90% IRF5 degradation in blood at doses with a favorable safety profile.

We will also assess PD activity using ex vivo stimulation assays to understand the impact of IRF5 degradation on key inflammatory pathway biomarkers upregulated by TLR7, 8 and 9 agonists, including Type 1 interferons, proinflammatory cytokines and inflammatory pathway gene transcripts. We have guided that it is our expectation that we should see between a 50-80% reduction in these biomarkers across the three TLR pathways assessed if we’re engaging IRF5 effectively, which would suggest the potential for IRF5 degradation translating into clinical activity in subsequent patient studies with KT-579.

Looking ahead, we plan to advance KT-579 into a study in lupus patients soon after the healthy volunteer study is complete. Despite recent scientific advances, most lupus patients continue to cycle through therapies without achieving sustained disease control, underscoring the need for differentiated treatment approaches.

Before I wrap up my remarks, I'd like to briefly highlight a few additional leadership updates.

We were pleased to announce at our recent Annual Meeting, Felix Baker has assumed the role of Chairman, succeeding Bruce Booth. Felix has been a member of our board since 2024, and we look

forward to his continued leadership and partnership. We are also grateful for Bruce's many contributions since our founding, and we're pleased that he continues to serve on the board as a director.

We also recently welcomed Penny Carlson to lead our development operations and Liz Laws as Global Program Lead for KT-621. Penny joins Kymera after a long and successful career leading global clinical development, most recently at Takeda. Liz joins us from Sanofi where she was highly involved in the development of dupilumab. Both Penny and Liz bring extensive experience leading complex clinical development programs, with direct relevance to Kymera, and their expertise will be invaluable as we advance our pipeline and continue building the capabilities needed to support a growing, late-stage clinical portfolio.

And, last but not least, as mentioned earlier in the call and disclosed last week, I could not be more excited to introduce Terence as Kymera's new Chief Medical Officer. Terence joins Kymera with what can only be described as the ideal set of experiences, expertise, and knowledge as we continue on this journey to transform the immunology market. He held senior leadership positions at J&J and Lilly, where he helped build and advance leading immunology franchises including ICOTYDE™, STELARA®, and TREMFYA®. His extensive experience across clinical development and portfolio strategy is highly complementary to Kymera and will help guide the continued advancement of our pipeline.

I wanted to allow Terence to share a few thoughts with you, and we will also have him join the Q&A. Terence?

Terence Rooney:

Thank you, Nello.

I'm excited to be joining Kymera at such an important time in the company's evolution. With multiple programs advancing across the portfolio and significant opportunities ahead, it's a privilege to be part of the team as we enter this new chapter.

By way of background, I've spent some 17 years in the biopharma industry focused on the development of treatments for immune-mediated inflammatory diseases. Most recently, I served as the Head of Portfolio and Asset Management for Immunology at Johnson & Johnson, where I led strategy and asset development for a broad immunology portfolio. Prior to that, I've spent time throughout the pharma R&D value chain including stints in early and late-stage clinical development, in business development, and end-to-end R&D leadership.

Before biopharma, I spent 12 years as a physician in academic clinical practice and research, specializing in Rheumatology and Internal Medicine. So, I've had the chance to work across academic medicine, translational research, and global pharmaceutical R&D, all focused on advancing innovative therapies from concept through clinical development, to approval and beyond.

What drew me to Kymera is the opportunity to help unlock the potential of targeted protein degradation to transform the treatment of disease. The science is highly compelling, the pipeline is strong, and I'm joining a great team that I'm convinced is well positioned to deliver meaningful innovation for patients.

To wrap up then, I'll echo what Nello said earlier about KT-621. Kymera's STAT6 program represents a truly transformational opportunity not only for the company, but most importantly, of course, for human health. And I couldn't be more excited to be joining and to help Kymera fully realize the potential of this and our other assets.

Nello Mainolfi:

Thank you, Terence.

In conclusion, with KT-621 advancing through Phase 2b development and KT-579 expected to enter its first patient study shortly, we are sharply focused on building the team and capabilities needed to execute potentially more than 10 Phase 3 studies over the next 2-3 years. We have also begun building our commercial capabilities to deliver on our vision of becoming the global leader in innovative oral immunology medicines.

Importantly, we are doing all this from a position of financial strength. With a robust balance sheet and cash runway extending into 2029, we are well-capitalized to advance our pipeline.

Looking back to the first half of the year, we have executed across our portfolio, from accelerating the development of KT-621 to advancing KT-579, while also progressing our preclinical pipeline and partnered programs.

With multiple catalysts ahead, we remain enthusiastic about the opportunities in front of us and look forward to sharing additional data and updates in the months ahead.

With that, I will turn it over to Bruce to review the financial results before we open the call for questions. Bruce?

Bruce Jacobs:

Thanks, Nello.

As I walk through the second quarter results, please refer to the tables included in the press release, which was issued earlier this morning.

Collaboration revenue for the second quarter of 2026 was $65 million, reflecting a $45 million option exercise fee related to the Company's collaboration with Gilead Sciences and a $20 million milestone payment associated with Sanofi starting the Phase 1 study for KT-485. We have recognized all deferred

revenue, so we do not expect additional revenue this year. Any future revenue would be tied to milestones achieved in either the Sanofi or Gilead collaborations in 2027 and/or beyond.

Turning quickly to operating expenses, R&D expenses for the quarter were $119.5 million, including approximately $10.4 million in non-cash stock-based compensation. Excluding stock-based compensation, adjusted cash R&D expense was $109.1 million, an increase of 22% compared to the first quarter of 2026, primarily reflecting continued investment across our advancing clinical portfolio.

G&A expenses for the quarter were $21.1 million, including approximately $8.6 million in stock-based compensation. Excluding stock-based compensation, adjusted cash G&A expense was $12.5 million, a decrease of 4% compared to the first quarter of 2026.

We ended June with cash, cash equivalents, and investments of approximately $1.5 billion, providing runway into 2029 and positioning us to execute on a number of important value-driving milestones over the coming years.

Our balance sheet supports the completion of the KT-621 Phase 2b trials in AD and asthma and the progression of KT-579 fully through our planned proof-of-concept study in lupus. Within our runway, we also expect to fund the beginning stages of the Asthma Phase 3 study for KT-621, and most of the KT-621 Phase 3 study in AD. At the same time, we will continue investing in our research pipeline and building the capabilities needed to support our transition into a later-stage development and commercial organization.

Overall, we believe we are well positioned financially to execute on our strategic priorities while maintaining a disciplined approach to capital allocation.

With that, we'll pause briefly while we make our way to the conference room and assemble the question queue, at which point we'll open the call for Q&A. Thank you.

Moderator:

Thank you. At this time, if you would like to ask a question, please click on the raise hand button, which can be found on the black bar at the bottom of your screen. If you have joined by phone, please press star nine on your keypad to raise your hand. When it is your turn, you will receive a message on your screen inviting you to join as a panelist. Please accept and wait until you are promoted to panelist. Please unmute your audio, turn on your camera, and ask your question. As a reminder, we are allowing analysts one question today. We will pause now a moment to assemble the queue.

Our first question is from Thomas Smith from Leerink Partners. Please unmute your line.

Thomas Smith:

Hey guys, good morning. Thanks for taking our questions. Congrats on all the progress and let me add my best wishes to Jared in his retirement. Given the really strong enrollment here into BROADEN2, just wanted to come back and ask if there was any color you could provide on the baseline characteristics for

these patients that were enrolled into the study. Anything that you can say relative to some of the other large contemporary Phase 3 biologic studies would be helpful. And then just thinking about the bar for success for BROADEN2, I know you characterized this as clinically efficacious and well tolerated, but just wondering if you could provide a little bit more specifics with respect to the profile relative to dupi and what some of the injectables have shown and what maybe some of your market research is pointing to in terms of a clinically meaningful profile. Thanks so much.

Nello Mainolfi:

Thanks, Tom. I love you had two questions in one. So, let's start with the first one. So, baseline characteristics, obviously one of our main goals of the study, and as I mentioned earlier, and we've done it in the past, was to actually prioritize quality of execution over speed. And in fact, our initial timelines were actually reflecting those expectations. We expected that some of the systems we had put in place would direct the speed of execution towards those kind of expected timelines. And obviously with that, we still saw a pretty fast enrollment, which again, we can comment as I commented earlier, we believe driven by the excitement around the program, the oral opportunity, the data that we generated, etc.

But going back to your first question, yes. The goal has been to ensure that we have the patients with the right severity on the study. As you know, when the first biologic in the space was developed, dupilumab, at this point I think Phase 2 was more than 10 years ago, there wasn't any approved systemic drug at that time for a broader population. So, the severity of the baseline of those patients was probably higher than what we've seen in more recent studies. I think again, all the things that we put in place was to ensure that, while we believe it's difficult, if not impossible, to replicate that type of severity at baseline, that the patients in our study would follow at least what has been seen in the past few years. So, I'm not going to comment on where we landed because I think it's unnecessary, but you'll see when we release the data, but we feel good about where we landed with the baseline characteristics right now.

On your second answer, the bar, I think there are maybe two ways to answer your question, and I'm sure this is a question from many others, so maybe I can be really good at answering once. So, what we've learned, so when we started this program, obviously we were very science-focused, and we were showing for the first time ever that actually you can block STAT6 signaling through degradation of STAT6. And what we saw in our laboratories was basically a downstream blockade that mimicked what we had seen with dupilumab. And so our narrative and science around this program has continued over the years to continue to demonstrate the science behind STAT6 degradation and how effective it is to block IL-4 and IL-13.

So what we obviously learned along the way by interacting with the medical community, including patients, is the really strong need of an effective oral therapy. That's the feedback we've continued to hear. This is the feedback we've heard. I've been in every investigator meeting.  I've been in every medical meeting. I've talked to a lot of KOLs myself, and really the main consistent feedback we've heard is the really, really important need of an effective oral drug. And that's what we want to deliver patients.

So we believe success, both clinically and commercially, is to deliver what patients want, which is an effective, safe, well-tolerated oral option that right now doesn't exist. Not only doesn't exist in AD, but actually does not exist across all these comorbidities. So that's, I think, maybe the north star.

Now, when we go to the science of it, we've continued to see over the past 6+ years that degrading STAT6 seems to be just as effective at blocking, as I mentioned, IL-4 and IL-13, as dupilumab. And so the data has shown, both preclinical and early clinical, that we seem to be able to block the pathway and affect the downstream biomarkers as well as initial clinical endpoints in a similar way as dupilumab. So our, let's say, data expectation will continue to be, in this study, to be in that ballpark of what dupilumab has shown at Week 16 in the previous AD studies, understanding that obviously different studies, different populations.

Thomas Smith:

Super helpful. Thanks, Nello.

Nello Mainolfi:

Thanks, Tom.

Moderator:

Thank you. Our next question is from Tazeen Ahmed from Bank of America.

Tazeen Ahmed:

Hi guys, good morning. Thanks for taking my question. I wanted to ask about ‘579. I think that's an exciting potential next big drug for you guys. Wanted to understand what you're hoping to learn from the healthy volunteer data that you're expected to show in the fourth quarter, especially since you've already picked lupus to move into Phase 1b there.

Nello Mainolfi:

Yeah, so thanks Tazeen. So, we're very excited about IRF5. This is, again, we believe, unless we're mistaken, I don't think so, this is the first drug targeting IRF5 that has gone in the clinic. And so, as we've done in the past, we feel the responsibility to actually demonstrate the power of this biology. So, the underpinning excitement is around the human genetics that tell us that when you have this point mutation or activation of IRF5, you see many of these subjects develop diseases like lupus, RA, IBD, etc. But actually, I would say even more importantly, what we've shown, is where is this biology coming from, right?

And so what we've shown in preclinical species is that actually it comes from the fact that IRF5 is a central node for three key pathways. It's B-cell autoantibodies production, it's inflammatory cytokines like 12, 23, TNF, etc., and it's Type I interferons. And obviously when this pathway is activated, and we believe in some of these diseases it's extremely relevant, we see IRF5 being this master regulator of

immunity. So, in this Phase 1 healthy volunteer study, the necessary but not sufficient step is first to show that we can degrade IRF5 robustly with saying at least 90% or more and safely. And then I guess the next step will be to show that by blocking IRF5, we can modulate these three important key pathways.

And we have, the team has developed, we believe, some really good assays to actually measure all these pathways. And so we expect that, while this is not a patient study, the fact that, even in healthy volunteers we can interrogate the relevance of these three pathways by blocking IRF5 signaling I think should give us that confidence to move into, as you said, lupus, but actually we're planning and discussing internally what other indications we could start as well in the relatively immediate future.

Tazeen Ahmed:

Thank you.

Moderator:

Thank you. Our next question is from Andy Chen from Wolfe Research. Please unmute your line and ask your question.

Jason:

Hi, good morning. Thank you so much for taking my question. This is Jason taking it for Andy, and I just wanted to ask if you guys have any plans around unveiling your degrader molecules maybe in the next year or two, and how many molecules we should expect, and are they likely to continue to be in immunology indications or with derisked mechanisms? Thank you.

Nello Mainolfi:

Yeah, so obviously we have a very productive research engine, and hopefully this is also appreciated externally. We have a general goal/guidance of having at least one new molecule entering clinical development every year. And so we continue to be on track to have development candidates in 2026 that will be entering the clinic in 2027 and beyond. So, for sure, we obviously want to be able to share more programs.

Obviously we have two important clinical readouts in the second half of the year in 4Q. So we're figuring out whether the next clinical program to be disclosed will be either this year, early next. Again, based on everything that we're doing, that decision's still to be made. Likely obviously it'll happen because we're on track with these programs, but we're still deciding exactly what the timing will be. And 80% of our effort preclinically is in immunology, so it's extremely likely, if it's not almost 100% sure, that at least the next program, next couple of programs will be immunology programs with highly validated targets without oral options.

Jason:

Got it. Thank you so much. I appreciate it.

Moderator:

Thank you. Our next question is from David Dai from UBS.

David Dai:

Hi everyone. Thanks for taking my questions. So, regarding the STAT6 degrader, KT-621, given the high placebo response we're seeing in some of the AD studies, what assumptions were used around placebo response-and-effect size when powering for the BROADEN2 Phase 2b study? And have the recent enrollment dynamics changed your confidence around those assumptions?

Nello Mainolfi:

Yeah, I mean, thanks, David. That's a great question. We're not going to go into the details of your question, although it's a very good one. So, we understand the STAT6 biology well. We powered the study within a range of expectations of what this biology can deliver clinically also with the Phase 1b data in hand. We obviously know that the placebo rates have increased, and we've accounted for that. Obviously there has been a couple of cases where, or at least one, where we had some crazy high placebo rates in the 70s. Obviously no study can plan around such a high placebo rate. But otherwise, if you look at the recent studies that delivered, let's say, positive data, and we've seen many in the past few years actually, and we've seen increased placebo rates, those have been accounted for in the power of our study. The enrollment has not really changed, the speed of enrollment has not changed our assumptions. When you have a high-enrolling study, you often end up enrolling slightly more than you plan just as the mechanics of the study will force you to do. But we haven't done anything with regards to changing our power assumptions.

David Dai:

Thank you so much.

Moderator:

Thank you. Our next question is from Ellie Merle from Barclays. Please go ahead.

Jasmine Fels

Hi, Jasmine on for Ellie. Thank you for taking our question and congratulations on all the progress. Can you give some more color on how enrollment is going in the Phase 2b asthma trial? So, with the acceleration of the enrollment in AD, how should we think about whether we can see this in asthma as well? And what are, I guess, some of the different dynamics playing into the AD enrollment versus asthma that could contribute to why the timelines are shaping up to be different? Thanks.

Nello Mainolfi:

Thanks for the question. So, I would say what is common between these two studies is the excitement around this mechanism. I think, as I said earlier, one thing that might be appreciated or not is the fact that this pathway is really well understood by investigators all around the world. This is probably the most drugged pathway in immunology. We have so many agents targeting IL-4, IL-13, both of them, etc. So I think that actually is a common theme of we understand the pathway.

We, the team, I should say, have done, I think, an amazing job educating the public about our mechanism and STAT6. So there is a level of confidence about where STAT6 fits in that pathway. And then you combine, let's say, this sense of, let's call it comfort with our program, and you combine it with, as I mentioned earlier, this really strong need of an oral option. I think what is common when we talk to both dermatology investigators and respiratory and allergist investigators, I think what's common is these two aspects.

Now, the two studies are quite different. For the AD study, obviously there are more AD patients, let's start there. And also we are enrolling all moderate-to-severe patients. For the asthma study, if you recall, we have some specific criteria with regards to feno- and eosinophils at baseline, which is looking at a subset of asthma patients. I like to call them, we should all call them, Type 2 patients. Some people call them eosinophilic asthma patients. Anyway, that's maybe for another day. And so that is a subset of the asthma population. So naturally, the denominator is smaller in this case. And because we have this, again, very specific entry criteria, generally we would see higher screen failure rates.

So, while I think we have also in asthma an opportunity to move the program enrollment at a good, fast speed, I don't think we can match what we've seen for atopic derm. But anyway, we are enrolling well, there's a lot of excitement, we're even in more regions than we were for the AD study. And so, we're confident that we'll be able to deliver a fast, but obviously high quality, execution. And within the timeline that we said.

In terms of changing timelines, as we've done for AD, we're only going to do it if and when we complete, or I should say when, we complete enrollment, but not before because these are highly variable parameters and it's really difficult, a priori, to start changing expectation timelines while you're in flight for these studies.

Jasmine Fels:

Yeah, helpful, thank you.

Moderator:

Thank you. Our next question is from Brian Cheng from JP Morgan. Please go ahead.

Brian Cheng:

Hey guys, thanks for taking our question this morning. Just want to touch on the BROADEN2 OLE portion. Can you give us some color on the latest there? How is the rollover rate looking? And are patients allowed to dose-adjust across the three doses into OLE? Thank you.

Nello Mainolfi:

Yeah, thanks, Brian, great question. So, we're not going to comment on that. We might offer, when we release the BROADEN2 data by the end of the year here in '26, we might maybe offer a bit of an insight on that. But right now, we feel it's too early to comment on the percentage of patients rolling over into the OLE.

On the dose adjustment, I think we've said this publicly, patients are all going on to one dose. And I think I'll leave it at that for now.

Brian Cheng:

Great, thank you.

Moderator:

Thank you. Our next question is from Faisal Khurshid from Jefferies. Please unmute your line and go ahead.

Faisal Khurshid:

Hey guys, thank you so much for taking the question and congrats to Terence on joining a great team. Just wanted to ask your latest and greatest thoughts on the competitive landscape. And I'm wondering both within the STAT6 class, I guess, we had Pfizer confirming that their STAT6 is active in Phase 2, and Nurix and Sanofi dosing their program. And then beyond that with bi- and tri-specifics as well, we also heard some interesting comments on that yesterday as well. So yeah, would just love to hear your latest and greatest thoughts on that landscape.

Nello Mainolfi:

Yeah, so I can share a few thoughts. So, on the STAT6, I mean, as you know, this is a highly interesting target pursued by, I think, probably at this point, the majority of companies that are in immunology for all the reasons that we're all here today. So, our focus is obviously on execution and making sure that we execute with high quality and speed in order to maintain the advantage that we've created in terms of timelines.

We know obviously there is Pfizer in Phase 2. We don't know much about their molecule. Obviously there's been flow of information from their study about starting and then pausing enrollment and then restarting. There are other parallel studies ongoing to study the molecule, the formulations and DDI studies. So clearly obviously Pfizer, from what I can see from where I sit, is trying to understand more about this molecule, the performance and the behavior in humans. I think they're reporting to complete the study in early '28, which seems like a very long time. So again, I don't know what that means. I can only go by what's publicly disclosed.

As you know, we don't believe small molecular inhibitors will be competitive with degraders, driven by our ability, with the degrader, to block the pathway completely at very low doses and low concentration over 24 hours, which we believe is needed to match this pathway blockade that you see with upstream agents.

Then yes, we've seen the Nurix-Sanofi program starting. Again, I think we'll be in Phase 3, if everything goes well, by the time some of these programs might have some Phase 1 data. So, we feel good about '621. So far all I can say, it's an amazingly well-behaved molecule, well tolerated. And we're focused really on ourselves, but aware of the overall landscape. With regards to other mechanisms, we're really excited by our oral options. We're also excited about learning about new biology. I think the bi-specifics and tri-specifics are an opportunity for everybody to learn where some of these mechanisms can deliver enhanced activity in a still relatively heterogeneous population, which especially AD can be.

Obviously there was some early data from a small company recently with a bi-specific. Obviously Pfizer is going ahead with its tri-specific, doing a head-to-head study with dupi, which makes sense. I mean, our view is, again, as I said earlier, KT-621's position is, from our perspective, a first-in-line drug for the millions of patients that have moderate-to-severe atopic derm that are not doing well with topical steroids, which is probably the majority of patients given how difficult it is actually to be responding well to topical steroids. It's not even how they work, it's just how complicated it is for patients to use them.

So, if we think about '621 as the first-in-line drug, then I think many of these bi and tri will probably be later lines once you don't respond to this, I think, single-mechanism drug that addresses most patients in AD and other indications.

So, we said, also, in the past we're interested by novel mechanisms, we're thinking about combinations of STAT6 with other mechanisms. So, we're obviously very curiously also observing these new mechanisms out there.

Faisal Khurshid:

Awesome. Thank you so much.

Moderator:

Thank you. Our next question is from Brad Canino from Guggenheim. Please go ahead.

Brad Canino:

Hey, good morning and thank you. And thanks to Jared for the collaboration over the past five years; I've definitely learned a lot from our conversations. My question is about moving STAT6 to the late-stage development phase. How are you positioning the company to extend the benefit to pediatric AD patients? I know for dupi, it took several years to expand the label from adults to different cohorts of pediatrics. Are you doing anything today with a goal to try to accelerate that? Thank you.

Nello Mainolfi:

Yeah, Brad, thank you. So yes, we're heavily focused on how to accelerate pediatrics development. I mean, the one thing that we've done, as you know, is incorporating adolescents in our study. We have 12 and up, which actually are pediatric patients.

Now, going younger in age is obviously something we're not in full control of. This is a conversation with regulatory agencies. All I can tell you is that we're heavily focused on it and we will update you all when we know more. For sure, we will know more after this Phase 2b data. You shouldn't expect any update between now and then.

Brad Canino:

Thank you.

Moderator:

Thank you. Our next question is from Judah Frommer from Morgan Stanley. Please go ahead.

Judah Frommer:

Yeah, guys, thanks for taking my question. Congrats to Terence and Jared as well on their moves. Just curious if you could help us with cash runway guidance remaining into 2029, given the acceleration in timelines for ‘621. Any changes to spend or trajectory over the next couple of years as you think about that?

Bruce Jacobs:

Yeah, no, thanks for the question, Judah. I think it's still intact. We had a runway into 2029, even with the acceleration of timing it's not enough to pull the runway in closer. So, we're still into 2029 with that. So you can assume there had been a bit of a cushion there as well.

I think clearly as we get to the end of the year and see the data and, I guess, solidify all of our development plans, not only for the core indication, but some of the others that we're contemplating, we'll talk about whether there's an update warranted. But we're in good shape, over 10 plus quarters of cash even with the accelerated runway. So, we should be able to, as we said in the past, our runway will incorporate obviously all the Phase 2s running today, the full execution of the IRF5 program, and then as well getting underway with both the Phase 3s. And we should come pretty close to finishing the AD study within our runway if all goes well.

Judah Frommer:

Great, thanks.

Moderator:

Thank you. Our next question is from Geoff Meacham from Citi. Please go ahead.

Geoff Meacham:

Hey guys, thanks for the question. I just want to say congrats to Jared on the retirement and Terence, welcome to the team. So Nello, on ‘621, as you guys look to Phase 3 design in AD and asthma, I guess, how important is the washout or prior therapy experience? I know there are multiple pathways that drive Type 2 inflammation beyond STAT6, but are some tilted pathways more heavily treated patients or maybe patients that have gone for a longer duration or maybe those that have worse baseline? Just trying to think of the entry criteria and the probability of success as you look to a Phase 3. Thanks.

Nello Mainolfi:

Yeah, no, Geoff, that's a great question. It's actually a topic of discussion. I just want to separate the two. So obviously the washout is critical, no matter what the entry criteria are, how you parse out whether you're going to allow every previous biologic patient or not. Obviously based on the half-life of the drug, you need to completely washout patients to actually retain the integrity of the data of the study.

Then maybe the other question in your question was, do we allow a biologics-experienced patient in the study? And as you know, we've done that in our Phase 2b with a caveat of not allowing the previous non-responders to this drug. But for Phase 3, I think it's a topic that we're still discussing. Obviously we need regulatory interactions as well. But we expect that in some shape, biologics-experienced patients would be part of our Phase 3 studies. There is just some nuance about what their experience was on biologics that we might have some kind of criteria around. But that's still something that we're discussing both internally, and we'll have to align with regulatory agencies.

Geoff Meacham:

Thank you.

Moderator:

Thank you. Our next question is from Derek Archila from Wells Fargo. Please go ahead.

Derek Archila:

Hey, good morning. Thanks for taking the questions. And Jared, good luck in the next chapter. Terence, welcome. So just actually following up on a prior question on the pediatric development. Are there any gating factors to a sprinkle formulation of ‘621? And I guess, how would you frame the pediatric opportunity relative to adolescents and adults? Thanks.

Nello Mainolfi:

Well, we actually had a five-year-old child at Kymera a few weeks ago with his parents talking about the life of a child on an injectable biologic. I won't say which one. It's not that difficult to guess. And I think it

crystallized for us what it is that these kids have to go through. And what is not only the opportunity, but I would say the responsibility to deliver for children an easy-to-take, effective, and hopefully obviously well-tolerated drug. So, we are doing, as I mentioned, all we can.

From a formulation perspective, we actually are in a very good place already. Again, I don't want to talk about specifics, about what is it that we need to go into younger patients. And then it's really about when we will be allowed to run those studies. And as I mentioned earlier, obviously having data from a global placebo-controlled, randomized Phase 2 study, especially with regards to both efficacy and safety, we feel is the important data set to initiate those discussions with regulatory agencies.

But all I can tell you is from Kymera's perspective, we will be ready to go as soon as we have completed this study.

Derek Archila:

Great. Thanks, Nello.

Nello Mainolfi:

Thank you.

Moderator:

Thank you. Our next question is from Alex Thompson from Stifel. Please go ahead.

Alex Thompson:

Hey, great. Congrats on all the progress. Appreciate you taking the question. Maybe another one, Nello, I'm not sure you will answer, but I just wanted to ask about, you've had a lot more experience now dosing patients to 16 weeks plus across AD and asthma. Can you comment at all about blinded safety and your confidence in the continued clean profile of '621 heading into the data later this year? Thanks.

Nello Mainolfi:

Yeah, no, thanks, Alex. It's a great question. I think you get it right. It's hard for us to comment on an ongoing study, blinded safety. So, it's only actually a few months away where we can actually all look at the unblinded data and then share it with you. So just a little bit of patience. I feel the same way. I like to know everything, but we have to wait.

Alex Thompson:

Awesome. Thank you.

Nello Mainolfi:

Thanks, Alex.

Moderator:

Thank you. Our next question is from Jeff Jones from Oppenheimer. Please go ahead.

Jeff Jones:

Morning guys, and congrats, Jared, and welcome, Terence. Question on the IRF5 program with KT-579. As you look at those results coming late this year, is there anything that'll guide you towards or away from particular indications beyond lupus?

Nello Mainolfi:

Yeah, great question. And so we have this translational hypothesis around KT-579 around being this master regulator of three pathways when IRF5 is activated. So B-cell, Type I interferons and some of these inflammatory cytokines. I think if we don't see a profile that reflects our understanding of this biology, obviously we will have to rethink what is our clinical strategy besides the translational one. I would be surprised if we are surprised by the data, but we'll see. I think we'll have to see what the biomarker data looks like.

Jeff Jones:

All right, thank you.

Nello Mainolfi:

Thanks, Jeff.

Moderator:

Thank you. Our next question is from Biren Amin from Piper Sandler.

Biren Amin:

Yeah. Hi guys. Thanks for taking my questions. It's interesting in your slide deck, you mentioned that the BROADEN2 trial data could support trials in GI indications. So Nello, I wanted to understand that. What data from BROADEN2 could support development in GI? And also, is the interest in GI due to STAT6 activation in ulcerative colitis? And since I'm talking about GI, would you potentially look at IRF5 in IBD given the expression of IRF5 in IBD? And I think there's some proof-of-concept data with anti-TNF reducing IRF5 in IBD. Thanks.

Nello Mainolfi:

All right. Biren wins, he asked three questions. So quickly, so you caught an important point actually that requires nuance on our slide. So first of all, when we talk about the GI, we talk about EoE, eosinophilic

esophagitis, which actually should not even be called eosinophilic, but that's another point. So, for that, the question is how's the Phase 2 study going to help us? So, we believe the Phase 3 dose that we will get from the AD study should be applicable in all the other derm indications. Could it be directly applicable to EoE? Maybe or maybe not. And actually for EoE, we might have a slightly different plan, which we will be disclosing at some point after the Phase 2b data next year. So, it will definitely be informed, but the development in EoE might not be the same as what we might do for CSU or other derm indications. For IRF5, we're definitely interested in IBD. We have shown some really exciting preclinical data, so stay tuned as we share more information on that.

Biren Amin:

Great, thank you.

Nello Mainolfi:

Thank you.

Moderator:

Thank you. Our next question is from Brian Abrahams from RBC Capital Markets. Please go ahead.

Kevin:

Hi team, this is Kevin on for Brian. Thank you for taking our questions. So maybe another one on ‘579. Just as you think more about lupus, the heterogeneity of the disease, and just maybe some of the challenges prior agents have had and novel ones which could be approved in the next few years, what are your latest thoughts on what the addressable opportunity could be in lupus and would the ambition here also be to have biologic-like efficacy or is this paradigm a little bit more complicated or more nuanced than what we typically think about with ‘621 and atopic derm? Thank you.

Nello Mainolfi:

So maybe I'll start, and given that Terence is a trained rheumatologist, maybe you can help me on this one. So, the quick answer from me, who I'm not a trained rheumatologist, is that what we're seeing in lupus is what the patients need, which is effective therapies that are acutely needed in this population. So, I think we need different mechanisms. We need different options. The reality is there are no good, right now, oral options approved. There are some interesting ones in clinical development. So, I think we believe that we can, again, continue to serve patients that want effective oral options. Terence, anything you want to add on this space in general?

Terence Rooney:

Yeah, thanks, Nello, and thank you, Brian. I would just underscore that there remains tremendous unmet need in lupus, including for oral medicines with a favorable benefit-risk profile. Brian, you flagged

the challenges of lupus drug development. That's absolutely acknowledged. Key to that is going to be patient selection, site selection, careful trial design, and careful oversight. So, you can imagine that's exactly what we're working through at the moment.

Nello Mainolfi:

Cool. Thanks Terence.

Moderator:

Thank you. Our next question is from Marc Frahm from TD Cowen. Please go ahead.

Marc Frahm:

And thanks for taking my questions and congrats, Jared, on a great career at Kymera, and best wishes for the next step. This is mostly for Terence. The company's talked in broad strokes about being interested in combinations across the pipeline, but STAT6 and IRF5. But obviously, Terence, at your prior shop, that was a big push early on. How do you view the optimal time to start those? What do you need to see from some of these earlier trials to really start working on combinations?

Terence Rooney:

Thanks, Marc, and thanks for the welcome. I think you're right. There's tremendous opportunity combining established mechanisms in particular in pursuit of greater efficacy. Nello had some remarks earlier on about the field there, and we're learning as we go. In some instances, we have combinations that have faltered, and in recent instances we've seen some encouragement. I think the learning therefore for an oral drug developer is which combinations in future might be rational for somebody like ourselves who's developing an asset against one proven target. I'll probably leave it there in terms of when to decide to combine STAT6 with another oral asset, but safe to say that it's a topic we're looking at carefully.

Marc Frahm:

Does it need to be oral to be interesting, or would you be interested in novel combinations that maybe aren't all oral?

Nello Mainolfi:

Maybe I can help there. I mean, I think it depends on what we're trying to do. I think there is a biological understanding of synergies or additivity, and then there is market positioning. We continue to believe that advancing oral options for patients is where the future needs to go towards. Would there be a case where we try to understand the mechanism with an injectable biologic? That might happen, but I'm not sure that's our north star right now. All right. Thanks, Marc.

Moderator:

Next question is from Jeet Mukherjee from BTIG. Please go ahead.

Jeet Mukherjee:

Great. Thanks for taking the question. Just for the BROADEN2 data that's coming up by year-end, could you comment if there's a cap on the number of patients who are on prior biologics, and will you ultimately break out the data by patients who are on prior biologics versus those who are not? Thanks.

Nello Mainolfi:

No cap on prior biologics. With regards to how we break out the data, we'll have to analyze the data, so let's wait on that.

Jeet Mukherjee:

Okay, great. Thank you.

Nello Mainolfi:

Thanks.

Moderator:

Thank you. Our next question is from Mayank Mamtani from B. Riley Securities. Please go ahead.

Mayank Mamtani:

Yes, good morning team. Thanks for taking our questions and best wishes to Jared, will be missed, and look forward to getting to know Terence. So, on the BROADEN2 program, if you could comment on what proportion of patients come from maybe ex-US or specifically Eastern Europe sites. And sorry if I missed the screen failure rate. I don't know if you've commented on that, Nello, and how that's maybe similar or different than recent biologic trials, and if your expectation as an oral pill would be for discontinuation rates to be actually better than the biologics, if you could comment on that. And then just on timing, just clarifying, last patient in was before the end of 2Q. So, can the four-month efficacy data drop maybe ahead of the IRF5 program or they're both tracking in parallel? How do you plan to have that disclosure?

Nello Mainolfi:

Yeah, maybe I'll start with the last one. I think it's extremely likely that we'll have IRF5 data first and then we'll have the BROADEN2 data. With regards to many of the other questions, all I can say our sites, as you know, you can look at it now on ClinicalTrials.gov, there's a global presence. There is US, Canada, Australia, Japan, South Korea, Europe. So, the majority of patients will obviously come from outside of

the US, but we have a big, obviously as expected, US presence. I'm not going to be able to go into a breakdown of patients at this point. We're not going to comment on screen failure rates. We might, when we release the data, if helpful to anybody, maybe our competitors. And then we'll talk about some of the other points you made about discontinuations, etc, when we release the data.

Mayank Mamtani:

Could you comment, sorry for one follow-up, if I may. Could you comment on when you plan to have an end-of-Phase 2 discussion focused on AD, and would that need to wait until the asthma data? Thank you.

Nello Mainolfi:

Oh, no, we'll have an end of Phase 2 meeting with the FDA, obviously as soon as we can after we have Phase 2b data in AD. So yeah, no, we will not wait for the asthma data.

Moderator:

Thank you.

Nello Mainolfi:

Thank you.

Moderator:

Our next question is from David Hoang from Deutsche Bank. Please unmute your line and go ahead.

David Hoang:

Hi there. Good morning. Thanks so much for taking my question. So, I just want to ask, to what extent could we look at the ongoing launch of the oral IL-23, which is ICOTYDE™ in psoriasis as an analog for how commercialization of KT-621 in atopic derm might go? So, what lessons can we learn there and what reasons would that be or not be a good comp for KT-621? Thanks so much.

Nello Mainolfi:

Yeah, obviously we have Terence here that was obviously involved in that program, so he probably will have a lot to say that he can't even say. But what I will say is that I think what is in common is the fact that from where I sit, the IL-23 peptide is a validated mechanism with a novel way of drugging that particular target or pathway with a molecule that I believe has actually behaved quite well in terms of efficacy and safety, and is showing that even in a saturated market, I guess the difference is the market, right. Psoriasis, I think from my point of view, a much more mature market with several drugs that have been approved over the years, several biologics, several orals. And even with all of that, you're seeing some really impressive adoption of that drug. I think in the first quarter, there were more than 100,000

[11,000] patients on the drug, if I'm not wrong. So, with KT-621, hopefully, I think that what we can match is the ability to drive efficacy and safety of an oral drug in a validated pathway. And I think what's different is that AD is a very early, immature market with very low penetration overall. And I think our expectation is that we should be able to launch the drug and have even a superior success than what ICOTYDE™ is seeing if we do a good job and if we obviously retain the profile that we want, because there are so many patients that don't have any oral, safe options. While for psoriasis, I think there are oral effective and safe drugs, maybe ICOTYDE™ is slightly better, but still there are options there while in AD there are no options.

David Hoang:

Thank you.

Moderator:

Thank you. There are no more questions at this time. I'd now like to turn the call over to Nello for closing remarks.

Nello Mainolfi:

Well, I want to thank everybody for attending our call. All the analysts for attending and asking questions even though we're in the middle of the summer, so we appreciate you taking time away from your vacation if you are. And we continue to be super excited about where we're going. Please stay tuned. We are going to have some of the most interesting data sets probably for the industry in the second half of the year, given these are both first-in-class programs in highly super relevant indications or populations. So stay tuned and I look forward to seeing many of you at our conference in September and then, after that, at our calls to disclose the data. Thank you.