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# EDITED TRANSCRIPT

PFE.N - Pfizer Inc Pfizer Pflash: A Spotlight on the PD-1 x VEGF Bispecific Antibody

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**OVERVIEW:**

Company Summary





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## PRESENTATION

**Operator**

Good day, everyone, and welcome to Pfizer Pflash, a Spotlight on the PD-1 x VEGF Bispecific Antibody. 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, Senior Vice President*

Thank you and good morning, everyone. I'm Francesca DeMartino, Pfizer's Chief Investor Relations Officer. On behalf of the Pfizer team, thank you for joining us for our 6th Pfizer Pflash webcast. Today's call will be recorded and will be available for replay on our IR website at pfizer.com.

As a reminder, our Pfizer Pflash series is intended to serve as an educational deep dive into our pipeline, products, and people. Each call will spotlight a specific product, therapeutic area, or growth initiative and give you an opportunity to hear from and interact with our business leaders.

Today's session will begin with a short conversation, followed by a live Q&A. As a reminder, this call is intended only for the investment community, including our sell-side analysts and institutional investors. If you're unable to join for the entirety of the event, you can find the replay available on our IR website.

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, which are all available on our IR website at pfizer.com.

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. With that, let's get started.

As you know, oncology is a key area of focus for Pfizer. We have recently announced the closing of a licensing deal with 3SBio for Global, excluding China rights to the bispecific antibody SSGJ-707 or 707 for short. Today, we will discuss why this agent is a strong strategic fit for Pfizer, the clinical data recently presented at ASCO and provide insight into the tumor types we believe 707 has the potential to be an important therapeutic option for patients, as well as a key driver of growth for our oncology business.

Before we move to the main discussion, let me take a moment to introduce our speaker, Jeff Legos, Pfizer's Chief Oncology Officer. In addition, Johanna Bendell, Chief Development Officer for Oncology; and Arati Rao, Thoracic Oncology Development Head, will participate in the Q&A.

Jeff, Johanna, and Arati, welcome and thank you so much for joining us today. Can you please start by introducing yourselves and giving a brief overview of your current role and experience, and Jeff, I'll turn it to you to start us off.

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**Jeff Legos** - *Pfizer Inc - Chief Oncology Officer*

Thank you, and it's a pleasure to join you today, Francesca.

I'm Jeff Legos, Pfizer's Chief Oncology Officer. I'm responsible for leading the end-to-end R&D for all of our cancer therapies, from pre-clinical through late-stage clinical development, including medical affairs. Prior to joining Pfizer earlier this year, I served as Executive Vice President and Global Head of Oncology and Hematology Development at Vardis. And prior to that, I was Vice President and Global Medicine Development Leader in Oncology at GSK.

Now I'll hand it over to Johanna.

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**Johanna Bendell** - *Pfizer Inc - Chief Development Officer, Oncology*

Thank you, Jeff.

I'm Johanna Bendell, Chief Development Officer for Oncology, and I'm responsible for Pfizer's strategy and execution of late-stage oncology clinical development. I also recently joined Pfizer, coming from Roche, where I served as Global Head of Oncology, leading Discovery and Early Clinical Development.

On to you, Arati.

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**Arati Rao** - *Pfizer Inc - Thoracic Oncology Development Head*

Thank you, Johanna.

I'm Arati Rao, Thoracic Oncology Development Head. I'm responsible for the oversight of clinical development strategies for all small molecules, ADCs, and immuno-oncology assets for lung cancers and head and neck cancers. I joined Pfizer via the acquisition of Seagen, where I served as Vice President of Early Stage Development, overseeing clinical development strategies for multiple ADCs and immunotherapy programs.

And with that, I'll turn that back over to Francesca.

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

Thank you for your introductions. Jeff, to get us started, can you tell us what 707 is and how it fits within Pfizer's broader oncology pipeline?

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**Jeff Legos** - *Pfizer Inc - Chief Oncology Officer*

Sure, 707 is a bispecific antibody that targets both PD-1 and VEGF. PD-1 is a receptor on immune cells that typically acts to prevent them from attacking cancerous cells. VEGF plays a key role in tumor blood vessel formation. When we look at this agent in the context of Pfizer's oncology established strategy, the fit is seamless.

First, it's a biologic, which is one of three core modalities where we have deep experience and industry-leading capabilities, including multi-specifics. Second, we have a strong focus and franchise presence in many of the cancer types where PD-1 x VEGF specific may have a significant impact, such as thoracic, genitourinary, and gastrointestinal tumors. And lastly, there's an opportunity to look at combinations with our industry-leading ADC portfolio, including the vedotin class of ADCs where we have a growing body of clinical data showing potential synergy with anti-PD-1 agents.

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

Thanks, Jeff. And beyond the strategic bit, can you provide an overview of why you chose to invest in 707, specifically, as well as the terms of the agreement?

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**Jeff Legos** - *Pfizer Inc - Chief Oncology Officer*

Absolutely. So first and foremost, I'm quite excited and we're confident in the mechanism of action. Anti-PD-1 and PD-L1 checkpoint inhibitors are amongst the most broadly impactful oncology drug classes in history.

The recent Phase 3 clinical data has shown that by combining PD-1 and VEGF inhibition within a single molecule as 707 does, there is a potential to achieve superior efficacy versus PD-1 inhibition alone. If this can be proven out in clinical trials alongside an acceptable safety profile across treatment settings and tumor types, we believe these results can be transformational for patients.

And looking at 707 specifically, we believe it has the potential to differentiate itself within the PD-1 and VEGF by a specific class. It's structurally distinct from other agents with pre-clinical data suggestive of best-in-class antiangiogenic activity as well as high affinity for PD-1.

Importantly, we are seeing this pre-clinical activity translate into the clinic with compelling Phase 1 and Phase 2 data demonstrating 707's anti-cancer activity as both a monotherapy and in combination with chemotherapy in a variety of cancer types such as non-small cell lung cancer and metastatic colorectal cancer. Based on these early results, we believe 707 has the potential to establish the cell as immuno-oncology backbone for multiple indications.

With regard to the deal terms, the license provides Pfizer with an exclusive global ex-China rights to develop, manufacture, and commercialize 707. In addition, this agreement provides Pfizer the option to extend the license to include exclusive development and commercialization rights for 707 in China.

In consideration of these terms, 3SBio receives a $1.25 billion upfront payment, a $100 million equity investment, and is eligible for up to $4.8 billion in milestones and tiered royalties on sales. We believe these terms strongly align with the value of 707 while leaving us well positioned to apply our global capabilities toward continued value creation for patients and for Pfizer.

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

You mentioned our global oncology capabilities is key to Pfizer's ability to create value. Can you speak briefly about these?

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**Jeff Legos** - *Pfizer Inc - Chief Oncology Officer*

Sure, as we show on slide 6 here, Pfizer Oncology has many capabilities that underpin both excellence and development of new cancer therapies as well as broad global expertise. Chief among these are deep expertise and global scale that collectively enable innovative clinical trial design, agile regulatory strategies, and accelerate the development of potential new therapeutic options for cancer patients.

The cornerstone of our expertise is our team. In particular, across our clinical development and medical organizations, we have more than 50 medical oncologists who bring unparalleled depth of clinical oncology experience and are driven to fundamentally change the trajectory of cancer diagnoses for patients around the world.

In our global scale, clinical operations is also key. We run our clinical trials in over 45 countries with 4,000 clinical research sites participating in our ongoing clinical trials, and we have 10 manufacturing and clinical trial supply sites across three continents. All of this allows us to execute clinical development with incredible speed. As an example, in our ongoing pivotal study evaluating sigvotatug vedotin in a second line non-small cell lung cancer study, we achieved our overall enrollment target of almost 700 patients in less than 15 months.

In addition, we have a robust pipeline of novel therapeutic agents. Currently more than 50 programs that span our three therapeutic modalities of small molecules, ADCs, and biologics, including multi-specific antibodies. Our pipeline and our portfolio of approved medicines each has a diversity in modality and mechanism of action, expands the range of potential combination treatment opportunities that we could explore with 707.

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

Thanks, Jeff. Speaking of multi-specific antibody development, could you briefly define what a multi-specific antibody is and then share some examples that highlight Pfizer's experience in this field?

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**Jeff Legos** - *Pfizer Inc - Chief Oncology Officer*

Yes, Francesca, and if we can move to slide 7, please.

Typical antibodies such as monoclonal therapeutic antibodies are those made by B cells in the body, selectively recognized and bind to a single target. In contrast, multi-specific antibodies are engineered biologics that selectively bind to two or more targets. Making all of this possible are the many advances in protein engineering technologies that have opened the door to design biologics with novel form and function.

Pfizer has a deep technical expertise in protein engineering and antibody design, which is crucially important as we evaluated 707 amongst the broader PD-1 x VEGF specific landscape. Shown here are a few examples of Pfizer's multi-specific antibodies. ELREXFIO is a bispecific antibody that recognizes and binds to B and T cells via BCMA and CD3 respectively and is currently approved in triple class exposed multiple myeloma.

In our I&I portfolio, we have two potentially first in class trispecific antibodies currently in Phase 2 development for atopic dermatitis. Our experience developing these multi-specific agents have provided us with added confidence in 707's potential to be successfully developed across multiple solid tumor indications.

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

We know that there are multiple PD-1 x VEGF bispecifics in various stages of development. How is 707 potentially differentiated?

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**Jeff Legos** - *Pfizer Inc - Chief Oncology Officer*

It's a great question. And if we move to slide 8, I'll walk us through some of the key characteristics of 707.

From a structural design basis, there's several ways in which 707 is differentiated from other PD-1 x VEGF bispecifics, including both the location and the geometry of the PD-1 and VEGF binding regions, as well as the FC region.707 specifically is a tetrabody or tetravalent antibody, which means it has four binding domains, two each for PD-1 and two for VEGF.

Each of the two arms of the antibody can bind to one VEGF in the tumor environment and one PD-1 on the surface of an immune cell. The relative location of these binding agents is not only differentiated but also provides some robust cooperative binding. This means that binding of one target enhances the binding of the second.

When 707 has bound VEGF, for example, in the tumor microenvironment, the strength of its binding to PD-1 is increased approximately 100-fold. This cooperativity enhances target binding and could potentially help drive synergistic anti-tumor activity.

This feature may also provide for activity that's localized to tumors due to the intrinsically high levels of VEGF, thereby potentially improving both safety and tolerability. Furthermore, because VEGF exists as a dimer, it can bind up to two molecules of 707, we believe this daisy chaining effect has the potential to augment the binding of T cells via PD-1.

If we talk about the Fc region of 707, it is also differentiated as is derived from IgG4, an immunoglobulin class with innately reduced ability to induce immune activation, thus potentially limiting undesirable inflammatory immune responses. Many other PD-1 x VEGF bispecifics utilize the FC region that require engineering to remove or reduce the potentially strong pro-inflammatory effective functions of other immunoglobulin classes.

If we take a holistic view of the existing preclinical data, we believe that 707 has potentially best-in-class antiangiogenic activity coupled with very high affinity for PD-1, all of which increases our optimism about the possibilities for clinical development.

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

Thanks, Jeff. If we could talk a little bit more about what makes the PD-1 x VEGF mechanism of action so compelling.

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**Jeff Legos** - *Pfizer Inc - Chief Oncology Officer*

Certainly, and if we can move to slide 9, please.

The clinical benefits of anti-PD-1 and anti-VEGF monoclonal antibodies as independent therapies have been well understood for some time. This, in turn, spurred investigation into their use in combination treatments. However, combining these agents produces a side effect profile that has hampered generally their broad use.

Intriguingly, engagement of PD-1 and VEGF by a single agent may not only lessen some of the side effects seen with the combination of anti-VEGF and anti-PD-1 therapies but also provide new biologic properties that could enhance anti-tumor activity. The PD-1 and VEGF bispecifics offer a potentially transformative mechanism of action by combining these two powerful well validated mechanisms into a single novel therapeutic agent.

The anti-VEGF activity inhibits angiogenesis or the growth of tumor blood vessels. Tumors have a very high energy requirement and produce chemicals that tell the body to expand blood circulation into the tumor in order to deliver nutrients that support rapid cell growth. If access to those blood vessels is taken away, cancer cells generally die and tumors shrink.

If we talk about the anti-PD-1 activity, sometimes referred to as immune checkpoint inhibition, this blocks the interaction of the proteins between PD-1 and PDL-1. Tumor cells often express the PDL-1 protein which can bind to PD-1 on immune cells and instruct the immune cell to ignore the cancer cell.

However, when this interaction is blocked, such as with anti-PD-1 antibodies, the immune cell can now recognize the tumor cell and therefore trigger an anti-tumor immune response. The anti-PD-1 activity is of particular interest to Pfizer as it has the potential for clinical synergy with our leading portfolio of vedotin ADCs.

Novel mechanisms of action are quite exciting, but the supporting data is really what drives value creation, and the field is already generating proof of concept data for the PD-1 x VEGF mechanism of action, specifically statistically significant improvements in progression through survival versus anti-PD-1 in monotherapy in Phase 3 studies across non-small cell lung cancer.

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

That's great. Thank you. Perhaps now we can transition to the clinical experience with 707 and the data that were recently presented at ASCO. Can you please walk us through those data and where you believe they can take us?

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**Jeff Legos** - *Pfizer Inc - Chief Oncology Officer*

I'd be happy to, Francesca. If we move to slide 10.

The data presented at ASCO is from a Phase 2 study conducted in China, where 707 was administered as a first line treatment in PDL-1 positive advanced non-small cell lung cancer patients. The study of design was fairly straightforward, enrolling patients with actionable without actionable genomic alterations, and ECOG status of either 0 or 1, and PDL-1 tumor proportion score or TPS greater than or equal to 1%.

2 cohorts of patients were enrolled corresponding to the two major non-small cell lung cancer histologies. So we included both non-squamous and squamous cell patients.

The study evaluated multiple ascending doses on a once every three-week schedule of administration. The primary endpoints of this study were both safety and an objective response rates. Data from the 10 milligram kilogram group showed particularly encouraging efficacy, and that's where we'll really focus today's discussion.

If we look at the efficacy from the 10 milligram per kilogram group shown here on slide 11, it is suggestive of both deep and durable responses. The depth of response is shown in the waterfall plot in the left panel. These plots represent tumor shrinkage from baseline with long -- longer downward bars reflecting larger decreases in tumor size.

These data correspond to a confirmed objective response rate of nearly 65%, or approximately two-thirds of the patients, with one patient pending confirmation of partial responses at the time of the data cutoff. The depth and the durability of responses is shown on the spider plot on the right. As with the waterfall plot, lines going down are better.

In addition, as you can see, over time, many of the lines continue to decrease further and ultimately stay there, providing an early glimpse around the durability and the depth of responses. The safety profile of the monotherapy 10 milligram per kilogram dose described on the next slide, was manageable.

The most common treatment related adverse events are shown on the left-hand side of the slide, with the maximum frequency being approximately 30% and only three of these having events of Grade 3 or higher. The aggregate frequencies are shown on the right, including Grade 3 plus treatment related adverse events which occurred in about 23.5% of the patients, and the treatment related serious adverse events in approximately 20% of patients.

Importantly, the treatment related adverse events leading to drug discontinuation were quite low, and there was no treatment related deaths. If we move to slide 13, please.

We were very pleased to see that the objective response rates and disease control rates or the fraction of patients who have stable disease or better, was generally comparable across patient subgroups at the 10 milligram per kilogram dose, inclusive of the challenging to treat squamous and PDL-1 low patient populations. In the left-hand side of the slide, you can see similar benefit rates across both squamous and non-squamous histologies.

Specifically, 75% of the patients with squamous cell disease and nearly 65% of patients with non-squamous disease experience a confirmed response. The disease control rates were very high for both histologies. This is encouraging because many therapeutic agents may perform well in one histology, but typically not in both.

Similarly, in the right-hand side of the slide or the right-hand panel, we see a similar benefit rate across PDL-1 expression levels, which are measured by TPS scores. These data are encouraging because patients with tumors having lower TPS scores are typically associated with lower response rates. Here in particular had response and disease control rates that were very close to patients with higher TPS scores. If we move to the next slide, I'll wrap up the discussion of the clinical data with a couple patient vignettes.

If we look at the top panel on slide 14, this shows sequential CT scans from a 50-year-old male with squamous metastatic disease and a TPS score less than 50%. One of the target lesions at baseline, as indicated by the red arrow. If we look out at week 6, the patient had achieved a partial response and by week 24 on the study, the patient's bilateral target lesions showed a net 76% reduction relative to the size at the start of treatment.

In the lower panel are images from a second patient. In this particular case, a 62-year-old male with Stage 4 disease and a PDL-1 TPS greater than 50%. In this particular patient, the tumor burden of target lesions, again, indicated by the red arrow baseline, was reduced by 54% by week 24 on treatment.

Here, it's also important to note that there can be a cavitary nature of responses in cancer such as non-small cell lung cancer. For example, if we look at the bottom panel, you can see that at week 6 of treatment following a reduction in the tumor size and opacity, there is what appears to be an empty bubble in the space previously occupied by bulk tumor.

Put simply, when a tumor in the lung dies or is killed by rapidly by a therapeutic intervention, it may leave behind what appears by CT scan to be a cavity with this black hole. However, the remaining cavitary lesions continue to be measured on the scan and compared to tumor size of baseline.

In other words, sometimes the measurement of lesion size by CT scan may not accurately reflect the extent of anti-tumor activity, and perhaps not even showing sufficient tumor shrinkage to be considered a response. This suggests that a focus purely on response rate may actually be underestimated the true degree of clinical benefit for 707 in non-small cell lung with cavitary lesions.

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

Thank you, Jeff. The data are certainly very encouraging. And as we wrap up, could you provide a brief summary of current 707 status and how you're thinking about near term prioritization?

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**Jeff Legos** - *Pfizer Inc - Chief Oncology Officer*

Absolutely. And if we can move to slide 15, please.

Look, myself, my colleagues on the call joining us today, and the rest of the development teams are quite excited to push forward and have been building out data-driven global clinical development plans for 707. All of this derived from the very encouraging Phase 1 and Phase 2 efficacy and safety data that has been observed to date as both monotherapy and in combination with chemotherapy in patients with non-small cell lung cancer and colorectal cancer.

But we look forward to providing some additional details and specifics over the coming months. I could share that our near-term objectives including include jumpstarting, global Phase 3 development in non-small cell lung cancer, and in other solid tumors. These are our top near-term priorities for 707.

We're also keen to explore 707 in multiple other tumor types and have already begun building up the data packages that will inform opportunities and indication where investment in future pivotal studies can add to the overall value narrative for patients and for the molecule. And of course, part of this development plan will be to look at the inter portfolio combination opportunities, particularly with some of our ADCs where we've been generating evidence that combinations with anti-PD-1 agents can deliver potentially practice changing results.

There's a lot we'd like to do, and we're excited to jump in and really get going. We then move to the next slide, please.

To put a finer point on the topic, here on slide 16, we framed that at a high level some of our initial thoughts on the potential development opportunities for 707. We've also overlaid this with US epidemiology data to give you an overall sense of the potential addressable patient populations. These include opportunities that directly map to our key franchises, specifically thoracic, genitourinary, and gastrointestinal cancers.

However, we believe that these represent just the tip of the iceberg in terms of the cancer patients who may potentially derive benefit from 707, and we're looking forward to evaluating 707 not only in these cancer types, but others as well.

All of this conveys that there are significant new opportunities that we believe have the potential to reinforce Pfizer as a continued leader in the development of innovative treatments for cancer. We're looking forward to sharing updated development plans, clinical data, and when relevant, our plans for commercialization.

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

Thanks, Jeff.

To summarize our conversation today, I would say that 707 fits perfectly into Pfizer's oncology portfolio and strategy. Pfizer has a proven track record in developing multi-specific antibodies. Some are currently in development, and some, like ELREXFIO, are already on the market. Furthermore, the target applications for 707 align with our current therapeutic areas in oncology.

Second, this licensing deal demonstrates continued execution of Pfizer's business development objectives that are aiming the current $10 billion to $15 billion deal capacity as drivers of growth for the end of this decade and beyond.

Third, we're encouraged by the Phase 2 data that were recently communicated at ASCO. Based on the totality of Phase 1 and Phase 2 data, we believe that 707 has the potential to become a backbone treatment option for multiple indications in the future, subject to clinical trial and regulatory success.

Lastly, as we communicated previously, we are working on a detailed development program to unlock value for 707, including pivotal Phase 3 studies and multiple indications. These studies, along with earlier stage trials, will evaluate both single agent 707, as well as in combination with other agents, including Pfizer's leading portfolio of ADCs. We will begin to communicate some of these details later this year. And as we mentioned in our press release, Pfizer plans to manufacture drug substance for 707 in Sanford, North Carolina and drug product in McPherson, Kansas.

We'll now begin the Q&A session with Jeff, Johanna, and Arati. As a reminder, our Pfizer Pflash series is designed as an educational deep dive into our pipeline programs. I'll therefore kindly ask participants to keep questions focused on 707 and the data and opportunities discussed today. And to avoid those 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. Thank you for your understanding.

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

## QUESTIONS AND ANSWERS

**Operator**

(Operator Instructions)

Geoffrey Meacham, Citi.

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

Hey, guys. Good morning. This is Nishant calling for Geoff. Thanks for taking our questions and your useful presentation.

I wanted to ask about the development plan. Do you believe that the upcoming summit therapeutics data from Harmony trial is a getting factor in determining how broadly your -- which indications you would develop this molecule further?

And then second, you mentioned that you know you plan to this molecule with some of your ADCs. Maybe can you highlight a couple of agents that you think have strong potential to be developed in synergy with this molecule? Thank you.

**Jeff Legos** - *Pfizer Inc - Chief Oncology Officer*

Now you're very welcome, and maybe I'll start and then I'll pass it over to Johanna.

I think as I mentioned during my presentation, right, I think these two mechanisms of action have been very well validated in some of the biggest practice changing impact on patients across a variety of cancers. If we look specifically at the PD-1, PDL1 checkpoint inhibitors, there's almost 30 approved indications just with the monotherapy alone, in addition to what you had mentioned with the combinations that I've already undertaken within summit. So this gives us a range of different cancers where we could potentially explore this based on pretty well validated, biology and immuno-oncology principles.

And in terms of combinations, we already have some great experience combining our vedotin ADCs with Pembrolizumab and urothelial cancers, but that's just the tip of the iceberg. But maybe Johanna, I'll turn it over to you to share a little bit more as to how you're thinking about the landscape and how you would develop this molecule.

**Johanna Bendell** - *Pfizer Inc - Chief Development Officer, Oncology*

Thanks so much, Jeff, and I completely agree with you. I think we take the totality of data that's out there not only from what we're seeing with other by specifics that touch PD-1 and VEGF, but also in other areas where we've seen activity of PD-1 inhibitors and VEGF inhibitors, and these will help flush out our clinical development plan.

And including the data that we have with our urothelial cancers with PADCEV as a potential combination ADC, we also have some great data that we're excited about with sigvotatug vedotin and PDL1V whereas Jeff mentioned, we've also looked at these in combination with Pembrolizumab and look forward to seeing how these will also work with the 707 molecule knowing that we also have data with these agents in non-small cell lung cancer in particular.

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

Great. Thank you. Operator, if we could move to the next question, please.

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**Operator**

Evan Seigerman, BMO Capital Markets.

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**Malcolm Hoffman** - *BMO Capital Markets - Analyst*

Hi, I'm Malcolm Hoffman on for Evan. Thanks for taking our question.

You noted PD-1 binding affinity that increased hundredfold in the presence of VEGF. Can you provide some context as to whether other PD-1 x VEGF agents you undoubtedly reviewed prior to the deal also demonstrated this increased affinity, or is this increased affinity or is the magnitude higher than any other agent you have looked at? Thanks.

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**Jeff Legos** - *Pfizer Inc - Chief Oncology Officer*

Thanks for the question, Malcolm.

Obviously, we're here today to sort of highlight the profile of 707 specifically, and we're quite excited about this sort of 100-fold increase in binding activity that we see for our particular bispecific. Can't really comment much on the other competitors in the marketplace.

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

Thanks, Jeff. Operator, let's go to the next one, please.

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**Operator**

Tim Anderson, Bank of America.

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**Tim Anderson** - *Bank of America - Analyst*

Thank you. A couple of questions, if I can.

So you say you're highly confident in the molecule in the approach, can we take that to mean that you're also highly confident that the upcoming ASCO and summit data this year will show a clinically meaningful survival benefit in lung and not just that big PFS benefit that it's shown? I'm guessing your answer would be yes.

And then a second question, just realistic launch timing ballpark in your first indication, ex-China, what would that be? Thank you.

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**Jeff Legos** - *Pfizer Inc - Chief Oncology Officer*

Thanks for the two questions, Tim.

And yes, I would reiterate kind of the statement that I made during the presentation. Based on the totality of the data that we've seen, 707 and within the bispecific PD-1 x VEGF class, that definitely gives us the confidence in the molecule and the overall approach.

With respect to speculations on a case of summits, overall, survival, you could ask them the specific question. But what I could say about our studies is we would design them with the appropriate power and design to look at both overall survival as well as progression-free survival.

With respect to the earliest possible approval and launch timelines, it's probably something that we'll share a little bit more details later on this year as we sort of take everyone through our overall development plan.

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

All right. Thanks, Tim, for the question. Operator, we'll go to the next one, please.

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**Operator**

Trung Huynh, UBS.

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**Trung Huynh** - *UBS AG - Analyst*

Hi, guys. Thanks for putting this on. Just a couple on the data from me.

So first on the dose response in that ASCO data, we did see the 20-milligram and 30-milligram doses underperform the 10-milligram on efficacy. I know you're not moving forward with these higher doses, but do you have a theory on why they underperformed? Is this due to lower dose intensity? I think you also noted that there was one unconfirmed response at the time of that data. Is that now confirmed?

And then second, 3SBio shared some chemo combo data in non-small cell lung cancer in their corporate deck that had an ORR of 58% in that first line non-squamous lung population with the 10-milligram. That seems below that ASCO data in the monotherapy where you showed 65%. Just what do you think drove this delta? Thank you.

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**Jeff Legos** - *Pfizer Inc - Chief Oncology Officer*

Thanks for the question, Trung.

And maybe just in general, as the molecule has been looked at, across a range of those levels, both below 10 milligram per kilogram and above10 milligram per kilogram. Maybe I'll ask Arati to comment specifically on the dose response as well as any updates that we are able to share on the follow up for the unconfirmed response as well as any initial early insights on the chemo combination. So Arati, over to you.

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**Arati Rao** - *Pfizer Inc - Thoracic Oncology Development Head*

Thank you, Jeff.

So the observation that the that the 20 and the 30 milligram per kilogram was slightly lower than the 10 milligram is correct. And we are -- there's some PK data and dose intensity that's being worked on, but we are looking to see, working with the agency to see whether the 10 milligram per kilogram would be the right dose to move ahead with.

In terms of the combination data that we -- that you've seen in the deck from them with in non-small cell lung cancer, this is true. The combination data is looking encouraging both in squamous and non-squamous first-line non-small cell lung cancer, and we're looking to present this in the future in some coming meeting -- in some upcoming meetings.

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

Okay. Thank you. Time for the question. Operator, we'll go to the next one.

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**Operator**

Steve Scala, TD Securities.

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**Chris LoBianco** - *TD Securities - Analyst*

Thank you. This is Chris on for Steve. We had a couple of questions on the diligence process.

How many PD-1 x VEGF assets did Pfizer conduct in-depth diligence on before deciding on 707? How challenging was it for Pfizer to get comfortable with data generated only in China? And what were the three most important questions during your diligence process for 707? Thank you.

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**Jeff Legos** - *Pfizer Inc - Chief Oncology Officer*

Thanks for the question.

Yes. Obviously, we can't comment on our overall kind of BD&L process, per se, but obviously, we did an in-depth due diligence including visits to 3SBio in China to specifically make sure that the mechanism of action, the pre-clinical data, the clinical data that was generated thus far and presented in the public domain as well as the manufacturing processes, were robust and met Pfizer's standards to take on such a deal like this.

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

Okay, thanks for the question. Operator, we'll go to the next one. Thank you.

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**Operator**

Mohit Bansal, Wells Fargo.

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**Mohit Bansal** - *Wells Fargo Securities LLC - Analyst*

Great, thank you very much for taking my questions and thank you for this presentation. I have two questions.

One is, so your competitors have talked about formation of Daisy Chain, and that could be the reason why they are seeing probably better efficacy and better safety. Given your structure, do you think that is -- that could be an issue here, or you, like, how should we think about that? That's number one.

And number two, it does seem that in the combination prior trials with the PD-1 combinations, VEGF toxicity was an issue. So to that extent, higher potency of VEGF, do you think there could be detrimental effect there and how should we think about the higher potency in the context of a VEGF PD-1 bispecific here? Thank you.

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**Jeff Legos** - *Pfizer Inc - Chief Oncology Officer*

Thanks, Mohit, and I think we covered a little bit during the presentation around kind of the unique structure and the benefits that we see in terms of both high affinity and the role that we believe that the Daisy Chain can play. And I'll let Arati comment a little bit more specifically regarding that, as well as share a little bit more detail on data regarding the safety, and the adverse events that have been observed today. So, Arati.

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**Arati Rao** - *Pfizer Inc - Thoracic Oncology Development Head*

Yes. Thanks, Mohit.

So the -- I agree, I think the mechanism of action was a key piece in figuring out the kind of molecule that we wanted to go after, and I think the engagement of both dimers of VEGF and the Daisy Chaining is an important feature of molecule is a tetrabody. But we do have preclinical evidence that suggests that the mechanism of action in which it engages VEGF and the cooperative mechanisms of engaging VEGF and PD-1 are similar or better than some of our competitors.

So that's all I can say. I can't give more data and provide more than that.

The second piece with regards to the AEs, specifically the VEGF related adverse events that we see with this class of agents, I would say that the AEs were comparable to the what we're seeing with the other agents in this in this class. This was in the Phase 2 data, I will say that the patients that were included in the study probably were more like the real world.

So for example, patients who were on anticoagulants, patients who had large cavitary lesions around large blood vessels were included in the study. And thus, we feel like we will be working with the agency and working with KOLs to come up with the right criteria as we go ahead to design our trials and ensure safety of patients.

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**Johanna Bendell** - *Pfizer Inc - Chief Development Officer, Oncology*

Thank you so much, Arati.

And also, to talk a little bit about why would we be excited about increased VEGF activity with this particular molecule is one thing that's very special about these PD-1 x VEGF bispecifics is the way that the binding occurs. It occurs primarily in areas where we have high concentrations of VEGF, which would be more localized to tumors. So we're excited about the possibility that we could have increased antiangiogenic activity and at the same time have it more localized to the tumors, which we are hoping translates into an even better toxicity profile.

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**Trung Huynh** - *UBS AG - Analyst*

Awesome. Thank you.

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

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

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**Operator**

Terence Flynn, Morgan Stanley.

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**Terence Flynn** - *Morgan Stanley - Analyst*

Great, thanks so much for taking the question. I guess it's kind of two-part.

Can you just talk about how you think about translatability of the ORR data and also safety tolerability in a Chinese population versus going to US, European population. Any puts and takes that you considered as you thought about mapping that data out and getting confidence that what we're seeing in a Chinese specific population across a number of these agents will translate through to similar results in European population. Thank you.

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**Jeff Legos** - *Pfizer Inc - Chief Oncology Officer*

Well, thanks for the question, Terence.

And obviously, when we think about, ORR it's not in isolation. And if you look at both the waterfall plots, and the swimmer plots or spider plots that were shown, what kind of gives us the confidence around the translatability is depth and durability of those responses.

And if you go one step further as you think about future time to event endpoints, stable disease is also a very important clinically relevant response here for these patients if we're talking about TFS or ultimately for overall survival. I think for this particular kind of class of drugs, we've already seen early data where that it does translate from patients ex-US or China to US and globally around the world. And as we think about the next steps of our development plans, those studies will be designed as global multinational, pivotal registration directive trials.

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

Thank you. Operator, looks like we have one question left and we'll take that final question, please.

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**Operator**

Rajesh Kumar, HSBC.

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**Rajesh Kumar** - *HSBC Securities (USA) Inc - Analyst*

Hi. Can you hear me?

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

Yeah, we can now. You were a little faint. Yeah, we can hear you were a little faint and it's coming. Go ahead.

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**Rajesh Kumar** - *HSBC Securities (USA) Inc - Analyst*

Thank you very much for taking the question.

So the first question is on the sequencing of the development program. You clearly have shown a lot of ambition and various indications that you would develop. Can we just go through which -- what do you need to see in your first development program that you can give a green flag to the others?

The second one is just on the timelines. When can we realistically see start of Phase 3 trials in approval?

And finally, just in terms of thresholds versus (inaudible) or the BioNTech Bristol program, you'll be potentially the third to the market. So I'm assuming you want to come up with a competitive profile. You said you're powering the trials appropriately to tell benefit, but what sort of benefit do you aim so that it becomes competitive enough to your peers who are slightly ahead of you in the curve? Thank you.

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**Jeff Legos** - *Pfizer Inc - Chief Oncology Officer*

Thanks, Rajesh. A lot of great questions and most of those, obviously, we can't answer. But I'm glad that you acknowledge both the ambition and the enthusiasm and looking at the early kind of development program and the sequencing of those.

Maybe I'll ask Johanna to comment a little bit more where she can regarding anything that we need to see or have already seen that has given us the confidence to move forward with this development plan. So Johanna?

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**Johanna Bendell** - *Pfizer Inc - Chief Development Officer, Oncology*

Thanks so much, Jeff.

And I think what gives us the confidence in how we're moving forward is what we actually saw within the due diligence and data that's already been shown of this molecule with both as a single agent and combination in it with chemotherapy. I think we have a lot of confidence in areas that we want to clearly play first, including non-small cell lung cancer. But I think also as we continue to move forward, we will be doing some signal seeking, as well as in other tumor types that will help us further design where we are and how we're going to invest in more of the pivotal studies as we move forward.

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**Jeff Legos** - *Pfizer Inc - Chief Oncology Officer*

I would just conclude with, Rajesh, right, if we think about kind of some of the other reasons why this is such a great strategic fit for Pfizer, I think it comes down to scale and speed, right? So if we use the most recent non-small cell lung cancer trial for SV in second line plus non-small cell lung cancer patients, right, that trial was enrolled in less than 15 months for 700 patients.

If we think about scale and depth of expertise, we operate in more than 40 countries. We have 4,000 clinical sites and we have a range of expert oncologists around the world which, I think, gives us the ability to sort of move very quickly as a multinational sort of Phase 3 pivotal program to help reduce the time to market.

Francesca, maybe I'll turn it back to you for any concluding remarks.

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

Yeah, it actually looks like we've got Trung from UBS with a follow-up question. So Trung, we'll take you and then we'll -- operator, if you could turn it back to me and then we'll close it down.

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**Trung Huynh** - *UBS AG - Analyst*

Sorry, just a very quick one. So apologies here. It just looks like there was a death in the 20-milligram arm in the ASCO data at 20 months, just wondering what drove that. Thank you.

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**Jeff Legos** - *Pfizer Inc - Chief Oncology Officer*

Arati, any particular comments to Trung's question regarding...

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**Arati Rao** - *Pfizer Inc - Thoracic Oncology Development Head*

Yes. The death in the 20-milligram patient, it was a patient from -- it was a patient who had passed away from a lung -- who had had prior chemo radiotherapy to the lung and to the lesion and had come on to the study quickly after and then had some hemoptysis and other complications. So this is the kind of patient who probably would not have entered a clinical trial over here given the recent radiation to that site, and there's usually a washout period that we have between radiation and getting onto our studies. So that was the reason for some bleeding and other complications in this patient.

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

Okay. With that, I first want to just thank my colleagues, Jeff, Arati, and Johanna, for taking the time to do this today. And then secondly, thank you all for joining. I know the invitation was short notice given we just announced the closing, but I want to thank you for joining, as well on a summer Friday.

And lastly, we will be hosting our next quarterly earnings on Tuesday, August 5. We will see you then and we'll pause on these Pfizer Pflashes to get through earnings and then we're -- we will come back to them in the fall. Hope you're all enjoying your summer and we will talk to you in a couple weeks. Thank you so much.

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**Operator**

This does conclude today's program. Thank you for your participation. You may disconnect at any time.

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