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GLPG.AS - Galapagos NV 1690 Halts Disease Progression in IPF Patients in FLORA Phase 2a Trial Call

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PRESENTATION

Operator

Good day, everyone, and welcome to the Galapagos FLORA Results Webcast. At this time, I would like to turn the conference over to Elizabeth Goodwin. Please go ahead.

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

Thank you, and welcome all to the audio webcast of Galapagos' FLORA Study Results. I'm Elizabeth Goodwin, Investor Relations, and I am very proud to be hosting today's event. This recorded webcast is accessible via the 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 this following telephone number. There are several numbers in the press release, here's the number for Belgium, that's 32-2404-0659, the access code is 2084135.

I would like to remind everyone that we will be making forward-looking statements during today's webcast. Those 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; and Piet Wigerinck, CSO. Onno will set the scene and Piet will go through the FLORA study design and results. You'll see a PowerPoint presentation on screen, while they are 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 would like to hand over to Onno, to kick things off.

Onno van de Stolpe - *Galapagos NV - Founder & CEO,*

Thank you, Elizabeth. I am very excited that I can do the introduction here for these exciting FLORA results. If we look at 1690, the molecule we're talking about today, it hits on the target called autotaxin. And autotaxin is the target that Galapagos identified through our target discovery platform. So after the JAK1, the target for filgotinib, it's the second molecule that has proven concept now in patients all the way from identification of the target to the drug in patients. So it's really a hallmark event for the company. We're extremely proud and pleased and excited that these data come out so nicely. And it's up to Piet to present them in further detail.

We have always said that the differentiating factor of Galapagos is our target discovery platform with which we can identify new mode of actions that are the basis of our research programs and this is a clear validation of that approach again, we have much more in the pipeline, more new mode of actions coming for various diseases. So the future really looks bright. But as also a very important of this program is that 1690 is fully proprietary to Galapagos. We don't have a partner here. We aim to develop this all the way to the market ourselves. We have the financial means to do so. We have the expertise in-house and if not, we'll get the expertise in-house, but we are committed to move this program forward as rapidly as possible to the patients.

We have obtained orphan drug designation in the EU as well as in the U.S., which will give us additional advantages in the clinical programs towards the market. So we're very pleased with that as well. And we're not only vetting on 1690 for IPF, we have 2 other mode of actions that we are developing in the same disease space in IPF, idiopathic pulmonary fibrosis, that will move forward and you'll get more information on their dose at a later stage.

If we look at idiopathic pulmonary fibrosis as a disease and as a market, it's clearly a substantial market opportunity with a very high unmet medical need. There are about 200,000 cases in the EU and the USA, and we got about 50,000 new patients every year. It's a very serious disease, where the diagnosis -- after diagnosis, the average time that these patients survive is between 2 and 5 years. So clearly something needs to be done about this, so that these patients have a future.

At the moment, there are 2 drugs approved for this indication and although they slow down the decline in lung function, they don't stop it and patients are still facing death at not-too-far future. On top of that, both of these drugs have serious side effects, which is difficult for these patients to stay on these drugs in certain cases.

But sales are doing well. The combined sales of these drugs are EUR 1.3 billion and growing rapidly. So the market opportunity is clear -- still they're clearly there. And they're priced at about $100,000 a patient a year. So a lot of room for a better product, more efficacious and safer product to come in and to provide solutions for this very severe disease. Piet will discuss now in detail how we believe that 1690 can play a role in this IPF space. Piet, floor is yours.

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Thank you, Onno. Good afternoon or good morning, everybody. So we designed the FLORA study as a mid-stage Phase II study, while we were generating the chronic toxic data. It was a 12-week study, within 3 to 1 randomization between active and placebo. In total, plan to include 24 patients. So the IPF patients who are diagnosed with HRCT, centrally confirmed. And in case of that, an available biopsy had to make the goal there. We targeted the mild-to-moderate patients and that's then reflected in the inclusion criteria. This was a multi-therapy study, in the sense that patients were not allowed to be on neither pirfedinone, neither nintedanib and if they have been dosed before being off the drug for at least 4 weeks. In practice, in fact, patients were recruited in Ukraine where those drugs are not available. And also in the U.K. and Italy, where the drugs are available, we only recruited patients that were waiting for reimbursement and so participate in the trial prior to get access to those 2 drugs.

And as final inclusion criteria was that they could have no exacerbation 6 weeks before or during screening. Next Slide. So the objective of this drug is to make a bridge between the 2-week data in Phase I and then the longer 6-month and 1-year studies we plan to do in the future. And so we wanted to explore the safety of the compound in IPF patients during 12 weeks. On top, during Phase I, we have generated plasma PK biomarkers data who wanted to see that confirmed and wanted us -- and confirmation of the PK. Secondary parameters, we measure the pulmonary function and we also took a deep dive into the lungs with the FRI measurements, and I will explain that later.

On top, we measure the number of biomarkers and the quality of life that we will report on those data later at other scientific conference. So in terms of the efficacy, primary there was to do this spirometry, which is an active measurement of the lung function and where you really measure how much air the patient can blow and so the critical parameter for IPF patients there is the FVC, or the forced vital capacity. The deep dive is the FRI. In this -- a technique where we take a number of CT scans while the patient is breathing and it will reconstitute a complete image of the lungs of the patient and on top, will simulate how the air flows into all parts of the lungs. This allows us to have much more specific measurement of parameters in the lung and in the sub-compartments of the lungs, as you will see later.

Let's now look to what type of patients we could include in the trial. So these are patients within mean age of 65 years old, a little bit more males than females, and more than half of them never smoked before. So this is in line with the classic inclusion criteria or patient data that they accumulate in other IPF studies.

Next slide, please. IPF patients won't live too long with diagnosis. So on average they had IPF diagnosis for 1.7 years. Well balanced between the 2 groups. And then as well the baseline FVC, as in total volume or as predicted of actual really shows that we recruited the patients in the mild-to-moderate region of the total population.

Also, the DLCO parameters there is exactly what we wanted to see in the study. Let us first look to the PD market, with this one we had shown up to 80% drop of LPA in plasma, and we fully confirmed this in the FLORA study. So you can see the orange curve dropping to almost 80% after 12 weeks. Showing us, indeed, in patients we got the right exposure, we got the right level of target inhibition as we have seen before in the Phase I study. In the placebo, of course, nothing happens. And as soon as we stop treatment, the LPA species which are -- which are made by the autotaxin enzyme return to the normal value there.

Let's now look to the FVC because these are the data that gets us all very excited here. So we were extremely surprised and pleased, not only the team internally, but also everybody externally we showed these data. Everybody was extremely impressed by the facts that over 12 weeks in the active group, we don't see any drop, honestly, while the placebo really performs there completely in line with all of the IPF studies published recently. So there in the recently published studies, the placebo group shows a 12 weeks drop between 50 mL and 100 mL and with 87, we are exactly within that range. And for the active group, in fact, this is the first study, we believe, that shows that over 12 weeks, there is no drop of FVC in the patient group. So we are extremely pleased with these FVC data. So how do these FVC data now compared to the published data? So if you -- on this graph look to the first breakthrough there in IPF or the pirfenidone Phase III results. Pirfenidone was the first drug that shows a decline of -- or a drop in the speed of decline of FVC, they got 3 months already than the box we have indicated you see that there is a difference between active and placebo, but also there are patients on pirfenidone expect there's a decline of FVC. And in contrast, if you then look to our data of 3 months or 12 weeks, we don't see any drop of FVC. Same picture has been obtained with nintedanib in Phase III, exactly the placebo drops as in the pirfenidone trials and after 3 months again, there also in the active treatments there is a clear drop of FVC, a start of the drop there while in our study we don't see that. And that's why we really believe that with this first autotaxin inhibitor in IPF just showing that there is no drop in FVC, we see a lot of hope and potential as in next future treatment. FVC is a way of measuring, and it's a standard way of measuring. We did deep dive into the lungs, let's say it like that, we did for the FRI.

As I've said before FRI, we take a number of CT scans while the patient is lying down, but we synchronized the taking of the CT scans with the breathing of the patients, so we exactly know when the patient inhales and exhales. And this allows you, as you can see on the picture, to reconstitute a complete image of the lungs. And now, you can go and measure the specific airways, the specific lobes, the upper lobes, lower lobes and all of that. On top of that, with advanced mathematics you can simulate the flow of air in these lungs. On the graph below, what you see there is data generated in IPF patients, and on the x axis as you see, the FVC, the most important parameter. And as the FVC drops, you can see that the airway radius will increase. So I will explain it on the next slide. But really there is a correlations between the drop in FVC and an increase in airways. So how do we explain that? That's on the next slide. So there you see a cartoon on the left of the healthy lung. And in the healthy bronchus or in the healthy adults, when patient breathe, especially the small airways and the alveoli that will change in volume and that will make sure that there is pressure in the lungs. If you now on the right look to the IPF patients, there's lots of scar tissue there, and there's an increased stiffness, so those small airways and the alveoli don't move that much. And it's in fact, the upper airways and the distal airways that will start to move and cause the pressure which is needed to make the air circulate in the lungs. So and that explains why there is an increase stiffness of the small airways into the upper airways that tend to increase or have increased as proven on the previous slide.

So if you look now with this very specific measurement, a very detailed measurement. In fact, if you go into long term with -- and compare healthy with IPF patients, in fact, you will be able of measuring smaller lung lobes but that's on the longer-term parameters that move earlier are the larger airway dimensions and a decrease in airway resistance. So then these are really the more sensitive parameters for the IPF patients, so if you see an increase or you can block the increase of the airways is that the first sign of activity, if you can block the decrease in airway resistance that's a second parameter, which we looked at.

In general, we expect the disease, you will see more changes in the lower lobes and it's quite clear and as also you can see it on image is that is due to the fibrosis in the lower part of the lungs. But then general and a message to remind that the FRI will move faster than the FVC.

Let's now look to the FRI data. So on the left we have specific airway volume. In the placebo, indeed, you see the further increase of the airway volume. So the upper airways tend to enlarge a bit, while we see a complete stop of that and enlargement in the actual sequence. So that's a very nice support of the fact that, indeed, 1690 is doing something special in the lungs. But as you can see on the graph as well, we had a very nice p-value for this disease.

Second parameter, where we got a very nice p-value is the airway resistance. There you see a further drop in the patients on placebo, while again 1690 stabilizes this effect. So there is no effect or there is no change with 1690 of the specific airway resistance.

Not on the slide, but this were really driven by the changes in the lower lobes in the lungs. Here we have given the values of the total lungs on the graph.

Now over to safety, because primarily it was a safety study. So we are extremely pleased with the safety we have observed as we have in 1 to 3 randomization, so we represent them as percentages and they're quite comparable between actives and placebo. So we had -- on the patients that had stopped based on treatment, there was 1 patient, in fact, it was a very unlucky patient because he was only couple of days in the study and was then diagnosed with a metastatic cancer due to something else. So and that patient just can't achieved both as a serious TE -- severe TE and a permanently stopped TE. So that's all the same patient sample. And so far, for the rest, in fact, safety was very in line with the expectations, just some mild headaches as being the most frequently reported.

Brings me to a comment of our principal investigator. So as soon as we got clarity on the data, shared them with Toby Maher of the Brompton Hospital in London. And he as well really got excited within a couple of minutes when he saw that the placebo confirms that we are fully in line with what we expect in these patients. I guess, for the first time, there is a drug that for a period of 12-week at least completely blocks the disease progression as measured by FVC. So we share that excitement with him and we're happy to bring that over to you today.

Onno van de Stolpe - *Galapagos NV - Founder & CEO,*

Thank you, Piet. So the conclusions. Clearly, we see a very nice effect in IPF with 1690. It's the first autotaxin inhibitor that actually shows the fact, we really are in the lead here. And as I've said at the beginning, thanks to our target discovery platform that we identified this. We saw a stabilization of the lung function over 12-week period, measured by FVC and confirmed by FVC data that Piet just explained, that even showed a statistical significance with such a small group that is quite stunning and generally well tolerated, very nice safety profile. We were very pleased with that because that's a, of course, very important to move this forward. And clearly, these results support a very rapid move towards a late-stage trial. And what that late-stage trial will look like, that Piet can answer in the questions. So with that, I would like to hand it over to Elizabeth because I'm sure there are a lots of questions. Elizabeth?

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

Yes. Thanks, Onno. So that does conclude the presentation part of the call. I'd now like to ask the operator, Lori, to connect this to any callers with questions for Onno and Piet. Go ahead, Lori.

QUESTIONS AND ANSWERS

Operator

(Operator Instructions) We'll go first to Anastasia Karpova, Kempen.

Anastasia Karpova - *Kempen & Co. N.V., Research Division - Research Analyst*

Two questions from my side. Can you please specify which serious adverse events you're seeing in the treatment arm? And secondly, I see in the FVC data that 4 patients discontinued in the treatment arm over the course of 12 weeks. Can you specify what were the reasons, whether they are due adverse events or other factors?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Okay. Anastasia, thanks for the good question. So first question was on the treatment emerging SAE. So the SAE, as I said -- mentioned a couple of times, the serious treatment emerging AE. The severe treatment emerging AE and the permanently stopped treatment is that same patient that he or she was only couple of days in the study was diagnosed with a cancer and due do that then discontinued treatment. So in terms of serious, severe and permanently stopped, in fact, we don't link this to the medication, it was a complete other disease. So then if you want to go further in adverse event, it's more the moderate, and then it's more driven by the headache that this was driven. Then your second question on the FVCs. So patient came to the center on a monthly basis. Patients were allowed to take bronchodilators, but if you did an FVC, there had to be a number of hours between taking the bronchodilator and the measuring of the FVC. And as part of the documentation, it was clear that the patient had taken the bronchodilators too close to the FVC measurement, that measurement is not included. So if you see a variable number there. The second reason why we did not sometimes take up the FVC, it's when there was a lack of quality during the FVC measurement. So, indeed, that number varies a bit because at some time points patient have taken bronchodilators, or either the measurement was not of sufficient quality to be included.

Operator

We'll go next to Matthew Harrison, Morgan Stanley.

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

I have a couple of questions. So sorry if there's a lot here. But so first, Piet, can you just comment on how you dealt with missing or interpolated data? And how, if that was -- if there was any data interpolation done in this study?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Okay. We go question by question, Matthew. No problem.

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

Yes, is that okay?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Yes, we did not do last observation carried forward. So what we report here are the actual measured values on the specific time points. So does that clarify your question?

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

Yes, that clarifies. So you didn't do any interpolation for any of the data then?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

No. Correct.

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

Okay. All right. Perfect. And then the second, I guess, 2 related questions. Can you comment on what the data would look like if you used medians instead of means, just trying to understand if there are any outliers that drove any of the performance on FVC? And then related to that, in drug arm it looked like you had a pretty significant rise in the first 4 weeks and then a decline in the second 4 weeks, is there a mechanism-related rationale for that? Or do you view that as noise in the measurements?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Okay. Medians or means were quite similar. In fact, medians would have given even a higher number. So but as we typically report means, we stick to means. So we didn't try to blow up the numbers here. And then the second, you ask, yes, the increase over the 4 weeks, I think we are extremely pleased to see a stable FVC over 12 weeks. And we don't think it's the increase at 4 weeks that drives the data at 12 weeks. I think it's just what it is there.

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

Okay, perfect. I have 1 last one and then I'll let some other people ask questions. Can you also comment -- did you see any hospitalizations, any exacerbations or any significant changes in laboratory findings over the course of the study?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Okay. Lab abnormalities, we haven't seen any there. So extremely similar placebo versus active, so really nothing to report as we have seen there. And we have had no exacerbations during the study neither.

Operator

We'll go next to Peter Welford, Jefferies.

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

Just curious with regards to the temporary stopping, I think there were 2 patients that temporary stopped treatment on 1690. Can you just explain, I guess, what it was that's -- that results in those patients temporarily stopping the treatment? And then a follow-on on the data just with regards to the dosing. I guess, is there anything in the data you've seen so far that would confirm the 600 milligrams is the best dose? Or I guess, how do you think about dosing now going into future studies? And then finally, one for Onno therefore, I guess, which is related to that. Is that given this is only a single dose, what sort of future trial should we think about? Is there likely to be a Phase IIb dose range finding study, perhaps, a similar design, first? Or would you go straight into a pivotal study with multiple doses? And I guess, what's your initial feel this -- how much financial commitment we're talking for Galapagos for this program to get to the end goal?

Onno van de Stolpe - *Galapagos NV - Founder & CEO,*

Thank you, Peter. I have Walid with me here to talk about future trials. I'll give the word to him. I don't think we have, at the moment, a clear calculation yet on what the total cost were going to be to get this to approval. But with the amount of cash that we have, I don't think that's at all a concern to Galapagos. So whatever is going to cost we'll be able to carry it. With that, I would like to hand it over to Walid to talk a little bit about what's next with regard to this program.

Walid Abi-Saab - *Galapagos NV - Chief Medical Officer*

This is Walid Abi-Saab, I'm the Chief Medical Officer. So thanks for this question. I think we're very excited about these results as this was our first trial. We used, as often is the case, 1 dose to test the hypothesis and essentially see if there is activity. With these exciting data that we have to date, the next step will be to evaluate more than 1 dose in subsequent trials. The subsequent trials will be of long enough durations. I'm thinking at least 1-year long and we'll evaluate as I said more than 1 dose. It's good that we also had the foresight to interact as well with health authorities, I think I'm allowed to say that. And we have actually discussed with them already a study plan that is 52-week long, which would be robust enough to be considered a later-stage trial, and potentially pivotal. So that's kind of the news that we have to date. As you can imagine, the team is very actively working right now to fine tune some of these things. And get us ready for the next step, which will involve also a rapid start of the study, but as well looking at other potential studies and interaction with health authorities to have a full package that will allow us to move forward as quickly as possible. And I think you had a question earlier, which probably Piet will take.

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Yes. Thank you, Walid. So Peter I'll come back to the 2 patients that were temporarily stopped. So 1 of those patients was, again, our bad luck patient there with the diagnosis of cancer. He first had a temporary stop and then later was permanently withdrawn from the study. The other patient had a skin infection and was take -- sorry, a shin infection and was taken off medication for a couple of days and then back in on treatment. And then on the dosing, so the 600-milligram dose was selected because that was the dose that in Phase I give us in plasma 80% to 90% reduction of LPA. With lower dosages we get there at the max, but not at 24 hours. So for the next study we will include a lower dosage but there we need -- we will then have somewhat less of target coverage and we'll see how that then translates into clinical efficacy. Did I answer your questions?

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

Yes. That's great. But just to confirm on the next study doses. So are you saying -- so is the next study pivotal? Or is the next study likely to be a multi-dose finding study over at least 1-year Phase II, if you like, over at least 1 year? I guess, it's not quite clear to me, is the next trial going to be the pivotal study?

Walid Abi-Saab - *Galapagos NV - Chief Medical Officer*

Well, I think this is a rare disease. And as such, I think doing a 1-year long study that's adequately powered, even if you evaluate more than 1 dose, it could very well be a pivotal trial. I don't see why it wouldn't. Will it be the only pivotal trial? I don't think so. And that's why, I think we need to have a little bit more discussions and evaluating the program, but we're keen on first starting with that very important and pivotal study as soon as possible.

Operator

We'll go next to Sandra [Cowenburg], KBC

Unidentified Analyst

Very nice study. I was first of all, wondering if you see any other possible clinical indications besides IPF like opportunities in liver fibrosis, for instance? And secondly, I would like to understand a bit better, the compound's properties itself. So you already touched on plasma binding, but if you could remind us on the kinetics of the compound, brain penetrants, potency, selectivity, these things?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Okay, Sandra. On other clinical indications, so as we said before, we drive -- it's our strategy to do more than 1 proof of concept clinical study with every novel mechanism of action, so we are actively looking currently which is going to be the second indication. It will be too soon to report on that today but I can tell you, we are running a number of animal models. And over the coming months, we'll let you know which is the second indication. So then on the compound properties, in fact, it's a selective autotaxin inhibitor so it does not bind to any receptor selective in terms of enzyme inhibition as well. Orally well absorbed with a half life of 5 hours and does not penetrate into the brain as far as we know. So we know we have the complete 6-month chronic package for it and so that has been discussed with the authorities over the past month. And so as what it said, we are fully ready to move forward into the later stage clinical studies.

Operator

We'll go next to Christopher Marai, Nomura Instinet.

Christopher N. Marai - *Nomura Securities Co. Ltd., Research Division - MD and Senior Analyst*

I was wondering first, if you could comment on (inaudible) on a per patient basis, if the biomarkers any on function measures really trended in the same direction, that is, on a per patient basis. Secondarily, I'm wondering if you could help us perhaps understand if there are any reasons to suggest patients may have been unblinded to receiving therapy? And then finally, based on preclinical and other findings, maybe remind us of some of the on target side effects that you've been looking at (inaudible) of interest?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Okay. Christopher, thanks for the question on the biomarkers. So the full set of biomarkers , it is a large set of biomarkers, we are still analyzing and will report those at specialized scientific conferences. [That's part of] the call today. What I said is indeed, the plasma LPA was important for us to see that patients, we had the expected response in terms of inhibition of LPA synthesis. Then the second question on the blinding, no. There is no -- if patients take treatment, there is nothing specific that tells them that they are on active, it's not a central reading. And also, the sites that kept completely blind in there so there's no reason -- and that's where we monitor centrally, so there's no possibility that they were unblinded. And the final question was on, oh, only 2 questions...

Christopher N. Marai - *Nomura Securities Co. Ltd., Research Division - MD and Senior Analyst*

So, just -- yes, they were regarding based on preclinical and other findings as expected on target (inaudible)? What may have you expected, anything of interest that you're watching right and then going forward? And I have a follow up.

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Yes. Well, the bigger picture of the autotaxin is in fact, quite clear. So we've seen in line with what we know is that this will be used in pregnant women, that's excluded so that there are no specific changes that are included. Our measurements are included because of the clearer package.

Christopher N. Marai - *Nomura Securities Co. Ltd., Research Division - MD and Senior Analyst*

And then just thinking about the potential next trials and potential arms. Would you be looking at trying this on top of currently approved therapeutics for IPF? Does that make sense mechanistically? Do you expect to see synergies? How should we think about the potential for this to be used in combination with currently available treatment?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Walid?

Walid Abi-Saab - *Galapagos NV - Chief Medical Officer*

Sure. Thanks, Piet. Yes, I think you have to follow both the science and the unmet medical need. As you heard from Onno, this is a disease that's pretty severe, about -- the median survival is about 2 to 5 years after diagnosis. So you have to first evaluate it as quickly as possible on top of -- but at the same time, I think our data and the initial trial were as a stand-alone. So I think going forward, it will robustly evaluate both populations. That's kind of how we're thinking about it right now, whether that will be done in the same trial or on 2 separate trials remains to be decided. But again, I think it will be driven mostly by the data and the unmet medical needs.

Christopher N. Marai - *Nomura Securities Co. Ltd., Research Division - MD and Senior Analyst*

Great. And then folks, finally, data venue, potentially sort of presentation of the results, and that's my final question.

Walid Abi-Saab - *Galapagos NV - Chief Medical Officer*

Where will be present the results?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

We will present the data at ATS in May, next year.

Operator

We'll take our next question from Vamil Divan, Credit Suisse.

Vamil Kishore Divan - *Credit Suisse AG, Research Division - Senior Analyst*

Just a couple on the patient population here, and you showed the demographics on Slide 10. Just I noticed, there's fewer males, more never smokers and then also higher baseline FVC in the drug group. So how do you think about that sort of the amount of (inaudible) studies? I'm just trying to get a sense of is that of any concern or how do you think about that -- those issues?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Thanks, Vamil. Well, it's in view of the number of patients we have in the study total and 1 to 3 randomization, I think, the differences between the two groups are perfectly acceptable to come to the right conclusions here. So we're not worried at all that there is a difference that could drive any of our efficacy outcomes. I told the total question, yes.

Vamil Kishore Divan - *Credit Suisse AG, Research Division - Senior Analyst*

Okay. And my second question is built on one of the earlier questions which is in terms of the future development plans. So what would you -- like, what's your goal here in terms of the late stage trials. Do you need to show superiority over the currently approved agents? Or if you can just show superiority of your placebo with a better safety profile, would that maybe cross-trial comparison suggesting that (inaudible) procedure? Is that sufficient? Just trying to get a sense of how you view the -- what you need to show to be successful commercially.

Walid Abi-Saab - *Galapagos NV - Chief Medical Officer*

Yes, I mean, as I mentioned before, I think this is a severe disease. So I think you want to attack it with whatever tools you have. So I think the most logical step first is to go on top of what's available out there. At the same time, we want to evaluate it as a stand-alone. And then based on these data, if it makes more sense for the drug to be used by itself because it has a much better efficacy and safety profile, then that's -- ultimately that's what will guide us to where we need to go and that will be -- the medical care will be shaped by that. If it turns out that actually, patients do benefit when you add it on top of what's available out there, then I think that's probably the better way to go for the patient. Remember, they have a median survival of 2 to 5 years, so we don't have a lot of time to try things with them. You want to hit it with as much as you can to try and slow the disease progression. We will be guided by the data. I think these are very good questions and ones that we're asking ourselves, but we need to generate more data with our molecules to be guided as to what will be the best bet.

Operator

We'll go next to Dane Leone, BTIG.

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

So a few for me in the same vein as some of my colleagues. First, I guess, is there -- can you just maybe give us an understanding of whether there would be a different expected disease progression over a 3-month study for patients that have had IPF for 1 year versus 1.9 years? Specifically, I'm referring to the balance in the baselines of these characteristics.

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Dane, I think within 12 weeks you don't expect any differences there. So the median survival is limited to a number of years but then you need to -- if you want to see the difference, you need a study over a number of years. So I think if you're measuring for the first 12 weeks that does not make a difference.

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

Okay. And then the next question for me would be, when you're thinking about the pivotal studies that have asked, I think we're all trying to understand whether you think you would need to use an active comparator arm versus what you've used as a placebo in this trial. To a certain degree, it might be an ethical question, I guess. And any thoughts there?

Walid Abi-Saab - *Galapagos NV - Chief Medical Officer*

Yes. I mean, again, this is Walid. I think this is the first thing that we're going to be going after quickly is an add-on on top of standard of care which includes nintedanib or pirfenidone. So in that case, that would be a straightforward. We will be looking to study the drug again in the same population that we have studied now compared to placebo. And those will be in places where probably neither pirfenidone nor nintedanib are

available or people are waiting to get it or maybe they're intolerant to either one of them. So we will evaluate things in that way and then we will decide as to what would be the next step.

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

I'll wrap the last ones up together here. So with this -- just a follow on that. Would this be the type of, given it's a rare disease and there could be active comparators where a non inferiority study would be possible? Or do you think regulators in terms of your early discussions with if you're looking at an active comparator arm would want to see statistical superiority across some of the measures? And is there -- I thought it was generally kind of standard to look at the 6-minute walk test as well. Is that something that we would see in later studies or add-on studies to the current one?

Walid Abi-Saab - *Galapagos NV - Chief Medical Officer*

Yes. So for the concept of the non-inferiority study or non-inferiority in a tiered analysis, looking at a superiority also is a potential study design that we are starting to think about. But this was not part of our initial discussion with the regulators so that would be part of the next time we meet with them based on these data. The 6-minute walk test and any other functional -- and other functional endpoints will be included in the next study, yes.

Operator

We'll go to Phil Nadeau, Cowen and Company.

Philip M. Nadeau - *Cowen and Company, LLC, Research Division - MD and Senior Research Analyst*

A couple on the FVC data. It looks like the follow-ups in the placebo group happened between weeks 0 to 4 and then there was a stabilization in FVC in the placebo group from weeks 4 to 12. It's a bit of a different pattern than we've seen in the other trials. Did anything happen in the placebo group in those first 4 weeks? Did any patients get sick or anything to kind of explain the relatively rapid fall off in stabilization.?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

I think within this 12 weeks study and I think it's a message we try to bring over for both the active group and the placebo is that at 12 weeks is I think the placebo is exactly where we expected in this population. So you expect the placebo group to see an FVC decrease between 50 ml and 100 ml. So with a measured 88 ml, you are exactly there, so we have included the right patients to watch them week by week or month by month. I don't think that makes lots of sense also.

Philip M. Nadeau - *Cowen and Company, LLC, Research Division - MD and Senior Research Analyst*

And then the second question, in the biomarker evaluation, there were 21 patients at the 12-week data point and then the FVC evaluation, there were 17 patients. What would the day look like if you had all 21 patients who were evaluable at week 12? How would the curves differ?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Well, we are calculating here bit so the effect we've used for FVC at week 12, the available data and not impute any of the previous measurements. So if you did FVC, there was a quality check whether the FVC did fulfill. And that's the only right way. And we then check as well the rate of broncho dilated just before they do the measurement and exclude those values. So the only correct way of looking to that and that's how we did it, This was predefined like that (inaudible) and juggling around with the what-ifs. We didn't spend our time, honestly. In the PD measurements, we see

-- well, 15 on active, Some measurements simply don't work, our samples got lost, but there as well with those 15 patients, we feel pretty comfortable that those numbers reflect what happened in the total population.

Philip M. Nadeau - *Cowen and Company, LLC, Research Division - MD and Senior Research Analyst*

On the FRI measure, I guess, I'm a little less familiar with that measure. Can you give us some sense of what the magnitude of changes that you saw? How meaningful those are, like a 3 ml per liter change in airway volume or 0.03 KPA per second change in airway resistance? So there were big changes, small changes? Again, I apologize. I'm just not familiar with that measure.

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Well, that's normal. FRI is new. The company Fluidda had generated a number of data in IPF patients. Honestly, the first time that they see even the follow up of patients over time, the stabilization of the area volume is the first time they see the stabilization of the resistance so they were as well, quite impressed with what they've seen. And those numbers, they -- well, they change over time. So over 12 weeks it's a limited difference but it's a more sensitive parameter and the other parameters you expect to move fast. And that's why we're so happy that they fully confirm the picture what we have seen on FVC.

Philip M. Nadeau - *Cowen and Company, LLC, Research Division - MD and Senior Research Analyst*

Great. And then just last question for me. The week 12 FVC difference between drug and placebo, based on the slide, it doesn't look like it was statistically significant. What was the p value? Was it was close to 0.05?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

I don't have it by heart because the trial was not powered to measure this, there's quite some variability there but it's clear that it was not close to 0.05.

Operator

We'll go to Adam Walsh, Stifel.

Adam Anderson Walsh - *Stifel, Nicolaus & Company, Incorporated, Research Division - MD and Senior Analyst*

My question goes back to Slide 13 and the FVC changes that are shown there as mean changed from baseline. I was still trying to get a better sense of this version for individual patients. Could you perhaps, provide a little bit more granularity on those individual responses? Perhaps give us the range of responses in terms of the drug arm, what was the best responder and perhaps, the worst responder? Anything you could give us to get us a little bit more granularity on how the actual individual responses played into the mean response you're showing on the curve.

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Thanks, Adam for the question. So it's a great question. What I know is what we did was really code for every patient over time, the decrease per visit and then when there was a decrease of FVC it was red and if there was an increase or no change, the color green. And I can tell you that the majority of the patients on active really were always in the green area. So when we have patients improving they improved over time and that stays like that. So as a total is around zero. Most of the patients really were in the green while in the placebo it was all red color so all of the patients there more or less show a drop of FVC consistent over the study as well. It's not that if you take month-by-month, just so variable that you can't come to a conclusion, it was a consistent picture for all of the patients over time. Thank you.

Adam Anderson Walsh - *Stifel, Nicolaus & Company, Incorporated, Research Division - MD and Senior Analyst*

That's helpful. And then just 1 other more theoretical question here. I know that literature have shown that autotaxin appears to produce the majority of LPA, but some literature sources note that several other sources of LPA are known to exist. And I guess, my question is, whether or not you believe the other sources of LPA production are important. Could they perhaps lead to a waning of effect of 1690 over time? Just help us understand how the other sources of LPA production might play into the efficacy of this drug over the long term.

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Great question, and thanks for asking. So indeed, autotaxin is considered to be responsible for 80% of the production of most LPA species. We've done especially around the (inaudible) of autotaxin as a main driver is also other enzymes that produced. They mainly produced the longer chain LPA (inaudible). I must say that both in the Phase I and now in this study, we see a consistent drop of LPA species. We don't see a second source taking over. So as we've analyzed those data as well quite carefully put patients over time doing Phase I and as well as part of the study which we don't see -- well, we can only speak for us as far as we've mentioned during in the healthy volunteers over 2 weeks we did not see any. And the same (inaudible) study, we don't see any decrease of LPA or LPA species over time in the study. So we don't see any reason to believe that the second source of LPA takes over and would then in longer-term hamper the efficacy or 1690.

Operator

We'll go next to Tim Woodward with Goldman Sachs.

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

Could I ask you to remind us where you are on your IP position please? And where you think this could be passing protected too? Also, where you are on the manufacturing and what more work you might have to do on the tox side. And then also, are you -- do have orphan designation for this yet or should we expect that imminently from the FDA? I know you have it in Europe and then what do you think your chances are of getting fast tracked around or breakthrough?

Onno van de Stolpe - *Galapagos NV - Founder & CEO,*

Okay. I'll start with that. We have orphan designation both in the U.S. as well in Europe. For the patent, we have protected compound of matter we file for the results in this trial as well. It looks to be all clean.

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

And then in terms of the manufacturing, we are ready to supply the upcoming study there. So we have tablets available. We've done the Phase I to show they are bio equivalent to the capsules we use. So all of that is well under control and we are ready to move to the next steps of the clinical development. We have the clinical tox package discussed that with the authorities. So all of that have been reviewed as well. So we don't see anything stopping us to move forward here.

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

Is there anything you can say on prospects for fast track or breakthrough?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Fast track approval. Walid, I'll leave that over to you.

Walid Abi-Saab - *Galapagos NV - Chief Medical Officer*

We are evaluating all these options now based on the data. So I think it's premature for me to take a position on it. We're still discussing.

Operator

(Operator Instructions) We'll go to Peter Welford, Jefferies.

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

Just back on Slide 13 for minute, a famous slide I think of FVC. Just wanted to ask you about the placebo patients of weeks 8, 12 and then in the follow-up. Can you just confirm, are these the same 4 patients that were measured at week 8, week 12 and then also in the follow-up? And I guess, there's a reason for asking because there's obviously quite a deterioration in FVC during the 2-week followup from week 12 to what I presume you call week 14. And I wondered if you could comment, I guess, on that. And also, the erosions of the FVC we see 1690 in that the 2-week follow-up of 55 or if you consider potentially put that I guess, in context for perhaps when the patients come off drug?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Okay, thanks for the questions on the FVC. So as I said before, the FVC may be measured at every visit, but there is a quality assessment there on the one hand side and then secondly is checked patient will not take bronchodilator just prior to the measurement. And so when we have N=4, N=3, these are the number of values that passed on the quality check. They're not always the same patients, so especially for the quality check, this is valuable per patient over time, so (inaudible). Then the erosion after stopping treatment, I can only speculate. I don't like to speculate in calls here, so (inaudible) what I think you should take away a drug that stabilizes lungs. Okay, you might expect something whether the amount we see indicates something that's a bit too difficult for me to answer today.

Onno van de Stolpe - *Galapagos NV - Founder & CEO,*

And then an addition to Tim's question regarding the IP. We have a combination of matter patent 'til March 2034, and that can be extended with 5 years. So we have a very long patent life on the compound itself.

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

Sorry, I didn't (inaudible) but just a quick question on the placebo arm, I guess. The patient that permanently stopped treatment, the 1 patient there. When did that patient on the placebo arm permanently stop?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Looking to my -- he stopped day 77, the placebo patient. That was day 77.

Operator

We'll go next to [Alan Voloski], Roth Capital.

Unidentified Analyst

Any comments on the recent [pamrebluman] results from [Fibrogen]? And perhaps in the context of BTGF versus autotaxin as a target?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Okay, thanks for a great question and the congratulations. Well, our mission here as scientists and as a company is to improve the life of patients. So the fact that also other approaches show promising data, we are enthusiastically about that and the more options for patients the better on long term. It's impossible, I think, to compare autotaxin and CTGF. We've published a post which is on our website which shows that by blocking autotaxin, we in fact downstream on the CTGF level so one you can -- and I want to refer to that post to show that those approaches are not coming from a different planet. It's more we try to tackle the disease effect the same way which you could consider from a similar angle.

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

Operator, do we have any other questions?

Operator

There are no other questions at this time.

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

All right, thanks. We had a great question-and-answer session. So I'd like to thank everyone who participated and I'd particularly like to thank our shareholders for their support all these years in helping us to get to our second success out of the platform. Paul van der Horst is in Europe and I in the U.S. will be available after this call to take any additional questions you might have. Our next financial results webcast will be on the 27th of October when we present our Q3 '17 results, and we look forward to speaking with you then. Goodbye, and thank you.