Galapagos NV

Galapagos NV presentation delivered at the 39th Annual J.P. Morgan Healthcare Conference on Thursday, January 14, 2021 at 9:10 AM

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James Gordon: Good morning and good afternoon. I'm James Gordon, J.P. Morgan European pharma and biotech analyst. Today at the J.P. Morgan Healthcare Conference, it's my pleasure to introduce Onno van de Stolpe, CEO of Galapagos, for this presentation. We're going to have a 20-minute presentation from Onno. Then we're going to have 20 minutes for your questions.

We're going to be joined by the board of Galapagos management team. We've got the whole gang so look forward to that. You can immediately start registering your questions. The way you do this is with the Q&A function that's associated with this presentation on the conference website. I'm going to be reading your questions on the presentation from there.

With that said, I'd like to welcome Onno and Galapagos to the conference. I look forward to the presentation.

Onno van de Stolpe: Thank you, James, pleasure to present here and thanks for the invitation, if you look at the first slide, the disclaimer, I would like to refer to the forward-looking statements that we have in the SEC filings. Then, we go to the real slides.

Slide number three -- ready for an exciting future, of course, as you know, we had a tough 2020 with the CRL that we received from the FDA regarding the filgotinib launch in rheumatoid arthritis. As a consequence, the stock dropped quite a bit. There was a lot to do around Galapagos, but we are in fighting spirits to regain shareholder confidence.

We have a true science-based organization, which has created a really exciting pipeline that we'll discuss today. At the same time, we're building out a commercial presence. We have regained all the rights in Europe for filgotinib. We believe that it's a great opportunity for us, and we have the capital with over 5 billion in the bank to make all this happen.

If you go to the next slide, we're going to talk about the inflammation franchise and, after that, the

fibrosis franchise. We'll start with the inflammation where we have our first marketed products in Europe on the market and in Japan on the market.

This is very important for us, after 21 years, to finally have a product that we have developed all the way from target discovery to launch. We're very excited by Jyseleca, but it's not only Jyseleca in inflammation.

We have provided more details on our Toledo program, and I'll give you more details on it later in this presentation. You also have other mechanisms in the pipeline that are moving towards the patient.

Let's have a look at that inflammation franchise in slide five. Filgotinib is on the market in RA, but is still in development in Crohn's disease, where the phase 3 is ongoing. We submitted for ulcerative colitis in Europe and are shortly going to submit in Japan.

We are continuing with filgotinib worldwide and introducing that through Gilead in the various marketplaces. There's more to it than just filgotinib.

We have our SIK3 inhibitor, part of the Toledo program, now in five different proof-of-concept studies. I'll get more details later. We also announced today that we have a program TYK2 inhibitor in psoriasis that is number 3667, an exciting target, an exciting program that I'll give some more details on.

We have an old JAK1 inhibitor from our pipeline that was for years in the hands of GSK back within Galapagos. We're progressing that in osteoarthritis where it's in Phase 1b. Another SIK program in SIK3, selective inhibitor already in the clinic moving forward, and other programs moving towards the clinic and towards the market, so a very broad information franchise that we're continuing to invest in heavily.

Let's first look at Jyseleca. On slide six, you see one of the marketing slides of Jyseleca. It's our first product. We're commercially launching that in Europe for RA with the potential expansion into UC and Crohn's disease.

If you go to the next slide, you see the renegotiated deal that we have with Gilead around Jyseleca, where we have now the full European rights, where we're going to transition the commercial rights and the commercial organization this year. By the end of the year, it should be completed, and we are going to launch it in all countries in Europe.

This year, we'll share the P&L of the launch of filgotinib with Gilead. Then after that, it will be at our cost. Also, the income will be ours, but as we will expect it to turn positive in '24 we'll start paying a royalty to Gilead between 8 and max 15 percent, but the fifteen percent would be at the very high sales in Europe.

We will get no further milestones or pay any further milestones to Gilead, and Gilead is going to pay us 160 million as a compensation payment for this. Gilead retains all the rights outside Europe. There we're getting further milestones, as well as royalties, as in the old agreement between 20 and 30 percent.

The broader R&D collaboration, important to note, is unchanged. We signed this big deal with Gilead in 2019. That deal still stands as was originally negotiated, with all the options of Gilead has on the programs and the opt-in that they already have on one of the programs.

Let's look on slide eight on the European market for inflammation. You see that the big five have a potential of almost six billion -- substantially lower, of course, than the United States, and that's mainly caused by the difference in pricing in Europe versus US, but still a substantial-market opportunity.

We have given our guidance ambition here to reach about half a billion in sales in this market. That would mean an 8 to 12 percent market share for Jyseleca.

If you look at the JAK class in slide nine and Jyseleca particularly, you see some major advantages in the treatment for patients here. Look at some specific JAK advantages of treating these indications, like RA, lasting activity. It's oral, it's a very fast onset of action. You see activity within the first two weeks.

You can use it as a monotherapy -- very good efficacy levels there, robust responses. Specifically for Jyseleca, a very differentiated safety profile and, at the moment, the only JAK inhibitor where two dosages are approved in Europe and Japan, which is for doctors a big advantage when they can play around with the dosage for their patients.

With Jyseleca in RA on the market, we are continuing with filgotinib development. In Crohn's disease, that trial is ongoing. We hope to fully recruit that later this year. We had already good data in an earlier trial. You see on slide 10 the data of the FITZROY trial in Crohn's disease.

You see there a small trial that we recently unblinded -- the DIVERGENCE 1 trial -- where you see a similar effect of the treated population with filgotinib versus the placebo. A big advantage over the treated population is in CDAI remission in week 10.

A good step in this exploratory trial and a confirmation for us that we are on the right track in Crohn's disease to show an advantage of filgotinib in this area where there's a high unmet medical need and a need for new innovative drugs. We hope to be on that market in a couple of years' times.

Let's switch gears here and look on slide 11 at Toledo. Toledo is a program that we started years ago, when we identified a set of targets that we believe are really a game-changer for treating inflammatory diseases. These are based around SIK targets, the three of them.

What we do, and when you inhibit these targets, you actually have a dual action on the inflammation. That's where the big benefit lies compared to all other therapies in these disease areas.

We have seen preclinical models with very strong activity that made us very excited to progress this as fast as possible and as broad as possible. We now have '3970, our first molecule, in five different proof of concept trials in parallel. Why is this important?

Looking slide 12 about the mechanism, you see that up to now all the treatments for inflammatory diseases are based on the reduction of the pro-inflammatory cytokines. You see that here with the balance. The disease is a dis-balance between the immunoregulatory cytokines and the pro-inflammatory cytokines.

You see that all the treatments like TNF and IL-6 reduce the pro-inflammatory cytokines, which is helpful for the disease but causes also side effects, like infections.

What we do with Toledo is actually increase the pro-inflammatory cytokines, as well as the immunoregulatory cytokines, and thereby making that balance back into control. We're not increasing pro-inflammatory cytokines. We're reducing them a little but increasing the immunoregulatory cytokines.

Therefore, the balance is restored. Therefore, we believe that this is a better way and a more complete way to treat inflammatory diseases like RA, Crohn's, and other areas.

In slide 13, you see multiple selectivity profiles that we have identified in the Toledo program. At the moment, we are progressing a number of molecules that are selected for SIK2 and SIK3, as well as one molecule that is specifically selected for SIK3.

We have 10 chemical series investigated, and we now have, in the patents, covered about a thousand compounds. A lot of work has gone into it, many, many FTEs, with very successful. This program has never failed us in progressing as expected, and it has surpassed any activity that that we could have predicted with these molecules.

If you look at slide 14, you see a very broad panel of diseases where, in animal models, we have tested our molecules. Except for osteoarthritis, we have seen activity in all other diseases where are these molecules were tested, so not only in inflammation but also in fibrosis models.

Depending on the selectivity, you see different activities. That's what we like to see. That means that they can develop specific molecules for specific indications, something that will help us in the further commercialization later on. For now, we are exploring this as broadly as possible. That's why we have tested these different diseases with our first molecule

If you look at slide 15, you see this dual activity confirmed ex vivo where we have analyzed the effect on TNF and IL-10, TNF being the inflammation cytokines. You see a very nice-dose reduction in the various green bars after already one day of treatment and continuing in day 10 of treatment, or day 14 in this case.

At the same time, you see IL-10, which are the good cytokines that you actually want to increase, going up on day 1 and continuing on day 10 and 14. This is quite spectacular.

We're very pleased to see this very nice dose response and the effect on the pro-inflammatory cytokines as well as the immuno-regulatory cytokines. This is why there is so much excitement within Galapagos for this whole Toledo program.

If you look on slide 16, you see the parallel proof-of-concept studies that we have initiated. We started with psoriasis. You see an NRA. These are short studies, six weeks, and it means that, of these three indications, we get data in the first half.

We will report that by midyear, and hope to get a good indication on the ultimate efficacy in patients, but also on the broadness of applicability of the Toledo program.

We also started longer trials in systemic lupus as well as Sjogren's syndrome, two indications where there's a high unmet medical need. We hope to see efficacy there, as well. We have developed a program that brings us as fast as possible towards phase III. We are planning already the phase IIb face to be immediately following the proof-of-concept studies of the first indications.

We don't want to lose any time here. We have a massive competitive advantage here, because there are no other SIK compounds in the clinic for inflammatory conditions. This is a unique position for us to come up with a completely new way to treat inflammatory diseases.

If you see the large space here, dominated, of course, by the biologics like TNF inhibitors, IL-6 inhibitors, and now also with the JAK family, there are no other oral mechanisms out there other than, hopefully, this SIK mechanism to bring to the market.

Let's switch gears again and look at slide 17. Here you see molecule '3667, as I said at the introduction, which is a TYK2 inhibitor that we have added to our portfolio. It originates from our own research activity. It's an inhibitor of the reversible kinase domain, very good PK profile.

We saw very good PD activity, and I'll show that in the next slide. We have moved it into a phase I with an indication in psoriatic arthritis where we think this molecule will work extremely well. It's an old target. We're excited to be in that class, and we're looking forward to clinical data.

If you look on slide 18 for the pharmacodynamic activity, you see a very good effect on INF-alpha as well as on IL-6, exactly as we would have predicted, a very nice dose response on day 1 as well as on day 10.

You see the IL-6 inhibition also on day 1 and day 10. This confirms what we were expecting based on the preclinical models, and we are ready to start dose-response finding studies in 2021, an important new activity in our pipeline.

So much for the inflammation franchise, let's switch to fibrosis. There, of course, we have our main lead program, ziritaxestat, in Phase 3 in idiopathic pulmonary fibrosis. We also have many other programs following that.

You'll see in slide 20 the franchise here. We want to build a broad program in fibrosis where we will have multiple products that reach the patient, led by ziritaxestat, which is an autotaxin inhibitor. We have about 1,300 patients enrolled in the 1,500-patient trial.

This year will be an important one where we have a fertility analysis mid-'21 which will give us a view on how this program is behaving in this Phase 3. That's not all. We have data presented last year on '1205, which is a GPR84 inhibitor. I'll show you that data.

We also have other programs, one that we license in, a program 4716, and other programs in there, including actually a Toledo program which is an SIK2/3 inhibitor. Multiple shots at goal, and we're preparing for a number of phase II trials this year.

If you look at slide 21, we are casting a wide net in IPF. We want to cover as much biology of fibrosis here as possible. Here you see the whole process of IPF, in a simplified version I must say, where we play a role in the immune response macrophages, in the fibroblast activation, as well as the extracellular matrix accumulation.

The last one where you see the [inaudible] targets is in-licensed from a Polish company. We're looking at internal generated targets and molecules, as well as programs that are outside and that they can in-license to complement this pipeline.

Many shots at goal here and I think it's the right approach for this disease with such a high unmet medical need. Average expectancy for your life when you get diagnosed is three to four years, so it's something that needs efficacious and well-tolerated drug [inaudible] .

As I said, ziritaxestat on slide 22 is our main asset that we're moving forward. It's in Phase 3. It's a 1,500-patient study, where one-third of the patients will be on nintedanib, one-third on pirfenidone, and one-third on neither.

Within those groups, we then have two dosages of ziritaxestat and one placebo. We will see the effect as a standalone as well as in combination with the two drugs that are on the market for IPF. The patients remain on standard of care as a global program all over the world.

The primary endpoint, as expected, is in FVC decline at 52 weeks, but we'll also look very important at other secondary endpoints like hospitalizations, mortality, quality of life, safety, and tolerability. All patients will stay on drug in the trial until the last patient completes the 52 weeks.

We'll get a lot of data on the longer-term effect of the drug in these patients. Of course, recruitment has been impacted by COVID. These patients are very prone for infection, so it's not easy to continue with the recruitment. We're confident that by half-year, we'll have all patients in

this trial, and so you can expect the data in '22.

In the meantime, we'll do the futility analysis as we had promised to the markets by H1 in this year. We'll have 30 percent of the patients at week 52 and 70 percent of the overall data of the total ISABELA trial.

It's a big chunk of data that will be analyzed, and a committee will look at it and decide if there is a likelihood of being superior to placebo on the primary endpoint. If one of the dosages meets that criteria, then we'll continue with the trial with both dosages. That's the requirement by the FDA.

It's an important moment for Galapagos in this important trial. If you look at other assets that we have here, the PINTA Phase 2 in IPF, we showed very attractive data last year on the FVC in that program. Here, we will go into Phase 2b dose range this year, a second indication, potentially as a standalone, but potentially also in combination with ziritaxestat in the future.

We go to slide 25. Strong R&D engine clearly at the basis of Galapagos has always been our motor to drive to identify noble targets as well as to drive these through chemistry to the clinic.

If you look in slide 26, you see what kind of numbers we have in our pipeline. 27 validated targets, 30 programs in lead optimization, 3 preclinical programs, and 11 clinical-stage programs. That is a pipeline that can compete with many mid-size pharma companies.

We will do over 25 patient trials with nine different molecules in 10 indications in '21. That is a massive clinical program, and hopefully going to deliver the results that everybody's hoping for.

We have the capital in slide 27 needed for this growth. Thanks to the deal with Gilead, we're having a very strong balance sheet. If you look at slide 28 around the R&D collaboration that we have with Gilead, we got $5.5 billion from Gilead in 2019.

We have a very good commercial deal, where we get 20 to 24 percent of the royalties when Gilead licenses a product and commercialize it outside Europe. We have the full European rights.

Very important to this whole collaboration is that we have a lot of interaction in the research phase, where we can access Gilead's expertise, their compound libraries, and there's a lot of interaction between the scientists to maximize the success on the targets that Galapagos discover. That should yield to very attractive new mode of actions going forward.

Of course, all this comes at a cost, all these activities, all these trials. In slide 29, you see our cash burn expectations for '21. It's going to be 50 million higher than last year, so we're going to go to a total of 670 million.

That is an increase of 50 million, which is solely due to the increase of the commercialization cost of Jyseleca. It's clear that we need to successfully and completely introduce this in the European market. We are fully committed to do so, and that comes with a cost.

For the rest, we are trying to keep the research organization stable at the moment. We have paused the growth, waiting for the success of ziritaxestat, and then we'll continue to grow again. We have more than enough money to support the engine for the years to come, so we don't have to worry about shareholder dilution and raising more money in future rounds.

If you look at Jyseleca and Europe -- slide 30 -- then we have guided for peak sales of 500 million. You see that we expect to have a margin of 50 percent, which is maybe not the 60 to 65 percent that you would expect. Clearly, this is based on one molecule in a commercial organization in Europe, but still a very attractive margin.

This is clearly an MPV, very positive opportunity for Galapagos. We'll have the full commercial structure in place by the end of this year, and we have breakeven predicted for '24.

We have a long patent still so we can, from '24, be profitable until the end of the patent in 2035. That's another 11 year after reaching profitability. That makes this a very attractive opportunity for our first commercial product.

I'm going to end with the news flow in slide 31. I'm going to highlight filgotinib. Of course, the outcome of the MANTA/MANTA-RAy study, which is the sperm count study that will determine the future of filgotinib, especially in the US.

We have all the confidence that that will be a positive study for us and that we can file for the IBD indications in the US. We're expecting to launch ulcerative colitis in Europe this year, and we also expect to be fully recruited in the Crohn's disease DIVERSITY study this year.

The other programs, I already talked about, ISABELA, fertility analysis, the read-out of the Toledo -- three proof of concepts, which is something we are very much looking forward to -- the TYK2 that we just announced at J.P. Morgan, and, of course, the read-out of the JAK1 in osteoarthritis,

the Phase 1b.

A lot to look forward to, we are committed to regain shareholder confidence and we hope '21 is going to be an exciting year for us and for the shareholders that are with us. Thank you.

[pause]

James: Thanks very much. We'll now kick off the Q&A part of the session. I believe we're joined by board of management from Galapagos. Just to check who we've got with us, we have CFO and CEO, Bart Filius. CMO, Walid. We have got CSO and CCO all with us.

Walid Abi-**Saab:** Hello?

Onno: Yeah, we're all there. Let the question come.

James: Hi, everybody. I've had some questions that have come in about Jyseleca already. One of the questions was about the peak sales guidance you've given for the product.

The investor says, "Given that Lilly was able to get to a $400 million run rate within three years for Olumiant with one indication, why are you only guiding to â‚¬500 million as the peak sales for all indications?"

Onno: Michele, that's a question for you.

Michele Manto: Sure. We are looking at these three indications and, of course, the competitive setup and the pricing setup has changed since the launch of Olumiant three years ago.

We are ambitious and also aggressive strategy to get and penetrate the market with the market share that Onno indicated. This range of 8 to 12 percent, which is important but in a competitive setup which is different from where Olumiant launched three years ago.

James: Thank you. Another question is about [inaudible] , also on Jyseleca, asking about what about outside Europe? The question is, "Have there been discussions, or do you have plans to acquire filgotinib rights ex-Europe?" The investor says, "Could that be risky and expensive? It would be helpful to provide reassurance."

Onno: Bart, do you want to answer the question?

Bart Filius: Yeah. James, you were dropping off a little bit, but I think your question was whether there is any plans to take on filgotinib outside Europe.

James: That's right, yeah.

Bart: I know. Clearly, the whole idea behind the restructuring is that we take all of Europe, that simplification and as well of a very good business case, as Onno was explaining.

Outside Europe, that's clearly Gilead's responsibility, and especially as long as the IBD opportunity in the US is fully alive and in front of us, that's going to stay this way. There's no plan currently to take on any obligations outside Europe.

James: Thank you. I've always been asked about how the European and Japanese launch is going. In particular, how are discussions with payers proceeding?

Michele: I'm taking that. The launch started after the approval in end of September, so the discussion with payers have been progressing. We started in Europe -- we and Gilead at the time -- within few business days after approval with all the submissions.

So far, we are on track with the planning and ahead of time, as compared to some of the competition, how the time went with the competition. As of return in terms of messaging and the messages that Onno alluded to, in terms of the strength of the profile of Jyseleca in the early country, our promotion is allowed.

Germany, the Netherlands, or Belgium, France, we get that resonating very well and also reflecting to the indicators we have, in terms of efficacy and safety return on messages from the markets.

Similar story in Japan, though that is a country that is managed by Gilead together with Eisai, which is a Japanese company, very strong in rheumatology, very connected with the experts there. They also were at the base of early [inaudible] launches 10 years ago. Also, their early indicators are posing the right direction.

James: There's also a question about the launch RAM and what's a good benchmark or analog. "Should we use Olumiant and Rinvoq as the benchmark? The other part of the question is, to what extent is COVID-19, the third wave we seem to be seeing right now, to what extent does

that disrupt things?"

Michele: Also taking that. Yeah, indeed the benchmarks, the reference point. We are looking at Adalimumab as one of the best launches that happen in rheumatology in the past five, six years, so we're looking at that, as well.

Also considering the fact that this is a different situation, we have seen the market that slowed down because of COVID happened across therapeutic areas. Alsos rheumatologists reduced the number of prescription they could do, the number of patients that could start on new therapies, so we've seen that, also a reduction in ability to access physicians.

On the contrary side, then this also will create a base of patients waiting for new therapies that we could access once the world returns to a more normal situation.

In terms of access to physicians, this has been our strategy from the beginning. To have a combination of face-to-face and also digital, that was in our go-to-market model from the beginning and that we're now revisiting continuously and accelerating that.

That was testified by our digital presence at conferences like you are. We also tested that at ERS, where we had really prominent success on our symposium digital presence that we can also deploy in the next European conferences and also the national ones.

James: Thank you. Question about IBD, UC, and CD. The question is, "How likely is it that Gilead moving forward with filgotinib reducing Crohn's in the US? Should we think that the US Is not going to happen, or could that still happen?

" Then in Europe, in terms of when you can actually file, could you file before you've got the maximum 52-week data from the MANTA and MANTA-RAy studies?"

Onno: Let's start with the first, we are still expecting Gilead to go forward with the filing, both in UC as well as in Crohn's. Of course, it gets somewhat more complicated if they have to wait 52 weeks on the MANTA data before being allowed to file. For Crohn's, that is not an issue, because the data will come after the 52 weeks.

For the European regulators and the Japanese regulators, the 52-week requirement is not being discussed or not on the table. This is a new request by the FDA that came after the initial OK on the trial design.

James: Thank you. There's also a question about the peak sales. "You're given a peak sales estimates for the European five, but then, how big is the Japanese Market relative to Europe?"

Michele: The Japanese market, as a general market, is relatively equivalent to 70 percent of Europe as a general statement. In terms of peak sales and ambition in terms of penetration, that's of course an answer that Gilead can address best, as this market there, they managed to [inaudible] .

James: Thanks. Maybe I'll take one more on filgotinib, and then there's some other questions, as well. One is that, "On your call in December, you mentioned that Galapagos had not necessarily given up on PsA, AS, and uveitis for Europe and Japan. Any update on those trials in terms of for Europe and Japan?"

Onno: Walid?

Walid: Thank you, Onno. We are trying to evaluate this. This team is now looking to see whether we can put together a plan that would satisfy the regulatory requirement for Europe and bring this valuable therapy, in our opinion, to patients in Europe. At the same time, we need to make sure that it's economically viable for us.

We're looking into it, evaluating it, and we will be communicating on this as soon as we get an answer. We're not there yet.

James: Thank you. Switching gears, Toledo, there's a question here. "Toledo looks promising with some novel activity, but we have seen the same in the past with OA and CF programs. On the learnings from those failures [inaudible] confidence to the preclinical evidence for Toledo will translate to benefit humans?"

Onno: Piet, do you want to answer that?

Piet Wigerinck: Thank you. I think it's hard to make a comparison between OA and RA. I think for the Toledo we see clear nice dose response, strong engagement of biomarkers like TNF that have proven with previous treatment that, if you can block that, you can go to efficacious treatments.

That is a given that's in the books now. We add to that the IL-10 increase. That's an unproven

rule. We will need to wait for that. Within view of what we know of how anti-inflammatory drugs work, I think this was a profile we aimed for for many years. We are quite confident that this is going to be a very nice class in the future.

James: Thank you. A question on Toledo, "Is there potential to combine other inflammatory assets which have different mechanisms of action to maximize the efficacy, or could that be a bit challenging given that Toledo has already got two mechanisms? Generally, when you stack lots and lots of mechanisms on each other, you could stack the safety issues, as well."

Piet: Step first is to understand how the Toledo on their own works, because it's indeed a dual mechanism of action in which diseases, we get best risk-benefit. We don't exclude going to combos, but that will happen later.

Clearly, if you look to JAK inhibitor, that would block the IL-10. That does not seem to be a smart combo on paper, but are other options available in the future. That is work for later, not for now.

James: Thank you. There's also question about IPF. The question is, "We recently saw a positive headline data from PINTA and IPF '1205. It's at the standard of care. How excited are you about that data? How do you see it relative to the FLORA data for ziritaxestat?"

Onno: Walid?

Walid: Thanks, Onno. I think the the PINTA data demonstrated that we do have efficacy that we see across the board, whether people were on no background therapy or on pirfenidone and Nintedanib. I think in this disease that's very much a very tough disease, there's a high unmet medical need.

As you've seen from Onno, that we're trying to target it from multiple pharmacology. '1205, in particular, sits in a different bucket than ziri. I think it lends itself also to being combined, so I think we're doing the next study, which will evaluate a couple doses, and larger study so that we can have a much better estimate of the effect that we're seeing.

We're going to have a much better sense of the risk-benefit at each dose level to then figure out where we go next. I think one of the things that in FLORA study for ziri that helps us feel more confident to move forward is that we identified a dose that demonstrated efficacy, but at the same time also had a very good tolerability profile.

In this case, with '1205, we did see a little bit of an increase in some of the adverse events that led to some discontinuation from subjects, particularly for those who were on background Nintedanib. That's why we felt it was necessary to do a better assessment of other doses with larger number of patients to have a much better sense and reduce the risk before we jump into phase 3.

Interesting activity that we've seen, we're following up on it to better understand it, and also we will be in better place to potentially combine with ziri and other molecules that we have in the pipeline.

James: Thank you. I can see we're almost out of time. Maybe I'll ask one closing question, which would be, you've got a very healthy cash balance. How much of it could you use for something beyond internal development? With shares trading where they are, what are the thoughts of actually buying back some of your own shares versus actually buying more for the pipeline?

Onno: Bart can answer that.

Bart: Yeah, I'll take that one. There's no option to buy-back shares. That's not in the plan. The cash balance that we have is dedicated for investments in research and development. That's also part of the arrangement that we have with Gilead, for the obvious reason that they want to have a benefit ultimately from programs that reach the end of phase 2b and then can be optioned by Gilead.

It's also our own desire, because we think we've got such a broad pipeline to invest in that we want to use our cash for research and development and not for share buybacks. We would, however, be interested to look -- and we are continuously doing that -- at external BD opportunities.

Last year, we've done a couple relatively small transactions. We will continue to look at those also going forwards if they are, let's say, a good strategic fit to the rest of our portfolio, to the strategic direction of the company.

James: Great. Thank you very much. I can see we're out of time. Thank you very much to everyone at Galapagos for taking part in the presentation and the Q&A. Very interesting, and I hope you have a great rest of the conference.

Onno: Thank you.

James: Bye, everybody.

Onno: Bye.

[music]
