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Q2 2021 Galapagos NV Earnings Call

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PRESENTATION

Operator

Good day, and thank you for standing by. Welcome to the webcast for the H1 results of Galapagos. (Operator Instructions)

I would now like to hand the conference over to Sofie. Please go ahead.

Sofie Van Gijsel ***Galapagos NV - Senior Director IR***

Thank you, and welcome all to the audio webcast of Galapagos' H1 2021 results. I'm Sofie Van Gijsel, Investor Relations, representing the reporting team at Galapagos. This recorded webcast is accessible via the Galapagos' website homepage and will be available for download and replay later on today.

We would like to remind everyone that we will be making forward-looking statements 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.

Today's speakers will be Onno van de Stolpe, CEO; Dr. Walid Abi-Saab, CMO; and Bart Filius, COO and President. Onno will reflect on the operational highlights, and Walid will speak through the top line results of our TYK2 and SIK 2/3 programs released in July. Then Bart will go over the financial results and end with expected news flow for the year.

You will see a presentation on screen. We estimate that the prepared remarks will take about 20 minutes. Then we'll open it up to Q&A with Onno, Walid and Bart, joined by the rest of our management board.

And with that, I'll now turn over to Onno.

Onno van de Stolpe ***Galapagos NV - Co-Founder, Chairman of the Management Board, MD & CEO***

Thank you, Sofie, and thank you all for joining this webcast in the midst of the summer. I would like to start with the year-to-date in review.

If we look at Q1, then clearly we had a big disappointment when our ISABELA trial was stopped because of tox signals and lack of efficacy of our ziritaxestat drug, that didn't meet up to expectations in the IPF trial. A big blow for the company, the risk of new mode of actions clearly illustrated here.

On the positive side, we had a good data on the MANTA - MANTA-RAy study, the testicular tox study that we're doing for filgotinib, where we had an interim analysis after 13 weeks. So that was a positive point. In Q2, Gilead decided to extend the lockup. We had requested that to answer some requests by shareholders, and Gilead actually extended the lockup on the shares that they have in the company up to 2024. So that is a nice certainty about the -- regarding the commitment of Gilead for the collaboration and for Galapagos.

Filgotinib was filed for ulcerative colitis in Japan in the second quarter. For your information, in Europe that was already filed by the end of 2020, and this was a big step forward for us in Japan. In the third quarter, we had clinical readouts, first from the TYK2 in psoriasis, which was the Phase Ib study, where we saw very nice data, which made us decide to move forward with our TYK2 in a Phase II study and that will be discussed later on.

And we also saw a very promising biological activity of our Toledo program, our SIK 2/3 inhibitor, both in psoriasis as well as in ulcerative colitis. Also, that will be extenuously discussed by Walid in today's presentation.

As a consequence of the setbacks that we have seen with filgo, the refusal together approved in the U.S., as well as the clinical setbacks in '1972 and ziritaxestat, we decided to do a strategic review, and we're implementing that review outcome as we speak. If you look at R&D, we have refocused the pipeline. My next slide will discuss that. In BD, we have said that we are looking for a substantial opportunity to strengthen our pipeline late stage, and we are in the full process at the moment to see if we can find a product or a company that would fit what we're looking for and hope to execute on it when that becomes clear.

Commercial, we are rolling out Jyseleca throughout Europe as a consequence of the renegotiations around the filgotinib deal. We obtained all rights in -- for Jyseleca in Europe. We've taken over a number of employees from Gilead in the process, and Jyseleca is now rolled out throughout most of Europe, and that will be discussed as well by Bart later in this presentation.

And then we also said that we would save on the financials, on the expenses as a consequence of the setback. That is now implemented, and that will show in the savings going forward in the second half and in the years to come, also to be discussed by Bart.

If we look at the pipeline, then clearly, it's less mature than we had until the late-stage failures, but clearly, still a very excited and differentiated pipeline focused on inflammation and fibrosis. A third therapeutic area, kidney disease, we have added in the first program. In that disease is now in a Phase II study. If you look at the pipeline, it's heavily focused on the Toledo program, the SIK 2/3 as well as the SIK 3 inhibition programs. There are other programs moving forward in discovery towards the clinic. So we hope to see a number of additional molecules reaching the patients in the years to come.

We also, as we said earlier, have a very exciting TYK 2 program that will go into psoriasis Phase II as well as in a UC trial Phase II in 2022. Then, I'm not going to discuss the other programs in detail. But clearly, we have still a lot of shots at goal here in these disease areas. These are all exciting new mode of action programs that we're moving forward with the risks associated with it, but we believe that we now have a balanced risk profile in our portfolio in diseases where we think we can make the best difference, and we're very excited with what we have now in development. Add to that what we have in research, I think that we have started to rebuild the company after the setbacks in the late stage programs, and we look forward with confidence.

With that, I would like to hand it over to Bart Filius, our President. First we go -- we go to Walid.

Walid Abi-Saab ***Galapagos NV - Chief Medical Officer & Member of Management Board***

Good morning, good afternoon, everybody. Thank you. I hope you can hear me well. So it's my pleasure to walk you through some excerpts of our data on the TYK2 and the salt-inducible kinase inhibitors. Just to put it in context, I will not be doing a full presentation of each of the 4 studies because otherwise it will take a lot of the time today. I refer you back to our PRs which include information on the safety dropout rates and so on and so forth, because I'm going to concentrate on specific elements on -- in some of these studies.

So if you can have the next slide, please. So on the TYK2, we're quite excited about this class. As you can imagine, it's also very hot in the field of inflammation right now. TYK2 signals through the pSTAT TYK2 pathway, and it affects a number of mediators like interferon,

IL-12 and IL-23. As a result, blocking TYK2 or inhibiting TYK2 has a number of potential in several autoimmune indications such as psoriasis, such as inflammatory bowel disease, lupus and other types of interferon diseases. So we're quite excited about this target, also based on the fact that it appears that this target also is -- has a promising safety profile. Our own '3667 is a proprietary selective TYK2 inhibitor, which, as you know, we've advanced to the end of the Phase I program.

If you can go to the next slide. Here, we report on data from our signal detection study. It's a 4-week trial where we randomized 31 patients in a 1:1:1 ratio. 2 doses of '3667, low, high dose as well as on placebo. What we found in this trial that '3667 was generally safe and well tolerated. We clearly saw a positive signal, as evidenced by the changes in the PASI score. We had 4 out of 10 patients randomized to be high-dose experiencing a PASI 50 or above response compared to 1 on placebo. But not only that, when you look at the additional endpoints, body surface areas and so on and so forth, that we look at in this indication, we clearly see consistent results across the board.

What we also know is based on data from competitors as well as the shape of the curve and our own trial, the plateau is nowhere near -- or the efficacy that we see at 4 weeks is nowhere near the plateau.

Now, we had previously planned based on an increase in the safety margin based on longer-term tox studies that were completed sometime early last year -- or early this year, I should say, in 2021, that we wanted to explore higher doses. And as a matter of fact, that study is ongoing right now. And this will enable us to evaluate the full dose range in the upcoming dose range finding study in psoriasis as well as in a Phase II study in ulcerative colitis, as Onno mentioned earlier, in 2022.

On the next slide, I'm trying to show here the essentially disposition of subjects based on the change of baseline and what they achieved on the PASI score. So this is at week 4. You can see at the x-axis, the PASI score, and on the Y axis, the proportion of subjects who achieve it. So you can imagine on the high-dose where we have 10 subjects, each uptick represents one patient having a response. And the dark green is the low dose of 3667, where we had 11 patients. So each increment is about 9%, so to speak. And you can see very clearly that we achieve -- so one patient clearly has a PASI greater than 75. We have another one that had a PASI of 74. We see them barely below the cut off of 75. And then if you do the cutoff at 50, you see that 4 people are on active, high-dose, and one on placebo, achieving that goal.

So overall, we're quite pleased in this -- with these results. Clearly, a signal was detected. And the overall picture supports further development of '3667 in psoriasis, but also in other inflammatory indication, ulcerative colitis, and potentially other areas that we're evaluating as well.

The next slide, I will talk a bit about our salt-inducible kinase inhibitors. We're very excited. This is a potential novel mechanism of action in inflammation. Salt-inducible kinase is signalled through 2 different pathways, as is depicted on this cartoon here. They affect both pro-inflammatory mediators as well as regulatory mediators. And that was the reason why we were very excited that, with our approach with inhibition of these particular kinases, we can both affect a reduction in the pro-inflammatory cytokines, such as TNF-alpha, but at the same time, an increase in some of the regulatory -- sorry, mediators such as IL-10. And that's a very promising profile that could potentially have a role in inflammation, both by increasing efficacy, but also reducing the long-term consequence of chronic immunosuppression that we observed with some of the treatment of these indications.

Galapagos has played a critical role and pioneering role actually in elucidating what the salt-inducible kinases can do in inflammation. We have a number of compounds with multiple level of selectivity for the type 2, type 3 and 2/3 together, as well as different level of potency, and we believe that we have potential for broad applications across the board here.

On the next slide, I'm summarizing a bit our experience with our '3970, which is the molecule that we took forward as a SIK 2/3 inhibitor in 3 signal detection studies that were 6 week in duration. Overall, we found '3970 to be generally safe and well tolerated, which bodes well for the platform in general. Now to be perfectly clear, '3970 was not the ideal candidate. We knew that by virtue of some dose-limiting toxicity, the margin that we were able to explore in the clinic was not that high. Nonetheless, we felt that it was very important for us to start generating some data that will help inform us and guide the future development of this whole platform, which, as you know, we've been investing heavily in.

So in the psoriasis study, the CALOSOMA study, which was 6 week in duration, we clearly saw evidence of clinical activity as seen by 4 out of the 13 patients achieving the PASI 50 versus none on placebo. These observations were very solidly observed across a number of secondary endpoints, which we've examined in the trial.

In the case of the SEA TURTLE study in ulcerative colitis, while we have not seen any evidence of efficacy with -- on the Mayo score, and I'll show you that in subsequent slides, when we look at objective endpoints, specifically, when you look at endoscopy and histology, and I'll talk about it later in more details, we do clearly see signs of biologic activity that are very exciting for us and definitely worthwhile further developing when we look at subsequent molecule.

In the case of rheumatoid arthritis, pre-clinically, this was actually the toughest indication for us. We knew that we needed much higher exposures to achieve efficacy based on the animal models. And in fact, actually, that's what translated in the clinic where we did not see any signal in RA, and I will not talk about it anymore today.

On the next slide, I'm showing the similar graph as I did for '3667, where you see the proportion of subjects at the end of the trial, week 6. In this case, I remind you, the -- we had a 2:2:1 randomization between drug and placebo, so 15 on drug and 11 on placebo. We had a couple of dropouts due to COVID or other reasons between drug and placebo. In the end, we end up with 13 on active and 10 on placebo. So you can imagine one patient will be about 8% on active compared to 10% on placebo. And you can clearly see how '3970 separates from placebo in this trial.

On the next slide, this is the primary endpoint for the UC trial. This is a trial, just to remind you, where we had 2:2:1 randomization, about 20 people on active versus 10 on placebo in this study, 6-week duration, which is relatively short for ulcerative colitis, but that was the tox coverage that we had at the time. When you look at the change from baseline, actually, they are relatively large on the Mayo score, and that tells you that we had a large placebo response in this patient population, which, by the way, were all biologic naive and JAK naive. And as such, we would not see any change between the 2.

However, when we -- if you go to the next slide, when we look at the objective endpoints, specifically, when you look at on the left-hand side graph, when we look at the definition of endoscopic improvement, which is a score of 0 or 1 on the endoscopic response, subscore of the Mayo, in the old days, this used to be called mucosal healing. You see that we have one out of 9 patients or about 11% of those who underwent endoscopy at the end of the trial versus 7 out of 18, which is close to 40% on active. So we think this is a clear signal. Again, it should be taken into the context of the fact that we did not see on the primary endpoint. But nonetheless, in small signal detection studies, we have to follow each signal.

And then you look -- which I'm showing on the right-hand side graph of those patients who had the endoscopic improvement, and try to look at their histology score. These are -- the numbers here -- the scores are the robust histology score. And you can see very clearly that those who are on drug have, for the majority, the largest drug. As a matter of fact, those who are at the bottom, the 4 or 5 patients who have the lowest score on the histology are the ones who actually virtually normalize their fecal calprotectin, which is an inflammation market for -- marker for ulcerative colitis. So all in all, when we look at the data from the ulcerative colitis, we are encouraged with these positive signs despite the fact that we had a large placebo response in this biologic naive population. But I think this bodes well for the platform as a whole.

So in my last slide, I think I'm -- I would like to summarize that we are quite pleased with the biologic activity we've seen with our salt-inducible kinase inhibitors in these short signal detection studies. It's not given that when you work on a novel mechanism of action, you will be able to translate that to efficacy in the clinic, and it was very clear that we've seen that evidence of clinical activity in psoriasis, which is an important inflammatory indication. We've also seen important signs of biologic activity. I hope you agree with us that there is something there in ulcerative colitis. We clearly know that '3970 doesn't have the necessary margin that will allow us to fully evaluate the inhibition of SIK2/3 in the clinic, and then we want to go back and work with the compounds that we have now in late stage discovery to come up with molecules with higher target engagement. And therefore, we conclude that we are very excited with the data that we have seen, that these data support further development of salt-inducible kinase portfolio and point to that potential inflammatory indication, and our goal is to bring one of our more potent and better pharmacologic profile SIK 2/3 inhibitor to the clinic as in healthy volunteer

studies in 2022.

And with that, I'll turn that over to Bart Filius. Thank you.

Bart Filius ***Galapagos NV - President, COO & Member of Management Board***

Thanks, Walid, and good afternoon, everyone. Good morning for everyone on the U.S. time zones. Let me conclude the presentation. I think I'll take about 5 to 10 minutes more, tackling 2 other topics, one being the commercial progress that we've made over the last quarter and then the financials for the quarter as well.

So first of all, we've made significant progress in the second quarter in terms of reimbursements, in line with expectations, but clearly, big steps forward. As you know, in Europe, it's not a one shop. You need to basically go from country to country to achieve reimbursement, which is again critical for being able to launch successfully. At the end of last quarter, we had Germany and the Netherlands in the markets, and during the second quarter, we've been able to add France and the U.K. as well as also reimbursement in whole range of smaller countries, Belgium, Luxembourg and Scandinavian countries: Austria and Ireland. So in total, we're now in 11 countries reimbursed in Europe with generally reimbursement per label, the exception there being France, where reimbursement is female only waiting the review of the MANTA data by the authorities, and then hopefully being able to implement also expansion of the reimbursement label in France thereafter. And very positively, in the U.K., we are the first advanced therapy that is recommended by NICE for the moderate and severe RA population. And that is obviously a big support to our launch there. Both of those countries have launched in the last month of the quarter. So the numbers at stake are still very, very small, but we're happy to see the progress there in terms of achieving market access. And still to come for the rest of the year is reimbursement in Spain and Italy. So by the end of the year, in all of the big European countries, we should have the product on the markets.

Just as a reminder, we're not booking sales yet in Germany and the U.K. in the first and the second quarter of this year. That's still booked by Gilead. But as of Q3, we will start to see German sales numbers in our own books. We've started supplying ourselves in July, and the U.K., and many of the smaller countries will follow by the end of 2021 so that as of 2022, all of Europe will be supplied by Galapagos and sales will be booked by Galapagos as well. So fully on track on reimbursement there.

Then a quick peak at market performance. Overall signal is that we are in line with our own expectations with regard to market penetration. It's still very, very early days. The only market where we are now a little bit longer active is obviously Germany, but maybe also the left is worthwhile to highlight how the JAK class as a whole is doing very well in Europe. And over the years, you see now that the class market share in advanced therapies has increased to 16%, here reflected in the green box in the bar chart, and anti-TNFs slowly declining, but other biologics as well. So the JAK class as a whole is doing quite well. On the right, perspective on how Jyseleca is doing in the German market, and this is an overview of the dynamic market. So those are all patients that are either switching from other advanced therapies, or are yet to be started on an advanced therapy, be it a biologic or a JAK. So this includes also, for example, patients starting on other biologics on TNF, for example.

And we see our market share in the dynamic market progressing rapidly to now north of 4%. So we're pleased with the results in Germany, and we're pleased with the results in Europe as a whole with regard to our overall class market share. And Michele Manto, our Chief Commercial Officer, is available for the Q&A to give further detail on the numbers as well.

Then let me switch over to the financials. First, a view on cash. Our cash balance at the end of the second quarter is a bit north of EUR 5 billion, and that reflected a cash burn of EUR 223 million over the first half of the year. As usual, we exclude a couple of extraordinary items from our cash burn. In this case, in the 6 months to date, these were positive influences, warrant exercises, the disposal of our fee-for-service business, Fidelta for EUR 29 million of cash impacts, and then some favorable currency translation effects as well, about EUR 30 million. Overall, that leads us to a very healthy cash position of EUR 5 billion at the end of the first half of 2021.

Then a bit on the P&L. There is definitely a much more detail available in our H1 report, and I invite everyone to take a look there on our website. Our revenues are EUR 277 million for the first half, mainly driven by revenue recognition elements for both filgotinib and the platform, resulting from our transactions with Gilead. And as of the end of June 2021, there is still approximately EUR 2.6 billion of deferred revenue available in our balance sheet that will be recognized over the months and years to come.

Operating costs slightly increasing on one hand. Filgotinib -- we've taken over a bigger chunk of the cost from Gilead with our transaction in December of last year. Toledo was higher relative to the comparative quarters in 2020 with the 5 clinical studies that we have been running in the first 6 months. And S,G&A is up a bit because of our launch efforts in the various markets in Europe. Our net loss is negative EUR 55 million, which includes the effect on currency and the disposal of Fidelta as is reflected on this slide.

A few words maybe on cash burn with a bit of perspective for the future, because it's been a topic that we've been discussing with our investors quite intensely over the last couple of months. And I thought it would be useful to give you some perspective on how our cash burn is built up today as well as where we see this heading. And this is not formal guidance because, obviously, we are not in a position today to look forward that many years in the future, but it is conceptually what our cash burn is looking like.

So let me start off with 2021, this year, which we anticipate, by the way, to be our peak cash burn year. So the numbers of cash burn will go down in future years. And out of that takes 100s, roughly 70% is what we call pure R&D burn, and roughly 30% is what we would call Jyseleca burn. Jyseleca burn in this case being not just the commercial field forces and medical affairs activities in the market, but also the burn for the Phase III programs that we're running for DIVERSITY, the long-term extension studies that we have for our RA programs as well as the MANTA study. And it also includes an allocation of our G&A expense that we are making in the company. But this is the way we look at it ourselves inside the company. Out of the EUR 600 million, which is the midpoint of our guidance for 2021, roughly 30% is connected to Jyseleca and roughly 70% is our R&D burn.

Then going forward. As I announced in the last quarter, we are pushing down our R&D burn expenses. The overall savings program is EUR 150 million. We anticipate to materialize another EUR 75 million as of full year 2022. So we do see R&D burn going down in the next couple of years to a level of, let's say, roughly EUR 350 million, which is obviously everything else being equal. As soon as we do, I don't know, something on BD, or if we have good successes on some of our pipeline programs, this number can fluctuate, but EUR 350 million is sort of your run rate if you look at it today, that we anticipate as of 2022.

Then we've also announced earlier that Jyseleca should obviously reach breakeven. Actually, we've always said that this would breakeven on a contribution margin basis. But we actually think that we can get pretty close to breakeven with a fully loaded cost view. So think about this as you go to 2024 time frame. The net burn for the company would then also go down to the R&D burn, which is roughly EUR 350 million. And then obviously, what we are doing it for is our -- is the outlook later on in the decade and in the years in the next decade, because Jyseleca has a very healthy patent life until 2034. And then in those periods of peak sales years, we can actually get a healthy contribution from Jyseleca to offset our cash burn. So if you think about the cash burn of the company, this is perhaps helpful in looking at how we would be spending our money, again, caveated by all events that could take place between now and these moments becoming reality.

Then, I'll conclude with an outlook for the rest of the year 2021, and then hand it over to -- or hand it back to Sofie for the Q&A. So what we've seen so far in the second half of the year is indeed the outcomes on '3667 and our SIK 2/3 program, and Walid has elaborated on those. Big for us obviously. Coming up is the decision by the CHMP and the European Commission regarding ulcerative colitis, and we're hopeful to get a positive decision there in the second half of the year.

And then trial progress DIVERSITY. We anticipate this to be finally fully recruited in Crohn's disease and also our study with '2737. Our kidney program should be fully recruited by the end of the year.

So with that, I conclude on the presentation and hand it back to Sofie and the operator for the Q&A. Thank you.

Sofie Van Gijsel ***Galapagos NV - Senior Director IR***

Thank you very much, Bart. That concludes the presentation portion of today's webcast. And I would now like to ask the operator to open the lines for Q&A. Thank you, Christina.

QUESTIONS AND ANSWERS

Operator

(Operator Instructions) And your first question comes from the line of Brian Abrahams from RBC Capital Markets.

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

Question on filgotinib, Jyseleca in Europe. I was wondering if you could maybe talk a little bit about the ongoing review there, in ulcerative colitis, the role that the MANTA data are playing there, how much you shared with them and continue to share with them and whether you've had any additional discussion with the regulators in Europe on that data set?

Walid Abi-Saab ***Galapagos NV - Chief Medical Officer & Member of Management Board***

Yes, the review is ongoing, as is the case. We have included the data from the interim analysis that we showed, including all the available data. As you can imagine, when there's an ongoing review, we provide with as much up-to-date information to the reviewing agency as possible. The review is progressing according to plan. And at this point, I have nothing more to add, except that we share the data with them, and we are in discussions with them. And I'm going to leave it at that.

Operator

Your next question comes from the line of Laura Sutcliffe from UBS.

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

Big picture question, please. You talked about sticking with your core expertise in immuno-inflammation and fibrosis after your strategic review. But is it all about large target populations for Galapagos, or would you consider some of the more niche areas within those therapeutic spaces, either with the assets you have, or from a BD perspective?

Onno van de Stolpe ***Galapagos NV - Co-Founder, Chairman of the Management Board, MD & CEO***

Well, let me start, and maybe Walid can add to that. We're clearly interested also in smaller indications and potentially also as first indications for some of our new mode of actions, but we're not necessarily directly -- go for the big ticket where we need very large trials with long duration. But if we can identify a disease where we can adjust the trial to -- a shorter period of time and smaller patient numbers, then clearly, that is of interest, and then potentially expand on it when we get positive data in that first trial. Walid, do you want to add to this?

Walid Abi-Saab ***Galapagos NV - Chief Medical Officer & Member of Management Board***

I think you pretty much covered it, Onno. I think the idea was for us to operate from an area that we know well and in the position of strength and know-how, which is the immunology and fibrosis space that we've been working in. But again, Galapagos always been opportunistic in our quest to look for opportunities. From a BD perspective, we will be expanding and evaluating specific, maybe niche areas or specialized disease areas within that space. But I think Onno covered this point also very well.

Operator

Your next question comes from the line of Dane Leone from Raymond James.

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

I guess, just kind of 1 for me to focus on the majority of investor conversations we've been having majority of this year now. Can you just maybe go into how you're thinking about revamping the process of taking research molecules into more development stage? And obviously, there's been a recent update with your salt-inducible kinase program, TYK2 program. Unfortunately, the investor reaction to that was not positive, evidenced by the stock movement. Is there anything that we should expect in terms of how you select targets to move forward to maybe increase confidence within the investor community, that what we're spending money on into these Phase I/II studies is going to be, one, a better use of time and also a better use of capital. I guess I'd put a point on that. Maybe specifically, when you look at the pipeline that you laid out at the beginning of the call, if you're looking at something like GLPG 555, a JAK1 inhibitor, we get questions of why you would even start a Phase I study in osteoarthritis, given the increased rate of venous thromboembolism with that group. So just -- we get a lot of questions, obviously, across how the selection process is being done. So anything you can help us with there would be appreciated.

Walid Abi-Saab ***Galapagos NV - Chief Medical Officer & Member of Management Board***

Well, look, I think it's a fair question. As you've been communicating for the past few months that we've been taking a long and hard look, critical look at the way we've been doing things. We've also been working with some external experts as well to help us with that. It's premature for us to come up with a finalized theme, but we will be communicating on that. But I can tell you that there are some initial themes that are emerging. I think it's very clear that as we are focusing on novel targets, I think it's going to be important that we spend much more time better understanding the link of these targets to our diseases and invest more time in derisking these going forward. I think one of the things that maybe we've been a bit guilty of is that you get excited about what we've been working on and putting more valence on speed and want to go quickly to the clinic with some of the molecules that we have. The problem is that when you're working with the novel targets, you're already working with a high risk. When you compound that with the fact that maybe you're not spending as much time to much better elucidate the biology and the link to the disease and then later take on molecules that might have a little bit more liability on their own for the molecule itself, now you're compounding your risk and you're decreasing your likelihood of success. So I think we're going to be taking all of these lessons. We're going to take a critical look at the way we do things. I think you will see that we will advance molecules, probably with better pharmacologic profile, better margins than what we've done before. And you'll also see that we will be doing studies that probably are more robust, maybe representing patient population that will be more in line with where we're going to be ending up marketing the drug and probably longer, more robust studies. I think you will see that our risk-taking is going to decrease a bit so that we can afford to continue working on novel therapies, which is truly what we are interested in doing because that's how we can address the patient's unmet need and be able to bring something that is meaningful. I'm sorry, I cannot give you a lot more detail because we're in the midst of it, and we're not ready yet, but I hope I gave you a flavor a bit as to our thinking and the direction we're taking.

Operator

Your next question comes from the line of Rosie Turner from Barclays.

Rosie Turner ***Barclays Bank PLC, Research Division - Research Analyst***

So 2, please. The TYK2 and '3667 going into UC, just wondering what the rationale for that was. Is this going to be pre-clinical studies that give you confidence in that indication? And then does that mean it's going to overlap with the kind of JAK1/TYK2 program you also have running? Will they both be looking at the same indication? And then '4399, I think we've now got confirmation it is SIK3. I'm just wondering if you're able to give us any more detail in terms of what indication is that being looked at in?

Walid Abi-Saab ***Galapagos NV - Chief Medical Officer & Member of Management Board***

So let me start with the last one, '4399. So this is a SIK 3 inhibitor. It's currently being developed in Phase I. We still haven't finished our Phase I program, and we should be able to give some more guidance on this towards the end of the year. Preclinically, I think we've talked previously about this molecule that it seems to work more in rheumatologic type of indication as opposed to IBD, as we saw with the SIK 2/3. But it's a bit premature to go into a bit more details. We will do that once we finish our Phase I with this compound. Regarding the TYK2, I think the data are pre-clinically quite robust, suggesting that the role of TYK2 in ulcerative colitis -- we talked about the role of signaling to IL-23 and how IL-23 could be inhibited by TYK2 inhibition. As you guys know, IL-23s do play a role and have been shown to have effects in ulcerative colitis. You also -- deucravacitinib from BMS is being currently evaluated in the UC study as well. So I think all of the data, pre-clinical and also through the IL-23 angle, suggests that the TYK2 can play a role in ulcerative colitis. As to the JAK1/TYK2 '3121 that you're referring to, this is a molecule that is -- works on both JAK1 and TYK2, but it's an oral molecule that releases locally in the colon. And here, the key question is can you with such a molecule, produce a significant local effect in the colon, particularly in ulcerative colitis without having significant systemic exposures, and as a result, reducing significantly the potential risk of systemically inhibiting JAK1 and TYK2. And as a result, you might have a much more beneficial risk-benefit profile in this case. Of course, that concept of releasing locally in the colon and producing a better efficacy, is one that needs still to be proven in the clinic, but the theory there is very plausible. And I think if the data from Phase I support the release profile that we're looking for, I think the next step will be to do the appropriate Phase II study in ulcerative colitis and look what the risk-benefit of this molecule would be like.

Operator

Your next question comes from the line of Matthew Harrison from Morgan Stanley.

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

This is Charlie Young for Matthew. How do you expect the FDA review of JAK safety to potentially impact filgotinib or impact on the new market from a commercial standpoint of view? And maybe just a quick second question regarding how any progress looking at the assets potentially license to buy? Should we -- is there something that we can potentially expect this year?

Walid Abi-Saab ***Galapagos NV - Chief Medical Officer & Member of Management Board***

Michele, would you like to tackle the first question?

Michele Manto ***Galapagos NV - Chief Commercial Officer & Member of Management Board***

Yes. So I'll take the first part. So what you have seen in Europe also in the past is that the influences will relate to the reaction of local European and national authorities assume that with olumiant which had a very good launch in the past years despite the situation in the U.S. Actually, we are seeing that also in this last month with our launch where actually the debate in the U.S. hasn't even acted the way that local prescribers and authorities have looked at the market.

Bart Filius ***Galapagos NV - President, COO & Member of Management Board***

Let me say then a quick word on BD. Always difficult to say a lot on BD when these processes are still ongoing. So I echo the comments from Onno before that this is clearly a priority for the company to work on BD, which could be licensing, it could be M&A. We're very active on that front. But today, there's nothing to report in terms of any transactions, but clearly, that's a priority for us at the management.

Operator

Your next question comes from the line of Jason Gerberry from Bank of America.

Jason Matthew Gerberry ***BofA Securities, Research Division - MD in US Equity Research***

So just on your TYK2, I was wondering if you could talk a little bit about how you differentiate from a pharmacologic perspective versus the other TYK2s that are a little bit further ahead in terms of TYK2 selectivity as we start to think about the unmet need that you'd be solving for in psoriasis. Just sort of curious if you can frame how you're seeing this molecule as you think about advancing into a Phase IIb dose ranging?

Walid Abi-Saab ***Galapagos NV - Chief Medical Officer & Member of Management Board***

Look, I think in our hands, our '3667 is selective based on our -- all of our assays that we have conducted. In the clinic, we do not see anything that makes us worry about our target activity. It's very difficult to compare to the others because we don't have all the data that they're basing it on. And if there is something that we've learned from the JAK world with our own filgotinib is that, there are many different assays that one can use and in different labs, and unless you do them all in the same way, it's going to be very hard to interpret. And in the end, it doesn't matter because what matters is what are the clinical data that you can attribute to activity or off-target activity. When we look at the data that's available from the most advanced compound, I would think that deucra is the only one. The others are virtually in the same area where we are in terms of how advanced they are in development. With deucra, the only data we have is what they've been publishing on the Phase IIb in psoriasis. The Phase III study, I don't think we've gotten a lot of the details of it. This will become much more transparent once the file has been approved, and the drug has been approved, you can look at the details of it. But one of the things that caught our eye is that, you can see in the Phase IIb study, they've used doses that are higher than what they've used in Phase III. And in Phase III, they had apparently a bit less efficacy than we have seen -- what they've seen in Phase IIb in psoriasis. So could that be because you go from a smaller Phase II b to a larger Phase III, or could it be that the doses that they use in Phase III, which is lower than what you -- in Phase IIb, is the one reason why that has less efficacy. Without knowing all the details and the rationale for why they didn't pick the highest dose to go into Phase III, it's really very difficult to compare, to be honest with you. And at the end of the day, the best way we can do this is to conduct a Phase IIb study that's very similar to the Phase IIb that deucra has conducted, and I think that's going to bring us the closest to being able to being able to see whether we have a competitive profile at the end of that trial. Beyond that, it would be just speculation on our part, to be honest.

Jason Matthew Gerberry ***BofA Securities, Research Division - MD in US Equity Research***

So is it fair to say that you probably don't really know how you stack up until you start to accrue large data sets, I mean, Phase III data sets? And as you kind of proceed, you just have to operate on the offices where you've got good selectivity and you hope that, that differentiates your molecule relative to the more advanced players?

Walid Abi-Saab ***Galapagos NV - Chief Medical Officer & Member of Management Board***

Yes. I mean, that's fair. I think the way you phrase it suggests that we're going blind, and we're going to see what's going to happen. That's not true. I mean I think our pre-clinical data clearly gives us confidence about our selectivity and our clinical data does not raise any concern about lack of that selectivity. So I think we are pleased with what we have seen. We think so far, so good. Again, with the caveat that we've just done a 4-week study. So our confidence in our potential efficacy and safety are limited by the small number. So we're taking the next step, but I think we're taking the next step very confidently based on our pre-clinical data that we have to date -- and our clinical data -- And then we think that at the end of that Phase IIb study, that would be truly the right time to look to see whether we have a competitive profile to deucra, but also to other competitors that advanced equally around that time.

Jason Matthew Gerberry ***BofA Securities, Research Division - MD in US Equity Research***

And, didn't mean to suggest the operating lines to the activity of the drug.

Operator

Your next question comes from the line of Lenny Van Steenhuyse from KBC Securities.

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

As of course, there's a lot of focus on the inflammation pipeline. I was wondering if you could provide us with a brief overview on the IPF portfolio and timeline going forward, specifically on '4716, which was expected to head into Phase IIB. What's the status on that one? Is there still some dose-finding or still some safety studies ongoing with that compound, and when should we expect another clinical study to commence with this compound?

Walid Abi-Saab ***Galapagos NV - Chief Medical Officer & Member of Management Board***

You're right. We spent a lot of time talking about inflammation. So IPF, I think that is a -- there were some big learnings that we got and actually a bit of disappointment, as you can imagine with ziri. What we are doing right now is figuring out how can we de-risk our programs going forward, short of doing a very large Phase II study that's going to cost millions of euros and take a number of years to be able to answer the question, and even there with a lot of uncertainty. As you know, FVC as an endpoint is a bit risky. So to be perfectly honest, we're still gathering actually all the data from ISABELA. We still haven't gotten all the data in. We're trying to better understand whether we can identify certain patient populations that would be maybe rapid progressors or there are certain signatures that we can use to identify and enrich our population going forward. We're also using the time to increase our confidence in the mechanism action of '4716 and chitinase inhibitors in IPF and other fibrotic diseases of the lung. And we are also doing some --necessary drug interaction studies because, again, those were the learnings that we obtained from the ziri program as well as the '1205 program that because of the use of significant concomitant medication before we deal with the program by doing the necessary drug drug interactions. So all of these preparations are happening this year. I'm expecting that in the early part of next year, we will be able to have a better idea about a Phase II study. I'm not sure if it's going to be right off the back going into a Phase IIb study or it's going to be more of a mechanistic Phase II study. This is something that we're still thinking about. And it's part of this rebalancing of how much risk we want to take as we engage, whether we can figure out a way to find biomarkers that increase our confidence and our success before we invest more heavily. But we're committed to the IPF space. This is a space which -- there's huge unmet medical need. And arguably, we have a lot of knowledge that we've accumulated as a result of the ISABELA program. We're going to put all of those to use and -- helpful -- hopefully, be smarter about the next step that we take with '4716.

Operator

Your next question comes from the line of Phil Nadeau from Cowen.

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

2 brief ones for us. First, on Jyseleca. Gilead didn't actually break out the revenue recorded in Europe when reported a couple of weeks ago. I was curious if you could let us know what was recorded in Q2 for Jyseleca revenue in the EU? And then second, can you remind us on '4399, how the potency of its target engagement for SIK3 compares to the target engagement of '3970 for SIK 2/3. Is it significantly more potent or is it more on parity?

Bart Filius ***Galapagos NV - President, COO & Member of Management Board***

And let me quickly answer the question on the revenues. It's correct, Phil. So we will be booking sales as of July, and we will be reporting sales ourselves as of the third quarter. Gilead is not, let's say, detailing those sales levels in their reporting. But once we do the Q3 updates, we'll make sure to give you a full perspective on sales of the compounds also on a year-to-date basis. Then maybe on '4399, I give that to Walid.

Walid Abi-Saab ***Galapagos NV - Chief Medical Officer & Member of Management Board***

So on '4399, I will -- maybe I can ask Piet to comment specifically on the potency, I believe it's in the same ballpark as '3970, but that's not the key point. The key point is whether the -- based on the half-life and the margin -- safety margin, to what degree we can inhibit the SIK 3 enzyme. And that will not be known until we finish Phase I, and we're able to figure out what are the doses that are safe and well tolerated that we can use. But maybe, Piet, you can say a couple of words, if you know about the potency of '4399 versus '3970.

Piet Wigerinck ***Galapagos NV - Chief Scientific Officer & Member of Management Board***

Indeed, as you mentioned, both '3970 and '4399, if you look both at the biochemical or cellular level, they are single-digit nanomolar potent compounds. So the big difference is that with '4399 you will only block the secretion of the pro-inflammatory cytokines. You don't play on the second angle where you help the system to rebalance quicker. So it will only inhibit the secretion of the Pro- inflammatory cytokines. It's a potent molecule from what we've seen in animal studies. It's going to be once a day, and we expect a good coverage of the biomarker in Phase I, but that's an ongoing study. So -- and we will need to wait until the data to report out on those.

Operator

We have time for one more question, and it comes from the line of Wimal Kapadia from Bernstein.

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

Can actually asked about '2737, please, for PKD. So I know the asset was previously investigated cystic fibrosis, met on the primary, didn't show any significant improvement in pulmonary function. And I guess, I'm just curious what gives you the confidence to start the Phase II trial? Just to get a sense of your conviction will be great. Just given some of your earlier comments on risk appetite within drug development.

Walid Abi-Saab ***Galapagos NV - Chief Medical Officer & Member of Management Board***

Yes. So I'll start, and if Piet wants to chime in based on the animal data, but this is a compound that, as you know, we've known for some time, and we were working on it in cystic fibrosis. And through the action on the CFTR channel, we were able to surmise that it's going to work in this indication. Our preclinical data actually were quite solid, supporting this, both on its own and using also tolvaptan as an active control, but also in addition to, and based on those, elected to do the study. Now of course, this is a long disease. The studies are long. There's a huge unmet medical need, and this is our first foray into that space. So there is an element of learning involved there, but I think the pre-clinical data are quite supportive. Piet, do you want to add some more color to the preclinical data?

Piet Wigerinck ***Galapagos NV - Chief Scientific Officer & Member of Management Board***

So '2737 is osmotherapy acts as a CFTR blocker. And so the disease, we are investigating, ADPKD is a disease where processes go wrong in the kidney where then cyst swell. So we expect the compound to block the swelling of those cysts and to complete block one of those channels that make those grow. In that sense, we've seen in the clinic signs of target engagement in -- during the CF program, and we believe that it's going to be sufficient to engage the target as well in the kidney in those ADPKD patients. We've tested it in multiple in vitro systems and in vivo systems. And we've always coming up with an efficacy which is close to the only approved drug currently. And that's where we stand today.

Sofie Van Gijsel ***Galapagos NV - Senior Director IR***

Thank you. So that's all we have time for -- on today's call. Of course, please feel free to reach out to the IR team if you have any further questions, we're happy to help. Our next financial results call will be the Q3 2021 results on November 4. Thank you all for participating today, and have a great day. Bye.

Operator

This concludes today's conference call. Thank you for participating. You may now disconnect. Speakers, please stand by.