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GLPG.AS - Q2 2017 Galapagos NV Earnings Call

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PRESENTATION

Elizabeth Goodwin - *Galapagos NV - VP of IR & Corporate Communications*

Thank you, and welcome all to the audio webcast of Galapagos' First Half 2017 Results. I am Elizabeth Goodwin, Investor Relations, and I'll be hosting today's event. This recorded webcast is accessible via that Galapagos' website homepage and will be available for replay later on today.

So that your questions can be included, we request that you call in to the following telephone number. That's 32 for Belgium, 2 400 6926. And the code is 4659682.

I'd like to remind everyone that we will be making forward-looking statements during today's webcast. These forward-looking statements include remarks concerning future developments of the pipeline and our company and possible changes in the industry and competitive environment. Because these forward-looking statements involve risks and uncertainties, Galapagos' actual results may differ materially from the results expressed or implied in these statements.

Today's speakers will be Onno van de Stolpe, CEO; Piet Wigerinck, CSO; and Bart Filius, CFO. Onno and Piet will go through the operational highlights. Bart will explain the financial results and the 2017 guidance. And then Onno will close with the expectations for this year.

You will see a PowerPoint presentation on screen while they're talking. We estimate that the presentation will take about 20 minutes, and this will be followed by a Q&A session.

And at this point, I'd like to hand over to Onno to start the presentation.

Onno van de Stolpe - *Galapagos NV - Founder and CEO*

Thank you, Elizabeth. A pleasure here to kick off the half year presentation. Clearly, we had a very solid year with a lot of results, both on the financial side as well as on the R&D side. Highlight is that filgotinib, that's clearly moves forward in the clinical programs. We showed the long-term extension study, DARWIN 3, in rheumatoid arthritis, which was very consistent with regards to the previous results. Piet will provide you more color on the datasets later in this presentation.

But we haven't stopped there. Apart from the IBD programs, filgotinib is now rolled out in multiple Phase II studies and more to come. Some are being executed by Galapagos, some are executed by our partner, Gilead. And clearly, we are blanketing the whole market here as broadly as possible to look at all possible indications for this remarkable molecule.

In CF, we're making very nice progress with our various components of the triple combination study. All Phase Is have been completed, and we're now moving forward with preparations for the start of the Phase III -- the triple-combo Phase II study.

In IPF, idiopathic pulmonary fibrosis, we are shortly expecting to show the data on 1690, the FLORA study. We also had an orphan status given to Galapagos for this program, so we're looking forward to progress in this program very rapidly.

In osteoarthritis, we started first dosing in patients in the U.S. with 1972. And today, we announced that Servier has executed its opt-in and has now licensed this program everywhere outside the U.S.

The research group has also come up with some remarkable results, and we have now 3 more PCCs, preclinical candidates, which brings the total of proprietary assets in our pipeline to 7. So clearly, you see a transition from a very partnered pipeline, development pipeline, to a more proprietary one.

That, together with the very successful offering on NASDAQ, we now have EUR 1.3 billion in cash. So we're well funded for the future, both on our internal activities, as well as potential acquisitions or licensing.

Also very pleased that we can announce that Michele Manto has joined us as the Senior VP, Commercial Operations. He is quite an extraordinary person. Was with AbbVie before, where he was responsible for the global marketing of HUMIRA, the largest selling drug in the world, and also responsible for the preparation of the launch of ABT-494, the competitor product for filgotinib.

If we can go to the next slide, I'll give you some color on the opt-in by Servier of the 1972 molecule. With this opt-in, Servier gains worldwide commercial rights in all indications except for the United States. There, Galapagos has the sole rights to commercialize and develop this molecule.

And of course, with the license, they also have an obligation to further develop this. We are getting a EUR 6 million license fee, which might look quite modest, but please remember that this deal was signed in 2010 when we had only targets and no molecules. So a very early stage partnerships with -- partnership with Servier. And at that time, this milestone was very appropriate.

We're getting more milestones down the road on this program. We have future milestones on 1972 of EUR 200 million. But far more important, of course, are the U.S. commercial rights we have and the royalties that we're getting on all sales outside the U.S. So we're very excited about this program, as we have reported previously. And this opt-in by Servier is a sign of confidence that we are partner that truly believes that this molecule has great opportunity in osteoarthritis.

With that, I would like to hand it over to Piet, who will give you much more details on all these programs.

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Thank you, Onno. Let me start with filgotinib. So as you all know, we have 3 Phase IIIs ongoing. We've commented on those Sufficiently in the past. But over Q2 as well, we have started 5 new Phase II studies.

Starting on this slide we just show that for first study we started in Q2 over ankylosing spondylitis, psoriatic arthritis, a lupus study and uveitis study. So this is only part of the total program we've planned, but this plan clearly shows that, together with partner, Gilead, we are extremely ambitious in exploring filgotinib to the maximum extent in a variety of inflammatory disease. So more studies will come over the coming quarters, and we will update you as soon as those studies come on clinicaltrials.gov.

During Q2 as well, at the occasion of EULAR, we, for first time, have shown both efficacy and additional safety data from DARWIN 3. DARWIN 3, as a reminder, is an open-label safety study. So almost all patients coming out of DARWIN 1 and DARWIN 2 got the invitation and wanted to participate in the long-term follow-up study.

And on this slide, we start with the efficacy. So we were extremely pleased to see that we already, at the end of DARWIN, had high ACR50 scores. But those ACR50 scores further increase up to close to 70%. And more importantly as well, this data show, on the left, that our choice to go for a once-a-day for Phase III was very valid to us. You can see on the left graph here that both bid and qd gives you essentially the same efficacy outcome as part of DARWIN 3.

On the right as well, an output I would like to illustrate is the fact the at start of DARWIN 3, we have both patients coming from the monotherapy study, DARWIN 2; as well as coming from the add-on to methotrexate study, DARWIN 1. And we've compared them on this slide in terms of clinical efficacy, again, ACR50. And there, again, you don't see any difference. So this will not influence our Phase III program, but clearly illustrate that with filgotinib as a selective JAK1 inhibitor late in the market, it could excellently play as a monotherapy for all patients failing on methotrexate.

I was very pleased with the efficacy that's also show over time. There is no decrease of the efficacy, in fact. So this is a sustainable efficacy, both as a monotherapy and as an add-on to methotrexate.

A few highlights on the safety. So filgotinib is the first and the only really selective JAK1 inhibitor in the field. And this is quite nicely illustrated by what is, in fact, the return to the midpoint of the normal values in hemoglobin. And this slide both illustrate the speed of that return, as the extent of the return of hemoglobin values, which are low because this is at start. And they returned quickly to the midpoint of the normal values.

Another illustration of the safety of filgotinib as part of DARWIN 3 are the platelets. As with most of the RA drugs on the market, also filgotinib shows, in fact, a return to the normal values. Those patients have high values, you see a quick drop during the first 12 weeks. And then as part of DARWIN 3, as some of the placebos after 24 weeks go on active, you see, in fact, from week 48 onwards, a stable value of platelets over time. So this is a classic picture you see with most of the RA drugs in the market. The only exception to this baricitinib, which in fact, shows an increase in these patients.

That concludes for me the update as part of this presentation on filgotinib.

Let's now look to the rest of the clinical pipeline. So in the CF field, nothing has changed. We are -- we have 3 potentiators in the game. And clearly, for triple, we are focused on 2451 and 3067, as clearly highlighted as part of the R&D update in June. Our C2 -- 2222 is well advanced in Phase II, both as a monotherapy and as an add-on to Kalydeco, and we expect to read out the first data this year.

And then we have 2 C2 molecules in the game. One in Phase I: 2737, which we'll move into triple patients during this year. And then a second one: 3121, which we'll move into healthy volunteers this year and then in patients next year.

On the IPF line, in fact, we have replaced the previous new PCC, 2938, by 3499. So this molecule is from the same family, acts on the same target, but it has really shown us much better in vivo efficacy data in the animal model. So we decided to start off the development with 3499 as a second mechanism of action in the IPF field.

So for GPR84 and 1205, we'll -- we are looking for a second indication and hope to announce as well before end of this year, what the second indication is, we will bring this molecule -- or test this molecule in the clinic.

The rest of the slide, in the middle there, did not change. But at the bottom, so we have added a new PCC in the inflammation area, an undisclosed novel target, 32 -- 3312.

And then we, as well, have a new mechanism of action in pain, which is #3535. So that's a molecule we will explore in the field of pain. Our ambition is, there, to see whether we can add anything to filgotinib or eventually in OA work, as pain is one of the most remaining complaint that patients currently have when treated with the RA drugs.

Over to CF now. So this is the same slide as at the R&D update. So we are progressing 3 triple-combos and plan to put them into clinic over the coming 12 months. So the first that will enter is 2222, 2737, 2451, plans is to go into Phase I in patients this year.

But on this slide as well is remarkable, in fact, that on the left in gray our dual platform, 2222 plus 2451 in these predictive HBE cells outperforms in a consistent way, the Vertex dual platform, the tezacaftor-ivacaftor combination. So we expect to start from a more solid dual basis to show the additional benefit of our C2s as part of the triple combination. So really there, we are looking forward to both gather dual and triple data over the coming year.

So the second C2, which -- the second triple which is planned to move into patients is the one with the other potentiator, 3067; and the same C1, 2222 and 2737. And then by mid of next year we, as well, hope to bring the next C2, 3221 into patients as planned. So that is, in fact, another mechanism of action and consistently shows somewhat higher efficacy in-vitro, so we are clearly pushing forward every C2 we have in hand and which we believe is a valid clinical candidate.

So in terms of patient studies on CF, and that is a question we get frequently. So the first study to read out and from which we will report our data this year is the ALBATROSS study. This is a dual therapy of 2222 in the G551D patients.

Then we have the second study we'll be reading out early next year, is the FLAMINGO study. It's the C1 corrector 2 2222 as a monotherapy in homozygous delta F508 patients.

And then we shift to the triple studies. So we will start soon a triple study where we add 2737 to Orkambi patients in the homozygous Class II patients. And then later, we'll start our first patient study with the internal triple-base 2451, 2222 and 2737.

So we are well on track with the plan we announced in June. So we are in -- we have submitted to the U.K. Regulatory Authorities the questions we have. And we will meet with them over the coming months to finalize the design and to kick off the triple study in patients.

And then early next year, you see the 3067 triple study in patients. And then by mid, the 3221 triple study also in patients.

So we, as well, are using here in the project, whatever triple believe can make it and can make a difference for patient, to bring that to patients and to test it in patients.

Also for this year, which will probably get more questions during the Q&A on the FLORA. So during Q2, we've completed the collection of all data, locked the database and we now, daily, get pieces of the total package in and we are analyzing. And so you can expect us to read out over the coming weeks.

And to tell to you how 1690 performed in this 12 weeks. It is base a 12 -- we did a 12-week study in IPF patients, where we did our best to do a study according to current standards with a centrally confirmed diagnosis in those patients. This is a monotherapy study. Those patients either did not have access to pirfenidone or nintedanib. Already in Western Europe, waiting before they could get some on their treatment. So it's a study with about 20 patients. And we will read out both on biomarkers and on the number of secondary endpoints, like FVC and others, when we report the data.

Then in the OA field. So today, we announced that Servier has taken the option they had to the ex U.S. side. We have the option to do both studies and to market the drug in the U.S. So as part of those facts, we have started the Phase I study which we discussed with Servier and agreed, in which we both extend the dose range and the age range of patients.

So these are OA patients. It's a study running in the U.S. So we've started our first read there have included patients where we will dose them for a month. We will follow, most importantly, is PK, safety, but as well as the biomarker. And then have a first exploratory look to some of the clinical endpoints.

So in the meanwhile, together with Servier, we are discussing how the Phase II will look like and how we will execute that study. This can be still 1 of 2 separate studies. That is under discussion with them.

So then the next readout of the data we expect soon is, in fact, on morphosis 106. And then with soon, I mean during Q3 this year. So this is the first time this will be the readout of both the single ascending dose in healthy volunteer, but more interestingly, the multiple ascending dose we'll be performing in patients. So we are still collecting the last data. So those, you can expect the data of this study by end of Q3, in fact.

So there, we will look as well, safety, PK, but as well, at measures and some of the classic scores in the atopic dermatitis field. And we will report out on how the MOR106 performs over time versus those scores.

That's, I think -- believe are the recent highlights of the Q2.

Bart Filius - *Galapagos NV - CFO*

Thank you, Piet. Good afternoon, everyone, or good morning if you're in the U.S. My name is Bart Filius, Chief Financial Officer, and I will take you through the numbers of the second quarter or the first half of 2017.

Firstly, as always, I'll start with our cash position, which is at the end of June, EUR 1.25 billion. A healthy increase from where we were at the end of '16, which was a little less than EUR 1 billion. As you know, we've done an equity offering in the U.S. in April which has netted EUR 350 million of proceeds, which is the big driver for this increase in the first 6 months of this year.

Taking you through the bridge which is on this slide. You see a EUR 17 million negative, which is a translation effect. And maybe to clarify this a bit further, we keep most of our cash in euros, but we have a portion also in dollars. This portion is roughly $250 million, which is to basically give a natural hedge to the expenses that we have in dollars over the next couple of years around the filgotinib program. But as we report, obviously, in euro currency, we get a translation affect. So this is not an expense, but it's really a translation of the dollar position into euros as a result of the decline of the dollar against the euro.

Then maybe more interestingly, getting to the cash burn. I've split it out in 2 categories: One is cash income from milestones, EUR 25 million over the first 6 months; and on the other hand, basically the cash expense, which is a net of some income, but mostly relates to R&D expenses of negative EUR 80 million, bringing us to a total cash burn over the first 6 months of a little over EUR 50 million.

We retain our cash guidance in the bracket of between EUR 135 million and EUR 155 million, as I pointed out when we announced our '16 annual results. We retain that guidance, which means that there is actually going to be a larger cash spend in the second half of this year, driven by 2 effects: On one hand, the milestones that we expect are bit lower than the ones in the first half of the year; but more importantly, cash expenses will go up in the second half of this year as the filgotinib program matures and the recruitment accelerates.

All in all, healthy position on the balance sheet, EUR 1.25 billion. And that's not even taking into account, which is in the footnotes, a little over EUR 70 million of receivable from the Belgian and French governments, which are going to pay out over the next 4 or 5 years, which are actually tax incentives.

On to the P&L. First, revenues. A healthy increase of revenues of 50% from the first half of 2016 to the first half of 2017. Two big drivers therein.

First of all, an accounting driver, which is noncash, which is the recognition of deferred revenues. This applies to our upfront license that we've received for filgotinib in early 2016 from Gilead which we've not recognized in full, but which we actually recognize in our P&L in proportion to the expenses that we make on the program. Which means that this will be recognized over a roughly, let's say, 4-year period. And which obviously then increases versus last year as the expenses are -- have also gone up.

And the other driver is that we've slightly higher milestones in the first half of the year, mostly around CF, where we've started multiple extra trials in the first 6 months of this year.

Then going to expenses. Operating expense is going up as well, mostly on developments. And that development expense is driven by, I will repeat myself, filgotinib on one hand and CF on the other hand, where the program, as Piet was explaining it, is getting broader and broader.

The total of the 2, of revenues and operating expenses, both increasing, leads us to a widening of our operating loss of EUR 8.6 million. Then if you look at our net results, that gap versus first half of last year is obviously much bigger because there is a couple of extraordinary events in there.

First of all, in the first month of 2016, we have recognized EUR 57.5 million positive in our P&L as a one-off accounting entry as a result of the Gilead transaction. And that is not recurring, obviously, again in 2017. So we need to rebase our net results to take that into account.

And the other driver is a EUR 15.5 million difference in foreign exchange of other financial income, which is the translation affect that I explained before, combined with a little bit of interest revenue and expense. So that translation affect also leads to a negative P&L impact there.

But the real underlying evolution in our P&L is a negative EUR 8 million on operations, which again, is a combination of increasing revenues against increasing expenses, bringing our first half year results to a negative EUR 50 million.

So far on the financials. I'll hand it back to Onno for the outlook.

Onno van de Stolpe - *Galapagos NV - Founder and CEO*

Thank you, Bart. Thank you, Piet. Before we going to the questions, an outlook for the remainder of the year. Clearly, filgotinib will continue in Phase IIIs in RA, in Crohn's disease and ulcerative colitis, of course, also continuing in all the Phase IIs and more to come in the second half in other disease indications.

We'll also see the start of the triple combo in patients in cystic fibrosis, something the market and ourselves and the patients are looking forward to. And hopefully, we will be able to generate as positive data or better data than our colleagues at Vertex.

We are going to show data from patients in IPF and atopic dermatitis in the coming weeks and months. So we're looking forward to present those data to you. All in the backlight of having a growing number of proprietary clinical programs, with our research organization continuing to come up with novel candidates that we move into development.

With Michele, we have started to build the commercial organization to commercialize filgotinib in the big 5 EU countries, in the Benelux, Belgium and Holland. And that will be the basis of further commercialization efforts by Galapagos for our proprietary programs in the future.

Our balance sheet, as Bart has shown, is very solid. We can continue to fund our own products, our own programs. And we have more than enough financial leverage to actually look at potential acquisitions or licensing that can strengthen our portfolio.

With that, I would like to hand it back to Elizabeth and we can start the Q&A.

Elizabeth Goodwin - *Galapagos NV - VP of IR & Corporate Communications*

All right. Thank you very much. This concludes the presentation portion of the call. I'd like to ask the operator, Ebony, to connect us to any callers who may have questions for our executives. Go ahead.

QUESTIONS AND ANSWERS

Operator

(Operator Instructions) Our first question will come from Debjit Chattopadhyay with Janney.

Debjit D. Chattopadhyay - *Janney Montgomery Scott LLC, Research Division - MD of Biotechnology*

So just start off with IPF. So given the relatively small number of patients in the study, would you expect to see a [stat seg] benefit in FVC? And what is your internal hurdle in moving the program forward?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Okay, Debjit, thanks for the question. So indeed, it's a very small study. As we've said, we had -- in Phase I a plasma biomarker, you want to see that confirmed. We've put in a number of exploratory biomarkers, but importantly, I think we will look to the respiratory parameters and hope to see that the activity is at least as good and, hopefully, be better than the placebo. So -- and finally, we don't want to see extreme drops in the active group that would to highlight that maybe some patients might improve but others are at risk of worsening of the disease. So it's going to be looking to the means but also the extremes of what you see in the patient population, which clearly that this study is not powered to show any difference between the active and the placebo.

Debjit D. Chattopadhyay - *Janney Montgomery Scott LLC, Research Division - MD of Biotechnology*

Great. So and then on the osteoarthritis drug, as you start thinking about the patient studies, are you considering excluding the KL3 and 4 OA patients primarily because the disease is probably so advanced in these and there are probably morphological changes to the bone alignment? So maybe they don't have cartilage or even if there is any cartilage, the rescue may not be enough therapeutically beneficial. So just wondering, how we should think about what kind of OA patients are most likely going to be in the study.

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Okay. On the OA design, indeed, we have a mechanism of action that is blocking the degradation of cartilage, so it's important that we include patients that still have cartilage, and that will, indeed, lead to limitation of which patients will go into the study, whether it's not exactly only KL2 and not 3 are different. I will not highlight today, but it's clear is one of the key points in the design of our study that we have the right patients there. It is not going to be an [o-com] study where we are at risk of having too much patients. That probably can't show any benefit.

Debjit D. Chattopadhyay - *Janney Montgomery Scott LLC, Research Division - MD of Biotechnology*

So if you could stratify the OA market then what percent of the OA patients are KL1 and 2 versus 3 and 4? Or I mean, is there -- I mean, clearly, for this drug to be a blockbuster, there is going to be some sort of a biomarker screening of patients much earlier on than that is currently used.

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Well, thanks for the question. With GLPG1972, we want to show that it's working and showing benefits, so I'm not too worried about how big the market is. Obviously, there are very sufficient patients that even if you only take part of the market, this is going to be a fantastic drug. So this is a field where not single drug has been approved over the past 15 years. There are really no disease modifying this in this field. So whether in the end, we will only have the patients, early disease, mid or end, I'm not too worried. And so I can't give you those numbers, but you can find them clearly elsewhere published by other groups.

Debjit D. Chattopadhyay - *Janney Montgomery Scott LLC, Research Division - MD of Biotechnology*

And just one last question. The testicular safety study, which is underway, in terms of timing and when we should expect a readout. And do you think that would finish early enough for you to recruit younger male patients in the U.S. in the GI studies? Or it doesn't really matter because you have plenty more patients ex U.S. from a labeling perspective?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Yes. So the safety study in the U.S. will help us start at a certain moment to extend the age range. So we've tested, as part of the of healthy volunteers, up to 55, and now are extending up to 75. And as you know, most of the OA patients are more in the elderly section of the population, but we can start this study. Earlier and as soon as we have those data, expand the inclusion criteria. So there is no limitation there. That's for the safety study, OA 1972. Maybe there was a question on the safety study on filgotinib. So that study has kicked off, and will not allow typically to expand the inclusion. So there we have a safety study as agreed with FDA, and the plan is that, that reads out in parallel and together with the efficacy studies that we're running at the moment.

Operator

And our next question will come from Dane Leone with BTIG.

Dane Vincent Leone - *BTIG, LLC, Research Division - Director and Diagnostics and Life Sciences Analyst*

So what I wanted to ask is -- and apologies if I missed this, but is 3499 an autotaxin?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

No.

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

So on the pipeline slide on the line for IPF, we have the autotaxin, and then the second compound is another mechanism of action different from autotaxin. So in that program, we have replaced 2938 by a new compound, which has shown first peer in vivo efficacy. And thanks for the question because -- so it allows me to clarify that, indeed, this is a new mechanism of action different from the autotaxin.

Dane Vincent Leone - *BTIG, LLC, Research Division - Director and Diagnostics and Life Sciences Analyst*

Do have a timeline in terms of when you might be able to characterize it a little bit more for us?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

So this now start preclinical development. Give us 15 months to bring it to Phase I. But to disclose the target, we typically wait until we bring this to patients, so this could take a while before we're going to disclose the target of this novel approach in IPF. Thank you.

Dane Vincent Leone - *BTIG, LLC, Research Division - Director and Diagnostics and Life Sciences Analyst*

Okay. And I guess, switching over to atopic dermatitis. As you move, we'll see the top line data of a healthy study in, I guess, this quarter. Going forward, what do you think the realistic challenges are for running a larger atopic dermatitis study in the moderate-to-severe population now that DUPIXENT is not on the market? I would assume eventually you would have to run part of the study in the U.S. Do you foresee any challenges? Would you think that you would be looking post someone coming off of DUPIXENT therapy? Or does it not matter given that you're looking at IL-17C?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Thanks, again, for the excellent question, which is how we will design on what types of patients we're going to include in atopic dermatitis Phase II study, which, if the Phase Ib is successful, is a logical next step. So it will all depend a bit on the risk benefit outcome, how much of efficacy do we see. But from a scientific point of view, IL-17C is independent of the IL-4 pathway of the Regeneron drug. So there is no reason why we would have ourselves scientifically oriented on that program because there is -- if you look forward five years from here, there is going to be a market of patients that did not respond, patients that have a moderate response. So you could think about an add-on design, you could think about a design where you focus on the failures. But I guess, the first study we'll be rather look into a monotherapy. [We'll stop saying] that (inaudible) file played in the market which where we'll try to get an idea in terms of efficacy, how the risk benefits looks with this novel mechanism of action in atopic dermatitis.

Dane Vincent Leone - *BTIG, LLC, Research Division - Director and Diagnostics and Life Sciences Analyst*

And the last one from me, is 2534 also an IL-17-targeted drug? Or is it a different mechanism of action?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Thanks as well for allowing me to clarify with our early path. I know this is a small molecule novel mechanism of action, which we will work on for a while, but have shown now. And we've looked in preclinical models into a number of inflammatory indications, but really in terms of efficacy in the animal models, atopic dermatitis was the best we've seen. And so it's going to be a small molecule novel target program that we started there. Thank you.

Operator

(Operator Instructions) And we'll move next to Matthew Harrison with Morgan Stanley.

Matthew Kelsey Harrison - *Morgan Stanley, Research Division - Executive Director*

Two CF questions, if I may, for me. I was wondering, first, if you could just comment on your reviews on the recent Vertex data and how you see that, what the implications are to your program from that data? And then secondly, any updates on timing from when you expect to finish receiving scientific advice from the U.K. and when you might be able to communicate what the program looks like after that advice.

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Thank you, Matthew for the questions. First of all, let me say we're pleased with the Vertex data and pleased for a number of reasons. First of all, pleased for the many patients in the CF clinics for which we had no clinical data that supports our ambition to bring effective treatments to them. I think with those new Vertex data in the minus population that dream of bringing treatment for more than 90% of the CF patients is coming much closer now, so that's very good. Secondly, we are pleased as well because it completely validates as well our approach that with the triple -- that the triple therapy is the way in the future to treat both homozygous and the heterozygous/minus patients. So we saw a nice validation of our platform as well. It finally takes away as well a number of worries when doing this -- yesterday, there was a clear sweat thing, there was a clear FEV.

So in terms of studies, we need to do early on to evaluate whether we have an effective and competitive treatment by using both sweat and FEV even with small numbers of patients and trials ranging from 1 to 3 months. We should get a good view on that. So that as well helps us early on to assess well the competitive value of our different triples. Finally, in terms of timelines, we've been watching the Vertex program for a while. It's all coming very concrete, but the timing they've indicated coincides with what we had in terms of planning for when we want to start the late-stage clinical studies for CF. I hope I covered most of your questions. Our scientific advice timing. So well, we've submitted the questions and we are on track there that we expect to file and to start patient inclusions in the half part of this year. So we're completely on track of what we told at the R&D day.

Operator

Our next question will come from Stephanie Put with Degroof Petercam.

Stephanie Put

Maybe first on osteoarthritis. With -- following the in-licensing by Servier, how do you see the path going forward, specifically for the U.S. going towards Phase II, Phase III studies? Will this be split up? Will you look for a partner eventually in the U.S.? Can you shed some light on that? Then on the CF, will you present the Phase I data in healthy volunteers of the different components at any scientific conferences? And then lastly, a small-detail question concerning the recently announced tax reform in Belgium. Do you have any idea if this might affect patent income deduction regime that you -- or rather, innovation deduction regime, that is called now, looking to filgotinib that I think is coming closer to market?

Onno van de Stolpe - *Galapagos NV - Founder and CEO*

I'll start with the first question on the Servier collaboration and the U.S., non-U.S. situation. So we have all rights in the U.S., and we can execute the Phase II in the U.S. independent of Servier. At the moment, we are discussing how we're going to move forward with our partner here, and that might result in a separate Phase II or actually a combined Phase II. That's still under discussion, and we'll inform the market as soon as that has cleared up.

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

On the tax reforms part?

Bart Filius - *Galapagos NV - CFO*

Yes, let me take the question, Stephanie, on the tax reforms because, indeed, that's one that has major impacts on the company in a sense. So first of all, for those that are less familiar with the Belgian situation, there's a ruling where we are allowed under a regime called BEPS. It's actually a patent box regime where we are allowed to deduct 80% of our income from IP from our taxable base. So as a result, we're only taxed on the remaining 20% of that income. There's new ruling in Belgium where -- there's a very new system called IID, which effectively comes to sort of the same outcome, it's slightly different. I won't go into those details there, but it's, if anything, a slight improvement over the old regime. And thirdly there is also some plans around Belgian tax reform around the Belgian corporate tax rates. It's a bit too early to comment, but it looks like the direction is down, so meaning the tax rates are going to go down meaningfully, while the IID regime after 2020 will be protected and sustained. So if anything, again, too early to comment in details, but if anything, it will be a beneficial outcome for us for future income coming out of IP-related assets such as filgotinib and CF and any other programs we're developing. So then for CF side, I will give to you, Piet?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Stephanie, thanks for the question, on the company effect. It allows me to remind most people on the call we have a policy as we publish all the Phase I data that we generate. So every clinical study, we execute, we publish those data either at a conference, either as part of a manuscript. And for those that have followed us in the CF field, they know that we've been quite present at every CF conference. We have a continuous flow of novel data. So you will see next flow early November at the North American Cystic Fibrosis Conference, and then the next flow will then come early 2018 at the European CF Conference. But every Phase I even the -- and as well for sure the Phase II studies, which we execute, we -- as soon as we have the data, we will publish them and comment on them at conferences. I hope I answered your question.

Operator

Our next question will come from Christopher Marai with Nomura Instinet.

Christopher N. Marai - *Instinet, LLC, Research Division - Analyst*

Number one, I was just wondering if you could further clarify, perhaps, some of the potentiator on the metabolite half life questions you may have received from regulators and any discussions that you may have had. And then secondarily, maybe remind us, is there a path forward to use your currently approved potentiators and just have your correctors on top of those should the small molecules be trickier than we all anticipate it to move forward?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Okay. Chris, thank you for the questions. The interactions with the authorities is ongoing, and we'll never comment on ongoing regulatory interactions. But what I can say is that we feel very comfortable that all of the data we've generated shows that 2451 and its active metabolite is going to be an effective and safe chronic treatment for the CF patients, and this will be part -- as of a triple therapy. So we are very comforted with all of the data we have generated and we have been generating over the past months. So -- but we never comment, in fact, on an ongoing regulatory interaction. So we'll not do that today. Then the second question, what can we combine with an approved potentiator? In theory, you can. On the other hand, there is quite a restriction on the availability of the potentiators. So if we would try to execute that theory that we can buy in the free market sufficient of Kalydeco to combine, I think we will immediately run simply as part of the clinical studies in an logistic nightmare because it's not that much available. And so that would immediately give us a very, very slow program to execute. So we've, from day 1, more or less excluded that option. Finally, as well, if you combine with a compound from the competitors for the final pricing, that's a nice discussion to have. And you might need a license for that. Don't forget that some of the combo treatment that have been brought into market have required licenses from the company that has brought it to the market. So there is not an easy, executable way of developing our own C1s and C2s, and it's not part of our plans. Thank you.

Christopher N. Marai - *Instinet, LLC, Research Division - Analyst*

Okay. That's helpful. And if I may, one follow up. Just, again, sort of thinking about your compounds and some of the data you see in your own labs versus the competitor compound in terms of just the triple. Could you perhaps comment on why you believe that you may have potentially the equal or better opportunity once you -- you're in patients given the data that we saw this quarter?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Thank you for the question. Well, also during this call as well as during the R&D Day, I showed some data that compare our dual platform other than 2451, 2222 or 3067, 2222 because you'd -- we'd repeat exactly the same data. Systematically, both validates internally with many external labs really, in vitro outperforms the Vertex dual platform. So we believe we will have an excellent dual basis to start from. And so we -- so the whole team have concluded, we believe that the dual platform is better than the Vertex platform so we have a better launching platform for triple therapies.

Quite honestly, we don't know what -- how the chemical structure is of the Vertex C2. So we can't do or perform any comparative studies in vitro currently in-house, so I can't comment on experiments we can't do. So what we can do is compare on published data. And there, we feel very comfortable that in terms of fault increases we see from starting from our dual, it is quite similar with what Vertex shows what they see in terms of increases starting from their dual platform. So if you start from a higher basis, we've already seen the same fault increases. We are hopeful that we will end up with a higher FEV values. But it needs to be proven in the clinic, that's clear. Thank you.

Operator

(Operator Instructions) We will move next to Timothy Woodward with Goldman Sachs.

Timothy David Ming-Tze Woodward - *Goldman Sachs Group Inc., Research Division - Equity Analyst*

Just to stay on the cystic fibrosis topic, I think a couple of patients on the Vertex side had elevated liver enzymes in that Phase II -- in those Phase II studies. Is that something that you've seen in your own early work? And is there a mechanistic reason that you are aware of that a triple-combination therapy could lead to elevate liver enzymes? And then a second question, if I may. It feels like you've waited on the cystic fibrosis program for drugs and data to come through so that you can pursue a once-daily dosing strategy versus Vertex, who have been using the twice-daily dosing. As you see it, how relevant is advantage of once-daily dosing versus twice-daily?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Thank you for the questions. On the elevated liver enzymes, if there is one thing everybody wants to know avoid early on in the development program it is really early liver enzyme increasings because typically, it never gets better over time with longer dosing and more patients. So if you would have seen elevated liver as part of the Phase I, that could have been, for us, a reason not to progress molecules further. So all I can say that as part of the portfolio, we haven't seen it and we are not progressing compounds where we have seen liver enzyme increases. But for the Vertex molecules, they have to judge themselves and see what is an effective and a safe dose and then take the decision what molecule at what dose in triple they will progress. But according to me, there is no mechanistic reason why C2 should or might increase liver enzymes. Then on the QD dosing, in fact, you're pointing to what differentiators are there left in the CF market. I think that's the big question many people have after having seen the first efficacy data of the Vertex triples. But I don't think it's any different to what any other program at this stage. So differentiation can happen either on efficacy basis, either on safety basis, either on a dosing or on a dose regimen basis. And for patients that have to take medication every day, lifelong, QD makes a big difference for them. So eventually, I think QD, if it will have the same risk benefit profile, would, for patients, be a good reason to change because it really affects their life if they have to take drugs twice a day or only once a day. So that is my answer to the QD dosing. Thanks for the question.

Operator

Our next question comes from Peter Welford with Jefferies.

Peter Welford - *Jefferies LLC, Research Division - Senior Equity Analyst*

I think I've got 4. Last -- firstly, just on the -- [sticking with] cystic fibrosis, is there any impact at all from the Vertex data on your choice of components within the triple, like which triple combination you perhaps preferentially would like to accelerate development of? I guess just trying to get a feel of whether or not you think that there's any differences between those that are worth pursuing more or less. Secondly, then just on filgotinib. I think it's been discussed with regards to platelet levels, but I just wondered if you would clarify the patient that did, in your studies, have a thromboembolic event, on what dose that patient had that event in the Phase II studies that have been conducted today. Thirdly, then on 1972, is the plan to wait for the U.S. study that's ongoing before kick-starting Phase II? Or will a Phase II start prior to those U.S. data? And then finally, a bit of a pedantic question on the financials, but the EUR 6 million license fee, is that recognized in the fall or will that be deferred over the next coming period from Servier?

Bart Filius - *Galapagos NV - CFO*

All right, Peter, I'll take the last question maybe first, and we'll continue with that quite quickly. We'll recognize that in full in our P&L immediately. And then, Piet, I guess, you'll take the other 3 questions on the science side.

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Yes. So let me start with filgotinib, and thanks for the questions so I can clarify. So as part of the placebo-controlled RA studies where we have different dosages of filgotinib, we have seen no thromboembolic event. So if you look to the placebo-controlled part in RA, our count is 0 there, and that's in fact the number that -- if people want to compare to what Lilly is reporting, they report a number of positive placebo-controlled trials that they've seen that as part of placebo-controlled RA studies. We have had no thromboembolic events. So that means that we've seen 3 cases up to now: 1 in the field work program where we have only dosed 200 milligram; 1 in DARWIN 3, where we also have 200 milligram; and then 1 other in the Phase II. So all of the events we've seen, and Norbert really explained very well yesterday how unlikely is that most -- that they are linked to filgotinib. All of them were up to 200 milligram dose. I hope that answers on the platelets and the thrombotic events. Then in terms of 1972, that's an easy question. So we planned to finalize the design earlier, the submissions as well. And as soon as we then have the data from the Phase Ib study, extend the age range in the planned Phase II studies. So we will not wait for the outcome of those studies to start the process to initiate and start the Phase II study of 1972. And then on the CF field, did the Vertex molecule make any -- does it have any influence on the choice of compounds? As I've said, we try to bring every triple therapy we believe based on all our data we have in terms of preclinical safety Phase I and preclinical efficacy. Every competitive triple, we will bring to patients. And then it's going to depend because time winds for the last one will need to be very fast there. So that's going to depend a bit for when Vertex plan to file for Phase III. But for the first 2, there is no change there. We are exactly with our plans within the -- on track in terms of progression, and we will start the pivotal study timely as we have said during the call. I hope this clarifies your questions.

Elizabeth Goodwin - *Galapagos NV - VP of IR & Corporate Communications*

All right. I'm just going to -- this is Elizabeth. I'm just going to intervene here. The time is up now, and I have the feeling there might be more questions. So Paul van der Horst in Europe and Elizabeth Goodwin, we'll be available offline after this call to take your questions. You can best reach me via my e-mail as I'm in Europe this week as well.

So this wraps up for today. And our next financial results webcast is expected on 27th of October when we present Q3.

I want to thank all of those who dialed in and listened in today, and I wish you a great weekend. Goodbye.

Operator

This concludes today's call. Thank you for your participation. You may now disconnect.