ASTRAZENECA

Moderator: Pascal Soriot
December 17, 2015
13:00 p.m. ET

Operator: This is conference # 8984139.

Operator: Welcome, ladies and gentlemen, to AstraZeneca's conference call on the majority stake investment in Acerta Pharma.

Before I hand over to AstraZeneca, I'd like to read the Safe Harbor statement. The Company intends to utilize the Safe harbor provisions of the United States Private Securities Litigation Reform Act of 1995. Participants on this call may make forward-looking statements with respect to the operations and financial performance of AstraZeneca.

Although we believe our expectations are based on reasonable assumptions, by their very nature forward-looking statements involve risks and uncertainties and may be influenced by factors that could cause actual results to differ materially from those expressed or implied by these forward-looking statements.

Any forward-looking statements made on this call reflect the knowledge and information available at the time of this call. The Company undertakes no obligation to update forward-looking statements.

I'll now hand over to Pascal Soriot.

Pascal Soriot: Thank you, Operator. Thank you, everybody, and good morning, good afternoon. Thank you for joining us today on short notice.

As we update you on our majority stake investment in Acerta Pharma.

Hopefully, you've had a chance to download the presentation we've provided. As a reminder, it's available on the investor section of our website.

On slide 2, you have the forward-looking statements. We read that to you already. So, I will invite you to move to slide 3.

today, I'm joined by Marc Dunoyer, CFO; Luke Miels, the Executive Vice President for Global Product and Portfolio Strategy, Medical Affairs, and Corporate Affairs; also, Sean Bohen, our Executive Vice President for Global Medicines Development and our Chief Medical Officer. As many of you know, Sean is a hematologist. So, this is an area close to his heart.

Also for the Q&A session, we're joined today by Mondher Mahjoubi, who is the head of our oncology product strategy group; Suzanne Galbraith, who is the head of research and early development at AstraZeneca Oncology; Nina Mojas , who is the head of New Products and business development in oncology; and also, we have a guest today with us, Jesse McGreivy, who is the Chief Medical Officer of Acerta. And today, Jessie is sitting in our office, together with Sean Bohen for this Q&A.

Today I'll provide a short overview of the investment before handing over to Luke who will outline the market opportunity, followed by Sean who will provide more detail on acalabrutinib. Marc will then go through the financial terms and I will conclude before we will open up for your questions.

So, if you move to slide 4, please. As outlined in the press release this morning, today we have entered into an agreement to acquire majority ownership of Acerta Pharma, giving us access to a potential best-in-class irreversible small molecule oral BTK inhibitor, acalabrutinib.

Acalabrutinib is expected to transform the treatment for B-cell malignancies, offering a potentially more effective chronic treatment option with limited side effects. This underpins our expectation of potential peak-year Product Sales in excess of $5bn.

As a late-stage potential medicine, acalabrutinib is in line for initial regulatory submission in the second half of 2016 and has an extensive development plan underway.

We will buy a 55% stake now for $2.5bn with $1.5bn deferred and also an option to acquire the remainder of Acerta in the future for ~ $3bn. This investment supports our return to growth and completes our transformation in Oncology. Finally, the investment complements and is synergistic with the Celgene partnership.

So, if you move to slide 5, you will remember that AstraZeneca is focused on three areas, based on our capabilities from biologics through to devices including small molecules.

We believe we have a very defined set of capabilities that set us apart in the industry. Personal healthcare is also a very critical part of our strategy and underpins all our efforts. Close to 80% of our pipeline is associated with the personal healthcare strategy.

This is also the lens for our targeted approach to business development as we seek to strengthen our pipeline and portfolio and create maximize value from our strong R&D productivity.

So, the agreement today is a clear evidence of our commitment to further focus the portfolio and to strengthen our broad capabilities. As we move forward, we will become even more focused on three therapy areas that are really getting critical mass and becoming very substantial. And it is very clear now that we will be a leader in each of those three categories in the years to come.

This will continue to be important in 2016, as we sharpen our focus on progressing key medicines within our three therapy areas. So, if you move to slide 6, the investments we are making today represent a major step forward in the completion of our oncology strategy and the transformation which as you are aware focuses on four areas of cancer: breast cancer, ovarian cancer, lung cancer, and haematology. AstraZeneca has a strong heritage in developing

cancer medicines and we believe oncology has the potential to be transformational for our future.

So, with this agreement, we are boosting a key area in our comprehensive oncology portfolio with the late-stage potential best-in -class medicine that could transform treatment for patients across a range of blood cancers as well as potentially some solid tumors and immunology too.

We have a clear strategy in the area of hematology. The investment in Acerta Pharma provides another best-in-class medicine which means that AstraZeneca now has cornerstone medicines within each of our four key areas of oncology.

Moving on to slide 7, the investment we are announcing today provides our Company with a small molecule presence in blood cancers to complement our existing immunotherapy based focus and our collaboration with Celgene, as well as offering potential innovative treatment options for solid cancer.

So, being able to benefit from the substantial expertise within this complex area of medicines enhances the expertise we already benefit from through our work with Celgene in immune-oncology.

Again, I'd like to repeat that our collaboration with Celgene is very important to us and they know hematology and we certainly are very committed to it.

So, with this, I will ask you to move to slide 8, and I will hand over to Luke, who will take you through the next section of the presentation.

Luke Miels, AstraZeneca plc - EVP, Global Product and Portfolio Strategy, Global Medical Affairs, & Corporate Affairs

Thanks, Pascal. So, clearly, we're very excited about this opportunity and the potential for a best-in-class asset. If we look at the overall background and the market itself, starting with hematology, you can see here the chart on the left is a combination of the sales today in hematology compared to solid tumors, the more common solid tumors, such as breast, prostate, and lung.

The sales today are comprised of the products in the table on the right-hand side, dominated by rituxan. And then, what's interesting and what was really attractive to us is if you look at the top of each bar, the relative growth opportunity that these segments offer.

Next slide. And specifically looking at CLL, there's a number of factors which are driving this which caused us to spend quite a bit of time looking at this opportunity and ultimately reached the conclusion that we've just announced.

So, the background medical need is substantial. Liquid tumors and specific diseases like CLL are disease of the aging. The background epi is still growing. These patients are complex. Many of them have existing co-morbidities, which limit treatment options to physicians.

But there is a demand for effective treatments with good tolerability. What was very interesting as we conducted our background diligence and market research; talking to physicians there was a very clear pattern. People were very excited about the BTK class. They're impressed with the efficacious nature of the product. The convenience of the oral regimen and also the potential to remove chemo for the first and potentially the second line of treatments.

And then, the opportunity to treat these patients for a significant portion of time.

And that's reflected in the chart on the right, which is a work by decisions resources independent of AstraZeneca. You can see the market today is dominated by antibodies. There's the presence of chemo and therapies are just under a quarter of the business as it stands in 2014.

However, looking forward, you can see that TKIs dominate the market and that's largely taken up by the BTK class.

Next slide. And the drivers for that are several. The first is this shift to using the BTK class earlier in first line. You can see that this first line population is growing over time. There's also rapid uptake of the first generation BTK in second line and significant penetration in first line already without the label.

And what's interesting of course is that you see this substantial shift in treatment duration from a relatively short six months with the antibody/chemo combo to a regimen which is approaching three years.

And again this pattern became very, very clear to us that if we're going to have a regimen in all the patients who are somewhat compromised then we would be looking for an asset, a compound, such as acalabrutinib which had strong efficacy, but also a very promising side effect profile.

And so, with that, I'll hand it over to Sean.

Sean Bohen: Thank you very much, Luke. I'll ask to be on slide 11, please.

As Luke mentioned, inhibition of BTK has revolutionized the treatment of CLL and other B-cell malignancies. That has also changed the treatment pattern for the disease from a short course of chemotherapy perhaps with targeted agents, to a very long duration of treatment.

The other thing we know about BTK inhibition is that maintaining presence requires continuous dosing and continuous suppression of BTK signaling. As a result, interruptions or dose reductions can lead to fast disease progression or the development of resistance to BTK inhibition.

In addition, in non-Hodgkins lymphoma, where combinations are required for treatment, often with chemotherapy agents with significant side effects, tolerability is a clear issue in order to make the combination more viable and achieve superior efficacy.

On the right-side of the slide, we highlight some of the tolerability issues that have been identified and are emerging with the current BTK standard of care. These include significant side effects such as afibrial fibrillation, rash, bleeding and bruising; as well as pain in the joints and muscles, which can be significant enough to lead to discontinuation of therapy.

We believe that acalabrutinib with its superior specificity will be able to address many of these issues.

If we could go to slide 12, please, slide 12 summarizes a publication on some of the rates and causes of discontinuation of the first-generation BTK inhibitor. What you can see is that over time, as the graph moves to the right, the number of patients who have to discontinue the medication increases. I'll point to some of the specific causes.

If we look at the red line, that's a relatively vague description of other event, but contained within many of those reasons for discontinuation are some of the tolerability issues that I have discussed previously.

If we look at the green line, this is a progression of CLL due to a transformation to a more aggressive disease, large cell lymphoma. This is called a Richter's transformation.

And then, the black line over time is progression of CLL therapy. This is the sign of the development of resistance.

Slide 13, please. I'd now like to turn to acalabrutinib itself. Acalabrutinib is a highly specific irreversible second-generation BTK inhibitor with best-in-class potential. About 1,000 patients have been treated to date with acalabrutinib, more than 600 of these patients receiving it as mono-therapy. It has better overall tolerability profile and low rate of discontinuations.

We have also seen a very low rate of richter's transformation in hundreds of patients treated to date.

On the right side, we describe some of the attributes that we believe will contribute to increased tolerability and improved efficacy. As I mentioned, acalabrutinib is selective. It is irreversible. It has a short half-life limiting side effects. Twice daily dosing allows for very potent target inhibition. It is better tolerated. And in combination, this leads to a lower rate of discontinuation and a potentially longer treatment duration.

If we could go to slide 14, please? Slide 14 summarizes recent data on acalabrutinib's efficacy in release refectory CLL. This data was presented earlier this month at the American Society of Hematology meeting and simultaneously published in the New England Journal of Medicine.

On the left side, you can see the response of patient treated over time. What you can see is that the response rate progressively increases over time. Many responses initially are a partial response with lymphno cytosis, meaning that the masses from the CLL shrink but you have lymphcytes floating in the blood. And what you can see is that over time that lymphcitosis goes away and you're response consolidates.

On the right side is the progression free survival that is seen in this group of patients. And I think that's quite remarkable evidence of efficacy and durability over time.

If we can go to the next slide? This is slide 15. This summarizes the heamatologic key points in hematology development program for acalabrutinib.

There's a quite broad CLL SLL program including two phase III trials. I will note as well that one of these trials is a head-to-head trial of acalabrutinib and in relapsed and refractory CLL. There are also programs in other B-cell malignancies including the diffused large B-cell lymphoma, follicular lymphoma, mantle cell lymphoma, multiple myeloma, Waldenström macroglobulinemia, and non-Hodgkins lymphoma.

We believe that regulatory submission for acalabrutinib will be possible as soon as the second half of 2016.

Moving on to slide 16, please. In addition to hematologic malignancies, there is emerging evidence preclinically supporting efficacy of BTK inhibition in solid tumors. This slide summarizes some of the ongoing clinical trials that are examining BTK inhibition with acalabrutinib in a variety of solid tumors, both in combination with immunotherapy and with chemotherapy.

In addition, there is evidence that BTK inhibition may have effect in inflammatory disease and this is being investigated in rheumatoid arthritis.

Slide 17, please. I'll now summarize development of acalabrutinib to date. There have been approximately 1,000 patients treated so far. This is across

multiple indications and includes some inbrutinib intolerance patients. More than 600 patients have been treated on monotherapy, with the remaining patients treated in various combinations.

The tolerability and initial efficacy data support the case for best in class BTK inhibition with acalabrutinib.

There are multiple potential registration approaches included in this development plan. Outside of hematologic malignancies, initial data in solid tumors is expected in 2016 and we plan for initial regulatory submission in the second half of 2016.

With that summary, I would now like to hand over to Marc.

Marc Dunoyer: Thank you very much. I'm going to describe in the financing terms for this transaction. First of all, AstraZeneca will acquire a 55% equity stake. There will be an initial payment of $2.5 billion. Then, there is a second payment which is deferred to the approval in the United States or 31st of December 2018 whichever is sooner. There is also a set of options for the Acerta shareholders to sell and for AstraZeneca to buy the remaining 45% equity for an additional but approximate $3 billion.

The completion of this transaction is expected at the end of the first quarter 2016, obviously subject to customary closing conditions.

The transaction itself will be accounted for as a business combination, which means that we will fair value the totality of the payments that are anticipated and the deferred consideration will be on the balance sheet also.

And finally on this slide, the funding of this transaction will be done by cash and by debt.

I turn to next page, which is on slide 19. we see this as an opportunity. It's a late stage development medicine with an expected launch in 2017. Potential sales in excess of $5 billion.

In terms of impact on our earnings, we see this as moderately dilutive to core EPS in the near term. We confirm once again a progressive dividend policy and we will provide on the 4th of February 2016 with the announcement of our results for 2015 our guidance for 2016.

With this, I will now hand back to Pascal.

Pascal Soriot: Thank you, Marc. So, if we can move to slide 20, I'd like to conclude by repeating that we continue to make good progress across all our strategic priorities, keeping us on track to deliver on our targets and that today 's agreement with Acerta, we are boosting a key area in our oncology portfolio with this late-stage potential best-in-class medicine that could really transform the treatment of a range of blood cancers but also has potential as highlighted by Sean in solid tumors and possible even beyond cancer in certain auto immune diseases.

So, we are really looking forward to working closely with the Acerta team. We have the highest respect for this team who have done a tremendous job over the last few years developing this tremendous medicine. We share the same passion for science, and we share the same passion for developing medicines that make a difference for patients. And Acerta will be an autonomous center within the broad AstraZeneca group of companies and will be considered a center of expertise in hematology.

Before I hand over to your questions, I'd just like to say I know it's been a busy year for everyone and I hope you have a restful and enjoyable festive period and I'd like to thank you again for joining us today, because I know this is a period of time when everybody is winding down and leaving for the Christmas break.

Before we take questions, I would like to remind everyone to ask one or two questions only so we can hear from everybody. We can always do another round if needed. Thanks a lot.

And now, back over to the Operator.

Operator: James Gordon, J.P. Morgan.

James Gordon: I'll do two questions, then, please. One is I think you're already running studies of ibrutinid with PDL1. I'm just wondering how much data you'd already seen for that combo? And was that a significant driver of the decision to get your own BTK?

And the other question was just a clarification on filing timelines. If I read correctly, you're filing in H2 of next year. What further data are you going to have by then? And what are the timelines for other filings? How much further out would other filings be?

Pascal Soriot: Maybe Sean, do you want to take his questions, knowing that there's some limit to how much we can disclose. James will appreciate this. But Sean, go ahead.

Sean Bohen: I'm going to start with the ibrutinid question which I think you're asking. And just to summarize, yes, we do have an ongoing research development collaboration around ibrutinib and durvalumab. That's in an early stage.

We are very excited about the potential to combine BTK inhibition with immuno-oncology. The choice to get acalabrutinib into the portfolio was really based on BTK inhibition and on the attributes of acalabrutinib itself.

So, the filing question. I think what we're going to do is we're not going to get into the details of which indications and what data the filing package will actually look like. We're studying a lot of hematologic malignancies with acalabrutinib. Acerta started, as I said, a broad programme and we think that there are some nice potential opportunities within that coming in the second half of next year.

James Gordon: Thank you.

Pascal Soriot: Simon Baker, at Exane. Simon, do you want to ask your questions?

Simon Baker: Just a couple of very quick ones on the structure of the deal itself. I wonder if you could tell us the proportion of the $2.5 billion that is in new debt? And also, just to be clear, while you have a call on the 45% of Acerta you don't

currently own, I just wanted to check whether Acerta has a put to you on that stake?

And then, secondly, on acalabrutinib itself, I wonder if you could confirm the IP protection on that? It looks like the patent coverage is until 2033, pre-patent registration. But I just wonder if you could confirm that for us?

Pascal Soriot: You are correct on the IP front.

On the call-put option, Marc, I'll ask you to comment. And on the structure of the deal, the debt. we basically raised debt recently and we're just going to cover this acquisition with a mixture of cash and the existing debt. But there's no new debt, right Marc? Over to you.

Marc Dunoyer: On the funding first off, as I said earlier on, it will be funded by cash and short-term debt. We're not planning to raise long-term debt for the time being.

You had a second question on the options. So, Acerta will have a put option on us, and we will also have a call option. So, there are put and call options which are of course dependent on the approval of the molecule and also on its commercial success.

Simon Baker: Great.

Pascal Soriot: Seamus Fernandez, at Leerink. Seamus, go ahead.

Seamus Fernandez: Just a couple here. The first question really is can you just sort of update us on the requirement around the $1.5 billion? So, more just kind of the scenario this is a non -- my understanding is this is a contingent payment based either on time or regulatory success. But the 2018 payment, will that $1.5 billion be paid out regardless of the potential outcome? So, worse case scenario would be a major safety issue emerges and that $1.5 billion would still have to paid out. So, I'm just trying to get a sense of what that contingency is.

And then a separate question is the acalabrutinib is also being studied in combination with pembraluismab. And I'm just kind of wondering how you

guys have your own PD1 in clinical development. I'm just wondering if that were to show a positive efficacy, would your interest be in studying this in combination with durvalumab or perhaps with your internally developed PD1? Because I think that might have implications for the collaboration with Celgene. So, I'm just trying to get a sense of how all of those things might interact with each other.

Pascal Soriot: Seamus, the first question, it's not actually a contingent payment; it's a deferred payment. And that payment will come either on approval of the product or alternatively at the end of 2018, regardless of approval. So, it's a deferred payment, not a contingent payment.

Seamus Fernandez: Okay.

Pascal Soriot: As far as the second question, I'll ask Sean to also comment in a few minutes. But I guess a couple of points I would make would be that we want to maximize the utility of acalabrutinib and therefore we are certainly supportive and committed to this development. The product will have to be used with a variety of other medicines in combination and certainly whatever makes sense to combine with acalabrutinib, we will consider.

And the other part of the question, the other comment I will make is that certainly we would also consider combinations with our own PD1 and the collaboration we have with Celgene is not with PD1; it's with PDL1.

So, Sean, anything you want to add?

Sean Bohen: it's a great question. I'll just say that this will be a data-driven set of decisions. And one of the extraordinary things about this particular deal is it gives us such great access to acalabrutinib and to be able to broadly combine; you've mentioned PD1 you've mentioned PDL1. We have a rich immune-oncology portfolio with other targets, and so, we are now in an excellent position to be able to explore those different options.

The decision as to how to move forward and what will be a data-driven decision based upon how we do in the clinic and how well we are able to make an impact on these diseases and these unmet needs.

With regard to the Celgene collaboration, that is, as Pascal said, durvalumab focused. And really does ask a different and complementary set of questions in hematology. So, we remain committed to our collaboration with Celgene, which is going quite well.

Seamus Fernandez: And if I can just follow up, the question was if you would move forward your internally developed PD1 which is not a part of the Celgene collaboration?

Sean Bohen: We haven't made specific plans for which combinations we're going to move forward right now.

Seamus Fernandez: Okay. And that was more the question, that you have a PD1 that's earlier in development versus durvalumab.

Sean Bohen: Yes, we do.

Pascal Soriot: Let me just make a sort of a general comment on this one, and the general comment is that we highly value our collaboration with Celgene. We really respect Celgene. We have a very good relationship. We will not do anything that would jeopardise this collaboration. The collaboration with Celgene is central to our strategy in hematology. So, there is nothing we will do that will threaten this. With everything we do will be in full collaboration with Celgene and fully synergized.

Seamus Fernandez: Perfect.

Pascal Soriot: We can move to Alexandra Hauber, at UBS. Alexandra, go ahead.

Alexandra Hauber: Congratulations on this transaction. Can I just ask whether this acquisition was the result of any sort of competitive process at all? Or, given the cultural fit you have alluded to, this was more like an exclusive negotiation?

Also, when you look at the competitive environment, I can see a handful of other early-stage BTK inhibitors specifically in hematology another handful in inflammatory conditions. How well -- how much data do we have of those

compounds that you can be sure that we're truly dealing here with a best in class molecule?

And finally, Sean, could you please in relatively simple terms explain the immune modulatory effects of BTK inhibition and whether this is potentially negative with the suppressive effect on MK cell activity and action, which we don't see with ACP-196?

Pascal Soriot: So, Sean, I don't know if there is a subliminal message; I guess in the request to explain "simply." but the last two questions will be yours to explain the immuno aspect and also the best-in-class aspect.

As far as your first question, Alexandra, this type of asset is always attractive to other companies and of course there were other interested parties in the discussion.

The one thing I would say is that this is a process that we've been working on for months and months. We established hematology as a key pillar in our oncology strategy a long time ago and we've been looking at what we could do in hematology, what would make sense for us, what kind of differentiated medicine we could bring on board.

And the discussions with our friends at Acerta started quite some time ago.

So, this is a result of not only process but also a process of building trust and aligning cultures and making sure we share the same view as to what we wanted to achieve as companies but also for this product.

Sean, over to you.

Sean Bohen: First was a question around data that has been seen, I think to support the best-in-class possibility. And I'll talk about two things. One is the tolerability of acalabrutinib from the data we have to date and the best thing to refer you to the is the New England Journal publication.

But the primary thing that was seen with acalabrutinib was headache, which was mild to moderate controlled with analgesics in the vast majority of cases.

And there was a decreased incidence of bruising and bleeding. Atrial fibrilation has been very low in the experience to date. I talked a little bit about richter's transformation.

I just remind you for some context that CLL is largely a disease of age. These are older patients, often with comorbidities and multiple medications that they're taking. So, we really believe the pathways to best in class are being able to maintain the treatment duration for longer in part due to this tolerability.

There's also the opportunity to suppress BTK signaling more potently by virtue of giving the drug twice a day and this tolerability maintaining the dosing which can also lead to a greater treatment effect.

Your next question is one that I'm afraid I can't really take on. You used the word "simply" and I'm glad you did that, because there is no simple explanation for BTK inhibition and immune-oncology. What I will say is that BTK we look at it as a B-cell target. It's clearly part of the B-cell receptor pathway. But as you alluded to, it's also active in signaling and myloid cells.

And so, the interaction between immune-oncology and BTK inhibition is an emerging area. I think pre-clinical data was quite interesting. It doesn't always translate, but that led us to do the clinical experiments and led Acerta, as well, to do the clinical experiments, and I think we'll use that data to really make our decision on how to move forward.

Alexandra Hauber: Thank you.

Pascal Soriot: And Sean, and maybe if Jessie who is with you in the room has anything that you want to add. jump in now.

Jesse McGreivy: I agree with Sean. This is a complex area, and there is significant potential for BTK to augment the effects of checkpoint inhibitors, both in B-cell malignancies and in solid tumors. And Acerta has initiated a number of Phase II clinical trials evaluating checkpoint inhibitor with our BTK inhibitor, and those are really based on the micro environment potentiating the favorable immune cells which attack and remove cancers and reducing the inhibitory

immune cells, which actually suppress our immune system and suppress our ability to fight cancers.

So, that's really the basis of those trials and they're underway. And we'll be taking a look at those results over the next year or so.

Pascal Soriot: Thank you, Nicolas, at Morgan Stanley. Nicolas, go ahead.

Nicolas Guyon-Gellin: I have two, actually. The first one is a follow-up on the strategic rationale of the deal and your commitments to a methodology. so, correct me if I'm wrong, but amongst other reasons I think you mentioned a lack of expertise in hematology disorders. When you out-licensed to Celgene. Previous comments also alluded to existing partnerships with where you have this one with . so, could you please help us reconcile what looks a bit contradictory to me? Or, putting it another way, would you do the Celgene deal now that you've done the Acerta one?

And the second is a quick financial question for Marc. You mentioned a moderate dilution to near-term core EPS. So, could you be a bit more specific? Are we talking about low-single-digit dilution? And when do you expect this deal to be accretive to earnings?

Pascal Soriot: So, Marc, I will hand over the second question to you in a minute. It's low-single-digit indeed, Nicolas, in terms of the dilution.

But the commitment to hematology it's very clear it's part of our strategy from the beginning. And as I said before, we certainly value our collaboration with Celgene and quite clearly it is very coherent with what we are doing here.

Celgene have their own hematology products. We will now have acalabrutinib in our portfolio. Together, we will work on immuno hematology and certainly continue to do this.

I certainly do not regret anything at all, because you saw the we announced four studies together with Celgene. There's absolutely no way we could have done that on our own. And we're going to get -- we potentially will be a leader

in immuno hematology together with Celgene, we could never have achieved this alone.

So, I don't see this as at all. We'd do the same again if we had to do it. We are making good process with Celgene. We'll keep working with Celgene. And in parallel to this, we will develop acalabrutinib, just like Celgene is also developing their own products in hematology and oncology.

Marc, the --?

Marc Dunoyer: First of all, talking about the moderately dilutive, the presentation points out a low single-digit type of dilution in the near term maybe about two to three years. I think we also need to understand that this is based on an expectation that we would get the product launched in 2017, as we have outlined before.

So, moderate dilution for a relatively short period of time is what we are now simulating.

Pascal Soriot: And as you can imagine all of this will depend on what kind of indication we get in the US if we can get approval, but also after listing and as a result of it the ramp-up. But we have good hope that the ramp-up for this agent could be quite rapid, because it is -- there is a clear unmet need in the marketplace that is developing rapidly which is those patients who are experiencing side effects and discontinuing their treatment. And there's a clear unmet need that needs to be addressed very rapidly. So, we expect a relatively quick ramp-up.

Let me move on to Peter Sehested, at Handelsbanken. Peter, do you want to go ahead?

Peter Sehested: Congrats on the deal. My question pertains to your comments regarding AstraZeneca completing the transformation in oncology. Could you put a little bit more flavor on that? Should we now expect that your acquisition spree within oncology is over?

And also, could you talk a little bit about how the whole development program for 196 will affect R&D costs over the next couple of years?

Pascal Soriot: So, a couple of questions. One is the

completion of the strategy and then the cost.

For the completion of the strategy, I guess what we meant to say here is that it rounds up our oncology portfolio, as I've explained, we have four areas that we are building the portfolio around. And clearly, the 196 is completing this.

You should assume that, indeed, we are now going to focus on the execution of our pipeline and transformation of this rich pipeline into benefit for patients and a commercial for the Company.

So, over the next months and years we're going to focus on execution from now on. It doesn't mean we will not consider additional deals that would make sense scientifically but not large deals like this, certainly more in the early-phase science phase. And the rest is really going to be focusing on the execution.

And the second question, Marc, you want to --?

Marc Dunoyer: The impact of -- when we talked about the moderate dilution I think it is obviously incorporates the R&D spend that we'll have to commit to in the years to come and yes in the programme that Acerta has already initiated and that we will continue. And depending on the results of these trials, we will obviously augment or change the orientation.

But we don't expect an enormous programme as we speak. Continuation of what has been done is more or less our strategy.

Pascal Soriot: We'll give specific guidance on the R&D spend in our 2016 guidance. But we'll certainly will have to manage in the context of the R&D budget as we will guide early 2016.

So, let's move to Credit Suisse.

Male: It's Tren, from Credit Suisse. I have a few questions here. Firstly, could you give us a bit more visibility on the $3 billion option to buy the remaining 45%? is this a

set amount, irrespective of any sales, any sales related to the successes or approvals or other indications?

And then, given that this was a competitive process, was there anything other than offering perhaps the most money that resulted in the conclusion of this deal?

Pascal Soriot: Well, I guess the I think an important part of this discussion as I said was really building trust and sharing views about the product and our cultural our company cultures. So, that has been a process that has lasted several months, in fact, of discussions and collaboration. We were looking at this asset and potential collaboration.

So, it's of course a financial issue, but building trust and getting to a point where you feel comfortable you're going to be able to work together. That's probably the only thing i can comment on.

But you could also imagine that this asset was of interest to other companies.

So, as far as the $3 billion, I don't think we want to be too specific there, but Marc, you may want to make a few comments?

Marc Dunoyer: as I said earlier on, the put and the call options are subject to approval. So, they can only be exercised if the molecule is approved. And it has to also -- it's also based on commercial success, not directly related to sales, but commercial success.

Pascal Soriot: Thank you, Marc.

Male: Thank you.

Pascal Soriot: So, I think we have no more questions. So, let me just summarize again. First of all, let me thank you for joining us today.

Let me summarize that we really look forward to working with our colleagues at Acerta what a tremendous team of scientists and clinicians. We share the same philosophy, the same goal, the same culture. And together we are going

to turn acalabrutinib into a tremendous medicine for patients around the world.

And the unique opportunity for both companies in in building our oncology portfolio. It really supports our return to growth, it completes our transformation in oncology, which means we are now going to really focus on execution.

Acerta will be a center of excellence for us, especially on the west coast. We are keen to have a presence there.

But the last point I would like to make is that I mentioned during the call, the partnership with Celgene will remain very pivotal to everything we do in hematology.

And so, the potential for this agent is $5 billion-plus. But we certainly are exploring other areas like autoimmune diseases .

With this, I'd like to again thank you and I wish you all a very merry Christmas and happy new year.

Operator: Thank you very much. That does conclude the conference for today.

END