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EDITED TRANSCRIPT

GLPG.AS - Galapagos NV DARWIN 2 Study 24 Week Final Results Call

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PRESENTATION

Operator

Good day, ladies and gentlemen and welcome to the Galapagos webcast. At this time, I'd like to turn the conference over to Elizabeth Goodwin. Please go ahead.

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

Well thank you and welcome, everybody, to the audio webcast of our DARWIN 2 final study results. I am Elizabeth Goodwin, Head of Investor Relations Corporate Communications.

This webcast is accessible via the Galapagos website home page and will be archived for one year starting later today.

So that your questions can be included we request that you call in to the telephone number given in today's press release. For those who don't have that at hand I can tell you that you can call 32 for Belgium, 2 404 0660 and the access code is 197 38 80.

I'd like to point out that the audio over the telephone runs ahead of the slide transitions on the site. To address this problem you could download the slides from the webcast player or simply listen to the audio of the webcast itself, which is completely in sync with the slides being shown.

I'd like to just remind everyone we'll be making forward-looking statements during today's audio conference. These forward-looking statements include remarks concerning future development of the 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.

We would especially like to emphasize that later research results could differ materially from the final results presented today for the DARWIN 2 study.

Today's speakers will be Onno van de Stolpe, Chief Executive Officer of Galapagos and Dr. Piet Wigerinck, Chief Scientific Officer. Onno will give a few introductory words and then Piet will take you through the DARWIN 2 final results data.

You will see a PowerPoint presentation on screen during this talk and we estimate that it will take about 30 minutes. This will be followed by a Q&A session with Piet and Onno.

So at this point, I'd like to hand over now to Onno to walk us through the introduction.

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Onno van de Stolpe - *Galapagos NV - CEO*

Thank you, Elizabeth. It's a pleasure to give the introduction today with the final data set of our DARWIN study, but before we dive in, a brief overview of the highlights of this Company.

Clearly, filgotinib, a JAK1 inhibitor in RA and Crohn's disease is prominent in our pipeline, but it's not a one-trick pony here. We've got a very interesting cystic fibrosis program with a number of molecules moving forward towards a triple-combo therapy.

We also have Phase 2 programs in inflamed bowel disease and idiopathic pulmonary fibrosis.

All fueled by a pipeline in which we bring novel mode of action targets into discovery and towards the clinic. This is all backed by strong financials and partnerships with AbbVie and other partners.

So clearly, a company that has a broad pipeline and has a very good basis to further grow out into an independent biopharmaceutical company.

If we then go to filgotinib and see the history of this molecule, you can ask is there still an opportunity for new drugs in the RA space? Well, in our view there clearly is. If the molecule is safe, if it's effective, if it's oral, clearly there's a great need for new molecules here that can help the lives of patients.

The two key aspects of new treatment should be: it has to be very safe; it's a chronic treatment; and it has to be very efficacious. We believe filgotinib meets both.

If we look at the history of filgotinib it goes back all the way back to 2005 when Galapagos discovered JAK1 as the target for rheumatoid arthritis.

We then embarked on a long journey of finding a molecule that bound to this target, that we optimized and designed, and ended up as filgotinib, that we moved into the clinic first Phase 1, and then three Phase 2 studies and that we have now concluded, after 10 years, with these DARWIN studies.

The molecule is now ready to go into Phase 3 in rheumatoid arthritis, which will start in 2016.

Clearly, we are also excited about the opportunity of filgotinib in other inflammatory diseases. We are ongoing in our Crohn's study, fully recruited, waiting for the data by the end of this year; and other inflammatory diseases in the future to be initiated.

At the moment, the clock has started ticking for AbbVie to take the license on this molecule and we are awaiting their decision in the coming months.

We are confident that they will execute this license, because of the data that we have shown previously and that we're showing today.

All in all, it's a fantastic story from 2005 to 2015 on the development of a molecule, all the way from a target discovery to completion of the Phase 2 package.

With that, I would like to hand it over to our CSO, Piet Wigerinck, to talk us through the DARWIN data. Piet?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Thank you, Onno, and all welcome to this call. It's a pleasure to present the DARWIN 2 24-week top-line results on behalf of a big team.

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This presentation will complete the Phase 2b top-line data flow in RA with filgotinib. The data nicely confirm the safety and efficacy observed at week 12 in DARWIN 2. And, similar to the 24-week data in DARWIN 1, will as well show a further increase in efficacy on the more difficult RA endpoints, like ACR70 and DAS28 remission.

But first of all, we believe that it's important that we work with the selective JAK1 inhibitor. The data of today will confirm that selective inhibitor JAK1 is a way to get to very high levels of efficacy, also as a monotherapy.

Secondly, the data will also confirm that with sparing JAK2, we allow the patient to benefit optimally from the reduction of inflammation by showing a positive impact on hemoglobin within the first week of treatment.

On the opposite side of this selectivity, it has been proven that by blocking JAK2, there is a clear increased risk of generating anemia, a risk we don't need to worry about with filgotinib.

Before showing the 24-week data, let me start with repeating our conclusions at week 12, the primary endpoint of the DARWIN 2 study.

We and many people with us were impressed by the fact that also as a monotherapy filgotinib shows a fast onset of action combined with high efficacy illustrated by the high ACR20/50 and DAS28 responses.

Also, the safety data were reassuring. In the absence of methotrexate the picture was clear: the low frequency of SAEs and AEs; an increase in hemoglobin; the absence of an effect on the lymphocytes or livers. All of these added to our high confidence in the different safety profile of filgotinib.

So before we go to the data, how did competitor molecules perform as a monotherapy within a 24-week setting? The monotherapy setting has been less explored during Phase 2, so fewer comparators are available.

Also, I wanted to remark that you can see that in the more recent studies the middle and the right studies, 2010, 2012, there is no placebo group any more post-week 12. So we still can do these trials as a monotherapy, but the placebo group stops at week 12. As a consequence, we can't calculate a significant difference at week 24.

For the anti-TNFs, like adalimumab, monotherapy leads to moderate levels of efficacy only.

The more relevant competition in these settings comes from the anti -- IL-6 antibodies, like tocilizumab. Tocilizumab in this Phase 3 trial reached in ACR20 in the high 60%; in ACR50, around 40%; and an ACR70 score between 20% and 30%.

Looking to the oral drug, tofacitinib, behaved similar on ACR20 and 50, but clearly has a lower ACR70 score.

So with the 24-week data, the key question for us was do we again see an incremental increase in the ACR70 score between week 12 and week 24 and can we get over the 20% hurdle.

A quick reminder on the design of the DARWIN 2 study. We included exactly the same patient population as in DARWIN 1. Patients with active RA defined by the same criteria. Big difference here, patients were actively washed out from the methotrexate at least four weeks prior to enrolment. Similar to DARWIN 1, patients could have not been on a biologic, so they are biologic naive and they are DMARD non-responders.

So this slide shows what happened to the different patient groups during the study, as defined by the protocol.

At week 12, by the protocol, all the patients in placebo group in gray were switched to a new group receiving a 100-milligram daily dose, in dark blue. This group is shown in dark blue and will be discussed as a separate group in the safety section.

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Fifteen patients that have started on the low dose of 50 milligrams once a day did not achieve 20% of improvement on tenderness on joint counts and therefore were, after 12 weeks, switched to a 100-milligram dose group, represented in light blue.

As this is a small group and did not contain any additional relevant safety data, we will not include this group in the safety report.

For efficacy, I will show one slide on these two new groups.

Let's go to the baseline demographics of DARWIN 2. The slide is a repeat of the previous webcast and illustrates that we have included patients with a highly active disease, illustrated by a DAS28(CRP) score of higher than 6. Secondly, the slide as well shows that the groups were well balanced.

Over to the data now. Let me first explain the layout of the slide. On the top line here, we have the data between week zero and 12 and the four groups. Below, we have the data between week 13 and week 24; and the placebo group now the same group has been switched to the 100 milligram.

The 50 milligram, those group is still there, but in the second part we only report on 52 participants, as we have left out the 15 that were switched. The 100 mg and 200 mg from weeks zero to 12 are the same in week 13 to 24.

During the previous update, at the primary endpoint at week 12, we remained blind for the different groups and only had access to the overall numbers. So now we can see also the exact numbers for every dose groups during the first 12 weeks.

First of all, the early discontinuation rate is low. We report here number of patients and of percentages. Seven patients dropped out in the placebo group, and this is the highest number we see in the full table. Four of those dropped out -- four of those seven dropped out for safety reasons.

All in all, for the filgotinib groups, we see low numbers and we see no dose response. For the second half of the study we see the same picture, low numbers in all the groups.

So in total for the study, 26 patients dropped out. This corresponds to 9% of early discontinuations, which is a low rate for a 24-week double-blind, placebo-controlled, dose-finding study and similar to the data reported by baricitinib.

The points to remember for the early discontinuations are few patients dropped out in filgotinib over 24 weeks, and we see no dose response.

Let's now have a look at the efficacy. First of all, I start with ACR20 and I show the performance of every group over time. We use the most conservative analysis here; they intend to treat NRI methods.

At week 12, the placebo group stops. As a consequence, beyond week 12, we can't calculate a significant difference with placebo and we don't have stars any more.

With dose, we see a nice effect on speed to get to ACR20, especially during the first 12 weeks, stabilizing thereafter. The 100 milligram group catches up with the 200mg daily group by week 16.

The 100 and 200 milligram groups perform almost identical during the second half of the study, with a response rate of about 70% of patients reporting 20% improvement in signs and symptoms.

At week 24, surprisingly, the two curves flip and we believe this is an artefact of the study rather than a [lead signal].

In the second half of the study, it's clear that the low group performs suboptimal, and so we consider the 50 milligram dose group as being suboptimal for this patient population.

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Let's now look at the final scores of ACR20 and we compare week 12 with week 24: on the left of the slide, the data at week 12; on the right, the data at week 24. As there is no placebo group, again there are no stars that we can calculate at 24 weeks. Both the high and the mid-dose group reached stable ACR20 of about 70%, a very nice result for a once-a-day oral monotherapy.

Over to ACR50 now. Patients that reach ACR50 experience a drop in signs and symptoms of at least 50%, a clinical-relevant hurdle to claim efficacy in the current competitive landscape.

Filgotinib already scores very high on ACR50 at week 12. Up to 43% of the patients in the 200 milligram once-a-day group did report at least 50% of improvement. At 24 weeks, the 200 milligram dose group confirms the excellent efficacy and also the 100 milligram group jumps over the 40% efficacy bar. These data further reinforced the efficacy as we reported earlier at week 12.

Let's now go to ACR70, the most difficult hurdle in terms of reaching efficacy. For all active groups, here we see a clear increase in response between week 12 and week 24. Both the 100 and the 200 milligram groups score clearly over the 20% hurdle.

Again, also in the monotherapy setting, filgotinib scores in terms of the highest efficacy amongst the drugs for RA.

As a conclusion, all ACR responses together at week 24 on one slide, the dose groups, 20, 50, 70. Filgotinib exceeded the efficacy expectations we defined upfront. It is the first one-pill-a-day monotherapy to show strong efficacy results in these advanced and severely affected RA patients.

In this study, we already achieved a maximum response defined as ACR with the 100 milligram dose group.

Over to DAS28, which is a complementary way of making efficacy using continuous scale. By design again, the placebo drops at week 12. The graph showing the change from baseline confirms our previous observation, fast onset of action within a week.

There's also further improvement after week 12 for all the active groups. The 100 milligram dose group catching up with the highest dose group by week 20.

At week 24, very robust responses for the 100 and 200 milligram daily doses again, showing that the one-pill-a-day treatment is a realistic option for these patients.

As a reminder for the next slide, please note that the 100 milligram group has a DAS28 drop of 2 points at week 12.

Let's now go to the next slide, where we have a single slide on those two new groups in the trial, on the one hand side, in dark blue, the placebo switches to 100 milligrams. And in light blue, the 50 milligram non-responders that get a double dose.

As you can clearly see from this slide, as soon as they were switched therapy, we see a much better response defined as DAS28(CRP). Also, within 12 weeks, the previous placebo group reaches the same efficacy as the 100 milligram group reaches during the first 12 weeks.

So a reinforcement of the 100 milligram group and as well an (inaudible) for these patients 50 milligram was clearly suboptimal.

Let's go to the next slide. How do the DARWIN 1 and DARWIN 2 data compare? Honestly, as they are separately trials, we can't make a correct comparison, but as we have the data, as shown here, the ACR50 responses in both studies and they look similar.

As a conclusion, both with but also without methotrexate, filgotinib is well positioned to become an excellent treatment option for RA patients, illustrated by the high efficacy rates at ACR50 on this slide.

DAS28 as well allows us to measure to what extent patients have few or almost no symptoms any more. Let me remind you that at the beginning of the study, these patients had a highly active disease with a mean score of bigger than 6.

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This is a quite complex slide, but let me guide you through this data. We start with the green bars and the green bars indicate the number of patients that achieved the lowest level of disease or ended the study with a DAS score below 2.8 as a percentage, the level defined for remission.

In practice though, these patients are almost symptom free with these scores, from almost 20% to up to 30% for the high dose. So in the severely affected patient population, the single-pill therapy with filgotinib induces remission in close to 30% of the patients; an impressive result.

If we add the orange and green bars together, we have all patients that achieve a DAS28 score below 3.2, or the definition of low disease activity. For patients, this is an important threshold, as they feel quite good, but also regulators in Europe ask us to report this as a critical endpoint.

Again here, the mid and high doses reach up to 50% response; a very nice result, and again, achieved as a single-pill monotherapy.

This slide as well illustrates the nice increase in efficacy starting from week 12 over to week 24 on this difficult endpoint.

So, as a conclusion, the data bring the field a big step closer to a one-pill-a-day monotherapy for this advanced patient population.

Over to the safety endpoints. For clarity again, we use the same layout with on top week zero to 12, below week 13-24 and the groups that are the same.

First, the first 12 weeks and including placebo. This slide reports on treatment in emerging events and you can see that we have a similar number of overall number of groups during the first 12 weeks; all compatible to placebo as well.

The frequency dropped of -- the number of patients reporting adverse events drops during the second half of the study. Again, no dose response visible there.

Looking to the SAEs as well, we have low numbers of SAEs across those groups and an absence of dose response.

Concluding with the serious infections, a few isolated cases reported over the various active groups.

Let's move to the slide on adverse event of special interest. In DARWIN 2, we had no cases of MACE, or major adverse cardiac events. We again see a slightly high incidence of infections, which slightly related to the mode of action. Again, we don't see an apparent relationship with those.

Special cases, we had one case of pneumonia at the high dose and one herpes zoster at the low dose. The increase is mainly driven by a slightly higher incidence of urinary tract infections and upper respiratory tract infections. For the rest, we see a scatter of all types of infections.

Important to note is that we had no cases of opportunistic infections; no cases of TB; no malignancies, meaning lymphomas; and none of the patients died in this study.

Over now to the relevant lab parameters; let me start with the hemoglobin levels.

On this graph, we show the evolution over time per dose expressed as mean percent change from baseline. Consistent with the blood sampling at the start of the study, we see a drop of hemoglobin during the first four weeks in three of the four groups. It takes about 12 weeks for full recovery in the placebo group.

Fully confirming observation DARWIN 1, for the high dose, we don't see any values over the whole study below the baseline. We only see increased levels. The values increased up to 4%, which is a level similar to increases reported in studies with the anti-TNFs.

So the increase in hemoglobin is a good thing, and is something that should happen under every good RA treatment. That's what we observe with filgotinib as well in DARWIN 2.

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The effect on hemoglobin is not limited to the high dose. Within four week, all patient on filgotinib have a net benefit from their therapy and have increased levels compared to baseline.

So in DARWIN 2, we have a nice confirmation of the clear advantage of the JAK1 selective [inhibitor].

The body can compensate within the first week the drop in [our] blood cells. Meaning in contrast to non-selective JAK1 inhibitors, the JAK1 selective inhibitor, filgotinib, does not put any breaks on the system and the clinical outcome is that the patient immediately and fully can benefit from the reduction in inflammation.

Let's now move to the lipids. Lipids is the last parameter where we see a slight difference between DARWIN1 and DARWIN 2. On the left, the percent changes in HDL and LDL in DARWIN 1; on the right in DARWIN 2.

In DARWIN 1, at week 12 already we had -- after four weeks already we had the stable increase of HDL and an increase which was about 2 to 3 times higher than the percent increase we saw in LDL. This picture in DARWIN 1 is clear and stable over time.

In DARWIN 2 on the other hand, both LDL and HDL show a similar increase on a percentage basis.

While in DARWIN 1 we could hope for a clear benefit on a cardiovascular risk factor, in DARWIN 2 we have unusual picture here. Where the difference comes from? We don't understand and we're looking into all the details we have gathered over the recent weeks and will continue to study that.

This brings me to a summary on the lab parameters. All in all, we have a very similar picture in DARWIN 1 and 2, allowing me to conclude from both studies together.

Thanks to the JAK1 selectivity, hemoglobin increases in a dose-dependent way up to 4%, a level also observed with anti-TNF treatment. We see no effect of filgotinib on lymphocyte. This is good, as lymphocyte protects from infections.

Similar to other JAK inhibitors, we see a small increase in creatinine. For both neutrophils and platelets, we see a small and well-controlled drop towards the mid-normal values. Both parameters are stable after a few weeks of treatment.

We have no safety [seal] on the liver. Important as well, we did not observe any safety seal on the male hormones in any of the two studies.

Finally, on a percent basis, there's still a higher increase observed on HDL compared to LDL over the two studies.

This bring me to the conclusions for the DARWIN 2 study, and let me start with efficacy. Filgotinib is an oral once-a-day pill therapy that shows a very rapid onset of action. We have seen a clear dose response and we have seen a sustained high level of efficacy measured at ACR20 and 50.

During the second part of the study, we have also seen a further gain in activity on the more difficult endpoints, like ACR70, remission and low disease activity.

More importantly, DARWIN 2 shows that a single-pill-per-day treatment generating a very high level of efficacy becomes a realistic option for many RA patients. With or without methotrexate, filgotinib works well.

Conclusions on safety on the next slide; we are pleased and encouraged with growing safety profile as well. The low rate of drop-out SAEs and serious infections gives us a lot of comfort.

On more safety endpoints, filgotinib groups scored comparable to placebo. The absence of any dose dependence on observed infections is also reassuring.

Finally, the differentiation we saw at week 12 versus other JAKs is now fully confirmed.

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So most importantly, with this final 24-week data from DARWIN 2, we are a big step closer to a safe and efficacious single day -- single-pill-a-day oral treatment for many RA patients.

Thank you.

Onno van de Stolpe - *Galapagos NV - CEO*

Thank you, Piet. It was a pleasure to hear these data presented, concluding the full package of the DARWIN 1 and 2 studies. But this is not ending the news flow and activities on filgotinib.

In the next two months we'll get the decision from AbbVie to license the molecule, take over filgotinib into further development in Phase 3 in RA and other indications.

At the moment of licensing, AbbVie will take over all cost, will pay us an upfront license fee of EUR200 million, and we are eligible for further downstream milestones, as well as royalties.

In the meantime, we are continuing with the long-term extension study, DARWIN 3. We have an incredible rollover percentage of patients after DARWIN 1 and 2 that continued on the drugs, so we're building up a large database of safety and efficacy data in DARWIN 3. There are already patients with over a year of filgotinib drug in that study.

We are looking forward to see the start of the Phase 3 in RA in 2016, being it by AbbVie or alternatively, if AbbVie would not take the license, by Galapagos.

And we are looking forward to the FITZROY study data, the Phase 2 in Crohn's disease, where we'll see the 10-week primary endpoint readout by the end of the year and the full completion of the study in Q1 2016.

So, a lot more data to come for filgotinib in the coming months.

With that, I would like to thank the patients, the investors, AbbVie and especially, of course, the Galapagos team, who has worked very hard of getting all this data together and presented to you.

I want to congratulate the team with the excellent results that we have been able to present to all of you; DARWIN 1, the DARWIN 2, the 12-week and the 24-week data sets. It has been a fantastic couple of months on providing that data, and I want to thank everybody who worked so hard to get that data set to us.

With that, I would like to hand it over back to Elizabeth for our Q&A.

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

Great. Thank you very much. That concludes the presentation today. I'd now like to ask our operator, Suzanne, to connect us to any callers who might have questions.

QUESTIONS AND ANSWERS

Operator

(Operator Instructions). Phil Nadeau, Cowan & Company.

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Phil Nadeau - *Cowan & Company - Analyst*

First, just one on the safety profile. Thanks for the mean values in the safety parameters. Were there any outliers in either creatinine, neutrophils or platelets, or were the changes relatively consistent throughout the population?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Thank you for the question. As you can see on the low dropout rate we had in the study, so if the dropout rate is low then that means as well that we had very few or no outliers. So the drop we've seen was a consistent drop of the limited consistent drop over the whole group.

Phil Nadeau - *Cowan & Company - Analyst*

Okay, great. That's helpful. And then second, on the dose. I appreciate your comments that the 50 milligrams seems not optimal for this patient population. How are you differentiating between the 100 and 200? Would you plan to move both forward as a monotherapy dose or is there one that you're favoring over the other?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Hello. This Phase 2B study at 24 weeks 100 equals the 200. That's illustrated by both the ACR50, ACR70 and DAS28 score. We are still fully analyzing. There is a clear advantage of the high dose in the onset of action, so with the higher dose you get to efficacy earlier. That is clear. But if you look to 24 weeks, they are the same, and so we are analyzing what is now the best of these two to take forward.

Thank you.

Phil Nadeau - *Cowan & Company - Analyst*

Great. Thanks for taking my questions and congratulations, again, on the data.

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Thank you.

Operator

Jan de Kerpel, KBC Securities.

Jan de Kerpel - *KBC Securities - Analyst*

I have two for Piet and one small one for Ono.

First, Piet, you seem to stress on the once-daily dosing, but if you compare the results also with the DARWIN 1, on twice daily it seems that twice daily dosing reaches higher results. How do you look at it? Is there any reason why you shouldn't go forward with the bi-daily dosing?

I'll take that one first.

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Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

All over is indeed, Jan, the numbers of BID are higher in terms of efficacy. That's the study in combination with methotrexate done, so the comparison with DARWIN 2 is more difficult there.

BID sometimes is, in terms of compliance, [later] for patients more difficult, that's why most groups prefer to go with the once a day. But again, there we are still analyzing and making up our mind, do we really see a difference? Does the BID only help us to get to efficacy quickly and later not any more?

So we're not there yet that we are going to say it's going to be either QD, either BID. But, in general, there is a preference from patients to have a once-a-day treatment.

Jan de Kerpel - *KBC Securities - Analyst*

Okay, thanks. Then secondly, in the DARWIN 1 you saw five herpes infections. Now, you see one in DARWIN 2. Do you think this will be a point of attention, these herpes infections?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Thank you for the question on the herpes infections. First of all, in DARWIN 1 the herpes infections were nicely balanced over active and placebo, so there we did not pick -- we didn't interpret as a signal that it was drug induced.

In DARWIN 2 the frequency as well, is very low. So also in DARWIN 1, if you compare the herpes rates to what was reported for tofacitinib, we are much lower than competition.

So we'll keep on looking what the herpes is but the current data gives us comfort that we will end at the lower end of the spectrum here, and it's not going to be a worry for us.

Jan de Kerpel - *KBC Securities - Analyst*

Okay, that's clear. And then, Onno, for you a small question if I may?

From where we are sitting it's difficult to understand if there would be any discussion between Galapagos and AbbVie, on AbbVie lifting -- if they would have the intention to lift the option. So is there some discussion still ongoing and on what is the, on what matters is that?

Onno van de Stolpe - *Galapagos NV - CEO*

Hi, Jan. There have been no discussions yet on the license. They were waiting 'til they got the full 24-week results of DARWIN 1 and 2. They will evaluate now the full body of results from both programs and make decisions, so including their own ABT-494, and make decisions regarding a further development.

So we are awaiting their decision and their communication. We are confident that the data are so outstanding that they will license. But as I have said the previous webcast, if they would not be enthusiastic about filgotinib and would not take the license, we're happy to continue with the filgotinib on our own.

But again we know from the FE individuals that they're very excited about filgotinib, so we are pretty confident that they will take a license.

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Jan de Kerpel - *KBC Securities - Analyst*

Thank you very much; and also, from my end, congratulations with these very nice data.

Operator

(Operator Instructions). Samir Devani, Rx Securities.

Samir Devani - *Rx Securities - Analyst*

Congratulations on data. It's just really going back to this question about optimal dose. On slide 25 where you've presented the LDL and HDL plot, I'm just wondering, what does the 100 milligram dose look like on those two plots?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Thank you for asking. In terms of lipids we had a nice dose response; so 100 milligram will be half of what we saw with the 200 milligram.

We've been -- we're still looking hard at the data. We've already come to the conclusion that the lipid data is a kind of un-blinding factor for the study, because if you see [HDL] moving up you can be sure the patient is on active, because we see such a nice response with dose.

So also there I don't think the 100 milligram will be the big differentiator.

In terms of dose as well, we're analyzing -- we analyze if AbbVie takes the option we'll have a good discussion on dose and how to move forward.

But it's a rich data set and we are really working ourselves to come to a final conclusion. But as I said, really, anything between 100 milligram and 200 milligrams is a good option to move into Phase 3.

Samir Devani - *Rx Securities - Analyst*

Just to clarify further, maybe I didn't make my question clear. But the discrepancy you see in the LDL and HDL in DARWIN 2 versus DARWIN 1, you see the same 100 milligram -- you see that same effect with the 100 milligram dose?

Piet Wigerinck - *Galapagos NV - Chief Scientific Officer*

Yes, we see the same effect; less of an increase, but a similar increase in HDL and LDL for the 100 milligram. That's correct.

Samir Devani - *Rx Securities - Analyst*

Okay, that's great, thanks.

Operator

There are no further questions over the telephone at this time.

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

With that then I think we'll close our question and answer session. Thanks, to everyone, for listening today and our webcast will be available for replay in about one hour from now. We'll also be putting a transcript up on the site a little bit later; tomorrow probably.

So thank you very much again and look forward to speaking with you next time. Bye, bye.

Operator

Thank you. Ladies and gentlemen, that will conclude today's conference call. Thank you for your participation. You may now disconnect.

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