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EDITED TRANSCRIPT

PFE - Pfizer Inc at Citi Global Healthcare Conference

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PRESENTATION

Andrew Baum - *Citigroup - Analyst*

So, I think we're going to start the session, so if everyone would like to take a seat, it's the last session of the last day. So, from here on in, it's home run.

So, look, thank you for joining us today. Delighted to introduce our guest speaker for this keynote, Mikael Dolsten. Mikael is President of Pfizer R&D, as many of you will know. We've also got Chuck Triano and colleagues who run the investor relations team with us in the audience today. I know Mikael wants to stay to give a brief overview, and then we'll just segue to the Q&A. So, thank you for joining us.

Mikael Dolsten - *Pfizer, Inc. - President of Worldwide Research & Development*

Thank you very much for inviting us here. Like always, we start with forward-looking statements that include cautionary language about our presentation. You can find a copy of the slide at our filings and our webpage.

So, we wanted to just have a kind of a quick glimpse, as we have look at our track record since 2010/2011, to here, to today. As we have kind of focused R&D on what we think are the most productive areas, we have built a portfolio with a mix of small molecule, biologicals and vaccines.

And I highlight to you some of the, about 20 key approvals since that starting date, of which about half are new medical entities. On average, three approvals per year, one to two of them being NMEs. And you note Ibrance, our leading novel cell cycle inhibitor for hormone-receptor-positive breast cancer. Prevnar 13, the number one vaccine in the world, including more recently adult indication, next to the previous infant. Inlyta, second-line renal cell carcinoma. Xalkori, the [already] poster child for treatment with precision medicine in lung cancer. Xeljanz, the first launched JAK inhibitor for inflammation diseases -- RA. Eliquis, the preferred-by-cardiologist novel oral anticoagulant for stroke prevention; venous thrombosis.

And to the right, two of the recently-acquired drugs we've either marketed for some indication like Xtandi in prostate cancer -- metastatic prostate cancer, and crisaborole, which is in late registration phase, PDUFA date early January next year, for atopic dermatitis.

When you look at the pipeline that deliver this flow of Phase 3 and approvals, we have about 90 projects, slightly more than 40% in Phase 3 registration, slightly less than 40% in Phase 1, and about 20% in Phase 2. About two-thirds are NMEs, and about 50/50 are small molecules versus biologicals. And that really is quite aligned with the goals that we set a few years ago.

2016 has been a productive, robust year for us. We added a second indication for Ibrance in US, the recurrent breast cancer, in February. And we got approval for both advanced and recurrent breast cancer in Europe, making it clearly the class leader in both continents.

Xalkori was approved for a second indication, ROS-positive non-small cell lung cancer, and we're actually exploring a third indication, MET-positive lung cancer, in our clinical development. We added a once-a-day formulation of Xeljanz. And Prevnar 13 got approved for infant in China, expanding it to a very important market for future use in prevention in pneumococcal disease.

We are in submission phase for a few different drugs. Inotuzumab and Mylotarg are two classical antibody drug conjugates for liquid tumors, both in filing Phase 4 US and Europe. Inotuzumab has also Breakthrough designation.

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In immuno-oncology, we were able to move our PD-L1 inhibitor to the first filed indication, Merkel cell carcinoma, where we have both Breakthrough status and Priority Review. And finally, we expect, project end of this year, ertugliflozin, to be filed in three different forms together with US Merck -- monotherapy, fixed dose with Januvia, and fixed-dose combination with metformin.

So, looking at the earlier to mid stage of the portfolio in 2016, I think we had a quite good momentum in building up a portfolio with a number of very meaningful and some really transformational drug opportunities.

In Alzheimer, we have now focused on secretion inhibitors and we have clinical Phase 1 data that are favorable, showing potency in reducing amyloid for both BACE and the first gamma secretase modulator that has a design and activity in clinical studies.

The other component in Phase 1 that I wanted to highlight is, to the best of my knowledge, the most advanced dual TYK2/JAK1 -- really interesting profile based on what we learned with Xeljanz. In particular, TYK2 extend activity to inhibit IL-12, IL-23 like cytokines, which we think will be critical for improved profile in diseases like psoriasis, Crohn's, alopecia areata, et cetera.

I'm very pleased to share with you that our C. difficile vaccine, which is a unique, high-technology, genetically-engineered two-component vaccine, has reached positive proof-of-concept criteria showing a very high percent of individual reaching the bar for meaningful immunogenicity levels against two toxins, and the levels reached were regarded as very high in a large number of individuals.

Recently, at ASH, we communicated that glasdegib, a SMO hedgehog inhibitor, had favorable data in a subset of AML. And we have shared, I think, six different Phase 3 studies in ertugliflozin at top line level, as we prepare for submission.

Xeljanz is now reaching its second step, going into new regions and new indications. Tofacitinib, the active ingredient, reported strong data in ulcerative colitis, psoriatic arthritis, and we are in advanced registration phase for the RA indication in Europe.

You can also see some of the early-phase assets. P-cadherin bifunctional antibody, a really novel agent which is now the number 10 immuno-oncology NME in our pipeline in the clinic. A JAK1 inhibitor dosed in atopic dermatitis -- we think it has a profile that would hit very nicely the IL-4, IL-13 cytokines that monoclonals have shown to be very important in that disease.

And then avelumab that I mentioned briefly for Merkel cell carcinoma, started six different Phase 3 across solid tumors, including one additional combo study with Inlyta for renal cell cancer. We think Inlyta has a really interesting profile as a targeted agent together with I/O compounds, as shared earlier when we combined it with Keytruda from US Merck.

Very briefly, when it comes to internal versus external, we really have our strategic ambition to be a networked R&D organization -- some strong capabilities internally, welcoming biotech and pharma partners, and you can see three example of that approach. Product acquisitions through M&A -- Medivation, access to Xtandi, talazoparib, among other things; Anacor, which was the company providing crisaborole, the topical PDE-4 inhibitor.

Next to that we have strategic acquisition of technology and early products. Bamboo is a company engaged in gene therapy, and provided to us what we think is the most comprehensive manufacturing of clinical gene therapy material in the United States, and also two projects for entering into neurological disease gene therapy scheduled 2017 and 2018 for first in humans.

And then a partnership with Western Oncolytics that provides us with an oncolytic virus, particularly interesting for cancer therapy of what we call cold tumors, with moderate to low immune activity.

Finally, Bind, as a small technology acquisition, provides us with nanoparticle technology. And very timely, we had a partnership with them exploring targeted agents in nanoparticle for prostate cancer. One of those agents seems to do very well, preclinical combination studies with Xtandi, so we were delighted as these two acquisitions very timely coincided.

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I will conclude on looking at what's next when it comes to novel-science medical entities in 2017 and 2018. We are taking the next step in immuno-oncology, moving the field from monotherapy to doublets and triplets, and you can see there is a whole cohort, what we think are really unique doublets and triplets with opportunity to be industry-leading in this next step of immuno-oncology. We expect up to three gene therapy clinical trials going in this time period; about 10 different Phase 2 studies with next-generation JAK inhibitors, across derm, rheum, and GI segments.

And with vaccines, I'll end and say, not just C. difficile that we will now plan in regulatory discussions to move to Phase 3 during the first part of 2017; we hope at the end of 2017 or possibly early 2018 to have Breakthrough-like design of an RSV vaccine that we think is unique and can be really important. And finally, we are now in clinical phase with a next generation of pneumococcal vaccine, covering up to 20 different serotypes.

I conclude on that, and thank you for your attention.

QUESTIONS AND ANSWERS

Andrew Baum - *Citigroup - Analyst*

Thank you, Mikael. So, maybe, before we get into a conversation about the individual assets in your portfolio, help us understand how you're thinking about building out Pfizer oncology from an organic basis, from an R&D function. Because within your business you have a vaccines part, a small molecule part, a biologic part. You have Rinat on the antibody engineering. And yet you're dealing, from an immunotherapy perspective, if we believe all roads lead to Rome -- a highly complex system to understand why some tumors are non-immunoresponsive; how to make other tumors. And it's going to involve carefully tying together across the organization lots of different parts.

So, are there any structural or organizational changes that you think are required, or do you feel the current separation that exists within Pfizer is something that is going to stay and is surmountable?

Mikael Dolsten - *Pfizer, Inc. - President of Worldwide Research & Development*

You know, you can never be sure about the future, but I would say I think we have a very well-aligned strategic structure. And let me say why. So, in La Jolla, we have our center for small molecule design in cancer. And it's the group that made all these great targeted agents, from Xalkori to the follow-on lorlatinib, to Ibrance, and are actually working right now with a next-generation cell cycle inhibitor that will address some of the resistance to molecules like Ibrance after the patient's been exposed for years, that we hope to have in the clinic within 12 to 15 months.

In the San Francisco area, we have our specialized immuno-oncology group that have worked on the recent 4-1BB and OX-40 checkpoint modulator, that brings us into what we think is leading doublet and triplet.

Going back to south San Francisco, we have our cancer vaccine, door to door with our small-molecule vaccine. And the cancer vaccine group has over many years built a significant capability for two platforms. One is a multi-antigen vaccine that is co-administrated with CTLA-4 antibody locally, and likely a PD1 Pfizer antibody systemically. It's now in a trial for prostate cancer, and we see early interesting signals. And we also have the oncolytic viral platform that I alluded to, through a partnership.

The third group, that is in Pearl River, outside New York, has deep expertise in functional genomics and help us to identify future targets that can augment activity of molecules like Ibrance, and can act in synergy with checkpoint blockade. There also are our skill center for antibody drug conjugates and nanoparticles.

So, I hope you got the sense that each of them have unique skills. Each of them are embedded in an academic network in either southern or northern California or in the metropolitan New York, that has been actually instrumental for us. For example, our genomics activity in Pearl River has been in close partnership with Cold Spring Harbor Laboratory, and we're pulling up some new targets that we're doing chemistry on now that we think can give us, in the future, epigenetic and cancer metabolism targets that, when combined with Ibrance, improve the long-lasting cell-killing

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substantially. So, this is the type of network we have put together to tap into the best of science, United States, but doing it in a complementary, rather in an overlapping way.

Andrew Baum - *Citigroup - Analyst*

So, moving down to the products, and if we take the premise that the next 10, 20 years are going to belong to immunotherapy, if not much longer, and the question is how you get more patients to respond, one area which I know Pfizer has a legacy of investment in is antibody drug conjugates. And inotuzumab, which is now resurfacing, but I know went through a period of investor disappointment, is obviously the most advanced of those. But I know you have a raft of interesting early-stage molecules, and we were talking about the PTK7 target before we came on. So, perhaps you could talk to what you have -- the advantages, tolerability, efficacy -- and then we can sort of segue into how we then leverage that through combinations with checkpoint blockade.

Mikael Dolsten - *Pfizer, Inc. - President of Worldwide Research & Development*

Yes. It's a great question. So, we have noted in recent chemo checkpoint combination studies that checkpoint modulators by themselves do pretty well in tumors that are what we call hot -- have high level of PD-L1, maybe more than 50% of patients are positive. While in corresponding similar lung cancer patients that are low, their efficacy is much more modest. Chemotherapy seems particularly in these cold tumors, be able to cause cell deaths and damage that leads to a subsequent immune response. And by combining chemo with immune blockade, you can get probably the best of the two. We thought that, going forward, systemic chemo will, over time, be a bit of the past.

And that's why we are quite excited about our antibody drug conjugate, because they carry, in a targeted manner, with better tolerability, chemo into the tumor. And I think the data you mention that you observed with PTK7 showed substantial response rate in very advanced ovarian cancer in the 30% range. Please remember, checkpoint inhibitors in this space would show about half, at most. We know that the key with checkpoint inhibitors is the long-lasting durability.

With antibody drug conjugates, you get good response rate, better tolerability than systemic chemo, but you are not likely to get that long-lasting response. What we have seen with PTK7 and similar construct in preclinical studies is that tumors that are really cold -- you find almost no immune cells -- quickly become enriched immune cells -- go into this immunogenic cell death. And that's why are very excited at the moment.

We are just in the final design phase, how a PTK7/PD-L1 type of trial will look like, and whether we go with PD-L1 only or, in additional studies, doublet of immune checkpoint inhibitors. We do think, whether you use ADCs or nanoparticles that are targeted to tumors, filled with selective agents that will have combined anti-tumor effect and favorable immune modulation on the microenvironment -- we think that is a really interesting game for cold tumors. While, for the hot tumors, we think you probably will do well with just double or triple I/O/I/O blockade.

So, I hope that gave you a sense for how strategically we have placed our best, and just -- and for the type of tumors where you have a very poor ability to evoke immune response. Usually tumors that have low degree of mutational burden, which is required for immune response -- those are the tumors that we will go with our CAR T platform and also with our bifunctional antibodies, because you circumvent the need for this mutational burden. And this story is really how we think at Pfizer we have built an immuno-oncology platform that will in the future have an ability to target a very comprehensive group of tumor patients, and possibly the most comprehensive available at this time point.

Andrew Baum - *Citigroup - Analyst*

So, on a similar vein but through a different route, increasing number of patients who are immunoresponsive -- you've obviously now taken Xtandi into your portfolio, and you touched upon mutational burden and neoantigens. I didn't see on your slide an intent to initiate a trial combining with checkpoint blockade, but of course many of your peers have done that -- Astra, Merck, and others. How are you thinking about that, and where you would target it -- any thoughts would be interesting.

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Mikael Dolsten - *Pfizer, Inc. - President of Worldwide Research & Development*

Great question. And I'll be brief, as these agents are new in our portfolio. We have noticed, there was actually a study with Xtandi and a PD1 inhibitor that did show added value. It was a small study, so this is an area we're now looking actively into -- how our checkpoint inhibitors could work with a drug like Xtandi for prostate.

I also wanted to mention that talazoparib, that was part of the Medivation acquisition, is used particularly for tumors that have high mutational burden as a PARP inhibitor. And we think that asset will do extremely well with our immuno-oncology portfolio. So, that's another area where we're looking actively, what would be the best strategies for both those two asset.

Andrew Baum - *Citigroup - Analyst*

And in terms of -- this is, again, picking up a conversation before we started, but perhaps expanding on the dual faces of chemotherapy. Because as -- to one end, you may increase epitope spreading. You may cause immunogenic cell death. But on the other hand, you're creating subclonal populations of mutated -- mutations in tumor cells which may in fact move you away from having a favorable effect to chemotherapy. Now obviously, we haven't seen the data. But just interested in how you're thinking about that, selecting patient populations using diagnostics in order to try and address that.

Mikael Dolsten - *Pfizer, Inc. - President of Worldwide Research & Development*

Yes. I think it's a great question, and that's why I think there could be some concern to use chemo/I/O for tumors that are already immunogenic, where you just need to release the brakes for PD1. There is enough of immune cells available, and you may not want to cause additional mutations at this stage. While for cold tumors, you really need to evoke immune response.

I think, over time, there will be more sophisticated approaches than chemo. That's why we're investing in a disease where we can deliver a much higher payload in the tumor, because mutations particularly appear when the amount of chemo is insufficient, and that's also why we added this nanoparticle platform, where you can load it with one or two targeted agents, which may be a better way over time to go than traditional chemo.

Andrew Baum - *Citigroup - Analyst*

Aside from avelumab, you have CD137 -- 4-1BB. You have OX-40. So, perhaps on OX-40, we have obviously saw some early data from Roche in combination with their PD-L1, which was underwhelming, frankly. So, whilst acknowledging differences between molecules and so on, as your scientists have looked at that data and thought about what to take home, what was their conclusions about whether it was dosing, scheduling, indication, or molecular? I'd be interested in that.

And then, just whilst I'm just on the other checkpoints, on CD137, notably it doesn't have the hepatotoxicity which Bristol has. Is that solely due to the lack of or the diminished effector function, or is there other differences on the idiotype end which make us more likely to contribute to the differential safety/efficacy?

Mikael Dolsten - *Pfizer, Inc. - President of Worldwide Research & Development*

A great question, and one of my favorite topics, so thank you for bringing it up. Both our 4-1BB and our OX-40 are designed as IgG2 antibodies with reduced risk for cell-mediated cytotoxicity, but with great agonist properties. So, we think they optimize for signaling through the cells to augment and sustain PD-L1-induced immune responses.

We have combined our 4-1BB with avelumab, and earlier, we combined it also with Keytruda. And data so far, which is early data -- we had a little bit more advanced with the Keytruda, but data that accumulates suggests to me that this is a productive combination, good tolerability, and seen

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to do better than the mono PD1/PD-L1 from emerging clinical data. I would like to see more, to be able to conclude where and how to use it. But I'm encouraged, what I've seen.

OX-40, as you've said, showed mainly disease stabilization, some signal responses, but also confirmed some biomarkers that it was having a different profile than 4-1BB. So, we are planning early next year -- you always want to have a good start of the year. Then we are ready to dose the first patient with a triplet of avelumab, 4-1BB and OX-40. We think it's the -- from a rational immune design, and from preclinical studies, it has all the bells and whistles to take the brake, augment cytotoxic and helper cell immune cells to play against the tumor.

The second kind of doublet/triplet platform we have is avelumab/4-1BB with IDO1. We are right now in clinical studies with our IDO1 inhibitor that we co-discovered with a Belgian biotech company, and we think for some tumors that will be a very interesting triplet. We're right now in mono and moving to doublets. Hopefully end of 2017, early 2018, we will have triplets. I think both those triplets will be led by Pfizer. That's our aspiration. And to the best of my knowledge, we are the only company that has internal access to these four different agents -- avelumab, 4-1BB, OX-40, IDO1.

Andrew Baum - *Citigroup - Analyst*

And presumably, I mean, when I look at that first triplet you've mentioned, you've got two active co-stimulatory molecules and a checkpoint. Presumably, at dosing, you're going to start very, very, very low, because of the tox risk. So, that will take a while to find the dose before you can -- is that the right way of thinking about it?

Mikael Dolsten - *Pfizer, Inc. - President of Worldwide Research & Development*

It's a very thoughtful question. We are planning the final pieces here. I don't think we need to start that low, because we have doublet data on both of them -- doublet of various combination of avelumab, 4-1BB and OX-40. Each of them have shown good tolerability. So, I don't think we need to start at some ultra-low, and should be able to dose-escalate. But in principle you're right. There are a number of dose step you need to take. But we certainly hope during 2017 to be able to get an early read on the clinical activity across the spectrum of tumors, and then move into dose-expansion cohort in specific tumor types.

Andrew Baum - *Citigroup - Analyst*

And your ICOS molecule -- is that in the clinic, or about to go into the clinic?

Mikael Dolsten - *Pfizer, Inc. - President of Worldwide Research & Development*

The one that's next to go into the clinic for us is likely going to be a GITR molecule. It's going to be likely in the clinic, second half of 2017. It adds to our armamentarium and offers another opportunity next to OX-40 to target the regulatory component of the immune system.

Andrew Baum - *Citigroup - Analyst*

And this is from Rinat.

Mikael Dolsten - *Pfizer, Inc. - President of Worldwide Research & Development*

It's also from Rinat.

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Andrew Baum - *Citigroup - Analyst*

Got it. Okay.

Mikael Dolsten - *Pfizer, Inc. - President of Worldwide Research & Development*

Rinat leads our checkpoint modulators and also together with Cellectis have a capability for CAR T cells that we're producing there. As you know, we're on clinical studies with our UCART19. It's an allogeneic platform, so you don't need all these cumbersome one patient, one harvest, one infusion. You can create an off-the-shelf product. The second indication that we hope in 12, 15 months or so to go into will be in myeloma, with another CAR T. Again, allogeneic; unique; likely first in industry for both of them.

Andrew Baum - *Citigroup - Analyst*

Segueing away from immuno-oncology just for a second, can we talk about CDK4/6?

Mikael Dolsten - *Pfizer, Inc. - President of Worldwide Research & Development*

Yes.

Andrew Baum - *Citigroup - Analyst*

Where obviously you are the industry leader and you -- with a -- clearly you have made a huge impact already on the disease. The competition is maturing, now, with Lilly and with Novartis. I'd like to get your impression of how much of a competitive threat from a scientific/safety/efficacy point of view you see ribociclib. Because frankly, we saw a drug which has got hepatotoxicity, a QTc, and is late. But I'd be interested in whether you've seen hepatotoxicity anywhere approaching what was seen in the clinical trial with Ibrance. There's no QTc; we know that.

And then if you could comment on Novartis' comments that they believe they will be first to market with a Faslodex combination. How much do you think that's really going to change the competitive landscape, given that Faslodex may become more the gold standard for management of these patients, and remind us where you are with a Faslodex combination.

Mikael Dolsten - *Pfizer, Inc. - President of Worldwide Research & Development*

Yes. So, I think you summarized ribociclib better than anyone else when you said it's late into the market, with liver and cardiac rates reported as adverse events. That's me quoting you --

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Andrew Baum - *Citigroup - Analyst*

Happy to help.

Mikael Dolsten - *Pfizer, Inc. - President of Worldwide Research & Development*

Not me stating it. Our drug has been now used by 40,000 breast cancer patients. It, based on PALOMA-2 and 3, has really good efficacy. It's well-tolerated. Patient seems to report good life quality. There has been some monitoring for neutrophils, but clinically, the patient seems to have few problems, and we haven't seen these type of issues that you reported from Novartis as being part of our profile to any significant extent.

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We think that the molecule has been out in the market, very well perceived by physicians and patients. Established market share across first and second line, exceeding 40%. And it gives us a very strong position. We are leaders when it comes to early breast cancer, which is adjuvant therapy to cure patients, or possibly delay the risk of recurrence, where we have two studies going -- PENELOPE and PALLAS, that will report around 2020.

Then we have combination studies in breast with a PI3K inhibitor. We're running a few studies in other tumor types -- head and neck; pancreas; subset of lymphoma, that we expect some data to share in 2017 and 2018. And then I earlier mentioned that we hope within 12 to 15 months or so to have another cell cycle inhibitor that can deal with resistance that inevitably unfortunately occur when patient's been on targeted drugs. And we have also developed insights on how CDK4/6 inhibitor move cancer cells to senescence, but they take a long time before they go from ageing to cell death, and we have identified the vulnerabilities and are planning to also address that with additional drug.

So, I just did that journey to give you a sense of, clinical, extremely strong position. Breast cancer -- early breast cancer, going beyond breast, and future combinations based on our insight. Coming back to Faslodex, which is the name for fulvestrant, we actually had data on fulvestrant in our PALOMA-3 study, while our PALOMA-2 was based on letrozole. And Ibrance combined very well with fulvestrant.

Andrew Baum - *Citigroup - Analyst*

But in terms of a registrational trial with a Faslodex backbone?

Mikael Dolsten - *Pfizer, Inc. - President of Worldwide Research & Development*

If I -- I think Faslodex is the brand name for --

Andrew Baum - *Citigroup - Analyst*

Yes, it is. But you -- so, then, do you have --

Mikael Dolsten - *Pfizer, Inc. - President of Worldwide Research & Development*

Fulvestrant. That's in our label.

Andrew Baum - *Citigroup - Analyst*

It's in the label. Fine.

Mikael Dolsten - *Pfizer, Inc. - President of Worldwide Research & Development*

The data in recurrent breast cancer is including fulvestrant, the AstraZeneca intramuscular-given drug.

Andrew Baum - *Citigroup - Analyst*

But that was the drug used in the trial?

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Mikael Dolsten - *Pfizer, Inc. - President of Worldwide Research & Development*

That was the drug used in the PALOMA-3 trial. Letrozole, used in PALOMA-1 and 2. We have additional trials looking at other hormonal blockade including, compared to chemo for certain patient type, a study called PEARL.

Andrew Baum - *Citigroup - Analyst*

So, we're going to be running out of time very shortly. But one question I wanted to ask is, given Christmas is around the corner, and when you're thinking about your wish list for 2017 in terms of business development, what areas are you thinking in terms of either platforms or therapeutic areas? I mean, you can be as -- I'm sure you won't be specific, but any indication you could give of where you think you would like to bolster your organic competence, as I'm sure the audience would find interesting.

Mikael Dolsten - *Pfizer, Inc. - President of Worldwide Research & Development*

Yes. Okay. Originally I was thinking to just have a less ambitious Christmas gift, and maybe as a new year wish, think about what drugs to the portfolio. On one hand, I would say we will continue to have appetite for our strategic investment areas. Right now, I think we have a very comprehensive immuno-oncology portfolio.

But we would always be open, if there is a new agent appearing. You may have seen that we have invested recently in novel CTLA-4 construct that are aimed to be local active in the tumor to circumvent the systemic tox seen with tremelimumab. We have two collaboration, OncoImmune and BioAtla. So, that will be an example of novelty strong checkpoint modulators that do better when active locally than systemically, and CTLA-4 is one example.

We have invested quite a lot in gene therapy. We have spectacular data together with our partner, Spark. Please check out the ASH presentation. It's just spectacular -- nine patients where you can give them synthetic factor IX that makes them independent of any further infusion, while they previously were dependent on infusions.

And we're going to Duchenne's muscular dystrophy. I would love to see us being able to change fatal outcomes for boys with that disease. And we'll certainly keep our eyes open if there is another gene therapy asset, as we have one of the, I think, best manufacturing facilities for clinical studies.

Maybe I should end to say that there are certain new science area. One is NASH in the metabolic space. We have -- you can see on my slide, we have two assets in clinical phase, a KHK inhibitor and an ACC inhibitor. Certainly that's, I think, a novel, exciting space -- one of the few areas of metabolic disease where you could come to the market without big outcome studies.

And I think it would be a disappointment if I didn't mention that you'll always be open on your eyes for breakthroughs in neurological diseases like Parkinson and Alzheimer. In the end, it's going to be a combination therapy, maybe new agents that deal with neuroinflammation, which seems to be a major culprit. That would be nice new year's gift.

So, I'm open in the corridor for anyone that have good suggestion how we make the new year's gift list come true for Andrew and myself, and we'd love to have good tips. Thank you for listening in today.

Andrew Baum - *Citigroup - Analyst*

I don't think I can say it better. Listen, many thanks, Mikael, for joining us. Thank you for the audience today, and if you have any follow-ups, please don't hesitate to reach out. Many thanks.

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Mikael Dolsten - *Pfizer, Inc. - President of Worldwide Research & Development*

Very pleasant to talk to you. Thank you.

Andrew Baum - *Citigroup - Analyst*

Likewise.

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