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Welcome to the Galapagos webcast. At this point, I would like to hand the call over to Elizabeth Goodwin. Please go ahead, ma'am.

Thank you. Welcome all to the audio webcast of Galapagos' Q3 2018 Results and Annual R&D Update. I'm Elizabeth Goodwin, Investor Relations, and I'll be hosting today's event. This reported webcast is accessible via the Galapagos website home page and will be available for replay later on today. Note that we will be posting the file copies of our webcast slides to the website as well later today. (Operator Instructions)

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

Let's look at the agenda for today. Today's participants will involve some prepared remarks from our executives. Today, we also welcome Dr. Philip Mease from the University of Washington who will be joining us from ACR in Chicago. For Dr. Mease, we will open up the floor and phone very briefly for a couple of questions immediately following his talk. But for the others, I request you hold your questions until the Q&A session at the end.

So with this point, I'd really like to hand over to Onno van de Stolpe, our CEO, who's joining us remotely from the Netherlands today. The folks here in the room can see him. And Onno, please go ahead and start our talk.

Thank you, Elizabeth. Pleasure to address people in New York and the rest of the audience on the webcast. Good morning, good afternoon. Happy to give an intro on what's happening at Galapagos.

And of course, we'll start with the announcement we did this morning or late last night regarding the revised agreement with AbbVie. There has been a lot of uncertainty in the market regarding what was going to happen with the cystic fibrosis program especially after the last press release, where we announced we were reevaluating the collaboration with AbbVie. Now we have come to the conclusion that in this space, AbbVie is the better partner to continue in the program than Galapagos. Galapagos is really a new mode of action company focusing on novel targets that we discover with our platform and has been moved

forward. And clearly, in the heat of competition that the CF space is in, it is more a pharma play than a biotech play. And I think both parties have understood that and we've come to a very good arrangement, I think, both for AbbVie as well as for ourselves regarding the future of the program with a consequence that all programs move to AbbVie. They pay us an upfront $45 million. We get nice milestones along the way if they reach their goal of getting triples into patients and to the market. And we also get very nice royalties ranging in percentage based on the number of candidates in there. All in all, I think we are very pleased with this outcome, and I think it's really the best for the program. We negotiated giving access to 2737, the candidate, for indications outside CF, and we hope to report on that program in the future to you maybe as in the -- at a future R&D Day.

So that's the -- actually the CF story for us at least as an active participant. And of course, we'll report to you when we get more milestones or information from AbbVie regarding the program with AbbVie's efforts in CF. We want to thank AbbVie for the collaboration here and wish them all the luck in coming up with a competitive triple because that's clearly in the interest of both the companies but also, of course, in the interest of patients.

We go to the next slide. Let's have a look at the target discovery platform because that is really what Galapagos really is all about to identify novel mode of actions, new targets that are studied [4 points] for drug discovery and ultimately can lead to drugs like our autotaxin inhibitor or the IL-17C inhibitor that we have come up with. And it's all based on the technology that we developed 18 years ago when we build a collection of viruses, adenoviruses, with small pieces of human DNA in there that when the adenovirus infects a cell but uses a so-called siRNA that knocks down one specific gene in the human cell. And by doing that, it can mimic what a drug does in the body, and we can do it very effectively for every important human gene that is druggable, which you can develop a drug going forward. That collection is about 20,000 viruses, and targeting about 6,000 different genes. And that today, after all these years, is still the basis of our target discovery platform. It's very versatile. We can basically apply to any disease for which we can mimic that disease in a cell almost of all diseases, either easy to mimic. With quite a serious number, we have been able to come up with development -- a cell model where we use primary human cells directly out of patients and use those as a basis. We provide trigger and device readout to do the first selection of targets that have a phenotypic effect on the disease and that can be used as a starting point for the progress.

Of course, along process of target validation, before we go into the next phase -- and that's actually indicated on the next slide, where we show our ambition in the R&D pipeline building, where we start with about 8 new targets. That's the goal coming out of the target discovery platform that should, of course, with the necessary attrition, lead to 3 preclinical candidates; 3 proof of concepts, very important step in the value creation; and ultimately, 1 Phase III start every 2 years. That's the ambition that we have laid back -- laid out a couple of years back.

And as you can see in the next slide that we have actually realized that very well over the last 1.5 years, where we've come up with a substantial amount of normal target in our 3 main therapeutic areas, inflammation, metabolic and fibrosis; quite a large number of preclinical candidates; leading to proof of concept in the IGUANA trial, the ROCCELLA trial and the FALCON trial; and of course, the start of Phase III with 1690 in ISABELA trial. So the very steep objectives that we have set with regard to the development of the pipeline, so far, have been very well developed.

If we go and look at where our focus is now today in research, then clearly, it is

still for the biggest part in inflammation and then, of course, includes Toledo, the program that we'll discuss in detail today at the R&D Day. We also have fibrosis and metabolic take up half of the discovery efforts within Galapagos, and then we've got some small efforts on hepatitis B and some other small efforts that we will report on hopefully on a future R&D Day. But it's very focused on these 3 main therapeutic areas, areas that still need a lot of improvement in the way these diseases are treated. And we think that's a great opportunity for Galapagos, focusing on new mode of action.

If we go to the pipeline in the next slide, you can see that our portfolio is moving forward very nicely. We came up with 12 preclinical candidates in the last 3 years, which is an incredible, productive research engine. And we obtained that by starting about 7 to 8 new targets every year that go into drug discovery. Of course, projects stop all the time. 6 to 7 projects don't make it to -- don't reach the candidate stage. That is not a problem. Of course, we would like to keep those numbers as low as possible, but we currently have a portfolio of about 20 to 25 projects, and that's about the numbers that we would like to keep going forward to continue to fill the pipeline and to become -- to continue to fill the pipeline of a fully integrated biopharmaceutical company.

Slide. If you look at the portfolio today in the R&D Day, we'll focus on filgotinib and idiopathic pulmonary fibrosis, where there's a lot to talk about. We had made major steps forward over the last 12 months. But also, as you'll be following Galapagos, we've had excellent news in atopic dermatitis, where IL-17C antibody, together with MorphSys, has shown very nice data. We're now in Phase II. We've reached an agreement with Novartis, who took over this program. And hopefully, this will be developed in multiple indications, an important program for Galapagos. And also, in osteoarthritis, we're excited that we have now started the Phase IIb trial. Galapagos is executing this trial in the U.S. We're recruiting about 300 patients. Our partner, Servier, is doing that for rest of the world with about 500 patients. That is going underway very well and of course, a long trial with 12 months of treatment would give high hopes that we can see a disease-modifying activity of our molecule there.

Already said, we have about 20-plus programs in inflammation and fibrosis in discovery, and that includes Toledo, where today, we'll be showing you our results so far in animal models. We're also going to talk about the strategy to maximize this opportunity in -- as broad inflammation diseases as we can find.

So to the next slide. And this was the part that I was presenting. That's also a good reason that I'm still in Netherlands and not have flown to New York. And it's really up to the rest of the team now to present the rest of the data. And we'll start with the Q3 results that, of course, were also in the press release last night. And I am happy to give the floor to Bart. Bart, good luck.

Bart Filius, Galapagos NV - CFO & COO [4]

Thank you, Onno. And good morning, everyone here in the U.S., and also good afternoon for those of you that are listening in, in Europe. I'll take the opportunity of this R&D Day to give you a quick snapshot on the Q3 results for Galapagos that we also reported yesterday. I'll do this rather quickly. I will have room for questions at the end of the session, but I'll do this rather quickly now so that we have enough time to focus on what really matters in terms of the purpose of

today, which is the R&D programs.

So maybe first, the delivery in our third quarter. And it has been a remarkable quarter for us. A hallmark quarter is what we called it in our press release because we had the first Phase III results of filgotinib in the FINCH 2 trial. And we'll go into that into a lot more detail later on. And those were very exciting to us, obviously, as a company. Then also in ankylosing spondylitis, we had the TORTUGA trial readouts. We had the first dosing in the ROCCELLA trial in osteoarthritis, our program that we partnered with Servier where we had the full U.S. rights. And also, in MOR106, we have started a bridging study for the subcu formulation. And as well in the negotiations with Novartis, we're expanding the development of MOR106 into other indications.

And at a corporate level, the closing of the Novartis deal has been in July. So that's been part of our third quarter as well. We had, in September, a follow-on offering, which raised EUR 300 million gross, and that has led us to a cash balance at the end of the quarter of a little over EUR 1.3 billion.

So let me get into the cash right away. This is a slide that I've been presenting quite a few times in the meantime. So most of you that have been tracking us know how to read this slide; on cash on the left, EUR 1.1 billion at the end of December and EUR 1.34 billion at the end of September 2018. And in between, there is the equity raise that I just mentioned with a net of EUR 286 million, but there's also the cash burden. Cash burden, the definition -- let me repeat that once more, is really a combination of cash coming in through milestones and upfront payments and, at the same time, the cash going out in terms of operating expenses. So the first 9 months has grossed EUR 100 million of cash burn. And we have, as a result also of the CF transaction that we have signed with AbbVie, lowered our expectation for the full year to a range of EUR 140 million to EUR 160 million coming from EUR 180 million to EUR 200 million. The EUR 40 million difference therein is exactly the same as the upfront that AbbVie is paying, which is $45 million in dollar terms. So EUR 1.34 billion of cash is a healthy balance to invest in our significant and broad portfolio.

In terms of key financials, I'll just go through this very quickly. On revenues, we've seen an increase on revenues of about EUR 100 million compared to last year, up to EUR 200 million over the first 9 months. A big chunk in there clearly is the recognition of roughly EUR 50 million from the upfront of the Novartis transaction, but there's also -- and that's something that's been recurring every quarter, roughly EUR 10 million per quarter of revenue recognition, which is the result of a change in accounting standards called IFRS 15, and that has helped us in our top line with about EUR 30 million. Operating cost, at the same time, has also gone up by roughly EUR 90 million compared to the same period of last year. As a result, the operating profit differential between the 2 is EUR 10 million positive. And the cost increase is really driven mostly by 2 programs, filgotinib, clearly, which is really in the height of its spending as we speak here in 2018, but also in 2019, with all the trials that we are doing there. But also, these are beta trial. The Phase III program in 1690 is consuming a significant portion of cash and operating costs.

Our net result is negative EUR 44 million, which is better than it was last year, on one hand, because of the improvement in the operating cost but also because of what I would call a balance sheet translation effect on our currency position. We are keeping part of our EUR 1.3 billion in dollars. That's a little over $200 million in dollars. And obviously, dollar fluctuations reported in euros leads to some changes in financial results, which are all paper changes because these are not materialized. But as a result, we are improving our net results compared to last

year by EUR 40 million.

So there, I'd like to already stop with the Q3 results, again, so that we can spend as much time as we can on the other parts of our Capital Market Day, filgotinib, the early programs and 1690.

Elizabeth Goodwin, Galapagos NV - VP of IR & Corporate Communications [5]

Thank you, Bart. And at this point, we are going to toggle back to bring in Dr. Philip Mease from the University of Washington in Seattle. Dr. Mease, are you on the line?

Philip Mease, [6]

I am. Can you hear me?

Elizabeth Goodwin, Galapagos NV - VP of IR & Corporate Communications [7]

Yes, we can. We can hear you. And we are looking -- there he is. Welcome, Dr. Mease. Thank you very much for taking the time to join us there from Chicago for your flight back to Seattle. I invite you to go ahead and tell us more about the results you presented.

Philip Mease, [8]

Okay, good morning. So you're in New York, and good afternoon to those calling in from Europe. My name is Philip Mease. I'm a rheumatologist based in Seattle, Washington. I direct rheumatology research at the Swedish-Providence Health System and a clinical professor at University of Washington.

If I could go to the next slide, please. Let me just frame the presentation of the filgotinib data on psoriatic arthritis that occurred this week at the ACR meeting. The first comment to make is that the abstract was chosen as a plenary presentation. There are -- on 3 days of the meeting, there are, what I call, plenary presentations where there are no other concurrent sessions. There are 6 abstracts chosen for each day, so 18 abstracts out of the many, many thousand abstracts that are presented at the meeting. So that -- I'm framing that perfectly because you can see that there was a lot of interest in this particular study at the meeting. I think it reflects also a rising interest in psoriatic arthritis in general in comparison to rheumatoid arthritis and other rheumatic diseases, and it also reflects an interest in the JAK mechanism of immunomodulation.

So this first slide that is being shown indicates the basic biology of how JAKs, at

the receptor level, mediate cytokine stimulation of the cell. JAK1, 2 and 3 mediate pro-inflammatory cytokine signaling as well as [JAK2]. There -- filgotinib is a very specific and selective JAK1 inhibitor. And we put in, in here that JAK2 has some off-target signaling as well, including hematologic. And when we get to the safety data, we're going to see some reflection of the fact that filgotinib does not inhibit or work through JAK2, so may have less in the way of hematologic side effect issues. And then JAK3 has an effect on gamma chain cytokines critical for lymphocyte function. So we'll be -- as we get into more and more selective JAK inhibitors, there are going to be 2 things that we're looking at. We're going to be looking at, is there any decrement in efficacy as we become more selective? And is there going to be [any pre in] -- or, excuse me, a decrease in adverse effects as a result of greater selectivity?

So let's go to the next slide. Here is the molecular model of filgotinib, as mentioned, a highly selective JAK1 inhibitor, 30-fold selective over JAK2. It has, via catalysis, shown here including that of the active metabolite and its basic mechanism of action. It has demonstrated activity in both Crohn's disease and rheumatoid arthritis.

Next slide, please. This is the study design, quite simple, Phase II study in which patients with psoriatic arthritis were randomized, receiving either filgotinib 200 milligrams per day take orally or placebo, roughly 65 patients per arm. The population coming into the study were predominantly patients who had been treated with a conventional synthetic DMARD, such as methotrexate. But there is a small proportion of the population, approximately 15% of both arms, that have been previously exposed to anti-TNF therapy. The way psoriatic arthritis trials are designed typically is that if the patient is on a background csDMARD, such as methotrexate, they're allowed to stay on it but they're not required to. So you get information both in monotherapy as well as add-ons to beat combination DMARDs.

Next slide, please. This is the primary end point study, ACR20 at week 16 by NRI analysis. This was achieved by 80% of the patients in the study versus 33% in the placebo arm. This is a -- one of the high results that has been seen in recent times in PsA trials and not only the overall threshold of ACR20 response achieved but also the effect size, subtracting of the placebo arm from the filgotinib bar. So this is a very sturdy result. I mean, at least, put it that way. If we look at the linear time course of the ACR20 response by 1 week, one can already see separation between the filgotinib-treated patients and placebo.

The next slide, please. Now we're looking at a higher threshold of response, ACR50. By the way, I'm assuming everybody knows what the ACR response is, but it's a composite of a number of different factors, including tender and swollen joint count, complication, pain, patient global function score and acute phase reactant. So this is at least a 50% improvement and what many patients find quite satisfactory, and 47.7% of the filgotinib-treated patients achieve this versus 15.2% of the placebo arm.

Before I go on to the next, I'm going to just mention a couple of other outcome measures briefly. There is much more shown in the presentation than I'm showing in here. But I also, at the very end of the talk, will steer you to The Lancet article in which all of this is published in detail. And one of the exciting aspects of this presentation was that simultaneous the presentation at the ACR meeting, the publication of the manuscript of this study was published in The Lancet along with the ankylosing spondylitis trial with filgotinib, which also showed -- it was a successful trial in terms of treating that particular condition. And by the way, we know that spondylitis occurs in psoriatic arthritis as well, and so we could use

those results there.

So just to mention the fact that patient pain improved very quickly with the -- a major improvement occurring in over half the patients. That's at least 50% improvement in pain. And part of the reason I mentioned this is that there's been some interest recently based on some of the results with other JAK inhibitors. There may be a specific and interesting effect on pain response that could be partly independent of treatment of inflammation. So that's just something for you to be aware of to track as you're looking at the data results.

The other important measure to mention is what's called the minimal disease activity measure. This is the composite which more holistically includes other aspects of psoriatic arthritis, including skin response and [uveitis] response. So that was achieved by 1/4 of the patients by week 16. So that's nearly complete remission of the disease.

Next slide, please. Now we're looking at a function score known as the health assessment questionnaire. And as you can see, this improved rapidly and significantly during the course of the trial. And on the right-hand side, you're seeing what's called a minimally important difference, and 2/3 of the patients achieved this trend, showed a 0.35 change in the HAQ score.

Next slide, please. Now we're looking at skin response. This is known as the PASI 75. At week 16, 45% of the filgotinib-treated patients achieved PASI 75 versus 15% in the placebo arm.

Next slide, please. Now we're looking at an enthesitis score. This measures the pain and inflammation that occurs where tendons and ligaments insert into bone. This is an important clinical domain in psoriatic arthritis, and there are several methods of scoring this. There happens to be one called the SPARCC enthesitis index, a little higher threshold index of 18 sites that we're measuring. And as you can see, there was a statistically significant mean separation, i.e. in the SPARCC score, and a numeric increase of complete resolution of the SPARCC. There is a another enthesitis measure also used, the Leeds enthesitis index, and that showed approximately 50% of patients in the filgotinib arm achieving a complete resolution of that particular index, a little slightly lower threshold in -- at week 16, so an important additional domain that sometimes takes a little longer or a little tougher to treat, [the synovitis] or skin disease.

Next slide, please. Now we turn to adverse events. And overall, the adverse event rate was relatively low not -- without surprising results. Compared to the rheumatoid arthritis and Crohn's disease data with this medication, there was one death in the trial due to a pneumonia incident only at the bottom of this table. There were no malignancies. There was no deep vein thrombosis, there was no pulmonary embolism, and there were no adjudicated major adverse cardiac events.

Next slide, please. I'd like to just mention briefly the -- some of the laboratory study results. And here, we're highlighting the hematology parameters. If we look at the top left, the hemoglobin results, what is shown is that there was actually an increase in hemoglobin in the filgotinib-treated arm, which likely is a result of treatment of inflammation, leading to improvement of hemoglobin. And there were no Grade 2, 3 or 4 decrements of hemoglobin noted. So this is, I think, as much an interesting point about biology as it is about safety, and that is that with more selective JAK1 inhibition, this -- it suggests that there may be less impact on some of the hematologic parameters. And if we turn -- look at the lower right-hand corner, the lymphocyte, there is no mean change on overall lymphocyte

count. There were 3 episodes of Grade 2 change, no episodes of Grade 3 or 4 change.

Next slide, please. So I would like to conclude and indicate that this was a successful trial with filgotinib in the treatment of patients with psoriatic arthritis. The primary and key secondary end points were achieved. And as you could see, there was rapid improvement noted with separation from placebo as early as 1 week. And overall, the safety profile was as expected compared to what we've seen with this agent in rheumatoid arthritis and Crohn's disease.

Next slide, please. And if you would like to have more details about the results of the study, I encourage you to go onto the online publication in The Lancet. As mentioned, it appeared earlier this week on Monday, along with the results of the ankylosing spondylitis trial in the same issue of The Lancet.

Thank you, and I'd be happy to address any question.

Elizabeth Goodwin, Galapagos NV - VP of IR & Corporate Communications [9]

Thank you, Dr. Mease. This is Elizabeth here at the Yale Club. Operator, could you please instruct callers as to how they can pose a question?

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Questions and Answers

Operator [1]

(Operator Instructions)

Elizabeth Goodwin, Galapagos NV - VP of IR & Corporate Communications [2]

Okay. And while we're waiting for people to dial in, are there any questions here in the room? I see a question here from [Sam].

Unidentified Participant, [3]

[Sam Wisely]. The -- you are a little bit late, at least, in the JAK space. The body of evidence on safety and effectiveness looks pretty good. Would you say that, that is because of your JAK1 selectivity? Or are there other aspects of your selection of the candidate? If so, what are those? Did you do some structure activity work? Or did you just perhaps -- it does happen. Did you just perhaps get lucky?

Philip Mease, [4]

Elizabeth, what might be best is some of the -- some aspects of that question are going to be best handled by internal representatives of Galapagos. A comment I might make just as an outside physician is that I'm not -- I want to caution you that these are Phase II results. We did see a very high ACR20 response, which is good. But as you know, sometimes, in Phase -- as you move into Phase III, results come down to earth a bit more. So it's a little bit tough to take this data and say that it's fairly -- that there is a signal for necessarily greater efficacy. I think a key point though has to do with the fact that there may be some differences in the safety.

Elizabeth Goodwin, Galapagos NV - VP of IR & Corporate Communications [5]

And is there a question on the line, operator?

Operator [6]

There are no questions at this time. So I'd like to turn the conference back to you, Elizabeth.

Elizabeth Goodwin, Galapagos NV - VP of IR & Corporate Communications [7]

Okay. Are there any other questions here in the room? Okay. Thank you very much, Dr. Mease. We hope you have safe travels back to Seattle. Thank you.

Philip Mease, [8]

All right. Thank you very much.

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Presentation

Walid Abi-Saab, Galapagos NV - Chief Medical Officer [1]

All right. Good morning, everybody. Good morning -- or good afternoon for those of us joining us from other parts of the world. Can you switch to -- perhaps I can address one element of the question. It will come through also in my presentation, but I want to say it here. I agree with Dr. Mease that again, this a Phase II trial and about 60 patients each. So we'll have to take those data with the limitations of the size of the study. But if you look at the totality of the program with filgotinib, I think the data are becoming more and more consistent, demonstrating the very high level of activity in terms of efficacy with a best-in-class safety and tolerability profile. And I think this is really borne out as a result of the selectivity. And I think this is a core area of focus in my presentation now. So I'm happy to take your question afterwards if you want to delve into more details after we open the Q&A session, okay.

All right. So talking about filgotinib and you look at this and you see generally that this is a pipeline in the drug. So we're, generally here, building a franchise with this compound. We have a number of studies that are going on in Phase III in rheumatoid arthritis and inflammatory bowel disease. But in addition, there are a large number of data in Phase II trials both in rheumatic diseases and inflammatory bowel diseases that are coming through. And now we've seen data from psoriatic arthritis, and I'll show you later data from ankylosing spondylitis. But we also have studies that are ongoing in Sjögren's syndrome, uveitis and lupus erythematosus. So we're very excited about the data coming up and the way this program is shaping up.

So looking at this, looking at -- about our ambition with filgotinib, I think we have -- I'm counting in my head. Previously, we have about -- well, actually, we have precisely 8 double-blind, placebo-controlled trials [that I have] with filgotinib from Phase II, Phase III, including some study in Phase II that Gilead has recently run in combination with their Syk inhibitor. And filgotinib, in the past, consistently performed as we've been expecting it based on the in vitro activity and the selectivity for JAK1, specifically very strong activity on efficacy, signs of symptoms of various diseases, both rheumatologic and also in Crohn's disease; a rapid onset of action, which is also sustained; safety profile, which -- up today, we have about -- more than 2,000 patient year experience. And the safety profile is shaping up to be best-in-class for the JAK inhibitors. So when you look at these 2 in combination, couple it with the fact that it's once daily and it has a potential for monotherapy, we think this is going to be a very important medication for many people living with rheumatic diseases and inflammatory bowel disease. And as a result, it will be also commercially very successful.

So where does the story start? It starts here with the high selectivity. You heard Dr. Mease highlighting this for the -- in the case of JAK2. These are in-house data. But also, those were independently validated by Professor McInnes, an independent and very well-respected investigator in the space. These are in vitro data that show, on the left-hand side of the graph -- the graph on the left-hand side that shows the selectivity for JAK1 over JAK2. And on the right hand of the side -- of the slide is JAK1 versus JAK3. And I think it's very clear that based on these in vitro data, the selectivity for filgotinib is very high compared to other JAKs in development. And then why is that important? It's important because this will have consequences clinically. In the next couple of slides, I'll show you some clinical data from the earlier studies. But as now, we're starting to build up more of our database with large [Phase III in vitro] studies reading out. The story is shaping up to support these assertions, and we've made this on the in vitro experiment. And I think that's really important. It's going to be a differentiating feature for filgotinib compared to the other JAK inhibitors.

So here, again, is a clear testimony of the effect of JAKs on off-target activity with

JAK2. So just to set the stage a little bit, chronic inflammatory conditions lead to anemia. And when you treat the underlying condition, people usually recover. The secrete EPO and EPO signal through JAK2 and you increase your hemoglobin. Drugs that effectively treat inflammation and do not interfere with the JAK2 signaling, like filgotinib, like adalimumab, as a matter of fact, you do see a very nice increase in hemoglobin as you treat the underlying condition even in short studies like this that we're showing 12 weeks. Again, to be very clear, those are not from the same studies. So we're just putting them adjacent to each other. So use the necessary precautions in interpreting this, but still, we think this matters a lot. You see that very clearly in the case of baricitinib, in the case of upadacitinib, where you would have a reduction -- dose-dependent reduction in hemoglobin over time.

And here on the next slide is a similar story with platelets. If you recall, if you have been watching the Advisory Committee for baricitinib, this is top of mind for the FDA, where they focus a lot on the role of JAK2 actually in platelet levels. Similar story here, chronic inflammation leads to increase in acute phase reactants, increase in hypercoagulability, increase in platelet levels. And when you treat with the underlying condition with the effective drugs, again, that do not interfere with JAK2, you have a reduction in platelets. Again, we see it with adalimumab. You see it with IL-6 inhibitors, and you see it also with the filgotinib and actually with tofa, which does not interfere with JAK2. However, in the case of baricitinib, you do see an increase. And in the case of upa, based on data that have been stated by AbbVie, there are no changes in platelets, whereas one would expect a decrease.

Moving on to the JAK3 story. Here you can see very nicely JAK3 affects the NK cells and downstream effects of it are the rate of infections and so on, so forth. And you will see on the left-hand panel with filgotinib that we don't see any appreciable changes in NK cells, whereas these changes are quite obvious with tofa and upa. So these are our data that we've been showing for some time on a regular basis, actually. Virtually every 6 months, we update EULAR and ACR. This is the long-term extension of our DARWIN 3 study. And often, we update it with updated data that our -- that the other JAKs are showing. Unfortunately, there's not been any recent update on the upa, but the bari and tofa data last week at the ACR continue to confirm what they have seen.

Looking at filgotinib, again, with more than 2,000 patient year experience, our data continues to differentiate themselves positively. Again, this is an open-label extension trial, so you have to use that judgment in looking at the data, but we're very pleased to see the rates of serious infection, herpes, DVTs and PEs and death, which are very clear here and trending quite low. In the case of DVT and PE, because I know this is something that is top of mind for a lot of you guys following this space, these represent 2 events actually in 1 patient, 1 DVT and 1 PE. And there's been, to be very clear, no change since the past year or so that we've been reporting on this. So the FINCH program, which is ongoing, is represented here. You will see, as I will discuss later the results of the FINCH 2, we've always said that we're very pleased, in fact, that we evaluate both 100 and 200 milligrams equally virtually in the whole program, and that will enable us to make very solid risk-benefit assessment for each dose when we look at the data. So these studies are ongoing. As you know, we reported on FINCH 2, and I'll go into more details today. FINCH 1 and 3 have been fully recruited and should be reporting early next year.

In the IBD, the programs are ongoing. Again, those are robust programs, both doses are equally evaluated in these programs. 1,300 patients in UC and 1,300 in Crohn’s, and both of these are now officially in Phase III. So without further ado,

and I apologize I'm going to go fast and I'm going to also select the data. Because if we wanted to go through all the data and you've heard Dr. Mease before, he added more than what was saying on -- what was shown on the slide, we will end up the whole day talking about filgotinib, which is not a bad thing, but we have other stuff also we want to share with you.

So let me start with, again, being very proud of the fact that we have not 1, but 2 Lancet papers appearing on the same day in coincidence with the plenary session on the psoriatic arthritis. So this is our study in ankylosing spondylitis. Again, we refer you to this. As you know, the Lancet and the appendix, there are a lot more details that you can go through and look at the data if you have further questions.

Study design, again, it helps to present this after Dr. Mease because it's virtually similar. In general, simple design, double blind, placebo controlled. Duration here is 12 weeks. These are patients with ankylosing spondylitis. About 60 per arm were randomized to either filgotinib 200

or placebo. These are patients who could have been on TNF before or are naive. The overwhelming majority actually were naive or about 90% people were naive, or they could have been on monotherapy or on conventional DMARDs, and about 40% of them were on conventional DMARDs. Primary endpoint at week 12 was the ankylosing spondylitis disease activity index. And this we show here, you see on the left hand side panel, at week 12, a robust reduction in the -- robust improvement in the ASDAS compared to placebo. And on the right hand side, you see also the time course, which again, is very consistent and this is a story you'll see it consistently from the psoriatic arthritis, ankylosing spondylitis and later, rheumatoid arthritis study that I will be discussing. The results are seen at the first endpoints we look at. In this case, it was 2 weeks and the results continue to improve and are sustained. Actually in this case, if you can see and you can appreciate from the curve, I don't think we reached a plateau by week 12. And I think it reflects also the more difficult disease that ankylosing spondylitis is. As you know, a number of compounds in this space, which have been active in RA or in psoriatic arthritis failed in ankylosing spondylitis, reflecting probably a different biology but also a more difficult to treat population. And as a result, a higher unmet medical need. So we're very excited about the data that we've seen. So this is the look at the ASAS20, this is the Assessment of the SpondyloArthritis based on the International Society, so another way of looking at it. You can actually derive the ASDAS from this 1, but this 1 takes out the CRP as part of the scale so that you don't kind of bias it for JAK inhibitors in general. But again, you see a very robust effect with ACR20 reaching 76% after 12 weeks of treatment. And again, you see this continuous increase which doesn't seem like it is plateauing, at least in this study.

Looking at the functioning, which is actually very important for patients, this is the BAS Functioning Index. And you see very robust changes, improvements that are seen early on in the trial and they're sustained throughout the trial and actually continue to improve. Again, the same story, I don't have a -- I don't have a feeling that we have completely plateaued at this point, so there's hope that with longer trials, we'll see even a stronger efficacy. And we'll look at spinal mobility with BASMI. Again, very robust effects that are observed starting at week 4, which is the first time we looked at it, and continuing to improve throughout the trial. When you look at MRI, and evaluate specifically inflammation, either in general or on the right hand panel, you see for the sacroiliac joint, which is the joint that's mostly affected in ankylosing spondylitis. Again, these are very robust effects that we are seeing in terms of efficacy.

Moving on to the adverse events. Again, consistent story throughout the day. The

rates of adverse events are not going to be any different between placebo and drug. Here, the rates of events over 12 week periods are about 30% for both. We've seen the same in ankylosing spondylitis. I think the rates there in psoriatic arthritis, the rates there were about 50% or 55%. But again there was no difference between drug and placebo. When you look at infections, they're equal. In terms of serious infection, we had 1 case of pneumonia at the filgotinib group, and that was grade 3. But when we stopped the drug to treat the patient, the patient recovered with no problems. When you look at other important infections such as opportunistic infection, herpes, tuberculosis, there were no such events seen in the trial. And the pneumonia is the 1 that I mentioned is the same seriously emerging adverse event, and that's the same event that's being represented. Malignancies, there were no malignancies seen including lymphoma. There was one case of deep venous thrombosis in the filgotinib 200 milligram dose. That was non-serious. This was a patient who had a predisposing condition, he was heterozygous for a condition called Factor V Leiden mutation. This a mutation that increases your risk of thrombosis and otherwise, is not recognized. These patients obviously -- often actually, realize that they have this condition once they have a thrombotic event that happens. In this case actually, this individual found out his diagnosis when his brother had a thrombotic event. And then the patient did the testing and turned out to have this condition. There were no cases of pulmonary embolism, no cases of major adverse cardiac events (MACE), and no death in this trial.

So to conclude, when we discussed these data with a number of experts in the field that were really impressed by the consistent effects that were seen with filgotinib in this patient population, not just only on symptoms but also on multiple domains in terms of physical function, spinal mobility and also, the joint inflammation as seen in MRI. So we sense a great level of excitement because of the consistent effects across multiple domains. And when we looked at the tolerability, we were quite pleased with the results that we're seeing. And again, we continue to build on the data that is supporting our position that filgotinib is looking like having a best-in-class safety and tolerability profile.

Let's go to the exciting FINCH 2 data. These data were released at ACR and a poster, late breaking poster. And the data contains more information that I'm -- again, I'm going to be able to show today. But we're very excited about this. This is, if you recall, this is in the most difficult to treat population, right? So we so we have FINCH 1, which is in the methotrexate IR or conventional IR; you have FINCH 2, which is in biological IR; and then you have FINCH 3, which is in the early RA population. So these are the most difficult to treat populations. Study design, 24-week, double-blind, placebo-controlled trial. And these guys receive placebo for the full 24 weeks, this a little bit different than some other competitors have done. About 150 patients are randomized 1:1:1 to either placebo, filgotinib 100 or filgotinib 200 milligram once daily. The primary endpoint was at 12 weeks and was based on ACR20. This is, again, what the regulatory authorities in the U.S. use and that's what was used in the trial. Patients after completing trial will enroll into an open label extension. And actually, that's the case for all the other FINCHes as well.

So these are the primary endpoints. On the left-hand side, you'll see a very robust effect that we see at the 100 milligram with 57.5%, meeting the ACR20 response rate, compared to 31% in placebo, but even a higher number with the ACR20 at 66%. And those are very impressive if you take into consideration this difficult to treat patient population. The right hand side, you see the time course, not only through 12 but also 24 weeks for placebo and the 2 active doses. And you see very nicely, again, a very rapid effect seen at the first time point we look at, which is 2 weeks. It goes up, I think it plateaus around 8 weeks and then it sustains

throughout the trial, with a very respectable 55% and 70% ACR20 response rates for filgotinib 100 and 200, respectively. And here, we look at the higher threshold, so to speak, the ACR 50, the ACR 70. So on the left-hand side panel, you will see the week 12 and the right hand side panel, you'll see the week 24. And you see, again, very nicely in ACR 50, of about 30% and -- 32% and 43% for 100 and 200 at week 12 and 35% and 46% for the 100 and 200 at week 24. ACR 70 continues to improve over 24 weeks and it's about 1/3 of the patients on 200 milligrams reached that threshold compared to 8% placebo. These data are quite robust.

So try to see how does this compare because we all compare. We try to put this and try to get the data from the similar trials that were done with other JAKs. So let me take a couple of minutes to kind of situate you here. The orange dot or semi dots as they're showing are the placebo response, the actual placebo response of the trial. The green -- dark green dots will be the active drug. And the numbers in the bar are the difference between drug and placebo. And so these are data from week 12. You see our data from FINCH 2 on the left-hand side panel and then you can see the data from RA-BEACON for baricitinib; ORAL STEP for tofa; and SELECT BEYOND for upa shown at the right hand side. And if we look at ACR 50, which is a little bit of the higher threshold and often where it's clinically looked at by rheumatologists, we're being told, you see again, that story, how it plays out here. Again, those are not from the same trial, so it's important for you guys to take that into consideration when you look at the data, but it helped us to figure out where our compound fits in this space.

So here, we talk about the patient reported outcomes and patient functioning with the health assessment and disability index. And again, a very strong effect at week 12 here, showing close to 0.5 for both doses compared to only 0.2 for placebo. These data are quite good. The American quite as in very good, the English is quite differently. And if you use other ways to measure, which is the low disease activity, defined as the DAS25(CRP) less or equal to 3.2; or remission on the right-hand side panel, with DAS28(CRP) less than 2.6. You see the responses with filgotinib 100 and 200 that are very robust in both of these cases.

So moving on to the safety and tolerability. Again, the story here is the same. If you look at the first row up top, that -- these are the rate of AEs. This slide is a bit busy, we tried to break it down with putting bold and regular text. But if you look at the top, you see that the rate of AEs are not different between active and placebo. And more importantly, there's no dose-dependent increase with filgotinib. I think this is an important point on the safety and efficacy, and I think that's a differentiating point compared to other JAKs that are either approved or are in development. What we have been seeing is an absence of dose-dependent increase in the safety and tolerability. And again, that is due to the high selectivity for JAK1.

If you look at the AEs leading to drug discontinuation, the numbers are quite small and not very different between drug and placebo, 2% versus 4% or 3.5%. When you look into serious AEs, again, we're not looking at appreciable differences and definitely no dose-dependent effect in the case of filgotinib. In the case of infection, I think there is a small uptick in the rate of infection, so we have about 26% on placebo and 34% to 36% on filgotinib. But when you look at the serious infections, that is not borne out, the rates are very similar between placebo and drug and certainly, absence of any drug-dependent increase with serious infections. There were 2 cases of herpes zoster, both of them were -- I mean, 4 cases, 2 on each of the active doses of filgotinib, there was none on placebo. There were no cases of opportunistic infection, no tuberculosis and no pneumonia in this trial.

Malignancy, including lymphoma, there were none in this trial. And there was no cases of deep venous thrombosis or pulmonary embolism in this trial. There was 1 subject randomized for the 200 milligram of filgotinib who suffered from a retinal vein occlusion. It's a thrombotic event but it's in the retinal vein. Looking at the major adjudicated cardiac events, MACE, there were 2 cases. One was on placebo and one was on the 100 milligram dose. There were no deaths in the trial. And again, we always try to compare and the next slide, I'm going to try to compare now the safety, and I'm going to do that by juxtaposing SELECT BEYOND with upa with ours. Let me set it up a little bit because SELECT BEYOND used placebo-controlled for only the first 12 weeks. Then, those who were on placebo, half of them got randomized to 15 of upa and 15 -- and 30 of upa. So for us to be able to add the numbers together and those are derived from the Lancet publication of SELECT BEYOND. And so we essentially added the n of patients who were randomized to upa 15 and 30 from the beginning, plus those who were changed from placebo to active in the second part. So that's how we arrived to the and at the bottom. And looking at the slide and looking at serious adverse events as well as adverse events of interest, I'm not sure I need to go through it in detail. Maybe I'll just show it to you here to demonstrate how the selectivity of our drug is translating into an adverse event that is shaping up to be best-in-class.

Now, these are not head-to-head, so you have to be very careful when you interpret this, but we show these data because we were impressed with the difference that we see between our drugs and others that are in development or on the market.

So to conclude on FINCH 2, we see a significant and rapid and sustained improvement in the signs and symptoms in this difficult to treat population. These are people who have failed more than 1 DMARD. Actually, many of them failed 2 or 3. We see a very robust efficacy at the 100 milligram and when you compare across studies that I've shown you before, you can see that very clearly. But the 200 milligram even did better than the 100 milligram, at least numerically in the trial. But that was consistent across the board, even when we look at the time course. I haven't shown you these data but they were in the poster. When you look at the ACR20 response rate and you compare whether -- depending on those who receive -- who failed 1 biologic to 3 or more, the response rate was not different between these patient populations. So the adverse event profile are consistent with the Phase II data. And again, based on our in vitro activity that we have selectivity for JAK1, as I said, we're very excited about the fact that we don't see evidence of increase in adverse event with those. So this is to us, an increase in safety between doses with no increase in -- increase in efficacy between doses with no increase in safety and tolerability as a differentiating factor and unique factor, actually, for filgotinib amongst the JAKs that are either approved or are in development. And I will leave you with this slide to tell you although we told you a lot of news on filgotinib, this is just the beginning. We are going to be having a series of additional data that we're going to read out from our Phase II program, also from our pivotal programs, and also news about a substantial Phase III start as well as filings, strategies and launches the coming few years. And with that, I'll turn it over to Piet to tell you much more about what's going to happen in the future. Thank you

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Piet Wigerinck, Galapagos NV - Chief Scientific Officer [2]

Thank you, Walid. And welcome to all the people in the room here in New York,

and as well to the people on the line. There's a good reason to stay a bit longer in the stock because I have a couple of scoops for you. So for the first time, I'm going to present to the outside world the project that we have coming and that's going to keep us busy for the coming years. I'll show you the thinking behind and I'll show you some of the impressive animal model data that really has convinced the whole company, in fact, that while we're so pleased with the efficacy we see with filgotinib, we need to remain ambitioned and we need to remain and try to push that up to the next level of efficacy. I also have a scoop in the IPF right there, where I'll highlight 2 new compounds in the pipeline and I'll show some data on that.

But let's start with the Toledo franchise. It's in fact, a Spanish town that we call it after this project. Some people ask me where is that name coming from and it's a very nice town in the center of Spain. And for all of you ever traveling into Europe, if you're out of ideas, it's always a good idea to go and visit that city. First of all, the question, does all these diseases still need next level drugs? And let me show here, over time, the evolution of how many patients fully can control or almost fully can control the signs and symptoms of psoriasis. You can see about 15 years ago in 2000, it was less than 10%. And with good science of many companies, we've now pushed this to 80%. So let's be clear, for disease, the room for further improvement is extremely limited and that's what we're after here. We are very pleased with this level of efficacy. This scoop shows the diseases and in green, we have RA. I was impressed and pleased as we are with our history data. If you look from the flip side effect, you take ACR70 as a measure that patients get almost full control of the disease. 40% is the really the max of today. There, we really see room for improvement beyond the JAK and other treatments that are currently in development. So there, we believe there is room to remain ambitious and to keep on looking for treatments that pushes the next level of efficacy.

Also in IBD in red, the situation is similar. Over the past year, the levels of efficacy have remarkably improved. We bring much better treatments to the patients than the ones we currently have. We should take the best drugs in development but still, less than 50% of the patients get their disease efficiently under control. So we really believe that there is still room for improvement there. And as filgotinib goes from PoC to PoC in Phase II and we have 2 studies, we'll see more diseases, where will filgotinib be bringing next level of control. There were still some of the patients, even those excellent drugs are not good enough. So being ambitious about next level of efficacy also puts a challenge on the table. And so we really took a couple of steps back and asked ourselves what type of a disease model do we need to design in order to have a chance of finding new drugs? And so let me take you to inflamed tissue. In fact, this is the patient with RA. And in fact, the patient suffers from the disease because there is an [im] cells. The immune system acts and the patient is not capable of controlling that action. So the immune activity goes out of control and causes local damage. So all of the red dots here are the cytokines and those are in excess of thro in inflammatory cytokines in the joints. And the cytokines bind with receptors and then they will sink through a JAK, and the yellow arrow there tells you that, that immune cell is on fire.

So current drugs then, we have different classes that do different things, but fundamentally do the same. They either block the cytokine, they either block the receptor or they block the JAK. In the end, so what they do is they dampen the overstimulation of the immune cells, so there's the consequence. If you're now a bit ambitious and you want to find something next, what we really want to have is a kind of treatment where we have less cytokines. We don't want to be the fireman anymore with our treatments, we really would like that we give the tissue the control back over the immune reaction. We want that the tissue is capable of reacting to the im cells but at the same moment, is capable of controlling that.

That's also kind of a challenge to the team. We said okay, let's now be honest and we can go for the next cytokine, we can go for the next receptors, the singling, whatever we can, optimize a couple of tissue properties. The effect if we really want to bring -- find something novel, we need much more complex systems. And that's where we designed this co-culture system. So it's a 2 layer system where on top, we have epithelial cells that may make the GI barrier in the tract. And in the same well but below, we bring in the sensory cells, the dendritic cells. And if you now add to this mixture a dark component, namely E. coli bacteria, we can put the whole system on fire and we can start to study the interplay between the sensory cells and epithelial layers. And so this types and it was only 1 way of many more of those, of co-culture systems that allow us to fundamentally go and look for more complex ways of inhibiting other immunity disease. One of them that we discovered is the Toledo. And the on the right, you could see dose response of the compound. In dark brown, we clearly show that when we apply it in the co-culture, we have a nice dose response. And even when we add bacteria, we can completely protect the GI barrier. On the other hand, we can run the model as well with a single cell, so no dendritic cells available. And at that moment, these compounds don't do anything, so kind of proof that really with these more complex models, to attain a more complex mechanism of action.

Dendritic cells are 1 type of immune cells, we've studied the Toledos in many more cells. Quite intriguing data and also, exciting data we got in the macrophages. What you see here is, in green, cytokines that drive the reaction. These are the pro-inflammatory cytokines. You see from left to right, a nice dose response and we suppress those, which brings you more or less into the same way of working as you would have with a JAK. What we didn't expect and we are very pleased to observe is that for the first time now, we see that we also as well with the same molecule and the same dose response rate, we can push up the anti-inflammatory cytokines. So we give the system much more control back to make sure it can respond to a challenge but it can control as well. And that's what when we saw that data, that made us start to dream to, wow, if you now can have a double punch system where we bring down the bad ones and we push up the good ones, we might have a good chance of bringing a next level of disease control to the patient. It all started in the GI tract and we extensively studied different animal models. On the right is the DSS model, this is the most frequently used model. In fact, the model where you locally irritate and where it's especially the local cells that play a role. And on gray on the baseline, these are the uncharged animals, you don't see any level of disease. The moment you start to challenge the animals in orange, you see the disease activity moving up. And so JAK -- some of the JAK compounds will score in the system, others for a reason, we understand don't do it well. And for us, we have another internal control, which systematically scores well and brings down more or less, the disease activity to a level we say can reach that disease as good as we've seen in this model.

For the compounds of this project for the first time, we have been really and compound after compound, seeing a much more at a much better disease control. So the amount of efficacy we bring and we observe in the model is something we've never seen before. And secondly as well, we see that extreme low dosages. So compound after compound effect, we see a better disease control and at a very low dosage. So really showing that for in this model, this compound nicely works. A bit more complex model is the T-cell transfer model. The epithelial barrier is not challenged and you bring in a piece of activated T-cells into the systemic stream of this immune compromised mice. From that, you will destroy as well the epithelial barriers. This is a model where for example, IL- 23 antibodies score well. Again, they're in gray if there is no T-cell activation, there's no disease. The moment you transfer those T-cells, you get a nice increase of disease activity. And again, there, our compounds of this project, they gives us much more control over

the disease than any other class we've studied before.

Finally, on the right, the MDR1 model. I don't know whether many people know this. In fact, it's an intriguing model. It's a model where we bring in a mutation in Pg people, and mice will develop over 12 weeks, a disease. So it's a whole bunch of research I'm going to really understand. Because also in patients, some of the mutations between Pgp may put patients at a higher risk of developing disease. But what exactly happens there is still not understood. We have the most complete setting of disease. And also there, we have the same picture, no mutation, no disease. With the mutation, the disease develops spontaneously after 12 weeks. With an abatacept, we can limit the activation of T-cells and partially separate this. With the Toledo, we almost fully suppressed any signs of disease. So clearly showing that from the complex model to a new way of controlling the disease by doing a double punch, pushing down the pro-inflammatory cytokines, pushing of the anti-inflammatory. We really have here, a whole cloud of compounds with a level of activity which is completely double and exciting to us.

Between that same concept as well, there is no reason why we're a bit lucky, this cannot work in other diseases. So these are now our first component we've tested in the CIA model. So the charge for the compound is a bit bigger, the dose of compound early needs to go to the joints, but as well show there, a level of disease control. So there as well, we see a -- start to see now a nice dose response where with the highest dose, we bring almost the disease fully under control. So this is a level of control we can reach with a JAK, we can reach with the best of the [ARAK] force. There's really an impressive level of disease control that we as well start to see. So with this target, we discovered in the GI model, we are now trying to expand this across a couple of other diseases.

So that's the ambition we have with the program. But the moment we've done IBD models, RA models, [skittles], psoriatic arthritis models, all of those disease activities, we start to see a very strong control of disease, and we plan to take this into lupus, osteoarthritis, OP, fibrosis, wherever we can. And the likes as well, we've been doing chemistry for 2 years and have the ambition of bringing a novel scaffold every other year. Because we've used scaffolds, we see different cell activities and as well, we see compounds that penetrate the different tissues into a different way. So we really want to maximally exploit any of the possibilities of this novel method of action. From there you see the local one. So we've proven that for the IBD models for ourselves, we have compounds that are systematically not exposed. We as well can completely control the disease activity. So over the coming years, you might expect from us a couple of compounds entering the pipeline, and they'll have numbers and they might be targeted at different diseases, thanks to the chemistry. Or they might be targeted to go on only if it's a local one.

So for our -- with this franchise the -- so the first compound has completed the preclinical top set, the 4-week IND enabling top study. We are writing the dossier and will submit this over the coming weeks and plan to start the first Phase I early next year. And then we hope to be in patients second half of next year for the first proof-of-concept.

Quickly behind will come an inhibitor from a different chemical class. And the plan there is by mid of next year, will be in the clinic as well. And then we're working on different chemistries on the local as well and they will follow soon. So our ambition is to bring 3 to 4, maybe 5 molecules into the clinic over the coming quarters, and then target them towards different diseases and try to prove in various proof-of-concepts and exploit the opportunities of this novel way of

controlling immune diseases. The strategy here is that I'm not going to tell you the target and that's of course, on all your minds, you would like to know the target, so -- but we're going to keep that hidden until we have our first proof-of-concept there to report out. And at that moment, we need to -- going to share with you the full scientific show.

The plan really is the biggest project in discovery, about 40 chemists working on it, about 30 people that explore the disease models in vitro and in vivo. So really continue for a number of years until we have full bases covered, all bases covered and then can push forward a number of molecules into proof-of-concept studies. And with all the experience we got from filgotinib, we can further to the market.

That's so far for the Toledo franchise. Let me now bring you to IPF. So I'll handle the earlier compound in the pipeline, and Walid will take over and explain to you how the Phase III program of '1690 is designed. Our science brought us to IPF. Honestly, when I joined the company 10 years ago, we did not have an IPF project. It was the autotaxin program when we looked for the best indication. That took us by our animal models into IPF. Only at that moment honestly, I started to realize that IPF really is a terrible disease. Patients that get the diagnosis, in fact, you can't get to us news for a couple of drugs. But we can't stop the disease, your life expectancy is limited, your lung function will decline and within a couple of years normally as a patient, you will die. It's a disease which in terms of size for a biotech is attractive, with 200,000 patients worldwide. As a company, we have the ambition here of developing these drugs through Phase III, bring it to the market and also do the marketing ourselves. So IPF really is a disease where we invest heavily because we believe we can make for us, to completely integrate this company.

From the graph there you see the annual survival rate, 5-year survival rate, where IPF is worse than most of the cancer, so that's a graph of the diseases.

Currently, 2 drugs are approved, the market is split 50-50. Good news for the patient is that there are drugs available. Both drugs slow down the decline of lung function, so patients should live longer. Unfortunately, both drugs come with serious side effects, and patients don't feel an improvement. And 25% of patients on treatment every year will stop due to all of the side effects. So that's really, if you look out there, even with 2 dugs available, 1 in 3 patients currently is not on treatment. So 1 of 3 patients also in the U.S. who are insured, while having access to the drugs, does not take these drugs because he doesn't tolerate -- he or she or she does not tolerate due to the side effects.

So we are progressing 3 different compounds for IPF. So the cause of IPF is fully understood. It's a kind of a black box disease. But what we know that is the start of the moment and fibrotic process locally takes over and accelerates the process. That's also why there is no golden bullet yet there. Nobody, I think, understands where you really need to intervene, and so our approach, we try to cover it from a couple of different angles. So 1690 has started Phase III. So 1205 is a GPR84 antagonist, has started Phase II as well, as we speak. And then the new one for today is 3499, I'm not going to disclose the date of it, but going to show you as well some of the preclinical data. I won't disclose the target, but I will show you some of the nice preclinical data and the thinking behind these molecules.

Let's start with 1205. For those of you who know us longer, 1205, it's a low number, so it's been long in the pipeline. We did a Phase II a number of years ago, and you see that failed. And afterwards, we started looking for a second indication. And that took us a while because you really want to be sure if you go for that second disease that you have solid data. For 1205, the disease where it

showed up in the animal models are quite strong and as well in IPF. On the left you see the bleomycin model. Bleomycin, I fully agree, it's a kind of a flex model if you don't know what's happening here. The way internally we run it when we select compounds for the clinic is that we really wait as long as we can until the inflammatory phase is over, and we dose in the second part of the model where the fibrotic processes are taking over. And so in that way, we really want to select molecules that show a strong anti-fibrotic activity. And 1205 has shown that in a consistent way in this model. And as a positive reference, we baked in a bit of IPF. So again, here in gray, if you don't have any bleomycin, the animals stay gray. As soon as you trigger, you get an Ashcroft score. An Ashcroft score is a composite of histopathology scoring. The big challenge is the window here. So experiment by experiment, this will vary quite a bit. But in our hand, and the way we run the model, most compounds never show activity, honestly, and only a few compounds will consistently show activity like 1205 and the reference compounds.

From the right you see a second model where we expose young mice to an X-ray in the lungs, grow them for a couple of weeks, randomize them and then we have an almost pure fibrotic process ongoing, and we'll start the treatment as well. In this model we, as well, can measure lung function parameters. I won't show them today, but they will come out over the coming months. Result in healthy and diseased to positive control, 1205 consistently, on this and other parameters, have shown good activity.

As for 1205, we had all of the preclinical data available. We had safety in patients for 12 weeks. We could immediately step into Phase II study. That study is called the PINTA study. It's larger than the FLORA in terms of that we take 60 patients. This year, the difference with IPF studies that will have patients on nintedanib, pirfenidone, and about 1/3 on what we call local standard of care which affects not any of the directed drugs against IPF.

So FVC will be primary endpoint, but as well here we will include FRI to really take a deep dive into the lung function, and that helps us as well understanding how good our autotaxin inhibitor, in fact, was working. The PINTA study has a bit of a challenge that we don't want to interfere with the 1690 program, so that's where we're exploring it in different countries, so that patients don't need to choose sites, don't need to choose between 1 of our 2 drugs. And that's why we're here exploring the countries where we've never been before, honestly.

Let's now move to 3499. 3499 is again another mechanism of action where we believe we are the first to bring this to patients in the clinic. So with 3499, we play on the stiffness, the contraction of the myofibroblast in the lungs. So what is known and known for a while, but also now we can measure this in both in vitro and in vivo is that if you have a stiff tissue, that makes disease progressing fast. So part of the disease trigger is the disease itself, so you really can have -- if you grow the myofibroblast on a stiff matrix, they'll cover its weakest to a fibrotic tissue. What you see here is, in fact, is a culture of the myofibroblast. And the darker the room, the better the picture. So I'm excusing myself a bit. But here, you have one of the triggers that causes it. And then from a light white circle, you get a more intense white color, which is the visual view of the myofibroblast that does contract. So you can really, in the lab, by just adding the compound, see that your drug is working on that aspect. So there's an external reference there but that is not developed at all. But our internal molecule are really nicely, and that's been showing a dose response from a nanomolar activity onwards, inhibitors contraction of the myofibroblast in the lungs.

So as well, why we said, wow, this is the first time that we really see a physiological effect on the cell type. We took this to the animal models. As well,

there, you see on the right side -- or left side, the bleomycin, strongest way of doing the model is waiting long enough until there is a fibrotic process already. And out of a large family of compound, in fact, 3499 was the only one showing consistent activity. So even the bleomycin is a black box, only very good compounds really will give you a consistent activity. Also, in the radiation model there, 3499 shows consistent good activity on these and a number of other parameters.

So I'm now going to give to Walid to show us the Phase III program of the other.

Walid Abi-Saab, Galapagos NV - Chief Medical Officer [3]

Right, Thank you, Piet, a very exciting pipeline both in the inflammation space as well as fibrosis. So I'm just trying to move forward with the sake of time. So 1690, as you guys know, we've published last year the results of the FLORA study in patients. Again, to remind you, this was a small study, 23 patients, randomized 3:1, placebo to drug. What was striking for us was that the patients on drugs seemed to stabilize and not have any loss and as we see over periods of 12 weeks compared to placebo where it performed, as you would expect, in this patient population in terms of disease progression. What was also important for us is that the adverse event profile of this drug appeared to be quite benign with no difference between drug and placebo in that patient population, which is an important factor. As Piet said before, the current drugs that are approved that are in the market right now are problematic and ask that patients actually elect not to take medication despite the fact that, that is a very bad consequence to the natural progression of their disease.

And in that same trial, when we used a more sensitive way to measure progression of the disease, this is a technique that employs high-resolution CT scan and couples it with low computerized flow modeling. You see -- over time, what you would expect to see is the disease progressing, which is an increase in a specific area of volume and the resulting reduction in the resistance and what we see with the orange bars, with 1690, is complete stabilization of this. And those were statistically significant.

Again, this is a biomarker. This company is using a technology that is developing, so we don't have a robust way of interpreting it. But still, the fact that the FVC data, the FRI data are pointing in the same direction. What we've also seen in the paper is also the results of home spirometry, which is done every day on average, now also show the same picture. We have target engagement with LPA in patients and in healthy subjects as well as some data from the quality of life, the St. George Questionnaire that trend in that direction. All of these made us feel quite confident about the results and quite excited. And perhaps, to the testimony, again, of the importance of these data, the results of the study were published in The Lancet as well, so now 3 Lancets today for you in our presentation.

More recently, we're excited about a development in this space, so let me take a minute to kind of situate things here for you. So this is a cartoon from a paper by [David Letter], who's one of our major collaborators on the 1690 program, where he walks through -- I'm not going to talk about all the other mechanisms of action here, but particularly the one pertaining to us where you have lysophosphatidic choline, which is essentially transformed into lysophosphatidic acid like autotaxin. LPA has been implicated in IPF. There are a number of trials looking at

increase in the L levels as well as in the tissues. And LPA then goes and works through a number of receptors. There is, I think, 1 through 6, if I remember correctly. And LPA1, in particular, is blocked by this compound, by BMS, and it is actually downstream from us. So an autotaxin inhibitor where we have shown that we reduced LPA in plasma, in healthy subjects and in patients, you would expect the results would be a reduction in signaling through the LPA receptors, which is important. So drug that works on the LPA1 and has been tested in this condition will be a good validation of our mechanism of action.

And these are the data from the Phase II study that they ran. This is a study that was randomized 1:1:1, placebo, 600 milligram; and 600-milligram BID. And you can appreciate on the graph a dose-dependent effect, reducing the worsening or the progression of FVC and reaching statistical significance at the 600 BID dose. So what I want to also mention is that this compound has toxic issues, which led to the premature discontinuation of the trial and also the drug. It has effects on the liver and also on the gallbladder. And I would presume that perhaps that precluded them from going to higher doses, which potentially could lead to better efficacy. But to me, seeing a dose-dependent effect here with a drug that works downstream from where we are, is a very good validation independently of what we're doing and makes us feel much more confident about our Phase III program.

And that's our Phase III program. So we've talked about it before. This is -- again, treating this disease, as you know, it's a very serious disease, has prognosis worse than many cancers. The way we do studies in it will be very much like we do studies in cancers. So you cannot tinker with people's standard of care. So you need to go on top of standard of care, this is very loud and clear to us by the FDA. And so we designed 2 identical studies, they will be run -- each one of them will be run in the U.S., in Europe and Latin America and also some of the Asia-Pacific region. Each study will have 750 patients that we call them ISABELA 1 and ISABELA 2. Patients will be randomized 1:1 ratio to placebo and 2 doses of 1690 that you see over here. The 600 milligram was used in the FLORA study. That was the top dose that was used. And 200 based on our PK/PD modeling and looking at LPA engagement is at the bottom end of the curve, where we still have an effect but not as robust as we see on this.

The idea here that we will allow patients to stay on their randomized treatments until the last patient finishes 52 weeks. Primary endpoint is 52 weeks change in FVC. However, the patients will remain on the randomized treatment until the last patient finishes, which means many patients will be treated much longer than this. We're anticipating recruitment duration would be about 24 months, give or take. And as a result, we will have much more data that we will be able to compare to in an, again, randomized controlled manner. And that's very, very important for the agency, especially that we have 2 identical trials, analysis could be pooled and -- prespecified in discussions with health authorities where we pool analysis between the 2 trials to demonstrate effect on the more rare, but clinically significant, event such as deaths, such as hospitalization due to respiratory exacerbation, such as reduction of FVC of more than 10% and look at that. And that was something that, I think, is a bit unique for our program.

The study is actively going right now. We have a number of sites in the U.S. that have been activated, and the first dosing is imminent. I would have wished to tell you this today, but we're still waiting on our first patient to come in. But we're very excited. There's a very good response from the patients, from the investigators, from the patient foundation as well. And we feel that we're well on our way to have a very good program going forward. And we'll be updating, of course, as this progresses.

And with that, I'll turn it over to Bart to conclude today's meeting.

Bart Filius, Galapagos NV - CFO & COO [4]

Thank you, Walid, and thank you, Piet, for those very interesting presentations. I'll make some closing remarks before we get into a Q&A, where I think we have about 20 minutes left for Q&A. I think there might be quite a few on your minds that you want to pose. But in terms of takeaways for today's meeting, I hope that you'll remember a couple of things. First of all, the pipeline that we're proposing here at Galapagos and the depth of that pipeline and the science behind that pipeline that we're very proud of, the platform that has now been proven in -- with multiple targets, in patients ultimately. Also, the news flow that is coming up regarding, especially filgotinib, but also the other compounds that we have in our pipeline is strong. And then, finally, we're also moving towards commercial stage, as we speak, we are couple of years away from launching in Europe filgotinib. We're going to do that together with our partner, Gilead. And we're going to build out our own commercial infrastructure also in those countries as the next step for Galapagos in terms of its growth as it brings us to our motto then, think big, and that's what we're after with Galapagos.

So with those remarks, I'd like to conclude. And I'll hand the floor to Elizabeth for a Q&A, and we'll take those questions between the 3 of us depending on the topics.

Elizabeth Goodwin, Galapagos NV - VP of IR & Corporate Communications [5]

Thank you very much, Bart. Operator, this is the moment you inform the callers again about how they can pose a question.

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Questions and Answers

Elizabeth Goodwin, Galapagos NV - VP of IR & Corporate Communications [1]

(Operator Instructions) We will now take the first question from Peter Welford from Jefferies.

Peter James Welford, Jefferies LLC, Research Division - Senior Equity Analyst [2]

Got a couple, I think, for a broader range of different people. Firstly, just with regards to the allocation of resources, obviously, a significant number of people were previously allocated to the cystic fibrosis programs internally. I'm just

wondering where those people and resources are now being allocated to and whether or not this could result in net savings, if you like, to the budget in 2019 or whether or not actually you're looking out to invest the potential savings for that in, if you like, in other projects and, therefore, actually capitalize on the savings that you expect? Secondly then, just a financial one, I guess, Bart, just with regard to the remaining upfront money from AbbVie. Should we see that now as accelerated recognition in the fourth quarter, given the collaboration is ended? And then, finally, just with regards to a comment that I think I caught at the very start, am I right in understanding that the tiered royalty on cystic fibrosis there is depending on the number of components and the identity of those components not as typically it seemed based on the level of commercial sales that are achieved?

Bart Filius, Galapagos NV - CFO & COO [3]

Peter, I'll briefly also repeat the questions in case some of the audience here, also in New York, had not fully understood. But I'll take those 3 questions. First one was around allocation of resources, whether taking people outside of the CF program would result in savings. Peter, we are -- as a company, we are growing rapidly. And over the last year, we have increased our staff from about 600 people to about 700 people throughout the various sites that we have in Europe. We have, indeed, a group of people that were active in CF, but we're going to be reallocating them to our other programs that we're starting. The number of programs that are in Phase II and in Phase III is so strong that we have very quickly a place for this group of people. So do not expect major savings out of the stopping of this CF program at the short term. Clearly, stopping CF in the medium and long term takes away also the expense that would have been associated with developing CF forwards. But for the short term, in 2018, we think the big impact is the receipts of the upfront payment of $45 million. That brings me then to your second question, how do we deal with that upfront. That is, indeed, expected to be received in cash in the fourth quarter. In terms of recognition of this upfront, that's probably going to be largely in the fourth quarter, but there might be small overflow into Q1 because there are some transition activities still ongoing over the next couple of months, and we'll need to recognize both the remaining milestones that were still in our balance sheets as well as this particular upfront over the period of future involvement. But this is definitely something which is short-term both in terms of accounting revenue and in cash as well. And then your last question was around the structure of the royalties. What I can say there is that these royalties are dependent on 2 variables. On one hand, sales levels, as was the case in the original agreements as well as on the number of components from -- that are currently developed, that have been developed by the collaboration previously into a future triple combination that AbbVie would put into the markets. And that ranges from 0 components to 2 components of the triple. And so royalties would, obviously, be lower when there's 0 components existing being used in any future triple combination. And they would go up as more components of -- that have currently been developed will be part of that combination.

Peter James Welford, Jefferies LLC, Research Division - Senior Equity Analyst [4]

Sorry. If I could do a quick follow-up on the 2737. Is the fact that you have carved that out as a potential drug that you could develop yourself outside of CF, given the results we've seen from the FALCON study, where, clearly, that C2 corrector seems to have minimal, if any, effect, does this -- is it a fact you think that, that drug has a potential, other mechanism or other activity that is perhaps was misunderstood, if you like, in the first place? And after the review for this drug, is there another indication? Or should we just read that some other way, given that, that drug has been significantly carved out, and that seems to be the component that failed, if you like?

Bart Filius, Galapagos NV - CFO & COO [5]

Yes, let me just make sure that I've understood the question correctly but -- because the sound is not always great, Peter, but I apologize. So 2737 is, indeed -- there is a carve-out option outside of CF for Galapagos for 2737. Today, we're not ready to talk about the details of where we think this component -- this compound can actually be effective, but we have some scientific ideas that we want to explore with 2737 outside CF. And in the case we would develop this further, some royalties will be due also from us to AbbVie.

Elizabeth Goodwin, Galapagos NV - VP of IR & Corporate Communications [6]

Yes, we can take a question here from the room now. Edwin Zhang?

Xiaodong Zhang, Stifel, Nicolaus & Company, Incorporated, Research Division - Associate [7]

Yes, Edwin Zhang from Stifel. Congrats on the very exciting quarter and all the progress to deliver an outstanding data on FINCH 2. So people are paying more attention to the time line of the potential registration, so your overall plan on this, are we still expecting for here, in the next year, a filing for RA? We know there's also a MANTA 50 study ongoing. Are there any updates on that trial? So my second question is on IPF. So what should we consider for a successful trial in term of the primary endpoints, FVC, in terms of trial design? Is it a non-inferiority design?

Walid Abi-Saab, Galapagos NV - Chief Medical Officer [8]

So I'll take both questions. So let's start with the filing and with RA. I think Gilead, later today, will have their own Q3 results. And I think they might give more color to this, but I think they have already shared some information on this from the perspective that, as you could imagine, the -- in order to be able to file,

you need a certain safety database for each dose. I don't know if you guys are aware of this, but this is a chronic disease you're treating with also an immunosuppressant agent, drugs that work very well, but still they're not trivial drugs. So the agency indicates that they want longer-term data for each dose that you plan to file on. So it's not good enough to just finish the trial, we need to accumulate those data. We need the FINCH 1 and 3 to complete and also have the full data set to enable us to have a strong risk-benefit assessment for each one. In addition, and that applies only for the U.S., not for Europe or Japan, the reading from the FDA of some of the histologic findings that have -- in some of the top studies was that we would need to do a clinical study in humans, a reproductive safety study, the MANTA study. And that the data from those studies will be needed -- from that study will be needed to be included in the overall package in order to support the adequate sustenance. So at this point, that's what we can say about it. As to the progress of this, look, the RA program went faster than anticipated, that puts a little bit more pressure on MANTA, I think. But Gilead is very much engaged in moving this forward. And so far we've been very impressed with the way they operationally have been able to execute on these studies. So that's as much as I can say about it. But I think it's better to ask them also later on today in there results. Regarding the IPF, this is on top of standard of care, so there's no -- it's not another inferiority study, right? So what we expect is that we're going to have a number of patients reflecting the U.S. population and, to some degree, also the European population where we have about 1/3 who are on nintedanib, a 1/3 who are on pirfenidone and 1/3 who are on neither of them. And the primary endpoint is to show a reduction in the FVC over time that is superior to placebo, reflecting a slower rate of decline with our drug. What we're expecting also, the aspirational is that we stabilize the disease if the data from our 12-week trial continues to hold for the remaining. The other important piece, which I alluded to, that's very important to us, it's not the primary endpoint but that would be an extremely important endpoint is if we can demonstrate differences versus placebo on mortality, on hospitalization due to that.

Elizabeth Goodwin, Galapagos NV - VP of IR & Corporate Communications [9]

Operator, do we have a question on the line?

Operator [10]

Yes, certainly. We will now take our next question from James Quigley from JPMorgan.

James Patrick Quigley, JP Morgan Chase & Co, Research Division - Analyst [11]

A couple from me. So on Toledo, it looks like it's -- the mechanism of action is it's blocking then the sick cells getting through to the epithelial barrier. Have you done -- or any analysis in the mice as to whether T cells can still get into the brain? And I'm sort of thinking along the lines here around Tysabri and PML list,

that's number one. Number two is in IPF. I'll be interested to know how 1690 stacked up in the bleomycin model and the radiation model versus nintedanib as the 3499 and 1205 look fairly similar. But obviously, we've seen the data from FLORA, which say 1690 could deliver you a disease effect. So just wondering how that's shaped up there. And then, finally, with filgotinib in FINCH 2, I may have missed this, but have you shown any data on the impact on pain alone? I'm just thinking more that GSK are positioning MOR103 as potentially having a strong impact on pain. I just wondered how filgotinib -- or how patients responded on pain with filgotinib specifically?

Walid Abi-Saab, Galapagos NV - Chief Medical Officer [12]

Who's going to start? So this is Walid. I'll start with your last questions on FINCH 2 and pain. So all the additional analyses have not been fully communicated, and there's a limit to what we can do. But I can -- and I realize -- I was just at ACR, and the focus on pain is large. The data that we have on pain are also very good. I just don't have the exact numbers that I can share with you, but those will be communicated as more data will be released on FINCH 2.

Piet Wigerinck, Galapagos NV - Chief Scientific Officer [13]

First question, if I understood the question well, is there any risk that the compound penetration to the brain as a consequence might have an increased risk for brain as a serious side effect. So that's -- we've gone through the talks, and that's not a lot or very less that we clear, honestly, with the chemistry we typically do -- we hardly see any brain -- any drugs that penetrate well into the brain. And that they are same up to now for what we've seen with this class of drugs, so that is not any of our concerns at the moment. And the second question?

Walid Abi-Saab, Galapagos NV - Chief Medical Officer [14]

The 1690 and BLM results. So I -- we haven't shown the data because, again, we've shared it before, but the data with 1690 and BLM are equally robust to the ones I've seen. I don't know if you can add more color to it, Onno?

Piet Wigerinck, Galapagos NV - Chief Scientific Officer [15]

Well, for 1690, we've performed the various forms of the BLM, the prophylactic, the therapeutic, both ways we did it. 1690 performed very well. We also did the radiation model, if that was a question also there, it performs across all the different models.

Elizabeth Goodwin, Galapagos NV - VP of IR & Corporate Communications [16]

Just want to add that we do have all of our preclinical work that we've published at conferences on 1690 on our website, clinical section, R&D. Okay, now we've got some questions in the room. Thanks, Phil -- thanks, James. Okay, we've got a question from Phil Nadeau.

Philip M. Nadeau, Cowen and Company, LLC, Research Division - MD and Senior Research Analyst [17]

All right. Phil Nadeau from Cowen. Two questions from me. First on IPF, you mentioned in your prepared remarks that you could look at combination regimens. Can you give us some sense when those combination trials could start and which of the candidates are likely to be used in them? And then second on CF, I'm just curious to hear a little bit more about your thinking as to outlicensing that program to AbbVie. Clearly, you don't need the cash, and it seems like a program you are pretty passionate about before, so I'm just wondering why you let them take control.

Bart Filius, Galapagos NV - CFO & COO [18]

Let me start with your last question, Phil, on CF. And the question was why it was licensed back to AbbVie. Essentially, and I think Onno alluded to that in his introductory remarks, we announced in June that we felt that the collaboration was not productive so we needed to find an approach that would either bring the molecules at AbbVie or at us. And we felt that, strategically for us, given everything that we have ongoing in all those other programs, in IPF, in -- with filgotinib, with OA, that we are -- that we have enough on our plates to work on. We think that the program will be in good hands at AbbVie. As Onno said, we are a company that's focusing on novel modes of action, first-in-class, and AbbVie will probably have an approach, which is more geared towards a best-in-class approach. And that's what they will be doing with the CF program going forward. So we felt that all in all, the package of the situation at Galapagos, the economics of this transaction was very compelling to sign up the agreement as we did yesterday, and we're very pleased with that outcome.

Piet Wigerinck, Galapagos NV - Chief Scientific Officer [19]

On IPF. I don't know it is the right question. I think you alluded to how and when do you plan to combine the different components we have in the pipeline. So combination of the drug components in this were our big ambitions. We have been looking hard and, in fact, the dynamic range in the animal model is too

limited, so that's not going to give us the answer, and that's not going to probably give us a push to accelerate that. So the plan there currently is, first, proof for each of them as a monotherapy and then, over time, and that will then probably depend on how far we are with 1690, first, as a drug on its own, combine them. But certainly, we don't plan currently for the coming year or 2 any combination of clinical studies where it's -- it's a long-term ambition because this disease really needs much better treatments. And as we don't understand it well, tackling it from different angles and bringing those together is a way forward. That's the case.

Elizabeth Goodwin, Galapagos NV - VP of IR & Corporate Communications [20]

Okay, we'll take another question from the phone, operator.

Operator [21]

Certainly. So the next call we will take is from Wimal Kapadia from Bernstein.

Wimal Kapadia, Sanford C. Bernstein & Co., LLC., Research Division - Research Analyst [22]

Wimal Kapadia from Bernstein. Just a couple on filgotinib, please. So do you believe that FDA will view the retinal vein occlusion as a thrombo event? And then tied to this, can you give us any color on what rate of thrombo events per hundred patient years? Do you think it will be the limit for FDA to consider a blind spot warning? I'm just trying to get a sense of how to think about the headline safety data when FINCH 1 and 3 are announced. Then I guess, a similar question on herpes zoster. Is there a threshold event rate that FDA could consider enough to turn a front page warning into black box warning?

Walid Abi-Saab, Galapagos NV - Chief Medical Officer [23]

Okay. Well, thank you for the question. So whether they will consider a retinal vein occlusion a thrombotic event, I think that is a thrombotic event, whether it's a deep venous thrombosis, it's not, and it's not a retinal embolism. I mean I think it depends how you want to look at it, but I think the -- while the potential consequence of having retinal vein occlusion could be severe with edema of the eye, potentially the loss of sight. It's not going to kill you, unlike, for example, preliminary embolism, which could be a result of deep venous thrombosis. When you look at the data from Barry, they divided things between arterial thrombosis and venous thrombosis. So the agency will look at these sub-qualifications and evaluate this. But even if you look at that, still the rate is very, very low. In terms

of the patient year exposure, I'm not -- I don't have the patient year exposure data from FINCH 2 or the others. What we have is what I showed you is that DARWIN 3 where the rate is 0.1 per hundred patient year currently, and it's trending very low. Regarding zoster, this is a well-known class effect for the JAK. So if you -- if some of you who were at ACR or followed us, you see this -- with all the JAKs, this is a known effect. And again, I think the data -- our data are looking very consistent where we're trending at the lower end of all the other JAKs that are out there. And again, we think it's because of our selectivity for JAK1.

Wimal Kapadia, Sanford C. Bernstein & Co., LLC., Research Division - Research Analyst [24]

Great. That's really helpful. Can I just follow up? I guess it's more on the lines of is there -- do you think there's a threshold for FDA to start to consider then starting to allocate these warnings on per hundred patient years?

Walid Abi-Saab, Galapagos NV - Chief Medical Officer [25]

I'm not aware of a specific threshold, to be honest. I -- yes, we -- I'm not aware of it. I haven't seen any communication from the FDA on that.

Elizabeth Goodwin, Galapagos NV - VP of IR & Corporate Communications [26]

All right, thank you. And I'm afraid that, that's going to be the end of our session today. I -- if you have a question, a burning question you'd like to pose, you can mail me at elizabeth.goodwin@glpg.com or ir@glpg.com, and we'll try to get your questions answered.

Thank you all, all the people who participated on the phone and the folks that came here to the Yale Club today. Our next planned financial results call is on February 22, 2019, with publication of the results the night before. I imagine we'll speak with many of you before then. So thank you again, and goodbye.