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# EDITED TRANSCRIPT

PFE.N - Pfizer Inc. Pflash A Spotlight on the Trispecific Antibody Tilrekimig

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**OVERVIEW:**

Company Summary



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## PRESENTATION

**Operator**

Good day, everyone, and welcome to Pfizer Flash, a spotlight on the Trispecific Antibody Tilrekimig. Today's call is being recorded. At this time, I would like to turn the call over to Francesca DeMartino, Chief Investor Relations Officer and Senior Vice President.

Please go ahead, ma'am.

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**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

Thank you, and good morning, everyone. I'm Francesca DeMartino, Senior Vice President and Chief Investor Relations Officer. On behalf of the Pfizer team, thank you so much for joining us. Today's event will be recorded and available for replay on our IR website at pfizer.com. As a reminder, our Pfizer Flash series is intended to serve as an educational deep dive into our pipeline, products, and people. Each event spotlights a specific product, therapeutic, or growth initiative and gives you an opportunity to hear directly from our business leaders. Today's session will begin with a presentation followed by live Q&A.

As a reminder, this call is intended only for the investment community, including our sell side analysts and institutional investors. I want to note that on today's call, we will be making forward-looking statements. I encourage you to view slide 2 in our presentation and the disclosures in our SEC filings, all of which are available on our IR website at Pfizer.com.



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Forward-looking statements on the call are subject to substantial risks and uncertainties, speak only as of the call's original date, and we undertake no obligation to update or revise any of the statements.

I am joined today by Mike Vincent, Pfizer's Chief Inflammation and Immunology Officer. Following my intro, Mike will speak about our potential first-in-class trispecific antibody, tilrekimig, review recent Phase 2 data in atopic dermatitis, and discuss the broader development plan for this molecule.

Christian Antoni, our Chief Development Officer for I&I, will join us for Q&A.

Inflammation and Immunology, or I&I, is one of Pfizer's R&D's four therapeutic areas of focus and a key part of our targeted strategy to address conditions with high unmet need and large patient populations where our science can deliver the greatest impact.

Pfizer's in-house discovery teams have extensive expertise across a broad range of core modalities, including small molecules, vaccines. and especially relevant for today's discussion, biologics such as multispecific antibodies.

Here's a select snapshot of our I&I portfolio, which includes small molecules and biologics at multiple stages of development across both new molecular entities and lifecycle management opportunities. This portfolio reflects Pfizer's established capabilities in I&I across both R&D and commercial functions.

While we recognize there is significant interest in several of these programs, the remainder of today's presentation will center on tilrekimig, and we ask that you focus your Q&A questions there as well. With that, I will hand it over to Mike.

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**Michael Vincent** - *Pfizer Inc. - Chief Inflammation & Immunology Officer*

Thank you, Francesca. It's a pleasure to be here.

As you mentioned, we've leveraged our expertise across small molecules and biologics to build a robust I&I pipeline and are eager to highlight tilrekimig today. tilrekimig is an investigational trispecific antibody that targets IL-4, IL-13, and TSLP, three established and complementary drivers of type 2 inflammation. Type 2 inflammation is a misdirected allergy-type immune response that causes the itching, swelling, and airway narrowing seen in the diseases I'll be discussing today.

Last month, Phase 2 results for tilrekimig and moderate-severe atopic dermatitis were featured in an oral presentation at the European Academy of Dermatology and Venereology Congress, or EADV. We believe these data provide proof of concept for tilrekimig and support the three pillars of its value proposition.

First, robust efficacy. At the top monthly doses studied at week 16, tilrekimig could achieve placebo-adjusted response rates of about 50% for EASI-75, defined as a 75% or greater improvement on the Eczema Area and Severity Index. Though cross-trial comparisons have inherent limitations, these results suggest the potential for superior efficacy versus dupilumab, today's standard of care biologic. Key secondary endpoints in an ongoing Phase 3 trial are testing this hypothesis directly in a head-to-head setting.

Second, favorable safety. At subcutaneous doses up to 400 milligrams every four weeks, conjunctivitis and injection site reactions in stage two of our trial were less frequent than the historical rates reported with dupilumab and comparable to placebo.

And third, a pipeline and a product opportunity. Atopic dermatitis is our potential anchor indication, but common biological pathways targeted by tilrekimig drive a host of other type 2 inflammatory diseases. Building on our strong phase 2 data, we've already started Phase 3 studies in both atopic dermatitis and asthma with an ongoing phase 2b/3 study in chronic obstructive pulmonary disease, or COPD.

I'll walk through each of these pillars before we open it up for Q&A. But first, let's review tilrekimig's design, mechanism of action, and the opportunity in atopic dermatitis.





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Tilrekimig was discovered internally at Pfizer and demonstrates potent binding to IL-4, IL-13, and TSLP.

Importantly, it's able to bind each of these targets simultaneously without interference, meaning affinity for one target is not impacted by the presence of the others. IL-4 and IL-13 are the central downstream effectors of type 2 inflammation.

Unlike approaches that block their shared receptor, IL-4Rα, tilrekimig binds the cytokines directly, minimizing clearance. TSLP is an upstream alarm that initiates and amplifies type 2 inflammation while also contributing to non-type 2 inflammation.

An engineered Fc extends tilrekimig's half-life to approximately 37 days, supporting once-monthly subcutaneous dosing. Targeting the ligands directly combined with the extended half-life may help maintain suppression of free cytokines throughout the dosing interval better than receptor targeting approaches, such as dupilumab.

Importantly, the biological pathways targeted by tilrekimig are clinically validated.

As shown in green, each one of the target -- each one is the target of an approved therapy. Dupilumab targets the IL-4Rα, which mediates IL-4 and IL-13 signaling. Tralokinumab and Lebrikizumab target the IL-13 cytokine, and Tezepelumab targets TSLP.

Tilrekimig is designed to target complementary drivers of type 2 inflammation with a single molecule. We believe this combination gives us a strong foundation and a clear basis for differentiation across indications such as atopic dermatitis, asthma, and COPD, subject to clinical trial and regulatory success.

Starting with atopic dermatitis, the leading contributor to skin disease burden globally, it's a chronic, relapsing inflammatory condition that carries substantial symptomatic, psychosocial, and economic burden, from persistent itch to disrupted sleep.

Approximately 9.8 million people in the US live with a moderate to severe form of the disease. Despite meaningful advances across the current landscape, many patients on advanced systemic therapy still do not achieve treatment targets. Real-world data at six months show that roughly 80% of patients missed optimal targets for skin clearance and itch.

Even against more moderate targets, more than a third missed skin clearance goals and two-thirds missed itch goals. And importantly, no biologic has demonstrated superiority to dupilumab, the current standard of care.

Next, I'll walk you through why we believe tilrekimig could potentially demonstrate improved efficacy versus dupilumab in our ongoing Phase 3 study.

It starts with potency. In preclinical in vitro studies, we compared tilrekimig with dupilumab and tezepelumab in a white blood cell expression assay. This assay is a functional measure of IL-4, IL-13, and TSLP signaling. The y-axis shows the level of admission achieved with a given concentration of antibody, with the measure of the concentration shown on the x-axis. Curves shifted to the left are therefore indicative of a more potent antibody.

As you can see, tilrekimig showed approximately 13-fold higher functional potency than dupilumab against IL-4 simulated signaling. In the middle, its potency against IL-13 signaling was approximately sevenfold higher than dupilumab.

And on the right, tilrekimig inhibited TSLP signaling at levels comparable to tezepelumab, an anti-TSLP antibody approved for severe asthma. We believe greater functional potency against IL-4 and IL-13, together with the additional mechanism of action targeting TSLP, may drive enhanced efficacy versus dupilumab.

Beyond potency, our hypothesis also rests on inhibition of multiple complementary pathways. To further explore the potential for additive effects of TSLP blockade, investigators stimulated human skin implants -- explants with IL-4, IL-13, or with TSLP, and then profiled the resulting gene expression. These data were presented at a poster in EADV.



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As the heat map shows, IL-4 and IL-13 and TSLP signaling drive both shared and distinct inflammatory pathways. IL-4 and IL-13 changed the expression of 910 genes by more than twofold. TSLP changed 179 genes. Only 44 responded to both, suggesting that the overlap is limited and blocking one pathway leaves much of the other active. This highlights an opportunity to achieve potentially additive efficacy by blocking IL-4 and IL-13 and TSLP signaling together with tilrekimig in atopic dermatitis versus blockade of either alone.

With that foundation, let me turn to the Phase 2 data for tilrekimig starting with efficacy. Phase 2 is a randomized, double-blind, placebo-controlled trial in adults with moderate to severe atopic dermatitis. It includes three stages with tilrekimig.

Stage 1 evaluated our highest dose, 450 milligrams every two weeks in biologic naive patients. Stage 2 was the dose ranging portion and stage 3 from which we've yet to report data enrolled a biologic experience population.

Today we're focusing on stage 2. In stage 2, biologic naive patients were randomized to placebo or to one of three monthly maintenance doses, 400 milligrams, 200 milligrams, or 50 milligrams, each following a loading dose.

The treatment period was 16 weeks. The primary endpoint was EASI-75 measured at week 16. The secondary endpoint measured skin clearance via the Validated Investigators Global Assessment, or VIGA, with responders defined as those achieving at least a two-point improvement from baseline and a classification of clear or almost clear skin.

An exploratory endpoint assessed itch, measured by the Peak Pruritus Numerical Rating Scale, or PP-NRS. Notably, background topical corticosteroids were not permitted, so these results reflect tilrekimig as monotherapy.

The trial met its primary endpoint across all tilrekimig doses. At week 16, about 59% of patients at the 400 milligram dose, 61% of patients at the 200 milligrams, and 48% of patients at the 50 milligrams achieved EASI-75, compared with about 9% on placebo.

Statistical significance was demonstrated as early as week 8, with response rates continuing to increase over time. Though cross-trial comparisons have inherent limitations, the Phase 2 tilrekimig results on EASI-75 at week 16 suggest the potential for superior efficacy versus dupilumab.

At the top monthly doses, from left to right on the slide, tilrekimig treatment led to placebo-adjusted response rates of about 52% at the 200-milligram dose and 49% at the 400-milligram dose. For reference, the placebo-adjusted EASI-75 response rates for dupilumab in its pivotal SOLO 1 and SOLO 2 trials were about 37% and 32%, respectively.

On the secondary endpoint of skin clearance, both the 200 and 400-milligram doses of tilrekimig achieved a placebo-adjusted vIGA-AD response rate of about 27% in the phase 2 trial. This was statistically significant versus placebo and similar to the vIGA responses reported for dupilumab in SOLO-1 and SOLO-2.

Two points of context. First, the SOLO trials used a sponsor-specific IGA scale, whereas our study used the validated IGA-AD, a more recently standardized scale. Second, consistent with the EASI-75 trajectory, we anticipate the vIGA response rate will continue to improve with longer dosing.

Turning to our exploratory endpoint itch, which is the most prevalent and burdensome symptom of atopic dermatitis and the main reason patients seek care. In an exploratory analysis, we evaluated the proportion of patients achieving a four-point or greater improvement in the Peak Pruritus Numerical Rating Scale. At week 16, placebo-adjusted response rates were about 44% and 35% in the 200 and 400 milligram groups, respectively.

For reference, placebo-adjusted rates in a cross-trial comparison for dupilumab in SOLO 1 and SOLO 2 were about 27% to 29%. Notably, tilrekimig's itch benefit emerged early, with statistically significant improvement versus placebo as early as week 4. We believe rapid and meaningful itch relief could be an important feature for patients and the physicians who treat them.



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Here you can see biomarker data from the trial that reinforces our efficacy findings and are consistent with tilrekimig's mechanism of action.

Starting with the panel on the left, TARC, or thymus and activation-regulated chemokine, is a well-established biomarker of atopic dermatitis disease activity. At week 16, tilrekimig significantly reduced TARC from baseline in the 200 and 400 milligram dose groups.

Turning to the right, the same dose is also significantly reduced eosinophils, which are key effector cells in type 2 inflammatory diseases. The observed reduction in eosinophils represents a potential point of differentiation versus dupilimab and may reflect the added mechanism of TSLP blockade with tilrekimig. These results, together with our efficacy and safety data, inform the design and dose selection for our Phase 3 program.

Now, trispecific was well tolerated with a favorable profile with no dose-dependent safety signals or serious adverse events related to the study drug. Treatment emergent event rates were comparable between trispecific and placebo. Injection site reactions were numerically lower with trispecific 400 milligrams once monthly than with placebo.

No eosinophilia adverse events were reported with either study drug or placebo, and rate of conjunctivitis were similar between study drug and placebo. While cross-trial comparisons have limitations, this profile stands out against historical rates with dupilumab. At doses of up to 400 milligrams once every four weeks, patients on tilrekimig in the Phase 2 trial had lower rates of conjunctivitis and injection site reactions than those reported with dupilumab.

This highlights an additional opportunity for potential differentiation as we advance our Phase 3 program. Our Phase 3 program in moderate-to-severe atopic dermatitis consists of two trials. One randomizes participants 2:2:1 to tilrekimig, dupilumab, or placebo.

The co-primary endpoints are EASI-75 and vIGA-AD, with tilrekimig versus placebo at week 16, which represents the traditional regulatory standard for Phase 3. Key secondary endpoints are testing superiority versus dupilumab. This will allow us to evaluate clear head-to-head evidence against the standard of care biologic.

Following a 24-week initial treatment period, a 28-week maintenance period is designed to evaluate continued treatment, extended dosing interval, and generate data on participants switching from dupilumab to tilrekimig. In parallel, a second Phase 3 study of approximately 375 patients is evaluating tilrekimig in combination with background topical therapy designed to reflect real-world treatment practice.

Together, these complementary studies are designed to support a potential BLA submission and demonstrate tilrekimig's potential differentiation in a rigorous, well-controlled setting.

Next, I'll speak about our plans for tilrekimig beyond atopic dermatitis.

With tilrekimig, we are applying a strategy we plan to use across multiple I&I programs designed to enable multi-indication molecules with development programs run in parallel.

First, identify a biological axis that drives multiple diseases with high unmet need, such as the type 2 inflammatory axis. Next, demonstrate clinical proof of concept and an anchor indication, a condition with a large patient population and clear need for new therapies. Finally, strategically expand to additional indications where targeting this axis is expected to drive significant clinical benefit.

As mentioned, the first two potential expansion indications for tilrekimig are asthma and COPD. These were chosen for the central roles of TSLP and IL-4/IL-13 signaling in airway inflammation and for the external clinical data validating both pathways as drug targets.

TSLP acts upstream, released by the airway lining in response to triggers like allergens and cigarette smoke. It activates immune cells that initiate type 2 inflammation, while also contributing to other non-type 2 pathways. IL-4 establishes and amplifies the type 2 inflammatory response, promoting the accumulation of eosinophils and the production of IgE antibodies that drive immune overactivity. And IL-13 is an effector. It drives mucus production, promotes tissue remodeling, and makes airways more sensitive and prone to constriction.



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As I alluded to earlier, approved antibodies validate IL-4/IL-13 and TSLP signaling pathways as targets in respiratory indications. Specifically, IL-4/IL-13 blockade has shown efficacy in type 2 high asthma and in COPD patients with eosinophil counts of 300 per microliter or higher. For anti-TSLP, efficacy in severe asthma was demonstrated across eosinophil levels in a Phase 3 tezepelumab study. And emerging evidence suggests a potential role for TSLP and COPD, particularly in patients with blood eosinophil counts of at least 150 per microliter.

Though recent biological approvals in asthma and COPD represent important advances, substantial unmet need persists. By a combined inhibition of IL-4, 13, and TSLP with tilrekimig, we believe we can treat patients with asthma and COPD who are not well-served by the current standard of care, subject to clinical trial and regulatory success.

These represent meaningful opportunities for patient impact, with approximately 3 million people ages 12 and up diagnosed with severe asthma in the US and about 2.5 million US adults diagnosed with COPD that remains uncontrolled.

Here you can see how tilrekimig's trials in asthma and COPD fit into our development strategy. In atopic dermatitis, we aim to demonstrate superiority versus dupilumab in a head-to-head setting. In asthma and COPD, we aim to demonstrate efficacy in a broad range of blood eosinophil counts. Our asthma studies enroll across eosinophil levels. In COPD, we are enrolling patients with bloody eosinophil counts greater than or equal to 150 per microliter.

This contrasts to dupilumab's pivotal COPD trials, which restricted enrollment to those with counts of at least 300 per microliter. This strategy is designed to clearly differentiate tilrekimig across all three of these potential indications and position it for substantial patient impact.

With that, I'll hand it back to Francesca.

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**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

Thank you, Mike.

To summarize, Phase 2 data from our anchor indications support tilrekimig's potential to be a first-in-class trispecific and leading biologic therapy for type 2 inflammatory disorders. We believe tilrekimig has the potential for superior efficacy and safety that compares favorably to the current standard of care biologic dupilimab and are evaluating this in Phase 3.

We also have ongoing late-stage studies in asthma and COPD, which together with atopic dermatitis are projected to represent a global market of more than $55 billion in 2030.

And now we will begin the Q&A session with Mike and Christian.

As a reminder, our Pfizer Flash series is designed as an educational deep dive into our pipeline programs. I'll therefore kindly ask that participants keep questions focused on the data development program and potential differentiation of tilrekimig that we covered today. Please avoid questions that would require us to provide forward-looking financial projections. While we're happy to clarify any information shared during the presentation, we will not be offering estimates beyond what has already been communicated.

With that, we are ready to take the first question. Operator, if you could please assemble the queue.

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## QUESTIONS AND ANSWERS

**Operator**

(Operator Instructions)



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Chris Schott, JP Morgan.

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**Ethan Brown** - *JPMorgan Chase & Co - Equity Research Analyst*

Hi, this is Ethan on for Chris. Thanks for taking our question. Just wanted to ask, on the trial, looking at tilrekimig head-to-head versus Dupixent, any color you can provide on the margin for superiority that the study has powered to detect along with the testing order. And then just more broadly, how you're thinking about the importance of superiority in order to gain kind of broad formulary access.

Thank you for taking our questions.

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**Michael Vincent** - *Pfizer Inc. - Chief Inflammation & Immunology Officer*

Thanks for that question. I'm going to give that to Christian Antoni to respond.

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**Christian Antoni** - *Pfizer Inc. - Chief Development Officer, Inflammation & Immunology*

Thank you, Chris, for the question. We are not currently disclosing the stats plan, we actually finalizing it. We are strongly powered against Dupixent in all the key secondary endpoints at two different time points. So we are very confident that we will have a chance to achieve it if the drug holds on.

The second part of the question, obviously, being superior to Dupixent will help the product tremendously, but we are not commenting on (inaudible).

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**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

Operator, we'll take the next question.

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**Operator**

Evan Seigerman, BMO Capital Markets. Please go ahead.

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**Unidentified Participant** - *BMO Capital Markets - Analyst*

Hi, this is Havan on for Evan. Thanks for taking our question. Could you maybe speak to the placebo response rate in your Phase 2 study? And then it seemed a bit lower than what we saw for Dupixent in Phase 3, so do you have any thoughts on what might be driving that? And then also, how do you think about this into your Phase 3 studies? Thank you.

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**Michael Vincent** - *Pfizer Inc. - Chief Inflammation & Immunology Officer*

I'll give this to Christian as well.

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**Christian Antoni** - *Pfizer Inc. - Chief Development Officer, Inflammation & Immunology*

Thank you, Evan. So the placebo rate is lower than we see in other trials, but it's a small trial where we have paid a lot of attention to the quality of the trial. When you go in the first phase of this trial, you also saw a little bit higher placebo response rate. And when then you look



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at the absolute number of the response rates, it shows that the drug is favoring where it should be. So we feel confident going forward into Phase 3, we learned a lot how to pay attention to the quality of the trials. And we are confident that we will keep the response rates on the Delta.

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**Unidentified Participant** - *BMO Capital Markets - Analyst*

Thank you.

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**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

Thank you. Operator, we'll take the next one.

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**Operator**

Vamil Divan, Guggenheim Securities.

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**Vamil Divan** - *Guggenheim Securities LLC - Equity Analyst*

Great. Thanks for taking the question and for hosting this. Maybe just two for me. One, I don't know if you can comment on this because it is forward-looking, but just as you think about the BLA submission and timelines. Maybe just what's your sort of current estimate on when you think this could be brought to the market for atopic dermatitis?

And then the second one, I'm just curious on, so the asthma COPD set, obviously, you guys have historically in [derm] and inflammation, but positive is going to be a little bit different. So I'm just curious how you're thinking about that, maybe more as a broader therapeutic area, especially if you're seeing positive signs of moving into this product, into asthma COPD. Thanks.

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**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

It's Francesca. So on the on the time to market, the only thing that I can tell you right now is just the PCD dates that are that are published on the on the various trials. So for the atopic, the AD trial that's going on right now, the PCD is the first quarter of '28. And then the third quarter of 28 for the one versus Dupi. And then on the asthma trials, those are more, first half of '29. So that's probably the best marker I can give you right now for the timelines.

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**Michael Vincent** - *Pfizer Inc. - Chief Inflammation & Immunology Officer*

And I apologize. I had a difficult time hearing the second part of the question. Could you repeat that, please?

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**Vamil Divan** - *Guggenheim Securities LLC - Equity Analyst*

Just more focused on sort of the pulmonary space more generally, if you're moving into areas like asthma, COPD. I'm just curious, is this turning into more of an area of focus for Pfizer, obviously you're in inflammation, you're in intra medicine and just curious more about this pulmonary space specifically, and if you see promise for this product moving into that space, would you, if the scenario you'd like to build up further?

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**Michael Vincent** - *Pfizer Inc. - Chief Inflammation & Immunology Officer*

Yes, I think that's absolutely the case with a molecule like tilrekimig to really realize its full potential. We need to expand into the respiratory space. And we look more broadly at that area as an area with a lot of unmet need as well as a good fit with some of our mechanisms.

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**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

Thank you. Operator, we'll take -- oh, sorry, Vamil. Are you good?

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**Vamil Divan** - *Guggenheim Securities LLC - Equity Analyst*

No, okay. Yes, I'm good. Thanks.

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**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

Okay. Thank you, Vamil. Operator, we'll take the next question.

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**Operator**

Geoff Meacham, Citibank.

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**Nishant Gandhi** - *Citi - Analyst*

Hey, guys. This is Nishant on for Geoff. Thanks for taking our questions and presentation today. Maybe one of broader questions. So your target profile for to recommend emphasizes superior efficacy versus GP. But market entry could coincide with less frequent biologics such as [zumilokibart] or maybe oral agents such as Kymera's STAT6 degrader.

What does your market research suggest physicians and patients value most among maybe incremental efficacy, safety, dosing and oral administration? And what minimum efficacy advantage would recommend me to overcome these convenience differences? Thank you.

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**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

Go ahead, Christian.

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**Christian Antoni** - *Pfizer Inc. - Chief Development Officer, Inflammation & Immunology*

Okay, so I take that one. So the question was the let's start with the patient's preference and the convenience piece. Obviously, a sub Q biologic is a very convenient drug. There are some patients who will prefer orals. But I think the difference is really also not just in the patient convenience piece. It's also the medical need. The biologics, especially here with the three targets, which you combine is a very strong therapy which will find its place among all these therapies which are coming. So we're very confident that the patient need and also patient preference for monthly dosing will be there. And can you remind me the first part of the question? I didn't really understand it completely.

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**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

Second one, yes?



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Nishant?

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**Nishant Gandhi** - *Citi - Analyst*

Yes, I mea --

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**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

We apologize. The AVs have been a little bit challenging, so we're just having trouble hearing all of you across the board. So if you wouldn't mind just everybody speaking up a tad, because yes, it's not just you, we just can't hear as well.

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**Nishant Gandhi** - *Citi - Analyst*

Yes, just your thoughts on market research, because like, it looks like tilrekimig would enter market like with multiple other drugs like zumi, which is like once every three months and six months, maybe talk about how the physicians like your interactions with them, give you any insight on like how this drug could be competitive versus, zumi, which could be every three, every six months, or the oral like stat six. Thank you.

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**Christian Antoni** - *Pfizer Inc. - Chief Development Officer, Inflammation & Immunology*

Yes. So, as I said earlier, patients preference will play a role and some patients will go to more extended dosing, no question. But at the end of the day, the benefit risk ratio of these drugs will, I think, the primary rule. And when you have a drug which is superior in the efficacy and very safe, I think it will find its place and dominate that convenience discussion.

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**Nishant Gandhi** - *Citi - Analyst*

Thank you.

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**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

Thank you, Christian. Thanks, Nishant. Operator, we'll go to the next question, please.

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**Operator**

Dave Risinger, Leerink Partners.

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**David Risinger** - *Leerink Partners LLC - Analyst*

Thanks very much, and thanks for hosting this session, and congrats on the phenomenal data. So I wanted to step back to a little bit higher level here. I'm very interested in the history of tilrekimig. So, obviously, this was internally developed by Pfizer. Could you talk about that history and discuss which research site gets the credit for it? Thank you.

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**Michael Vincent** - *Pfizer Inc. - Chief Inflammation & Immunology Officer*

Yes. Thanks for the question, Dave. This is a molecule that was conceived, discovered, and developed at the Cambridge Kendall Square site. And really goes back to our days at Wyeth when Wyeth was actually the first company to discover the IL-13 pathway and had a couple of IL-13 agents in the clinic over a decade ago.

When we conceived this molecule, part of the appealing strategy was to combine the different mechanisms into a single molecule. As you see with some programs today, individual monoclonals are sometimes given in combination. But from a complexity standpoint, in particular, deciding on the best dose, we thought it was much more straightforward to design in a fixed potency across the three molecules and then have a single program where you can just do a single dose ranging study to establish the most efficacious dose. So it's a molecule with quite a bit of history and came from the Cambridge site.

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**David Risinger** - *Leerink Partners LLC - Analyst*

And how long is the exclusivity?

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**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

Want us to get back to him. Dave, we'll get back to you. We'll look it up and get back to you.

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**David Risinger** - *Leerink Partners LLC - Analyst*

Wonderful. All right. Thank you again.

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**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

Thanks, Dave. Operator, we'll take the next question.

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**Operator**

Louise Chen, Scotiabank.

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**Hannah Liu** - *Scotiabank GBM - Analyst*

Hi, this is Hannah on for Louise. I had a question about the use of topical corticosteroids. So because of Phase 2 evaluated tilrekimig as a monotherapy without the background use of steroids, how should we think about the potential incremental response when combined with these topical therapies in the Phase 3? And do you believe that adding topical therapy would make it harder to demonstrate the drug's intrinsic differentiators?

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**Michael Vincent** - *Pfizer Inc. - Chief Inflammation & Immunology Officer*

Okay that to Christian.

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**Christian Antoni** - *Pfizer Inc. - Chief Development Officer, Inflammation & Immunology*

It's a very common. Trial design atopic dermatitis, that you have trials in combination with topical steroids and trials without combination of the steroids. And we have no doubt that that tilrekimig will fare as the other drugs and will show a very nice response against placebo and in the combination.

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**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

Thank you, Christian. Operator, we'll go to the next one.

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**Operator**

Rajesh Kumar, HSBC.

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**Rajesh Kumar** - *Hsbc International - Analyst*

Good morning. Just in terms of the readout, when you think about the house (inaudible) by not expected to be GP at the time. So in order to be able to get, decent level of type, what are the main things you would like to highlight about the trial design that gives you the confidence that you'll be able to differentiate?

The profiles with fire from the (inaudible) or some of the pipelines at the start of the day. What were the main considerations you would like? It is you factored in when you talk about the presentation.

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**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

Dial in on another line because we can't hear you at all. It was very choppy. So if operator, if you can go to the next question and perhaps work with Rajesh to get him back into the queue. We just, we can't hear. It's very garbled.

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**Operator**

Steve Scala, TD Cowen.

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**Steve Scala** - *Cowen and Company LLC - Analyst*

Oh, thank you very much. I have three brief questions. First, are the dupilumab doses. Being used in Phase 3 identical to Phase 2? Second, given the three mechanisms of tilrekimig, are drug interactions hard to prove or disprove and how are you attacking that? And then lastly, do you know if agents that combine these same three mechanisms are being developed by competitors, particularly perhaps in China? Thank you.

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**Michael Vincent** - *Pfizer Inc. - Chief Inflammation & Immunology Officer*

I'll take the -- the first question was.

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**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

-- On the -- yes, the dose.



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**Michael Vincent** - *Pfizer Inc. - Chief Inflammation & Immunology Officer*

Dupilimab was not included in our Phase 2 study. So, there's -- we'll be using these labeled doses of dupilimab in the Phase 3 study.

**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

And then the DDI was the second question.

**Michael Vincent** - *Pfizer Inc. - Chief Inflammation & Immunology Officer*

I mean, DDIs, generally speaking, are not a significant issue in the context of biologics. So we're not anticipating DDI issues as a component of the of the program going forward.

**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

Editors and if anything's emerging.

**Michael Vincent** - *Pfizer Inc. - Chief Inflammation & Immunology Officer*

I personally do not know of any identical or very similar molecules coming out of China at the moment, but I would not at all be surprised if there are.

**Christian Antoni** - *Pfizer Inc. - Chief Development Officer, Inflammation & Immunology*

Thank you.

**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

Thank you, Steve. Operator, we'll go to the next one.

**Operator**

Jason Gerberry, Bank of America.

**Jason Gerberry** - *Bofa Merrill Lynch Asset Holdings Inc - Analyst*

Hey, thank you guys for taking my questions. One, just as you guys contemplate the trial design, realize you're not speaking to the stats plan, but just curious if you can speak to the trend in the field, I think what we've observed on at least EASI-75 kind of Phase 2 to Phase 3 effect size compression, kind of in that 8% range, so just wondering if we're in the right ballpark thinking about how single-agent activity could evolve going from Phase 2 to Phase 3.

And then my second question is that in the Phase 2 asthma trial, wondering -- I don't know if you think this is the right comp, but Sanofi's IL-13 / TSLP didn't look like it was much better than Tezspire on cross-trial comparison. So, kind of curious what underpins your confidence with your trispecific approach showing benefit over Tezspire? Thanks.



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**Michael Vincent** - *Pfizer Inc. - Chief Inflammation & Immunology Officer*

I'll take the second question first. Thanks, Jason. In our hands, the Sanofi molecule is not a particularly robust inhibitor of IL-13. So we think it's difficult to draw any firm conclusions about the mechanisms from that particular agent. And the first question?

**Christian Antoni** - *Pfizer Inc. - Chief Development Officer, Inflammation & Immunology*

Well, first question I'll take. I can take this digital question. If we would have an erosion and the response rate from Phase 2 to Phase 3, atopic dermatitis, not as good as psoriasis, but in general, it doesn't erode that bad like other diseases like rheumatoid arthritis, et cetera, when you look across the spectrum. So we hope that we, your 8%, we think it's a little bit smaller than that one.

And what I mentioned earlier about the placebo response, the quality control, we learned a lot in this Phase 2 trial. Atopic dermatitis is now a good way you really have to look at each site individually. We look at each patient individually with the pictures before we include them. So we are confident that going forward we can control these erosions.

**Jason Gerberry** - *Bofa Merrill Lynch Asset Holdings Inc - Analyst*

Very good. Thank you.

**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

Thanks, Jason. Operator, we'll go to the next one.

**Operator**

Michael Yee, UBS.

**Matthew Hagood** - *UBS AG - Analyst*

Great. Good morning. This is Matt on from Mike Yee from UBS. Thank you so much for the presentation.

I wanted to ask if you could just expand a bit on how you feel the incremental contribution of TSLP is here and what gives you confidence that the TSLP component is really adding something versus just kind of hitting IL-4 and IL-13 more strongly. And just thinking about that in the context of the historical TSLP trials in atopic term. Thanks so much.

**Michael Vincent** - *Pfizer Inc. - Chief Inflammation & Immunology Officer*

Thanks for the question, Michael. As I showed in the slides through the presentation with our skin explant data, we see, as you would expect, based on where the receptors are expressed across different subsets of cells, that in fact, we do see highly complementary pathways activated by the two different sets of cytokines.

In the case of IL-13, it's expressed very broadly, whereas TSLP is much more limited in its expression to immune cells. As you point out, the monotherapy efficacy in atopic dermatitis with TSLP has been underwhelming, but I think there's a clear signal with TSLP and understanding that all of the pathways that are blocked by 4 and 13 still leave behind a significant number of type 2 pathways that are still active, we think is a very strong biological rationale and consistent with the greater benefit that we've seen, at least compared to historical results.



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**Michael Yee**

Thank you.

**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

Thanks, Matt. Operator, we'll go to the next one.

**Operator**

Courtney Breen, Bernstein.

**Woody Polglase** - *Sanford C Bernstein & Co LLC - Analyst*

Hi, guys. Thanks for taking my question. This is Woody Polglase on for Courtney. Just a question on drug presentation. We noted on CT.gov you were running a phase one combining the drug with hyaluronidase. Just wondering why this is necessary if the drug is already subcutaneous. Is this due to volume of administration and patient pain, or is this broader PK/PD or something else? Thank you very much.

**Michael Vincent** - *Pfizer Inc. - Chief Inflammation & Immunology Officer*

Yes, thanks for the question, Courtney. We're always looking for the most patient-friendly formulation and the study that's on ClinicalTrials.gov is just an additional assessment that we're doing to try to understand how we can bring the most favorable patient-friendly formulation to patients when we launch.

**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

Thank you. Thanks for the question. Operator, I think it looks like we have one last question, so we'll take that.

**Operator**

Akash Tewari, Jefferies.

**Manoj Eradath** - *Jefferies LLC - Analyst*

Hey, this is Manoj on for Akash. Just one question. So how do you think about the dose response you observed in Phase 2 and how important is that dose selection going into Phase 3, especially when you are considering a subcutaneous formulation going forward? Just wondering about that.

**Michael Vincent** - *Pfizer Inc. - Chief Inflammation & Immunology Officer*

Thanks for the question, Akash. There's really not much more important than dose selection coming out of Phase 2, so we spend a lot of time thinking about that. I think in some of the slides presented today, you could see a clear dose response, particularly with the biomarkers, which is often the most faithful representation of dose response.

And we see a clear plateau and feel very confident about the dose we're taking forward.



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**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

Okay. Operator, it looks like there's one last one we can just popped up.

**Operator**

Nick Esposito, RBC Capital Markets.

**Nicholas Esposito** - *RBC Capital Markets - Analyst*

Hi, team. Thank you for taking our question. We were just curious on when the Stage 3 of the Phase 2 data could read out. We noticed the PCD date is actually today on ct.gov and if that will inform anything for the Phase 3 studies. Thank you.

**Michael Vincent** - *Pfizer Inc. - Chief Inflammation & Immunology Officer*

Thanks for the question, Nick. Typical time to lock databases and turnaround might be on the order of a couple months. We've already looked at interim data internally from that study, and it's met our expectations in terms of the response rate in patients who've been previously exposed to biologics. So we don't really anticipate any program changing data coming from that study.

Christian, would you like to add something?

**Christian Antoni** - *Pfizer Inc. - Chief Development Officer, Inflammation & Immunology*

Yes, to be specific, it will be presented at one of the next scientific congresses.

**Francesca DeMartino** - *Pfizer Inc. - Chief Investor Relations Officer and Senior Vice President*

Thank you, Nick. Okay, so with that being our last question, I first want to before we close it down, I want to come back to Risinger's questions on exclusivity just for everybody's benefit. So the patent applications are pending approval, but right now the US and EU5 date would be February of 2043 for exclusivity. So Dave, thank you for that question. Hopefully that answers it.

With that, I would like to thank Mike and Christian for their time today. And I'd also like to thank everyone for joining the webcast and to our analysts for their questions. I know there's a lot of interest in this program. If there's any remaining questions, the IR team is always available to receive your questions. So please reach out.

And with that, thanks for joining and we wish everyone a great day.

**Operator**

Thank you. This brings us to the end of today's program. We appreciate your time and participation. You may now disconnect.



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