Q1 2020 Galapagos NV Earnings Call

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Q&A Analysts

We'll take our first question from Christopher Marai with Nomura.

Christopher N. Marai, Nomura Securities Co. Ltd., Research Division - MD & Senior Analyst of Biotechnology

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Q

First one is really on Toledo. And maybe if you could elaborate further on, number one, the target. But number two, it appears you selected one molecule for another 3970, if I recall, with that TOL2, TOL3 target-selective molecule versus the 3312 that you are -- it looks like no longer developing. And that may have been a PanTOL selective molecules. So could you maybe elaborate on what it was that helped guide the decision here in healthy volunteers to choose one over the other [ETR] business related to the selectivity of the compound or other molecule properties often observed in Phase I trials?

And then finally, just on your SSc program. Would love to understand if you think that autotaxin would have a differential manifestation on organs. We've seen some data historically on skin and lung, and it's sort of different across various compounds and modalities. So would love to hear your comments on that.

A

Piet Wigerinck, Galapagos NV - Chief Scientific Officer

Okay. Chris, thanks for the question on the TOL program. So you first ask on the target. Well, that's still undisclosed, so I'm not going to disclose that today. So where we have plans that when we're in the clinic, we will brief the whole field broadly on the discovery, the identity and the promise we see in this program. But that is not for today.

So you mentioned the 2 compounds, 3312 and 3970. So 3312 indeed was a PanTOL, 3970 a TOL3. 3312 was a compound we targeted for distribution in the column that's been taking a long time. So over Q1, we completed the multiple ascending dose part of the healthy volunteer studies with both 3312 and 3970. For both compound effects, we were pleased with the observed safety and the observed exposures. For 3970, we as well could include their plasma biomarkers and where Onno hinted to promising data on 3970. I think you should think about the plasma biomarker that really shows us that with that compound, we are very well on track.

So that's -- then when we had all the data, looking forward with the COVID-19 around, starting up novel clinical studies is really not still allowed because we can't get to the hospitals. We have a number of protocols approved. We are waiting until the hospitals open. We can go in there, and we can start those studies. And in all of that struggle, we decided to focus on a single compound in the hope to push that compound through in a number of indications rather than disperse of different compounds and not do anything good.

But it's not a choice for a profile. It's more that with 3970, the plasma biomarker really pushed us to sail. Let's put every effort behind that in a difficult landscape where we live in today.

A

Walid Abi-Saab, Galapagos NV - Chief Medical Officer

So regarding scleroderma, Chris, so the approach that we've taken in the trial is to have an exploratory Phase II study where we're evaluating essentially the effects on the disease itself not a particular subtype or a particular organ. We're taking a very broad approach, and this is our first foray into the space.

Scleroderma is really a tough disease. It's actually the #1 cause for death in autoimmune diseases actually. But the trials in that space are difficult. So based on animal models, we -- or at least the one we understand the biology, we cannot prioritize certain organ versus the other, but we have good reason to believe that the autotaxin inhibitor should have a positive effect in this disease, and that's what we're setting up to do in the NOVESA trial. And that's the approach we're taking. Thank you for the question.

We'll next go to Jason Gerberry with Bank of America.

Jason Matthew Gerberry, BofA Merrill Lynch, Research Division - MD in US Equity Research

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Q

Mine is just regarding the update on GLPG1690 and the time line shift on the futility analysis due to COVID. It was my understanding that you guys had enrolled the 1/3 of subjects necessary for futility in early 2000. So just sort of curious what's causing the push and the timing there. I'm wondering, is it, a, maybe trouble capturing the FVC endpoint? Or alternatively, are you just needing to upsize the trial, maybe a larger sample for the futility? Any color on that would be really appreciated.

A

Walid Abi-Saab, Galapagos NV - Chief Medical Officer

Thank you, Jason. This is Walid again. Yes. So actually, just bear with me. I'm going to get a little bit more technical here. So for the futility analysis, we said that we need about 1/3 of the patients enrolled, which you're correct. We've enrolled those. But we also said that we need about 70% information to be able to do the right statistical evaluation. And that 70% information is "derived" from those 33% of the patients who have been enrolled and have gone all the way through 52 weeks plus all of those behind them that are contributing to the various earlier endpoints, 9 months, 6 months and 3 months, to collectively help us estimate what the 1-year rate of decline in FVC would look like. And I think it's these other parts that are a bit delay.

The other piece is indeed as a result of the COVID-19 pandemic. We have given more leeway to sites to widen essentially the visit windows and obtain FVC in a safe way, whether they can do it at the sites or maybe they delay it by a month or 2 and do it at a later time point or put in place a system where we can get that by a special provider as well.

So those elements are leading us to have maybe a bit less information on FVC that we need from the additional patients that I mentioned but also those that are nearing the 52 week. And that's why we anticipate there's going to be a bit of a delay. I don't think that's going to be a big issue. I still am hoping that we're going to get the data in early 2021, but we did not want to make a promise that we couldn't keep, and that's why we're widening the window and saying the first half of '21. But I can be -- I can assure you that it's nothing due to any changes or any difficulties we're having beyond a bit of a slowdown as a result of COVID.

We'll take our next question from Evan Seigerman with Crédit Suisse.

Evan David Seigerman, Crédit Suisse AG, Research Division - VP & Senior Equity Research Analyst

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Q

I hope everyone is staying safe and healthy. Can you expand on your commercial strategy for filgotinib in RA in Europe if this is kind of your first launch into a competitive market? And assuming similar labeling to the competition, namely upadacitinib, how do you differentiate filgotinib? I mean have you kind of maybe continue to see plans for a more virtual launch assuming that things don't necessarily go back to normal right away even in the fall or come early next year?

A

Michele Manto, Galapagos NV - Chief Commercial Officer

This is Michele addressing that question. So there are multiple elements you put into this. So first of all, it's to confirm that we're really excited in energizing having our first launch, and this is really reflected in the number and quality of the talent that we have recruited in the past months and we keep recruiting in the country. So just to give a sense, we have completed the recruitment of the leadership teams in all the countries and in the headquarters supporting them. And namely, on the medical and access part, we are also fully then prepared there.

And in terms of sales management also in the countries that we'll have reimbursement this year, they are also -- they are completed. So -- and with all people coming from big experience in rheumatology and biologics are coming from companies that have been promoting and working on those markets until yesterday. So they are able to operate contact customers engage on day 1 as they join Galapagos. And that's what exactly how they have been doing.

Also these different times of COVID working virtually, we had a number of virtual adboards, engaged payers. So this is going, I would say, under the circumstances as we plan and as we would wish. So of course, we keep a flexible approach there in terms of expanding our full sales force recruitment depending on how the situation will evolve in terms of reimbursement and COVID situation.

On the label, on the strategy, well, we are, of course, very confident as well in the profile that we got and demonstrated in all the FINCH studies across all patient populations. And this is what definitely we're going to leverage counting on a differentiating label, of course. But anyway, having strong data that really positions filgotinib as the best-in-class jack with great efficacy and really differentiating safety profile, which, especially in these days, is coming up as a really needed feature.

Wimal Kapadia, Sanford C. Bernstein & Co., LLC., Research Division - Research Analyst

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Q

Wimal Kapadia from Bernstein. Just I'd like to get your thoughts on the upcoming UC data and just the context of what we've seen so far. So I'm thinking high teens 20% for the induction phase on what we've seen from some of your peers. And even in the maintenance rate, we're looking at a low 20% placebo-adjusted rate. So just to get your thoughts on your expectations heading into the data would be great.

A

Walid Abi-Saab, Galapagos NV - Chief Medical Officer

Yes. Thank you. This is Walid. Thank you for the question. Well, indeed, I mean, I think, as you know, for UC, we have no previous Phase II data with filgotinib. The SELECTION trial by itself was a Phase IIb/III trial. There was a futility analysis at one point that evaluated each dose versus placebo in each of the populations. We're studying the biologic naive and the biologic IR, the groups. And we pass that to make it into Phase III, so that's a good sign. But we have no basis specifically with filgotinib in UC to try and estimate what other effects will be.

So in the absence of that, we actually rely to a great extent on performance of filgotinib first in Crohn, which is sort of a closer disease to UC. And in the FITZROY trial, our data were very strong efficacy signal that we've seen in that trial. And that makes us feel very positive about it.

And then the other part is to look at the performance of filgotinib in other indications like RA, psoriatic arthritis and ankylosing spondylitis and kind of try to make this comparison, again, not within trial but across trials, how other JAKs have performed. And based on that, expect what we will see in UC. And I think based on that, I think you can agree that the data that we have seen in RA and

the other indications that I've mentioned, you would expect that filgotinib is going to be performing at the top line of all these -- from an efficacy perspective compared to the other JAKs, particularly upadacitinib and perhaps a bit better than TOFA where we have seen so far. And from a safety and tolerability, I still expect that filgotinib will continue to show this best-in-class profile in terms of safety. And those are our expectations going forward.

The numbers there in the coming quarter reported are probably in the ballpark of what we're thinking about. But that's the best way that we have going forward since we don't have the previous data with filgotinib.

We'll take our next question from Debjit Chattopadhyay with H.C. Wainwright.

Debjit D. Chattopadhyay, H.C. Wainwright & Co, LLC, Research Division - MD of Equity Research & Senior Healthcare Analyst

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Q

So I'm just curious about where you stand with the RA MANTA program and especially for any supplemental NDA for UC. Would that be necessary to file? And a follow-up on a prior question regarding the differentiated label. What kind of interactions have you had with the FDA now that there is unlikely to be an ad com to push for a label, which recognizes the PE DVT differentiation?

A

Walid Abi-Saab, Galapagos NV - Chief Medical Officer

All right. So again, this is Walid. As you know, the status of the MANTA program has not been disclosed yet. The most that Gilead has shared is that we should expect to complete recruitment in the second half of this year. That would be for all the MANTA program, both MANTA in the UC population as well as the MANTA in the rheumatology MANTA-RAy. That's the name of the study.

In terms of discussion with the FDA, again, these things, we don't comment on them. There's been a number of discussions, of course, that our partner, Gilead has had with the FDA. What we say is that we're very appreciative with the work that they're doing. We're very confident in the data package that we have developed for filgotinib in RA, and we look forward to having further discussion as we get closer to the PDUFA later this year.

Regarding submission in UC and whether MANTA will be needed or not, again, I cannot comment on this, but I think it's a little premature before we even have the results of SELECTION. So I think just any discussion with the FDA will have to happen on that indication after the results of the SELECTION come out later this quarter. Thank you for your question.

We'll next go to Brian Abrahams with RBC Capital Markets.

Brian Corey Abrahams, RBC Capital Markets, Research Division - Senior Biotechnology Analyst

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Q

I was wondering if you could talk a little bit more about your level of confidence in the regulatory time lines and in the filgotinib manufacturing given COVID-19. And then would love to hear a little bit more detail about the specific path forward for 3970, what your latest views on what proof-of-concept indications you might be considering and potential trial designs.

A

Walid Abi-Saab, Galapagos NV - Chief Medical Officer

Okay. This is Walid. I'll take the filgotinib question. So let's start with manufacturing. We have no concerns around manufacturing, and we've been talking with Gilead on this. And there's -- we don't expect any negative impact from COVID on that at all.

In terms of confidence and regulatory time lines, we've been in contact with both the FDA and the EMA on this. So far, we have no indication that there will be a delay in this. As you know, health authorities try to do their best to honor their PDUFA date. Always there are things that could happen that could delay this. And of course, now we're living in unusual circumstances, so I think we need to keep an eye out for this. But so far, we have not heard anything directly from them that indicate that there will be any delay, whether in the U.S., in Europe or in Japan for that matter. So we're still on target as of now.

Piet, over to you for Toledo.

A

Piet Wigerinck, Galapagos NV - Chief Scientific Officer

Okay. Thanks for the question on 3907. So as indicated before, we plan a number of studies that will come in waves. So those waves are triggered by the tox coverage that we have accumulated so far. So -- but we disclosed the designs and indication as they come online. So currently, I believe there is one which is published as psoriasis study, which is a Phase Ib study in patients. And the next one as soon as they are approved and they come online, we will share that with you. There is a wave which is staggered in duration as tox coverage has been obtained. So you first see the shorter one, then the longer one and eventually, one 1-year studies as well. But it's all in the autoimmune space. Let's keep it there today. Thank you.

And our next question we'll take from Emily Field from Barclays.

Emily Field, Barclays Bank PLC, Research Division - Research Analyst

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Q

I just had a couple of quick questions about COVID-19 actually. I believe that in the NIAID trial, remdesivir is being trialed with baricitinib. I was just -- if that trial were to show any promising efficacy, would there be the potential to combine remdesivir with filgotinib?

And then also, obviously, it's quite early on, but there's been some early evidence of patients with severe disease that have recovered that have significant lung starting in fibrosis. And given the depth of your fibrosis portfolio, is that something that you would explore potentially from an R&D perspective?

And I would like to sneak in another one this time, but I'll leave it at that for now.

A

Walid Abi-Saab, Galapagos NV - Chief Medical Officer

Yes. Thanks, Emily. This is Walid. Yes. I mean I think, as you know, there's a lot that we're learning in this field about the COVID-19, the pathophysiology of the [MS] data are popping up in a variety of places and whether the approach would be to target antiviral treatment but then at a later point, focus on also cytokine storms and so on and so forth. So we've had a discussion with Gilead. But as you can imagine, Gilead now is really all firepower is concentrated at least in that space, in the virology space, to push on moving forward remdesivir and generating data that would be very potentially useful for humanity as a whole as we're battling this pandemic.

Of course, as we look at data that will come out from the study that you mentioned and if it turns out that inhibition of JAKs could be helpful for this, of course, this would be something that would be considered in due time, and Gilead would be in a prime position to do just that.

Yes. Interesting data emerging about sort of some of the sequel -- people who are recovering from COVID and then end up with significant lung impairment. I think today, we don't quite know what is the past physiology of this. But as we start getting better information, if our compounds actually work on this, and you know at Galapagos, we're very much invested in understanding fibrosis and working on it, both pulmonary and other types of fibrosis, we would definitely be interested in testing our molecules, either ziritaxestat or some of our other molecules of development into that space.

I don't know, Piet, if you want to add anything to this. But I think right now, we don't quite know about the pathophysiology.

A

Piet Wigerinck, Galapagos NV - Chief Scientific Officer

No. Thanks Walid. So well, what I can confirm is that none of our development candidates has any direct antiviral effect. So we are not in that game. We are looking into that cytokine storm. So -- and there, we are currently looking which animal models, and we are running a couple of compounds in animal models just to see whether we can bring something to this battle which is not available out there. But with all of the IL 6s on the market, the chance for JAK1s to differentiate there is honestly quite limited.

Fibrosis, our research is really mainly focused on slowly developing fibrosis. So now repositioning those compounds into something which is as acute as a COVID-19 damage to the lungs. We have to look into that, but I don't believe there's any good animal model there. So that's a huge jump from a theory to patients that are in high unmet medical needs. So that is a huge step to take and a bit hard, I think, for compounds that are not approved yet. So -- but we'll see where we go from there. Thank you.

And we'll next go to Ellie Merle with Cantor Fitzgerald.

Eliana Rachel Merle, Cantor Fitzgerald & Co., Research Division - Research Analyst

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Q

Just a quick one on the ulcerative colitis trial. Can you tell us a little bit about your expectation in terms of the mix between TNF or biologic naive versus experienced in the trial, if you can comment on sort of what you expect to see in the patient mix?

And then in terms of the osteoarthritis trial, can you talk about any impacts from COVID that you're seeing in terms of missed visits, if at all? And if so, sort of what some of the provisions would be in the case of sort of any missing data that could happen as a result of COVID in that trial?

A

Walid Abi-Saab, Galapagos NV - Chief Medical Officer

Yes. Thank you very much. So the SELECTION trial actually is almost 2 trials or 3 trials in one, right, because there's an induction part and a maintenance part. But also for each of those, we are studying a cohort of biologic IR and another one in biologic naive. So essentially, the mix will be exactly 50-50 because they're separate cohort. So it's not open within the same trial to be -- for them to be a mix, I guess, is what I'm trying to say.

For ROCCELLA, the 1972 program in osteoarthritis, of course, we are seeing -- and we're also dealing with the effects of COVID just as we do for all of our trials, we're very quickly able to step up and connect with sites and provide feedback. Maybe I'll take a minute here to tell you a little bit about our approach.

Very early on, actually, we developed a task force that essentially meets on a daily basis. The task force is comprised, in addition to myself, the Head of Clinical Operations, the Head of Clinical Research, the Head of Medical Safety, Biostatistics, Regulatory Affairs. And we monitor information

both from our ongoing trials, from the sites, from our teams but also externally from regulatory authorities, the -- any scientific literature and so on and so forth. And the goal is to be able to provide the necessary guidelines to our teams, to manage things going forward.

Our approach has been priority #1, maintain the patient safety in the trial; priority number two, maintain the study integrity. And fundamentally, at the basis of that strategy is to trust our sites, that they are the ones that are best able to judge what should be done and what measures should be used to maintain the safety of the patients and also the integrity of the trial. So we gave them a number of opportunities to widen visit windows to do -- use local laboratories to get a blood work, to provide using maybe other sites, which might be open in case one site is closed nearby and so on and so forth.

So what we're seeing is that the impact in ROCCELLA is, at this point, minimal. We're not seeing anything major. I would imagine there will be a little bit of a delay because -- it's not a large delay but a little bit of a delay because we're widening the window of a visit to be able to allow patients to get their MRI, which is the primary endpoint at the end of the trial. So that could delay the closure of the study by a few weeks to make sure that we maximize the chances that everybody gets that in.

But so far, we haven't had any significant missing data. Again, as with everything COVID, we continue to monitor it very closely because it's a shifting environment. But I think it's good to say that right now, we're starting to see the tail end of it. At least in Europe, we're starting to see the light at the end of the tunnel. And in the U.S., also, they're starting to relax a little bit. And I would imagine that things will -- the sites will have more ability to gather the necessary information. So, so far, I'm not too worried about it. Thank you.

Our next question will come from Matthew Harrison with Morgan Stanley.

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

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Q

I just wanted to maybe just spend a moment on 1205 and Pinta. I guess could you just comment broadly, given that this will be the second IPF study that you read out, outside of the results specifically related to 1205, what this may tell you either from patient characteristics or being able to validate some of the more novel markers you're using, including the imaging? Will that -- will it help you at all validate or give you any more confidence around the early data that you have for your other IPF compound?

A

Piet Wigerinck, Galapagos NV - Chief Scientific Officer

Matthew, thank you for the question 1205. So 1205, the PINTA study is a proof-of-concept in about 60 patients and what is new compared to FLORA is that here, patients are on what you call standard of care, which is then 1/3 of the patient is on nintedanib, 1/3 is on pirfenidone and 1/3 is on a local standard of care where none of these drugs is approved. So in that sense, we are shifting from the

fully placebo-controlled local standard of care design in FLORA towards more the ISABELA setting, what is expected as well for Phase III.

In PINTA as well, we've included FRI, which we indeed will validate that as a more sensitive marker for stopping the progression of this deadly disease. So 1205, we have all patients recruited. And second half of the year, we'll have a good view, and it will help us as well understanding how, in a complex setting, trials are designed well and how the different groups compare in the progression over 6 months, which help us in the understanding. So the big offer is, of course, that it both would be active in view of the benign safety profile that we would really come within a combination treatment of the 2 compounds on the long term with a very clean safety profile and an efficacy which outperforms what we currently have in the market. Thanks a lot.

We'll next go to Phil Nadeau with Cowen & Company.

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

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Q

I did want to go back to the 3970 versus 3312 in the Toledo program question again. In the past, you had actually characterized 3312 as being one of the best compounds you had ever seen in your IBD preclinical models, and now it's been passed over for 3970. So I'm curious, why is that? Were the preclinical model somewhat not predictive of what you saw in the clinic? Or is 3970 producing simply better data on biomarkers than 3312 in the clinic? It does seem like something's clearly changed in your enthusiasm for 3312.

A

Piet Wigerinck, Galapagos NV - Chief Scientific Officer

Yes, Phil. Thanks for coming back to the Toledo program. So what happened is over Phase I, in fact, that -- this 3312, which is a colon targeting, you don't want to see anything in the plasma changing. So you want to see low levels, and you don't want to see any changes. And that's what we saw, but that doesn't prove that you do anything good in the colon needed because you don't measure it.

3970 is a different compound, will absorb, distributes well from the plasma to the different tissues. And with 3970, as I said, we had the opportunity of doing plasma biomarkers. And as Onno said, we saw promising data there. So we are quite pleased with what we saw there. And then you have the bird in the hand, as we say, with those plasma biomarkers. You have the exposure. You want to go for the safety that we say let's push this forward because it is more solid than seeing absence of things. So that's what made us choose for a 3970 at this stage.

And we'll next go to Dane Leone with Raymond James.

Dane Vincent Leone, Raymond James & Associates, Inc., Research Division - Research Analyst

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Q

So I want to actually go back to the IPF commentary that you made on the statistical modeling. I just had -- got some follow-up questions on that. So was the question on pushing out the futility analysis based on fewer data points than you had expected on the front end of the curve. And is that because you're using an MMRM model to try and model out missing data points? And has there been any issue with missing data points from dropouts in the study?

A

Walid Abi-Saab, Galapagos NV - Chief Medical Officer

Yes. Thanks, Dane. Thank you for the compliment. But that was a low bar anyway to cross being better than the (inaudible). No. Yes, I think you're absolutely right. We will be using statistical modeling. I'm not sure if it's MMRM specifically. I think our statistical colleagues would be much better at answering that point. But indeed, we rely on data from the earlier time points to be able to estimate the efficacy. It was actually the week 52 for those individuals. So those will add the power. We're not just taking only the 30% or 33% then across the finish line.

And I will tell you, we will -- we're not having any concerns about dropout in ISABELA. I think the ISABELA trial is really -- the way we designed it, we try to make it as much as possible closer to real world because we're going on top of standard of care. As such, we allow a much wider window of treatment. We allow change in the background medication. We allowed temporary stopping of medication, restarting because, again, we're interested in the overall treatment effect after a length of time. So, so far, we're very pleased with what we're seeing. We haven't had any concern in terms of dropout.

And since you're asking me about this, and I think it's important to do this, you can imagine those people are at high risk. They -- we worry about them. So we monitor this on an ongoing basis. We have a data safety and monitoring committee that actually looks at these data in an unblinded manner, and we've had a recent meeting with them where they gave us, again, the okay to continue with the trial with no changes.

In addition, we work very closely with our Scientific Advisory Boards and particularly with our lead PI, Dr. Toby Maher. And his take on this, and here I'm quoting, is to say, so far, in a large global database of IPF patients from the ISABELA program, we have seen a low event rate of possible COVID cases and no fatalities related to this at this point. So we're quite on top of it, marching very carefully. We're not worried about what we have seen so far or loss of data. And I'm very happy with how our patients are managed and they're able to be kept safe. So very pleased with this. Thank you.

And we'll take our next question from Laura Sutcliffe with UBS.

Laura Sutcliffe, UBS Investment Bank, Research Division - Equity Research Analyst

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Q

Could you just tell us with respect to Toledo whether we should think of this really as just 3970 for now given the current circumstances and some minor change in plans or whether you're hoping to get some of the other assets you have into the clinic in the near future?

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Piet Wigerinck, Galapagos NV - Chief Scientific Officer

Laura, thanks for asking on the portfolio of the Toledo molecule cell. So we have a couple of compounds following it. So we have -- we kicked it off with 3312, sequence 3970, and then we have 4399 coming as well. We plan to bring that one to Phase I this year. And in discovery, we keep on looking into all types of profile with TOL1, TOL1, TOL2s TOL2s, TOL3, TOL2, 3s whenever we can. So it's a very broad program, which we will then look where -- what are the best indications for the different profiles. So -- but if you have to do today's studies in the difficult environment, let us be clear then 3970 is well equipped to open up this broad program in the clinic. And with the following molecules, we'll see how they differentiate and whether we can push harder in certain diseases pending on the profile. Thank you.

And our last question will come from Lenny Van Steenhuyse with KBC Securities.

Lenny Van Steenhuyse, KBC Securities NV, Research Division - Financial Analyst

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Q

Just a quick one on the financials. We see some shuffling around of cash assets from cash in hand to financial investments back to cash in hand. So I was wondering if you could elaborate a bit on that one.

And perhaps a quick one on NOVESA. Walid, you mentioned the broader approach in this trial. Does this also imply that you would be looking at preliminary function readouts next to the primary endpoint of the MRSS?

A

Onno van de Stolpe, Galapagos NV - Co-Founder, MD, CEO & Executive Director

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Yes. Lenny, I'll take the question on the cash shuffling, as you call it. What we indeed have done in the first quarter is to go a bit further risk off in terms of our investments, and we've exited a couple of money market funds and have some further direct investments in deposits and bonds. That are --

again, are a little bit further risk off in the in the current environment even though we were already very risk off. But better safe than sorry, is, I think, the approach at the moment. So that's it for cash.

Maybe NOVESA?

A

Walid Abi-Saab, Galapagos NV - Chief Medical Officer

Yes. So thanks for the -- yes. So, indeed, we will be looking -- we'll be measuring FVC in that trial. However, we have not selected patients with scleroderma that do have interstitial lung disease. So this is -- we took -- essentially, this is a trial looking at scleroderma as a disease itself, not scleroderma with interstitial lung disease. But we will look at FVC, of course, and see what happens over time. I'm not very optimistic that we will have enough power to detect them all because I don't think we're going to have enough people at least who have interstitial lung disease represented in that trial, number one. And number two, usually people who have interstitial lung disease with scleroderma have a slower decline than IBS. Again, in this trial, I cannot -- I don't imagine that we're going to be able to pick up a signal. But we'll look -- so -- and we'll let you know how that comes out. Thank you.

Next scheduled call is going to be for the first half 2020 at 8:00 a.m. Eastern, 14:00 CET on the 7th of August.