Kymera Therapeutics Third Quarter 2026 Results Call | October 29, 2026

Operator:

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 Third 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's quarterly update call which will also include a discussion of the KT-579 Phase 1 healthy volunteer results.

Joining me this morning with prepared remarks are Nello Mainolfi, our founder, president and CEO, Terence Rooney, our chief medical officer and Bruce Jacobs, our chief financial officer. Following our prepared remarks, we will open the call for questions from our covering analysts. In order to have as much time for as many questions as possible, please limit your questions to one.

If you would like to ask a question, please use the raise hand icon, which can be found at the bottom of your meeting window. A replay of today's call, including a copy of the corresponding presentation, will be available in the investor section of our website soon after the call concludes.

As a reminder, today's presentation will include forward-looking statements about our future expectations, plans, and prospects. These statements are subject to risks and uncertainties that may cause actual results to differ materially from those projected. A description of these risks can be found in our most recent 10-Q filed with the SEC. Any forward-looking statements speak only as of today's date, and we assume no obligation to update any forward-looking statements made on today's call.

Before we begin, we have one housekeeping item. Starting next week, we will enter our previously planned quiet period ahead of our upcoming STAT6 data. As a result, we don't expect to provide additional commentary on our STAT6 program until we report those results. With that, I'll now turn the discussion over to Nello.

Nello Mainolfi:

Thank you, Justine, and good morning, everyone. Thank you for joining us today. This is a very exciting time for Kymera.

We're seeing our scientific vision take shape across our pipeline of oral medicines to address serious inflammatory diseases with KT-621, our STAT6 degrader for atopic dermatitis, asthma and more, and KT-579, our IRF5 degrader for lupus and potentially other autoimmune diseases. Together, they reflect our goal of developing medicines grounded in strong biology, with the potential to reach many more patients than served by available treatments today.

I'll start today with an update on KT-621 as we approach its Phase 2b AD readout before year-end. I'll then briefly cover the rest of our pipeline before we turn to KT-579 Phase 1 results.

Our STAT6 program continues to advance according to plans across both AD and asthma.

As shown here, we've advanced KT-621 through a series of studies, building on what we learned at each stage to guide the next steps. In Phase 1, we evaluated KT-621 in more than 200 healthy volunteers and observing deep STAT6 degradation, effects on downstream biomarkers and a favorable safety and tolerability profile. We then treated 22 patients with AD in Phase 1b, where we saw the first evidence of strong clinical activity in patients. With each study, we've built a stronger body of evidence and further de-risked KT-621, supporting its advancement into the ongoing Phase 2b studies.

In the BROADEN2 AD study, we are excited to report that we have completed the 16-week placebo-controlled portion of the study with the last patient just recently completing their final visit. We expect that the remaining activities to close out this portion of the study will enable us, as planned, to share the KT-621 Phase 2b data by year-end. At the same time, we are well into our preparations for the first Phase 3 study in AD, which we plan to initiate, assuming both supportive data and alignment with FDA, by the middle of 2027.

Before we move to an update on asthma, as we have shared previously, patients that complete the full 16 weeks of treatment in BROADEN2 are eligible to enter a 52-week OLE. Today, we are happy to report that 98% of eligible patients have enrolled into the OLE.

Now, turning to asthma, enrollment in the BREADTH study continues to progress as planned, with topline results expected in late 2027.

In total, these studies have already enrolled hundreds of participants, providing a robust dataset to assess safety and efficacy of this exciting novel mechanism. And, as a reminder, the BREADTH study also includes an open label extension, which together with the AD OLE, are designed to further support our long-term safety database for regulatory requirements and the potential registration of KT-621.

As we approach the BROADEN2 readout, I wanted to take a moment to put the opportunity for KT-621 in AD into perspective.

As illustrated on slide 7, published epidemiological reports show that there are approximately 6 to 9 million people in the US, 12 years and up, with moderate-to-severe atopic dermatitis. Despite this high prevalence, only an estimated 400,000 receive advanced systemic therapies, a penetration of only about 5% of the total moderate-to-severe patient population.

As we have been advancing KT-621, we have also been conducting market research to better understand the market dynamics of advanced systemic therapies. What we hear from both patients and prescribers is that the main reason for the limited adoption is that many patients don't perceive that their disease state justifies an injectable biologic or a medicine carrying a boxed warning. That perception creates a significant barrier to the use of currently available advanced therapies.

Our market research tells us that patients and physicians want a safe, effective oral treatment. In fact, when we ask what level of efficacy that treatment needs to deliver, we consistently hear that it does not necessarily have to match biologics. And we have seen that opportunity play out in other markets. Otezla launched more than a decade ago with roughly half the efficacy of biologics and went on to become a multi-billion-dollar franchise. More recently, Icotyde, despite efficacy that, by cross-trial comparisons, does not quite match IL-23 biologics, is experiencing one of the strongest launches in psoriasis, bringing new patients to treatment in an already more saturated market.

Going back to KT-621, a safe effective oral is what patients are looking for, and that is the profile we aim to deliver. Our focus is to serve the largest segment of the moderate to severe AD population: those patients who are either untreated or in many cases poorly treated with non-systemic therapies. If we're successful, we believe that KT-621 would have the potential to dramatically expand the number of patients accessing advanced therapies.

All that said and based on the preclinical and Phase 1 data we've generated so far, our scientific expectation remains that BROADEN2 efficacy should land in the ballpark of what dupilumab achieved at Week 16, as compared to what was reported in the in the SOLO 1, SOLO 2 trials, while recognizing the limitations of comparing results across studies.

Beyond KT-621, we are also tracking with expectations and timelines across our broader pipeline. Our partnered programs are both progressing as planned. Sanofi continues to advance KT-485, our second-generation IRAK4 degrader, in a Phase 1 healthy volunteer study that is also expected to enroll HS patients. And Gilead is advancing KT-200, our CDK2 molecular glue, into IND-enabling activities, with an IND planned for 2027.

With respect to our research pipeline, our undisclosed programs are tracking with our expectations and to support our goal to advance one program toward development candidate annually. Regarding the disclosure of our next program, given all we have to share in the fourth quarter with today's KT-579 data and the upcoming KT-621 Phase 2b readout, we've decided to hold off on that introduction until next year.

With all that said, before I turn the call over to Terence to share the IRF5 healthy volunteer data, I wanted to talk a little bit about the importance of today's IRF5 data release.

We have now successfully advanced multiple degrader programs from target selection and drug discovery through preclinical and into the clinic. This is in fact our 7th first-in-class degrader to enter the clinic and today we are sharing that we continue to demonstrate impeccable human translation. While each target and molecule is unique and must ultimately prove itself independently, this repeated

execution is indicative of the positive and compounding effects of all of our learnings and experience across our programs.

IRF5 itself is a highly compelling and genetically validated target that has historically been difficult to drug with conventional drug discovery approaches, and this study is an important first step in establishing our IRF5 thesis in humans. As you'll hear shortly, KT-579, which was well tolerated in this study, robustly degraded IRF5. Importantly, the potent IRF5 degradation led to meaningful reductions in multiple disease relevant biomarkers.

With this data in hand, we now plan to move the program, with confidence, into a Phase 1b lupus trial. The study is designed to establish whether KT-579 can demonstrate the biological and clinical activity we would expect based on this mechanism.

Overall, the KT-579 data represent another important example of our ability to target a highly compelling but historically undruggable target, in this case a transcription factor. It also demonstrates our ability to translate our preclinical work into humans in a predictable and compelling way. We've now done this across multiple programs, and with each success, we're building on a strong and increasingly repeatable foundation for selecting the right targets, developing differentiated degraders against them, and efficiently translating those programs from discovery into the clinic.

I know everyone is eager to get into the data, so with that, I'll turn it over to Terence to walk you through the results. Terence?

Terence Rooney:

Thanks, Nello. It's a pleasure to join you all today to review the positive Phase 1 results for KT-579.

As a rheumatologist by training, I'm particularly excited by what we've demonstrated with this program.

The translation from the strong preclinical profile into humans is an important milestone and validates our confidence both in the biology and the potential of IRF5 degradation as we prepare to move KT-579 into patients.

I'll start with an overview of the Phase 1 study design and the demographics of the healthy volunteers enrolled in the study. I'll then walk through the PD data, beginning with the depth and durability of IRF5 degradation in blood. Next, I will cover the functional consequences of that degradation across three distinct IRF5-dependent immune pathways assessed using disease-relevant *ex vivo* stimulation assays. And finally, I'll review the safety profile observed and then discuss how the totality of these data inform our plans to advance KT-579 into lupus patients. As we have been saying for some time, primarily for competitive reasons, in some areas of the presentation we're sharing only limited data.

As shown on Slide 12, the Phase 1 study was designed to characterize the safety, PK and PD of KT-579 following both single and multiple ascending doses in healthy volunteers. We evaluated a range of doses up to 600 mg in the MAD cohorts. In the SAD portion of the study, 8 subjects per cohort were randomized 6:2 to receive a single oral dose of KT-579 or placebo. In the MAD portion, 12 subjects per cohort were randomized 9:3 to receive once-daily oral KT-579 or placebo for 14 days.

While the primary objective of the study was to evaluate the safety and tolerability of KT-579, the study was also designed to establish proof of mechanism in humans. In both SAD and MAD, we evaluated IRF5 degradation across dose levels, and in the MAD portion, we also assessed the downstream functional consequences of IRF5 degradation using disease-relevant *ex vivo* stimulation assays measuring inflammatory biomarkers.

Together, these endpoints allow us to address the key questions we set out to answer in this study. First, can we safely achieve deep and sustained degradation of IRF5 with KT-579? Second, does that degradation translate into the expected functional impact on IRF5-dependent biology? And as you'll hear today, the answer to both questions is yes.

Turning to Slide 13, the demographics of the volunteers enrolled in the SAD and MAD portions of the study. Overall, there were no meaningful imbalances in baseline characteristics that would impact our interpretation of the study results.

Now, before turning to the MAD degradation data, although not shown in this presentation, we did want to mention that KT-579 exhibited the predicted PK profile with dose responsive exposures in SAD and MAD cohorts. Additionally, while we generated degradation data across both the SAD and MAD portions of the study, we are only sharing the MAD degradation data here, as repeated daily dosing most closely reflects the intended clinical use of KT-579.

Now looking at Slide 14, which illustrates degradation across MAD dose cohorts, with data shown at Day 7 and 14. The top five dose cohorts all reached median IRF5 degradation greater than 90%, with saturation of degradation by dose level 3 with blood degradation achieving 94%.

Importantly, while not shown, we also observed comparable reductions in IRF5 levels in skin, demonstrating target degradation in tissue as well as blood.

Overall, KT-579 demonstrated robust degradation of IRF5 at or above levels predicted by our preclinical work in both the SAD and MAD. This profile shows that the target remains continuously suppressed with once-daily dosing, providing sustained pharmacologic coverage of IRF5 expression.

Now, in addition to degradation, we also assessed the impact of IRF5 degradation on downstream biomarkers which we believe is an important indicator of the potential biological effects.

As a reminder, IRF5 has a role in the regulation of three major immune pathways relevant to several autoimmune diseases. These include:

First, inflammatory cytokines. IRF5 promotes the production of key inflammatory cytokines by myeloid cells, including monocytes and macrophages. And when dysregulated, these responses can sustain chronic inflammation.

Second, interferon response. IRF5 helps connect nucleic-acid sensing with interferon production and associated gene-expression responses. Dysregulation of this pathway can amplify immune activation and inflammation.

And the third pathway is B-cell biology. IRF5 contributes to B-cell activation and pathways associated with plasmablast differentiation and autoantibody production.

So, this breadth of biology is an important feature of the IRF5 mechanism. By degrading IRF5, KT-579 has the potential to modulate multiple interconnected drivers of inflammation through a single oral approach.

Now, to assess this biology in healthy volunteers, we used three orthogonal *ex vivo* stimulation assays that our team developed internally. These assays activate distinct RNA- and DNA-sensing toll-like receptor pathways and measure downstream changes in inflammatory mediators and gene expression. This allowed us to assess whether IRF5 degradation translated into functional effects across all three key areas.

In the following slides, we'll focus on the impact IRF5 degradation demonstrated in each pathway. In each graph, we'll show the median of all KT-579 MAD dose cohorts combined, compared to placebo.

Shown on Slide 16, IRF5 degradation translated into deep and sustained inhibition of core inflammatory cytokines, including TNFα, IL-12/23p40 and IL-1β. As shown here, median cytokine reduction across all MAD dose cohorts combined, ranged from 87% to as high as 94%. The kinetics of cytokine inhibition track with the kinetics of IRF5 degradation, and the magnitude of the effect is consistent or superior to what was observed preclinically. These reductions are particularly encouraging as this is a core pathway involved in lupus, through TLR7/8, and IBD, through TNFα & IL-12/23, two of our highest priority indications.

We interrogated the same inflammatory cytokine pathway more broadly with transcriptomics. The transcriptomics provide a complementary confirmation of the cytokine data, showing modulation of IRF5-dependent inflammatory biology. Though anonymized, the heatmap shows the changes in selected pathway genes with placebo and across all KT-579 dose cohorts at Day 14, in relation to their respective pre-dose sample, while the graph quantifies the overall modulation of a gene set. We observed strong gene-to-protein concordance, with genes like TNFα and IL12β moving in the same direction as the corresponding functional cytokine readouts, as described in the previous slide.

The second major axis we tested was interferon response, another important component of IRF5-dependent inflammation and a particularly relevant pathway in heterogenous diseases. The effect on this pathway cytokine is particularly pronounced. We demonstrated near complete reductions in TLR 7 & 8-stimulated IFNβ levels, with the MAD dose cohorts showing a median inhibition of 98%. Importantly, the transcriptional data independently support and extend this result showing modulation results in broad coverage across key IFN pathway genes. These data validate the pharmacology beyond the inflammatory cytokine pathway we showed in the prior slides, demonstrating that IRF5 degradation can translate into profound functional modulation of the interferon axis, which is well-recognized as overactive in the biology of several autoimmune disorders including lupus and Sjögren's disease.

Finally, as shown on Slide 19, the effects of KT-579 reach into the third major axis of IRF5, B-cell biology. Selected TLR9-driven transcripts are significantly modulated with KT-579, including key genes associated

with DNA sensing and B-cell activation – again, important steps in the pathogenesis of autoimmune diseases including lupus.

In summary then, KT-579-driven IRF5 degradation translates into coherent functional modulation across three major pathways we set out to interrogate: inflammatory cytokines, interferon response, and B-cell biology. That breadth of pharmacology is one of the key takeaways from this first-in-human dataset.

Okay. Moving to safety, KT-579 was well-tolerated and had a safety profile comparable to placebo. As shown on Slide 20, there were no SAEs, very few mild treatment related adverse events, no adverse events leading to discontinuation of study drug or from the study, and no clinically relevant changes in vital signs, lab tests or ECGs. There were also no imbalances in the rates of infections.

It is very encouraging that KT-579 was able to robustly degrade IRF5 in blood, and also functionally modulate disease-relevant biomarkers across three key immune pathways, while demonstrating a placebo-like safety profile.

The Phase 1 data represent an important milestone for KT-579, for our IRF5 program, and for Kymera. These results establish important human proof of mechanism for KT-579. We've demonstrated that we can achieve deep and sustained degradation of IRF5 with an oral medicine, with a favorable safety and tolerability profile. Importantly, we've also seen that IRF5 degradation translates into strong and encouraging downstream effects on disease-relevant inflammatory biology.

Taken together, this gives us increased confidence that we are achieving the profile we designed KT-579 to deliver and provide a strong foundation to move into patients.

As the program following our STAT6 efforts, IRF5 is yet another example of Kymera's ability to develop potentially transformational medicines for patients.

So, on that note, we now plan to advance KT-579 into its first patient study in lupus.

Lupus remains an area of significant unmet need where many patients continue to cycle through therapies without achieving adequate or durable disease control, and treatment often requires combinations of immunosuppressive agents with meaningful safety and tolerability limitations. We see significant opportunity for a new safe and effective oral therapy.

Lupus is a compelling indication for this program given there is strong genetic evidence linking IRF5 to lupus, and IRF5 plays a central role in lupus disease biology. We've previously shown disease-modifying activity in multiple preclinical lupus models. And as you've seen today, we have now demonstrated exciting biomarker data suggesting a strong connection between IRF5 degradation and three important disease relevant pathways. So, this gives us a high degree of confidence in the underlying biology and a clear rationale for evaluating the impact of IRF5 degradation directly in patients.

The planned Phase 1b study will allow us to begin answering the next important questions, whether IRF5 degradation and downstream biology we observed in healthy volunteers carry through to patients with lupus, and whether KT-579 can affect the disease pathways that are active in those patients.

In healthy volunteers, we measured pathway effects after stimulating blood samples *ex vivo*. While in patients, we'll be able to assess those effects in the setting of active disease, as well as look for early signs of clinical benefit.

We view this as an important next step for the program and toward establishing IRF5 degradation as a potentially new therapeutic approach for patients with autoimmune disease.

Here's a snapshot of the Phase 1b study design for the trial, which we have named LUMORA. We plan to enroll approximately 30 patients with SLE, randomized 2:1 to KT-579 or placebo, and treated for 16 weeks. Oral corticosteroid background therapy is permitted. In fact, our upcoming oral presentation at the American College of Rheumatology conference demonstrates that KT-579 shows significant activity alone or in the presence of background therapies in preclinical lupus models. The Phase 1b will evaluate the impact on inflammatory cytokines, interferon signatures, B-cell subsets, and other key immune pathways implicated in lupus. And we'll also explore measures of lupus disease activity, including changes in the mucocutaneous and musculoskeletal symptoms patients experience. In this small Phase 1b study, those measures will help us look for early signs of clinical activity alongside the safety and biomarker results.

So, with that, I'll turn the call over to Bruce.

Bruce Jacobs:

Thanks, Terence.

Turning to our third quarter results, I'll quickly walk through the income statement shown here.

As expected, we did not recognize any collaboration revenue in the third quarter. And as a reminder, we do not expect additional collaboration revenue for the remainder of 2026, as the milestones and our previous deferred revenue under our collaborations with Sanofi and Gilead were fully recognized as of the second quarter.

In terms of operating expenses, reported R&D expenses for the quarter were $128.5 million. Excluding approximately $10.9 million in non-cash stock-based compensation, R&D expense was $117.6 million, an increase of 8% compared to the second quarter of 2026.

Reported G&A expenses for the quarter were $18.7 million. Excluding approximately $7.9 million in stock-based compensation, adjusted cash G&A expense was $10.8 million, a decrease of 14% compared to the second quarter of 2026.

We ended September with cash, cash equivalents, and investments of approximately $1.6 billion, providing a runway into 2029 and positioning us to continue execute on a number of important value-driving milestones over the coming years.

Our financial position gives us the resources to advance both our wholly owned programs into and through their next phases of development. Specifically, it allows us to complete the KT-621 Phase 2b study in asthma and take KT-579 through our planned Phase 1b study lupus. We also expect to fund the

KT-621 Phase 3 studies in AD and the initial stages of Phase 3 development in asthma within our current runway.

Alongside these clinical priorities, we'll continue to invest in our research pipeline and build the capabilities needed as we move toward later-stage development and commercialization. We believe this puts us in a strong position to execute our plans while remaining disciplined in how we deploy capital.

I'll pause here so Nello can provide a few closing comments before we open the call to questions.

Nello Mainolfi:

Thanks, Bruce.

To wrap up, we have important milestones ahead for KT-621, building on the compelling profile observed in Phase 1 healthy volunteers and patients with AD. In dermatology, we expect Phase 2b AD data by year-end. Subject to those results and regulatory discussions, we plan to begin Phase 3 development by mid-2027, and that planning is already well underway. In respiratory, our Phase 2b asthma study is ongoing, with data expected in late 2027.

And for KT-579, today's results are an important step for the program. In healthy volunteers, we saw evidence that degrading IRF5 can modulate multiple disease-driving pathways. Along with the safety and tolerability profile observed in this study, these findings support our plans to advance KT-579 into patients with lupus by early next year. We also plan to expand beyond lupus, and we'll be sharing more on our broader development strategy next year.

These programs are emblematic of our strategy to build a world-class pipeline of wholly owned immunology programs that are directed at fundamental disease pathways with the potential to help large patient populations. In closing, I want to thank the study participants, investigators and our team for making all this progress possible. With that, we'll open the call for questions.

Operator:

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 dial 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 panelists. Please accept and wait until you are promoted to panelist. Please unmute your audio, turn your camera on, and ask your question. As a reminder, we are allowing analysts one question today. We will now pause a moment to assemble the queue.

Our first question comes from Brian Cheng from JP Morgan.

Brian Cheng:

Hey guys, thanks for taking our questions today. On the Phase 1b LUMORA trial design, I'm curious how you're thinking about the bar for success there and any color in terms of which doses you would pick to move forward into this study, just given that it's interesting to note that you're already saturating at

dose level three. And just one tiny question at the end is any opportunity for patients to also taper off steroids as well? Thank you.

Nello Mainolfi:

Thanks, Brian. So, I'll take a little bit of the question, then I'll pass it to Terence who's our expert. So, I think it's important to remember that the goal of this study is to demonstrate for the first time that in patients where there is obviously disease and we believe this pathway activation, we can modulate the biology of patients with the drug, obviously without the need of *ex vivo* stimulation. So, it's really demonstrating for the first time that IRF5 is relevant in the disease biology of lupus.

I'll let you, Terence, maybe talk about some of the other specific goals that we have. I just want to touch a little bit on the dose. So, as you pointed out well, Brian, again, we're so proud that another first-in-class target that's never been drugged before, we've been able to do it robustly, safely, and showing probably better than expected biomarker outcomes. We have many options in terms of doses because we've shown preclinically that 85% is actually enough to drive the biology, and you saw all of our doses at more than 85% degradation. So, we will continue the mystery around the dose for now, but obviously we have several options, and we'll pick the one that we believe will allow us to answer that question well.

Terence, on the Phase 1b, maybe you can add more color.

Terence Rooney:

Thanks, Brian. Good to see you. Success criteria. So obviously we'll look to recapitulate degradation in the setting where the target is active and much more highly expressed than in healthy volunteers. And then downstream of that, we look for movement of relevant biomarkers in those three key pathways that we discussed in the healthy volunteer study, inflammatory biomarkers, interferon response, and B-cell biology, and we'll be able to do that without having to artificially stimulate the pathway *ex vivo*. We'll also have the opportunity, obviously, to also look at clinical outcome measures, and so we'll take heed of that too. Steroid taper, I think was your final question. We'll get into a little more detail around trial design at a later date when we launch the trial and post on clinical trials, etc. But I think it's fair to say that across lupus studies these days, the expectation is that taper is incorporated in design.

Brian Cheng:

Great, thank you. Congrats on this data.

Nello Mainolfi:

Thank you.

Nello Mainolfi:

Next one.

Operator:

Our next 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 in these very nice IRF5 data. Just with respect to the IRF5 degradation, I was wondering if you could comment on how much interpatient variability you observed. Were there any patients who didn't achieve at least that 80% threshold you were aiming for? Was there any difference in serum versus skin? And then I was also wondering if you could just comment on some recent data we've seen from the TLR7/8 class and how that may influence your thinking around probability success in an indication like lupus. Thanks so much.

Nello Mainolfi:

Okay, so I'll take the first two. I do want to encourage people to try and focus on one because if you have three in one, we're not really doing one. This is for future questions. So, on the degradation, I mean, if you look at our graph, the slides should be up on our website, by the way, to everybody listening, so you should be able to see it. Our degradation profile has now, this is the sixth program we show data for, is always extremely robust and consistent. Our error bars are extremely small, so there are no outliers in the degradation profile in the subject in our study. The difference between blood to skin is minimal, if any.

The IRF5 expression in skin is extremely low. In fact, we actually did not set the expectation around skin because actually we thought we weren't going to be able to measure it, but we have an amazing team here that has developed unique assays, some of which we're not sharing because they're assays that we've invented and we want to protect as well. But we were able to actually measure even very low expression level we were able to robustly reduce with this drug. So that was another important milestone. So, there was a question around the competitive landscape. Terence, do you want to make a comment?

Terence Rooney:

Thanks, Thomas. I'd say the TLR7/8 direct inhibition gives us confidence and probability of success, but of course this mechanism goes beyond just TLR7 and 8. So that I think is a good guy for us too, and really looking forward to interrogating that profile in lupus patients very, very soon.

Nello Mainolfi:

Thank you. Next one.

Thomas Smith:

Thanks guys. Congrats again.

Operator:

Thank you. Our next question is from Derek Archila from Wells Fargo. Please unmute your line.

Derek Archila:

Hey, good morning, and thanks for taking the questions and congrats on the data today. So more of a market question in terms of atopic dermatitis, but the beyond opportunities for KT-621. So, I was wondering if you could help quantify how the atopic dermatitis market may expand with the introduction of a safe and effective oral. And I guess the bigger question is, could you expect similar expansions in other indications like asthma, COPD, and so on? Thanks.

Nello Mainolfi:

Yeah, thanks, Derek. So, actually, this gives an opportunity also to make another point, which sometimes we forget to do, which is KT-621, potentially the first safe effective oral for Type 2 diseases, is something that has never actually been accomplished before. So, we can focus on AD in a minute, not only this is a drug for AD, but it's a drug that will or should, I should say, be treating all comorbidities. One of the main reason for dupilumab success is not only the robust efficacy and pretty amazing safety profile, but it's the fact that it's the preferred options for most doctors because it treats comorbidities that most patients have when they have any one Type 2 disease. So that is something that we often forget to remind, but I just wanted to take the opportunity to remind, this is a unique drug also because of it.

Now, when we go back to your point about, we had a slide today earlier in our presentation that there is millions of patients with moderate to severe AD in the US, six to nine we said. This is actually a publication. This is not our numbers. And another data that is not ours, but it's a fact. It's about 400,000 are on advanced systemic therapy. So, the opportunity here that we have with potentially the first safe effective oral drug is to expand the market of patients that are sitting on the sidelines because they're not comfortable with the idea of taking an injectable biologic. So, we think this would be transformative almost regardless of where exactly the efficacy profile will land. Although as we said, we expect it obviously to be in the dupi ballpark. And that could be applied, as you were implying, to other diseases where actually either there is mostly, if not only biologics, for example, asthma or diseases where there is only one drug or two drugs. If you think about EOE, only injectable drug so far only Dupixent would probably have teze, we assume, approved relatively soon. CRS, again, these are dominated by injectable drugs that are not what people are looking for. So, we believe that the opportunity here, we continue to make the case, is not so much that we will have to compete with biologics, which I'm sure we will do as a highly competitive team here, but it's actually about expanding the market to new patients. And even in psoriasis, as you've seen with Icotyde recently, a much more mature market, 60% of new prescriptions for Icotyde are new patients, which is pretty impressive for a market that has been built for now almost 15 years.

Derek Archila:

Very helpful. Thanks guys.

Nello Mainolfi:

Thanks.

Operator:

Thank you. Our next question is from Geoff Meacham from Citi.

Nishan Ghandi:

Hey guys, this is Nishant on for Geoff. Thanks for taking our questions. So, looking at today's data from KT-579, are there any elements that most strongly support development beyond lupus, maybe like in IBD, Sjögren's or RA? Thank you.

Nello Mainolfi:

Yeah, maybe I'll let Terence take this question and then I might add one thing at the end. Go ahead.

Terence Rooney:

Thanks. So genetically, there are strong genetic links to each of those diseases for IRF5 overactivity. So that gave us confidence as a starting point. From the data today, obviously we looked at those three pathways, pro-inflammatory cytokines, Type 1 interferon, B-cell biology, and you can draw threads between each of those and rheumatoid arthritis and/or Sjögren's disease or IBD. So just as an example in IBD, obviously in the pro-inflammatory cytokine panel, things that we looked at included TNF-alpha and interleukin-23. Each of those have been successfully targeted as monotherapies and recently in combination. In Sjögren's disease, it's well recognized as an interferonopathy with strong interferon signature, and we saw good damping of Type 1 interferon in today's readout. And finally, it's also well-recognized as a B-cell-driven autoantibody-associated disease, and we saw signals there too. So, I think we've got good evidence of a broad oral opportunity.

Nello Mainolfi:

Yeah, and the only thing I would add, thanks, Terence, is that the lupus LUMORA study, which is bound to start soon, you should expect it to be one of multiple patient study that we plan to initiate around this program with the KT-579.

Operator:

Our next question is from Mike Ulz from Morgan Stanley.

Mike Ulz:

Great. Good morning. Thanks for taking the question and congratulations on the data as well. Maybe I could just ask a follow-up just on the upcoming BROADEN2 data, just given you completed the last patient visit in the study, just wondering if you can share any thoughts on baseline characteristics and how that might impact our thinking when we try and do cross-trial comparisons to the Dupixent Phase 3 studies. Thanks.

Nello Mainolfi:

Yeah, thanks, Mike. As we've said in the past, we're trying to just preserve all the patient baseline characteristics as well as obviously the data when we report the data later this year before year-end, as we've said. The only new update we gave today in our remarks was around the percentage of patients that went from the 16-week study to the OLE, just because we have completed that study and now the study is officially in open-label extension. And we said that of the eligible patients, 98% enrolled into the OLE. That's the only comment we're going to make around that study, and then the next update would be when we share the data. But thank you. Next one.

Operator:

Our next question is from Biren Amin from Piper Sandler.

Biren Amin:

Hi guys. Thanks for taking my questions and congrats on the IRF5 update. Maybe a question on KT-621 and BROADEN2. What measures did you take in the trial to mitigate against high placebo response, which typically we've seen that historically in other AD studies? Thank you.

Nello Mainolfi:

Yeah, Biren, thanks for the question. I'll let maybe Terence take this one.

Terence Rooney:

Thanks, Biren. So, the priority of the company in enrolling that study was to focus on quality rather than speed, hence it was actually a particularly pleasant surprise that the study enrolled a couple of quarters early. Measures included careful site selection, careful investigator selection, control of factors that can sometimes drive placebo response rate like previous medications or concomitant therapies coming into the trial, careful patient selection coming in, including some fairly novel measures to assure that patients had atopic dermatitis, that it was moderate to severe. So, despite that tight filter, as I say, there was a lot of enthusiasm. The trial enrolled approximately two quarters early. Some of that enthusiasm I think came from appetite for a safe effective oral in the space, comfort with the pathway, a high touch approach by the team, and a transparent scientific disclosure, but really looking forward to disclosing the results very soon.

Nello Mainolfi:

Thanks, Biren. Next question. I'm doing the operator as well today.

Operator:

Thank you. Our next question is from Alex Thompson from Stifel.

Alex Thompson:

Hey, thanks for taking our question. Maybe another on BROADEN2. What should we expect in terms of data beyond the primary and key secondary endpoints as it relates to subpopulations like those with comorbid asthma and as well as biomarkers? Anything there would be helpful. Thank you.

Nello Mainolfi:

Yeah, thanks, Alex. Obviously, we don't have visibility into the data yet, so it's really hard for us to commit 100% to beyond what we've already said. I think the goal of the first disclosure is to ensure that we share all the data that we believe is material to obviously the company, to investors to assess the safety and efficacy profile of the drug. Some things that we believe are additional interesting scientific information but are not critical, we love to keep them for eventual presentation and publication. So that's kind of the overarching philosophy. I think once we'll share the data, we'll be able to then explain what's in there and what will come at a later point.

Operator:

Thank you. Our next question is from Faisal Khurshid from Jefferies.

Faisal Khurshid:

Hey guys, thank you so much for taking the question. Just wanted to ask maybe a little bit different of a question. With regards to your degrader platform, can you talk to us, there's some interesting extracellular degrader programs and platforms that are out there. Can you talk to us about whether that's something that is within the Kymera repertoire or is that something that you could be interested in going into in the future?

Nello Mainolfi:

Yeah, thanks for the orthogonal question. Let me explain what we're very good at. I think we're the best at is I think identifying the right targets for the technology, identifying small molecule highly specifically ligand for novel targets, making exceptionally well-behaved and specific degraders, and I think designing and executing so far early to mid-development studies in immunology. And obviously that sounds like status quo, but actually that's really hard to do all in a completely novel company. Another thing that we're really good at is any type of heterobifunctional molecule chemistry. So that obviously could incorporate by just nature of the business, extracellular degraders, these RIBOTACs obviously molecular glues. And so, we as a curious and well-financed company, we look at other contiguous technologies as

well. What I will say is that I find it extremely hard to develop a technology, let's say an extracellular degrader solution that is not oral, meaning I think the power of heterobifunctional molecules is the ability to make these drugs, oral drugs that can replace or expand beyond these injectable biologics.

So, I don't think you're going to see us making extracellular degraders that are injected because I don't think there is a need for that. There are other technologies that can trap extracellular proteins much more efficiently or even now probably a lower cost than novel technologies. Thanks for the question. Next one.

Operator:

Thank you. Our next question is from Jeff Jones from Oppenheimer.

Jeff Jones:

Good morning, guys, and again, congrats on the really striking data. As far as Phase 3 planning for '621 beyond AD and asthma, just any updates on your thinking around additional indications and programs there? You've disclosed some thoughts previously, but just if there are any updates to your thinking?

Nello Mainolfi:

Yeah, thanks, Jeff. Great question. I'll try and address this quickly. So, our goal is to launch KT-621 in all indications that we believe are relevant to T2inflammation. In terms of priority indications, are the ones that we believe there is highest unmet need and opportunity for an oral drug to change the landscape. So those go beyond AD and asthma. I think we want to have that conversation post BROADEN2 data so that at some point early next year we can share a bit more comprehensively what our late development plan is because that will be obviously highly dependent on these upcoming data readout.

Operator:

Thank you. Our next question is from Brian Abrahams from RBC Capital.

Kevin Meli:

Hi, good morning team. This is Kevin on for Brian. Thank you for taking our questions. So, on the Phase 1b lupus study, will you enroll both interferon high and low patients? And for later stage development, do you think there is value in focusing on interferon high patients should the effect be more pronounced there? Thank you.

Nello Mainolfi:

Terence.

Terence Rooney:

Thanks, Brian. Thanks for the question. We will enroll both interferon high and low patients. It's premature at this stage, I think, to focus on a biomarker-driven subset. Obviously, we're well aware of interferon high and its potential for controlling placebo response rates in lupus trials, but just hearkening back to the mechanism of action here, obviously the MOA includes what goes well beyond just interferon inhibition. So, at this stage we're in learn mode. We'll enroll a well-controlled, well-defined lupus population, but not necessarily tightly focused on just interferon high.

Nello Mainolfi:

Yeah, I agree, Terence. And more importantly, because we want to learn whether there be a different biomarker response based on heterogeneity of baseline. So, I think at this stage it's very important to be more open-minded than not. Next one.

Operator:

Our next question is from Andy Chen from Wolfe Research.

Jason:

Hi guys, thank you so much for taking my question. This is Jason on for Andy, and we just had a quick question on the PD for IRF5. Did the data suggest any flexibility in dosing frequency or rapid reversibilities if any safety issues emerge? Thank you.

Nello Mainolfi:

So, our drugs, as you can see even from the biomarker responses, when you stop dosing the degradation, obviously the target comes back to baseline in a few days, and you can see it again in the biomarker response group whose kinetics is very similar to the degradation as Terence said. So I think we know how to obviously dose this drug, what doses we need to adopt. We personally, I should say maybe I personally don't expect that we'll have to react to some emerging adverse event based on mechanism, but even if that was the need of cessation of dosing, again, these are drugs whose half-life are in the hours, not in the days, and so you can see a re-uptake of pathway signaling relatively quickly, so we're not concerned.

Jason:

Gotcha. Thank you.

Nello Mainolfi:

Next one.

Operator:

Thank you. Our next question is from Morgan Lamberti from Goldman Sachs. Please unmute your line and ask your question.

Morgan Lamberti:

Thank you. This is Morgan on for Andrea Newkirk. Thanks for taking our question. On IRF5, how are you thinking about the translation of biomarker reduction in healthy volunteers to lupus patients in terms of whether you would expect to see similar or greater reductions? And then how should we think about the safety or the ramifications of achieving greater than 90% IRF5 degradation, just if there's any specific AEs you're monitoring there? Thank you so much.

Nello Mainolfi:

So maybe just quickly I'll try to answer and then Terence can help me. So just remember the *ex vivo* cytokine activation is, let's call it an artificial pathway activation, and this allows us to measure in the absence of IRF5, how much pathway inhibition you see when you have a robust activation. So, the fact that we're able to practically inhibit fully these pathways with IRF5 degradation, it's telling you that this target is central to these pathway activations when we use this stimuli. I think in patients you obviously have this pathway activated, but not with this type of stimuli. There is obviously disease-driven stimuli that might be often there would be probably less robust than these, and so it's always difficult to being able to predict. I think the fact that we can block them fully gives us confidence that no matter what the stimuli is in patients, we should be able to see robust pathway inhibition. What was the second part?

Terence Rooney:

Safety.

Nello Mainolfi:

The safety. Terence, you want to take that?

Terence Rooney:

Yep. So, thanks, Morgan. I think we expect to see movement in all three key pathways unstimulated in lupus patients, some of which are obviously clinically validated in that disease, including type one interferon and B-cell biology. On the safety side, we've been encouraged by the safety profile seen pre-clinically before this study and particularly encouraged by the preliminary safety data we've seen here in Phase 1, including a lack of any signal with respect to infection. We'll continue to watch carefully, but so far so good.

Nello Mainolfi:

Yeah, we know that there are redundancies in the IRF5 family, so while we'll have a watchful eye, we're not concerned about the long-term safety of this drug. Next one.

Operator:

Thank you. Our next question is from David Hoang from Deutsche Bank.

David Hoang:

Hi there. Good morning. Thanks so much for taking our questions and congrats on today's IRF5 data. So, before you head into the quiet period ahead of the BROADEN2 readout in AD, just wanted to ask you, could you remind us what is your view of in the ballpark of dupi for endpoints like EASI-75 and IGA 0/1? And then in terms of the scenarios that could occur, knowing what we know about STAT6 biology, is there a scenario where '621 could outperform dupi at 16 weeks? Thanks so much.

Nello Mainolfi:

Yeah, thank you. So, I mean the reason why we've continued to talk about this ballpark is because for all the data that we have generated so far for more than half a decade, we've seen that blocking IL-4 and 13 with an upstream biologics or blocking IL4/13 with a STAT-6 degrader seems to block all downstream signaling in a similar way. So that's where our hypothesis comes from. The other aspect is if there are differences between the two drugs, obviously that could be driven by many things. It could be, obviously these are different technologies, small molecules, antibodies. It's still an intracellular versus extracellular target, but it's difficult for us a priori to determine the impact of these nuances on the clinical effect that we will see. With regards to defining the ballpark, it's actually, I'm trying to define unscientifically something that is also unscientific. So cross-trial comparisons are technically not doable, and they have an inherent flaw in the approach. And so, it's impossible for me to tell you exactly what the numbers should be that we should be looking at. But I think for people that are students of the space, you should be able to have a reasonable interpretation when you see a data set across several endpoints between two studies that are around a decade apart.

So, you have to look at the placebo rates, you have to look at the absolute treatment effect, you have to look at the consistency across endpoint. And at the end of the day, it actually does not matter because we're not trying to replicate the dupilumab study. We're trying to develop one of the most exciting oral drugs for Type 2 patients that is right now in clinical development. And the important thing is that we demonstrate safety, efficacy, and tolerability. I know that there's a lot of focus on comparisons and we will do that comparison too. I'm not trying to be a purist here, but I'm just trying to say that actually the important thing is that we deliver on the profile that we believe would be commercially successful. And for the scientific curiosity, there are many tools that one can use to compare trials that will all be wrong but will give us a sense of what it looks like. This is the definition of ballpark, what I said in the past two and a half minutes maybe. Next one.

Operator:

Thank you. Our next question is from Tyler Van Buren from TD Cowen.

Tyler Van Buren:

Hey, great. Good morning, guys. Thanks so much for the ‘579 presentation. Regarding BROADEN2, should we expect to see itch data with the initial release? And can you discuss the potential to differentiate there given the IL-31 reductions observed? Our KOLs have recently brought this up in discussions.

Nello Mainolfi:

Yeah, we plan to have itch data, of course. Itch, sleep, sleep itch, quality of life. These are actually important measures of how well the drug is actually serving patients. If you do market research, the number one complaint for patients that are actually on therapy continues to be itch. So, with regards to differentiation, it's hard for me, and obviously I should be the most biased person in the world for the STAT6 program. It's hard for me to over-index on the IL-31 data. It's great that we saw IL-31 blood reduction in the BROADEN study. This is the Phase 1b. It is also true that hasn't been measured by others. There was a study with dupilumab in Japanese patients where they didn't see a reduction of IL-31. But you know again, we're 10 years apart. We have an amazing translational medicine team under Terence's guidance. I think that it's probably more likely that we're just better measuring than we are different biologically from Dupixent. But anyway, we'll know soon, so we'll definitely share the data. Next one.

Operator:

Thank you. Our next question is from Jeet Mukherjee from BTIG.

Jeet Mukherjee:

Great. Good morning. Thanks for taking the question and congrats on the data. It almost seems you guys make degraders of this quality in your sleep now. My question was just focused around the KT-579 Phase 1b lupus study. Could you just elaborate on some of those exploratory endpoints and what would tell you that you have a clinically differentiated profile here for KT-579? Thanks.

Nello Mainolfi:

Terence, you want to take that?

Terence Rooney:

Sure. Jeet, I assume you're referring to the exploratory clinical outcome measures. We look at all of the usual lupus clinical outcome measures, including composites like SLEDAI and BILAG. We look at the individual components, and obviously that'll sit alongside the biomarker data, which are the chief focus of the study. And we'll take the whole set of data together in making our decisions downstream of that.

Nello Mainolfi:

Yeah, I want to add two things. So first, the reason why we make drugs in sleep is because we don't sleep, so we actually can make drugs effectively. That's a joke by the way. And then on the other thing, I just want to remind everybody, the study's not powered for clinical endpoints. That's why we've said that they're more exploratory than the usual exploratory. So, everything Terence said, obviously I agree, but just remind everybody, this is a small study, biomarker focused to understand the biology in the right clinical context of IRF5 degradation. But obviously it's a 16-week study, so we'll keep an eye on any move on clinical endpoints, understanding that, as you guys all know, placebo rates in lupus are high, and so we got to be careful on how we interpret those data sets. Next one.

Operator:

Thank you. Our next question is from Jasmine Fels from Barclays.

Jasmine Fels:

Hi, Jasmine, on for Ellie Merle. So, congratulations on the IRF5 data. I wanted to ask a little bit more about dose selection and dose response. So, can you characterize the dose response you saw across the different MAD cohorts for the *ex vivo* stimulation biomarkers? So, did you see a similar trend as degradation for these with saturation at dose level three, or was there a wider range of dose response across the different biomarkers?

Nello Mainolfi:

Yeah, thank you. It is a great question. So, to be honest, the reason why we're not sharing what we usually share, Terence said it earlier, it's we want to retain some competitive advantage over the fierce competition that we've seen increasing over the past few years. We feel with KT-621, we probably taught the market too much. Actually, I shouldn't say the market, our competitors, and so we're trying to be a bit more thoughtful. So, the reason why we're not sharing, let's call it the dose response on biomarkers, because I personally don't want to share what is the level of IRF5 degradation that leads to the optimal biomarker changes that we're seeing. So, we have a really good understanding of what is the dose we need to use, and that dose selection has been based on what we think the level of degradation is and what we know the level of biomarker changes are. And for now, we're going to leave it at that.

Operator

Thank you. Our next question is from Seamus Fernandez from Guggenheim.

Seamus Fernandez:

Thanks for the questions and congrats on the IRF5 data. The question that I have on IRF5 and maybe at some upcoming meetings would be the potential links of the robust biomarker effects that you are seeing targeting IRF5 and some of the data that we're going to learn coming out of ACR, particularly as it relates to Sjögren's disease. Terence, what would you be looking for from other disease states given this

sort of B-cell versus T-cell dynamic, CD40 ligand versus APILBAF and the opportunity that you see in other disease states from some of those learnings?

Terence Rooney:

Thanks, Seamus. I think Sjögren's is a really good indication for the target. There's a lot of unmet medical need in Sjögren's, as yet no approved advanced therapies. The biology is a good fit. We've got good genetic links between IRF5 and Sjögren's risk, and some of the pathways are clearly activated. So, for example, we know Sjögren's to be a highly high interferon flavored disease, and we know that B-cell targeted therapies have shown efficacy, including one that's hopefully on the cusp of approval out of Novartis. So, I think we're very encouraged that Sjögren's could be a good indication and one we're carefully considering.

Nello Mainolfi:

Cool. Next one, we'll try and accelerate. I'm being told we're being too slow, so let's see if we can accelerate.

Operator:

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

Tazeen Ahmad:

Guys, good morning. Thanks for squeezing me in on a busy morning. A couple of questions. So, for this big data update that you have upcoming, as it relates to safety, in particular for conjunctivitis, what if you don't see any instance of it? Because I think mechanistically it should be something to expect, but if at this juncture, you're still not showing it, how should we be thinking about what that means? Could that differentiate from other treatments, for example? And then secondly, can you just give us a sense of what the top catalyst for 2027 in your mind would be as we think about next year and beyond? Thanks.

Nello Mainolfi:

Thanks, Tazeen. I love that we haven't gone through the biggest catalyst, but we'll talk about the next one. So, the next one, let's start with your second question. Obviously, we have big data with asthma, which will be a huge catalyst to initiate multiple Phase 3 programs in asthma and other respiratory indications. So, that from where we stand today is probably the biggest data set of '27. We have other things that we will be sharing more in the near future, but I think for now I will say this year... Next year was supposed to be both AD and asthma, AD has moved to this year. So, this year is AD, next year asthma and other things.

On your question about conjunctivitis, the mechanisms that, as was always said, biologically is coherent with IL-4 and 13 biology. So, I've said it for a couple of years, we expect to see conjunctivitis. If we don't

see it, obviously we could talk about potential reasons for it. But again, as we are saying, we're close enough and we'll have that conversation than what we see or don't see. Next question.

Operator:

Thank you. Our next question is Mayank Mamtani from B. Riley.

Mayank Mamtani:

Good morning. Mike on for Mayank. Congrats on the quarter and the data. When you report for BROADEN2, will you break out IAE's using the same conjunctivitis and keratitis clusters from dupi's label? This is just so can compare KT-621 with like for like. And also considering that Nimluvio is kind of seen as the itch benchmark, how do you think KT-621compares to Nimluvio when it comes to itch? Thank you.

Nello Mainolfi:

I think for both questions, we'll know soon. I mean, I don't know the data to know what's going to be put with what, but for sure we will present the data in a way that, as I said earlier, everybody should be able to assess the efficacy and the safety of the drug, as well as whether there has to be any comparator way of looking at also safety. We'll do it in the way that is interpretable. With Nimluvio, it's not a very active drug, but it does have an impact on itch by IL-31. We do have also a downstream effect on IL-31. I'm not sure Nimluvio would be our competitor, let's put it this way, but we'll see. Data will come up soon.

Operator:

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

David Dai:

Great. Thanks for taking my questions and congrats on the IRF5 data as well. So, you noted a similar IRF5 for reduction in skin and blood. So, I'm just curious, how does the skin tissue data give you the confidence that the peripheral blood IRF5 degradation will be a reliable surrogate for target engagement in disease relevant tissues in lupus and potentially IPD?

Nello Mainolfi:

Well, the reason why we always look for a non-central tissue and skin really is the only thing we can look at in healthy volunteers is to, again, de-risk the distribution and degradation profile of the drug beyond the central compartment. So, I'm a big believer through, again, 10 years of work here, that skin is actually the hardest tissue to degrade targets in. And that's why we always measure it because we know that if you degrade in skin, you're probably going to be fine for the rest of the body. We also have preclinical data that shows that we can consistently degrade this target in all the relevant tissues. So, this was just an additional validation of our profile.

Operator:

Thank you. Our next question is from Kripa Devarakonda from Truist Securities.

Kripa Devarakonda:

Hey guys, thank you so much for taking my question and congratulations on the data. So, you accelerated your atopic derm program because of the great high level of demand that you saw. Are you seeing similar demand with the asthma program as well, and has that led to increase in enrollment rates? And if not, why do you think there is a difference? And how predictive do you think the endpoint, the FEV1 endpoint, is of eventual exacerbation benefit?

Nello Mainolfi:

I'll take the first question so we can move more quickly. So, we are seeing the same level of enthusiasm in AD as we have in asthma. The main difference is that in asthma, our inclusion/exclusion criteria are much more strict and so the screen failure rate is much higher, and that's what determines how quickly or not quickly we're enrolling the study. Next question.

Operator:

Thank you. Our next question is from Joseph Holden from Canaccord Genuity.

Joseph Holden:

Hey everybody, this is Joe Holden on for Edward Nash. Thanks for taking my question. I just wanted to see if you could provide any guidance on expected R&D expenditure into 2027, given KT-579 Phase 1 in lupus and the expected Phase 3 program in AD. Thanks so much and congrats on the data.

Nello Mainolfi:

Bruce?

Bruce Jacobs:

Yeah, I'll just be brief because we don't give explicit guidance, but I would guide you to the runway, which is into 2029, and you should see, I would say, increases in operating expenses that are gradual but consistent to get you to the point that you're into 2029. Obviously, the expenses will accelerate as we start the Phase 3 study in AD, which we've guided to by mid-2027. So, we are also adding significantly to the team in preparation for those studies as well. So those will be the biggest factors in driving the cash year-over-year, I say, use of cash into ‘27 and beyond.

Nello Mainolfi:

I believe this was the last question. I want to thank everybody for attending the call and also staying to 9:40. Please take the opportunity to check out our website. The slides are there. We're very excited about where we're going with now two very exciting first-in-class programs in patients, and so we're happy to follow up with any of you in the next couple of days apparently, but not beyond. Thank you.

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