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INCY - Q2 2016 Incyte Corp Earnings Call

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OVERVIEW:

Co. reported 2Q16 revenue of $246m, net income of $34m or $0.18 per share basic and diluted.

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Barry Flannelly *Incyte Corporation - General Manager US*

Steven Stein *Incyte Corporation - Chief Medical Officer*

Dave Gryska *Incyte Corporation - CFO*

Reid Huber *Incyte Corporation - Chief Scientific Officer*

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PRESENTATION

Operator

Greetings, and welcome to the Incyte second-quarter 2016 financial results conference call. At this time, all participants are in a listen-only mode. A question-and-answer session will follow the formal presentation.

(Operator Instructions)

As a reminder, this conference is being recorded. It is now my pleasure to introduce your host Mike Booth, Vice President of Investor Relations for Incyte. Please go ahead sir.

Mike Booth - *Incyte Corporation - VP of IR*

Thank you, Kevin. Good morning, and welcome to Incyte's second-quarter 2016 earnings conference call and webcast. The slides used today are available for download on the Investors section at www.incyte.com.

Speaking on today's call will be: Herve Hoppenot, our CEO, who will begin with a quick strategic overview; Barry Flannelly, who leads our US organization, will provide some detail on Jakafi (ruxolitinib) sales during Q2, as well as reviewing the recent clinical data presented at ASCO and EHA; Steven Stein, Incyte's Chief Medical Officer, will give an update on our clinical portfolio, as well as our upcoming news flow for 2016; and Dave Gryska, our CFO, will summarize our second-quarter financial results, as well as the accounting treatment of the recent ARIAD transaction in Europe. We'll then open the call up for Q&A, for which we'll be joined by Reid Huber, our Chief Scientific Officer.

We'd like to remind you that some of the statements made during the call today are forward-looking statements, including statements regarding our expectations for 2016 guidance, the commercialization of our products, and our development plans for the compounds in our pipeline. These forward-looking statements are subject to a number of risks and uncertainties that may cause our actual results to differ materially, including those described in our 10-Q for the quarter ended March 31, 2016 and from time to time in our other SEC documents.

I'd now like to pass the call to Herve for some introductory remarks.

Herve Hoppenot - *Incyte Corporation - President & CEO*

Thank you, Mike. And good morning everyone.

Incyte is in an excellent position as we enter the second half of the year. Barry, Stephen and Dave will each provide greater detail on our financial and clinical progress, but first I would like to spend a minute talking about Incyte from a bigger picture perspective.

We have a plan in place for the growth of our Company and we are executing on that plan quarter by quarter. We are building something important at Incyte and let me summarize our recent progress. Sales of Jakafi continue to grow rapidly, driven by the increased awareness of the benefits of Jakafi in both its MF and PV indication. Our partner Novartis has also seen significant growth of Jakafi in actual sales, resulting in royalties to Incyte that are up almost 50% over the same period from last year. With more than a decade of expected patent protection for ruxolitinib in front of us I believe we are in very good shape with this existing franchise.

Our dynamic top line revenue growth may be further enhanced next year from Baricitinib royalties, should it be approved for the treatment of rheumatoid arthritis. The first regulatory decision for Baricitinib is expected in the first quarter of 2017, and if approved we believe that it will compete well in the rheumatoid arthritis market which has been estimated to be worth a total of $21 billion last year.

The ARIAD transaction concluded in June as planned, and our European team is now fully operational supporting and growing the Iclusig (ponatinib) brand. Furthermore, the team in Europe is already participating in the development of our clinical portfolio, further validating our decision to accelerate the European expansion.

On the clinical side we have achieved several very important milestones in recent months. Firstly, we initiated the first Phase 3 trial in our development program for epacadostat. The ECHO-301 study is now recruiting patients and is evaluating epacadostat plus pembrolizumab as a first-line treatment for patients with advanced or metastatic melanoma.

The second significant milestone was the achievement of Breakthrough Therapy Designation from the FDA for ruxolitinib in acute GVHD. There are currently no approved treatments for this patient population and we are preparing to initiate the pivotal program for ruxolitinib. I'm confident that our decision to pursue JAK inhibition as a treatment for GVHD has the potential to improve the treatment of this often deadly disease and provide further growth opportunity for our JAK franchise.

We have also made progress within our earlier stage portfolio. This morning we announced bladder cancer as a first indication for our FGFR program, and in June we entered a new clinical program with the initiation of the first trial of our anti-GITR agonist. The last molecule drug discovery alliance was timed with Agenus last year has already generated one clinical program and we anticipate initiating the second clinical program targeting OX40 later on this year.

The first half of 2016 has been a success for Incyte, and whether looking at the Company from a top-line perspective, at our portfolio or at our expanded capabilities, it is clear that we are making significant progress on our journey to become a world-class biopharmaceutical organization.

With that overview, I would like to turn the call over to Barry for an update on Jakafi.

Barry Flannelly - *Incyte Corporation - General Manager US*

Thank you Herve, and good morning everyone.

We continue to see a strong growth in Jakafi sales. Net product revenue for the second quarter was $208 million, an increase of 46% over the same period last year, and a 14% increase over the first quarter of 2016. Year to date net Jakafi revenue has been tracking towards the upper end of our previous guidance and given the strength of the clinical data underlying Jakafi and the strong performance year to date, we are therefore increasing Jakafi net product revenue guidance for 2016 from the previous range of $815 million to $830 million, to a new range of $825 million to $835 million.

The commercial success of Jakafi is built on a foundation of excellent clinical data as exemplified on slide 8 by the five-year analysis of overall survival from the COMFORT I study in myelofibrosis. These were presented at ASCO and EHA in June of this year and support the effectiveness of Jakafi as a long-term treatment for the patients with myelofibrosis. Specifically, the data showed global reductions in spleen volume and significantly longer overall survival in patients originally randomized to ruxolitinib compared to those originally randomized to placebo.

The Kaplan-Meier chart on the right of slide 8 is not adjusted for crossover and still shows a greater than 30% reduction in the risk of death for patients who received ruxolitinib versus patients who received placebo. This suggests that these patients may benefit from earlier treatment with Jakafi.

RESPONSE 2 is the second successful Phase 3 trial for Jakafi as a treatment for patients with uncontrolled polycythemia vera. The data on the right side of slide 9 were presented at EHA in June and show that Jakafi was superior to best-available therapy in PV patients without enlarged spleens. Patients on Jakafi had better hematocrit control without the use of phlebotomy, and experienced more relief from their PV related symptoms than those on best available therapy.

I'll finish by saying that Q2 sales have given us excellent momentum and recent clinical data serve to further reinforce the benefit that Jakafi provide the patients with MF and PV.

I'll now pass the call over to Steven for clinical update.

Steven Stein - *Incyte Corporation - Chief Medical Officer*

Thanks, Barry.

There's been a lot of progress in the clinical portfolio since our last call. I will go through the highlights in the next few slides starting with epacadostat. Slide 11 summarizes the key trials of epacadostat in combination with PD-1 and PD-L1 inhibitors. In June we were pleased to announce that the first patient was dosed in the ECHO-301 Phase 3 trial of epacadostat plus pembrolizumab for the first line treatment of patients with advanced or metastatic melanoma.

Updated data from the Phase 1 portion of ECHO-202, the combination of epacadostat plus pembrolizumab in a variety of tumor types has been accepted for presentation at the European Society of Medical Oncology in October of this year. As reminder Phase 1 data from ECHO-202 was first presented at SITC in late 2015 and the ESMO data this year will provide a further analysis of this data set with significantly longer follow up. Recruitment continues across the ECHO program and we are on track to meet our year-end recruitment goal of over 600 patients into the Phase

1 and Phase 2 trials of epacadostat in combination with PD-1 and PD-L1 inhibitors. As we have previously stated, data emerging from these trials will inform our decisions on next steps.

Slide 12 details our current plans for the development of JAK inhibitors for the treatment of Graft-Versus-Host Disease (GVHD). Receiving Breakthrough Therapy Designation from the FDA recognizes the severe nature of this disease, the clear unmet medical need of these patients, and the potential, based on clinical evidence to date for ruxolitinib, to address the urgent needs of patients with this life-threatening condition. We are pleased with ongoing regulatory discussions and are working towards the initiation of the pivotal program for ruxolitinib in Graft-Versus-Host Disease. The program of trials is expected to focus on the acute and chronic steroid-refractory populations.

The first study in the total program is expected to be a single arm trial of ruxolitinib plus corticosteroids for the treatment of steroid-refractory acute Graft-Versus-Host Disease. Additional studies of ruxolitinib, including randomized Phase 3 trials in both the acute and chronic Graft-Versus-Host Disease settings are also in preparation. The proof-of-concept trial of our JAK1 selective inhibitor '39110 in patients with acute Graft-Versus-Host Disease is now fully enrolled and we expect to have initial results from this trial before the end of 2016.

Moving now to slide 13. Today we declared the first indication within our FGFR program. This will be for the treatment of patients with bladder cancer harboring FGFR pathway alterations. The unmet need here is substantial and the trials are expected to be initiated in the next several months. The data generated within our FGFR program to date is summarized in the upper left of slide 13, and has provided us with a go-forward dose in bladder cancer. We look forward to sharing those data with you at an appropriate medical meeting.

The last program I like to highlight is our anti-GITR agonist '1876. We dosed the first patient in the clinical program in June, and the monotherapy dose-escalation trial is currently enrolling in a 3+3 design. This will be followed by dose expansion in several tumor types. The future development of '1876 in expected to focus on combination regimens and the chart on the right side of slide 14 illustrate the compounds potential for synergy with PD-1 inhibitors.

Slide 15 summarizes the whole portfolio which includes 14 compounds in development against 11 different molecular targets. Driven by innovative science in multiple indications, our portfolio includes numerous opportunities both as single agents and in combination. As I've outlined, we are making excellent progress on many fronts and I look forward to sharing our future progress with you.

I will finish on the newsflow slide and there are multiple potential value drivers for Incyte over the next 12 months. As mentioned, we are preparing to initiate the pivotal program for ruxolitinib in Graft-Versus-Host Disease and we will seek to provide you with proof-of-concept data from our JAK1 program in GVHD later this year. We also plan to initiate the 39110 plus osimertinib study in lung cancer. We expect to initiate the '54828 FGFR bladder cancer study later this year, and we will also provide you with initial clinical data from the FGFR and BRD programs at suitable medical conferences.

In immuno-oncology the updated data from the Phase 1 portion of ECHO-202, studying epacadostat plus pembrolizumab has been accepted at ESMO in October of this year, and will be initiating a proof-of-concept for our anti-OX40 agonist antibody '1949 in the second half of 2016.

Outside of oncology, within the next 12 months we should be in a position to present data from the ongoing study of topical ruxolitinib in alopecia areata, and very importantly, we're anticipating global regulatory approval decisions for baricitinib for the treatment of rheumatoid arthritis early in 2017.

With that I will pass the call over to Dave for the financials.

Dave Gryska - *Incyte Corporation - CFO*

Thanks, Stephen and good morning everyone.

Q2 was another strong quarter for Incyte. Jakafi continued to deliver strong growth and we are pleased with the performance of Iclusig in June. We recorded $246 million of the second-quarter revenue, this was comprised of $208 million in Jakafi net product revenue, $4 million in Iclusig

net product revenue, $26 million in Jakavi royalties from Novartis, and $8 million in contract revenue, including a milestone paid to us by Novartis related to ruxolitinib GVHD collaboration agreement.

Jakafi's net product revenue of $208 million represents 46% growth over the same period last year. Based on Jakafi's performance we are increasing our full-year Jakafi net product revenue guidance to a range of $825 million to $835 million. Our gross net adjustments for the second quarter was approximately 11% and we expected the growth and adjustment for the full year to be approximately 12%. Our cost of product revenue for the quarter was $12 million. This includes the cost-of-goods sold for Jakafi and Iclusig, the payment of royalties to Novartis on US Jakafi net sales, and $2 million for the amortization of acquired product rights relating to Iclusig product acquisition.

Our R&D expense for the quarter was $120 million, including $14 million in non-cash stock compensation. Looking at projected R&D expense for the full year we are updating our current guidance to a range of $620 million to $630 million. The reduction in projected R&D expense for the previous guidance is in large part due to slower-than-forecasted headcount growth and lower-than-expected development expense for the JAK clinical programs. In the first half of 2016 we recorded $277 million of R&D expense and therefore we expect to see a significant increase in R&D expense in the second half of the year.

Our SG&A expense for the quarter was $67 million including $8 million in non-cash stock compensation. We are on track to end the full year with our existing guidance range of $285 million to $310 million.

We have added a new expense line to our income statement which relates the change in the fair market value of the contingent consideration for the Iclusig royalty liability. We recorded $2 million in expense for this line item in the second quarter which consists of the month of June.

Turning now to net income and earnings per share for the second quarter we reported $34 million in net income or $0.18 per share basic and diluted. We now expect 2016 net income to be in a range of $30 million to $40 million which is an increase from our previous net income guidance.

Looking at our balance sheet we ended the second quarter with $629 million in cash and cash equivalents. During the second quarter our cash decreased mainly due to the $140 million payment for the acquisition of the ARIAD business unit which closed on June 1. We expect positive cash flow from operations for the remainder of the year and expected to end the year with our $650 million in cash and cash equivalents.

Now turning to the accounting for the ARIAD acquisition. We have approximately $269 million of intangible assets on the balance sheet related to the product rights for Iclusig. The amortization of these product rights is estimated to be approximately $11 million for the next six months and will be included in the cost of product revenues. We will amortize these product rights through 2028.

The goodwill and the balance sheet as of June 30 of approximately $156 million relates to the ARIAD acquisition. We have also recorded $294 million in contingent consideration, which represents the current fair value of the royalties that we expect to pay on future net sales of Iclusig in Europe. Each quarter we will record a change in the fair value of the contingent consideration as an operating expense. We expect the change in fair value of the contingent consideration to be approximately $15 million for the next six months.

To summarize, Q2 was a very strong quarter. Jakafi delivered strong revenue growth, we successfully closed the acquisition of the ARIAD business unit adding Iclusig European revenue to our portfolio, and we expect to generate positive cash flow for the remainder of the year while continuing to make significant advancements in our clinical-development programs.

Operator, that concludes our prepared remarks please give your instructions and open up the call for Q&A. Thank you.

QUESTIONS AND ANSWERS

Operator

We will now be conducting a question-and-answer session.

(Operator Instructions)

Our first question from Salveen Richter, Goldman Sachs.

Salveen Richter - *SunTrust Robinson Humphrey - Analyst*

Thanks for taking my questions.

Just wondering what your updated thoughts may be on presentation from the IDO1 and PD-L1 and PD-1 combinations and what it takes to make go forward decisions? I do know we will see the Merck cohort at ESMO, just any chance we can see data from the other three cohorts in Q4 or is that more of a 2017 event?

And then a second question, with the failure of Bristol studying for first-line lung, how does that impact your thinking on IDO, and any thoughts of adding IDO to I/O-I/O combinations?

Steven Stein - *Incyte Corporation - Chief Medical Officer*

Salveen, it's Steven. Thank you for your questions.

The ECHO program itself as I stated is progressing very well. We will enroll over 600 patients this year alone. The 2015 presentations at SITC and SMR on the Phase 1 ECHO-202 program obviously we now have a lot of additional scan data. This has reinforced our confidence in the activity of epacadostat plus pembrolizumab in melanoma and our decision to initiate that Phase 3 program.

As I also said the whole study will be presented as a poster discussion at ESMO on Monday, October 10, the abstracts for which will go live on September 28. And I will refer you to that to get that update.

Obviously the entire Phase 2 program is a lot larger, consisting of ECHO-202, -203, and -204 which have 12 tumor types. We look at least three aspects, obviously response rate, time to event data, how long people are -- in response in terms of progression free survival, and then the third component is a biomarker data - which is critical. That will all combine to help us make more informed decisions in terms of triggering further Phase 3's.

The first-line data that you asked me to comment, and the reason I brought up the biomarker piece at the end, is I think it speaks to the importance of using all three components to make decisions: response rate, time to event, and biomarker data. Obviously the BMS data set used a lower (PD-L1) cut off than Merck had used in their program and that is something we will look closely at going forward at our own data sets to help us make informed decisions.

As to when data will be available, through the end of this year and early next year we will look at the programs and find appropriate medical meetings to present it at, and that's all I can tell you right now at the moment.

Operator

Our next question comes from Eric Schmidt from Cowen and Company. Please proceed with your question.

Eric Schmidt - *Cowen and Company - Analyst*

Maybe I can ask Salveen's question a little bit differently. I think in the past you have expected to have go no-go decisions on epacadostat plus PD-1 inhibitors by year-end. If you are not able to hit that milestone, or maybe you still are I don't know, but what would you say to the skeptics

who say the inability to make a decision is an indictment of the program and that you are not seeing much in terms of activity or a clear path forward?

Steven Stein - *Incyte Corporation - Chief Medical Officer*

Eric, it's Steven again. Again thank you for your question.

Again, it is the three components that we need to make decisions. I will be repetitive. Response, the time of the end response: progression-free survival, and the biomarker piece. We are gathering across the program all three pieces of information. Again enrollment has been as expected or better with more than 600 patients enrolled by year end.

And then there are two components, the sit down, where we look at the data and make decisions, and then to present at an appropriate medical meeting and to find that meeting. We feel extremely confident in the program and opportunity and it doesn't speak to any lack of confidence at all, it's just finding the appropriate venues. The data is coming in as expected and again through the end of this year and early next year we will be able to share that in appropriate venues.

Operator

Our next question comes from the line of Geoff Meacham from Barclays.

Geoff Meacham - *Barclays Capital - Analyst*

Good morning and thanks for the question.

Not to beat a dead horse here on epacadostat, but when you look at data and other combo studies, is there a specific number of cycles that you are looking for to be the trigger for the next steps? I guess scientifically what is the thought of the onset of action for what you have seen so far when you combine epacadostat with PD-1? Thanks.

Steven Stein - *Incyte Corporation - Chief Medical Officer*

Jeff, it is Steven again. And Reid may make additive comments on the second part of your question on the scientific aspects.

I think you allude to one thing we did learn last year around the SITC/SMR experience is to have a little more mature data sets beyond first scan information. Generalizing across our program we generally do first scans at around nine weeks and second scans at 18 weeks and then from there on. It is around the time to event piece that is the most critical here because that tends to be the end point that is used in regulatory studies including our own ECHO-301 program where we're looking at progression free survival as well as overall survival.

I personally favor having more mature data sets to make informed decisions and then combining that as you have seen critically now with the recent BMS experience with the biomarker piece as well. So a long answer to your question, but the two scan at least piece of information is critical and beyond that in terms of hitting various benchmarks.

I don't know if Reid wants to make anything additive to the scientific aspect of having longer data in the Immuno-Oncology setting where you tend to focus beyond response on things like progression free survival and overall survival.

Reid Huber - *Incyte Corporation - Chief Scientific Officer*

Just one other additive comment Geoff, this is Reid.

We know now in immune therapy and we have come to expect that responses can occur later in the treatments so responses that are converted from patients with stable disease after a number of cycles is not uncommon. So it is important to gather that duration of treatment data so we can more accurately measure and quantify the benefit of a double regiment. So absolutely it is an important part of the program and as Steven alluded to is captured in the second bucket of data where you are looking at durability of response where you can also convert and capture those patients that are exhibiting responses a little bit later in the treatment.

Geoff Meacham - *Barclays Capital - Analyst*

Thanks guys.

Operator

Our next question comes from Michael Schmidt from Leerink Partners.

Michael Schmidt - *Leerink Partners - Analyst*

Thanks for taking my question. I had one for Barry, in terms of Jakafi growth can you break down volume versus pricing inventory movements?

And then one for Steven around epacadostat. Is it fair to assume that, in the Phase 2 program, is it fair to assume that it is biased towards indications where PD-1 is approved and how important is it for you to make a Phase 3 deal decision depending on what a partner is opting into cost share of the trial?

Barry Flannelly - *Incyte Corporation - General Manager US*

Thanks Michael, this is Barry.

As you know we had a 14% growth quarter over quarter in Q2 over Q1. And you know we took a 6% price increase at the beginning of the second quarter. We don't get all of that 6% of course because of mandatory government rebates and discounts and so forth, so that is about 5%.

We know the gross to net improved in the second quarter by 2.9% versus the first quarter of 2016. We had prescription demand increase by more than 6% in the second quarter versus the first quarter.

I will turn it over to Steven for the next part of the question.

Steven Stein - *Incyte Corporation - Chief Medical Officer*

Michael, it's Steven.

I don't think bias is the right word but if you look across the ECHO program in its totality and the four different studies we have 12 different tumor types there. You are correct in that many of them are considered the hotter tumors that already have approvals for PD-1 or PD-L1. Melanoma and non-small cell lung cancer, bladder cancer, and just recently head and neck, but additionally we are studying other areas where we will explore the biology of areas where it maybe is not so hot from an immune aspect, like triple negative breast cancer, ovarian, lymphoma, colorectal cancer etc.

And then we will study all those endpoints that I alluded to earlier in terms of making decisions. But we are looking at both the hot and cold tumors, so I don't think it is biased.

In terms of selecting partners for future collaborations and how to do them and whether opt-ins are important, I'm going to ask Herve to address that.

Herve Hoppenot - *Incyte Corporation - President & CEO*

We like to work together with the partner and to have the cost sharing program, so that is something we're trying to get. But it would never stop us from moving into the next step of the program if for some reason the partner was not willing to go and do the cost sharing. It is basically a good guy if we have it and if we don't we go ahead and do it anyway. If we believe that we have enough data to justify moving to the next step.

Michael Schmidt - *Leerink Partners - Analyst*

Great. Thanks so much.

Operator

The next question comes from Brian Abrahams from Jefferies.

Brian Abrahams - *Jefferies LLC - Analyst*

Thanks for taking my question and congrats on a good quarter.

Two questions. First off, I guess shifting gears to GVHD, sounds like the pivotal program, steroid-refractory patients, is kicking off. You mentioned we'll get some data for '110 (INCB39110) later this year. Just wondering, where might '110 potentially fit in? Do you see a potential role for that in sort of a different segment perhaps in prophylactic or front line patients? Any reason why selective JAK1 might work differently versus ruxolitinib or should we see that as proof of principle for ruxo?

And then for Barry, I'm wondering as we see continued survival data roll out for Jakafi are you seeing an evolution in clinical practices in terms of earlier intervention in MF and how much potential room for growth do you foresee in terms of moving toward earlier stage patients?

Steven Stein - *Incyte Corporation - Chief Medical Officer*

Brian, I will start off with Stephen and Reid may make additive comments.

So you are right in terms of Graft-Versus-Host Disease you have to be somewhat careful in separating out the different entities. There is the acute setting and then the chronic setting, and then within the acute setting there is steroid-refractory, first-line and the prophylactic setting. And we feel that both compounds may potentially have a role there.

In terms of the biology that may underlie that, we are obviously developing the clinical data now and look forward to sharing the '39110 proof of concept data with you hopefully later this year. But that is leveraging the JAK1/2 selectivity in terms of modest oppression in certain settings, and we feel that '39110's best role in that disease setting may be in first-line. So prior to developing the steroid-refractory and in the prophylactic setting where the potential in terms of the lack of mild suppression may be more helpful.

These patients are just post transplant and tend to have lower blood count, so that is one area we may be able to leverage the different biology, and Reid may want to make additive comments there. So both prophylactic and first-line, whereas for Rux itself, our current program is focusing on steroid-refractory acute and then the chronic setting, which by definition is also steroid-refractory. I will leave it to Reid if he wants to make any more comments around the biology.

Reid Huber - *Incyte Corporation - Chief Scientific Officer*

Brian thanks for the question.

The data and the translational of work around how JAK inhibition is having these productive effects in patients with Graft-Versus-Host Disease is still very much evolving. But to the extent that we studied it and other academics have studied that, they appear to be mediated largely by JAK1 containing signaling networks, so it's a very provocative scientific hypothesis to study a JAK1 selective inhibitor in these diseases.

As Steven said, ultimately the clinical data will tell us what profile benefits maybe of a JAK1 inhibitor versus the JAK1/JAK2. But there are certainly some emerging scientific bases to be interested in studying JAK1 inhibition in this disease.

Barry Flannelly - *Incyte Corporation - General Manager US*

So Brian, this is Barry, just to address your survival data in MF.

Strangely many of our prescribers are only now realizing the survival -- or the benefit of starting Jakafi earlier in myelofibrosis patients. Now we have two phase 3 trials, COMFORT-I and COMFORT-II, with five-year follow-ups and we have a more than 30% reduction in the risk of death by starting Jakafi earlier versus placebo followed by Jakafi, or best available therapy followed by Jakafi. We think we penetrated the prevalence in the United States by about 25% to 28% and we think we have a long way to go in myelofibrosis particularly to benefit those patients who might start therapy earlier.

Brian Abrahams - *Jefferies LLC - Analyst*

Thanks very much.

Operator

Next question coming from Alethia Young from Credit Suisse.

Alethia Young - *Credit Suisse - Analyst*

Thank you for taking my question. Congrats on the good quarter.

Just one around the -- that new ASCO SITC conference in February, do think that's a potential venue where we could get some more clinical information for epacadostat?

And the second one, I just wanted to talk a little bit about the duration of treatment for Jakafi, I just want to see if you are starting to see increased duration with some of the longer term data that you have been presenting. And also on P-vera I just wanted you to talk about the same trend there as well. Thanks.

Steven Stein - *Incyte Corporation - Chief Medical Officer*

Alethia, it's Steven.

We have the ability to present epacadostat at various meetings, that is an interesting meeting and our eye is on it. We in general will not speak to whether things will be at a meeting until the meeting itself releases its abstract data. So I will just leave it at that and pass it to Barry for the second question.

Barry Flannelly - *Incyte Corporation - General Manager US*

We talked about persistency for both MF and PV. It continues to get better year over year for both MF and PV. I think I said before that PV persistency is a little bit longer.

Obviously we look to our Phase 3 clinical trials as one signal and in fact in both RESPONSE and in COMFORT-I, COMFORT-II patients stay in therapy for a long period of time. We try to keep patients on therapy because we think they will continue to benefit.

Obviously the survival benefit that we have shown in myelofibrosis, and then in terms of symptom control and disease control that we've seen in RESPONSE and RESPONSE II. We think this persistency will only continue to get better.

Operator

Thank you. Our next question comes from Tony Butler from Guggenheim.

Tony Butler - *Guggenheim Partners - Analyst*

Thanks very much for taking the question.

I would love to get your view on the GITR agonist especially potentially in combination with the anti-PD-1, given the notion that there are a number of inflammatory events that may occur with PD1 alone, perhaps pancreatitis, etc. So the question really is do you think you may get, or obtain access 'itis, whatever 'itis, simply because you are adding effectively two agonists together? I would love to understand that view.

And then second, with respect to bladder cancer, on the FGFR inhibitor really around selectivity given that there are a couple of other competitors in the market with FGFR inhibitors and more importantly why bladder cancer first as a first-line. I recognize there are a percentage of bladder cancers that certainly have FGFR3 but the question really is why bladder cancer first? Thanks.

Reid Huber - *Incyte Corporation - Chief Scientific Officer*

Tony this is Reid I will take both your questions and Steven can chime in with additional comments at the end.

First with respect to GITR, we're learning a little more about the agonist space after this last ASCO including data presented on the OX40 agonist, but there is an outstanding question in the field up until these clinical data as to what the ultimate safety profile and tolerability may be of co-stimulatory agonist. And the data, at least emerging thus far from OX40 and maybe soon to emerge around GITR is encouraging in that respect.

So that is the first thing to note, and I think that's important for the field to recognize that these antibodies appear to be able to be delivered at least as single agents as safe treatments with early signs of pharmacologic or pharmadynamic activity.

As you know the ultimate utility of these antibodies based on preclinical data is likely in combination and you referenced the PD-1 based combinations which are clearly attractive combination approaches for both GITR and OX40. It's where our program is certainly designed to move to and we think that the most relevant go or no go decisions and true understanding of safety and efficacy is probably going to come from data emerging from those combination regimens.

Whether there is an inflammatory or an -'itis' type toxicity is yet to be determined but certainly monotherapy data is encouraging and then we are going to have to look for the actual clinical combination data to speak to that because preclinical data doesn't really read on safety.

On your next question with respect to FGFR '54828 was brought forward and designed to -- and was brought forward because it is a very selective inhibitor of three FGFR isoforms, FGFR1, -R2, and -R3. Those are the receptor targets and kinases that are often mutated or translocated and activated in a number of solid tumors.

Bladder cancer is one where the genetics are particularly clean with respect to FGFR3 involvement and we have a very attractive path forward there with a companion diagnostic approach to identify those patients. So it is an interesting one to accelerate the program around.

I don't think necessarily it is the only one and there are of course other tumor types which harbor FGFR amplifications which we may be able to pursue as the program evolves. But certainly out of the gate, bladder cancer is an attractive one for us.

With respect to the competition there are a number of other FGFR inhibitors out there. The first group of them were actually were very nonselective inhibitors and more recently over the last few years we have seen from Novartis, J&J, and AstraZeneca more selective FGFR inhibitors.

One important feature of our program that we were focused on in the Phase 1 setting was whether or not we would only be dose limited by on-target pharmacology, mainly phosphate elevation. Or whether, like some of those other inhibitors I mentioned, we may have off-target dose limiting toxicity. And we have been very pleased with the safety and tolerability profile thus far in Phase 1. We think that is allowing us to push to quite high levels of pathway inhibition, perhaps high enough to begin to differentiate across some of those other molecules in development. But obviously it is still early days and the clinical data will ultimately speak to the quality of the profile.

Tony Butler - *Guggenheim Partners - Analyst*

Thank you. That is all.

Operator

Next question today is coming from Simos Simeonidis from RBC Capital Markets.

Simos Simeonidis - *RBC Capital Markets - Analyst*

Hi. Thank you for taking the question.

This has been asked earlier in a different way but I wanted to get your thoughts on whether you're currently doing anything differently post the surprise to many people failure of the Bristol drug in front-line lung cancer in your trials with PD-1, PD-L1 inhibitors in lung cancer.

The second question is, I know Mike Booth has communicated in the past how you guys will talk to us about which combinations you might take into the next steps with epacadostat. Is it still going to be in a staggered fashion? Is it going to be all at once and then we get the data? Is there any changes to that?

Steven Stein - *Incyte Corporation - Chief Medical Officer*

Simos, it is Steven answering your question.

I think in terms of first-line lung cancer, and some more general comments, sometimes it can be advantageous to not be out front because you can learn from other data sets, not necessarily failures but just other data sets. And in that case I think at least in lung cancer, as opposed to

melanoma, the quantitative intensity of the staining may be more important than it is and other histologies. And where the cut off lie and how they inform your drug and its activity is going to be really really important.

The obvious answer is yes, we will be looking in depth at the biomarker parts of the lung cancer program and trying to understand where we differentiate on efficacy and where we add - is it at the same cut offs or different cutoffs? And that work is in progress at the moment.

In terms of combinations, I will let Reid add to this, it is really wide open. We will look at different levels of evidence. There's preclinical cell line evidence, then xenografts, and then clinical evidence either from ourselves or collaborators and where we will work in both hot and cold tumors and in combination in terms of enhancing other compounds.

The majority of the program right now is on PD-1 and PD-L1 combinations, but there are others in developments including triplets where we look at our own small molecules in different dosing schedules to see if we can modulate the tumor microenvironment by looking at paired biopsies and enhance immune responses. And then we have a vaccine program as well in combination with vaccines.

And it's not stepwise. We are willing to look at things as soon as the evidence is available and remain very confident in that opportunity. And then Reid if you want to add anything around combinations? Hopefully that answers your question.

Simos Simeonidis - *RBC Capital Markets - Analyst*

Great.

Operator

Our next question from Cory Kasimov from JPMorgan.

Morgan Haller - *JPMorgan - Analyst*

This is Morgan on for Cory.

I just wanted to ask a quick question on the data presentation at ESMO. You referenced potentially for us to see PFS data. Are we going to see median PFS or are we going to see any sort of first hand PFS at certain time points?

Steven Stein - *Incyte Corporation - Chief Medical Officer*

Morgan it is Steven.

Obviously "wait for the presentation" is a high level answer. Again it is on a poster discussion on Monday, October 10. If you look at the SITC and SMR data sets we will now have additional scan data and will be able to look at progression free survival over a much longer time interval. As for the actual data I will refer you to the abstract when it goes live on September 28 and then the actual presentation.

Morgan Haller - *JPMorgan - Analyst*

Great, thanks.

Operator

Our next question comes from Ying Huang from Bank of America Merrill Lynch.

Ying Huang - *Bank of America Merrill Lnch - Analyst*

Good morning guys.

I have a question on whether you have any preclinical data or clinical data yet that's not disclosed to show there's a role for I/O inhibitions for PD-L1 negative or even low expression of PD-L1 tumor types?

And then secondly I was wondering if you can provide more color on the position by partner Lilly to drop a couple indications including diabetic nephropathy and then move on for another two indications including atopic dermatitis and lupus. Thanks.

Reid Huber - *Incyte Corporation - Chief Scientific Officer*

I will start off with your first question. This is Reid.

On the preclinical data, IDO has been studied in a number of tumor models now and they really range across both what we think of as hot or more inflamed tumors and tumors which are characterized by less of an immune cell - a productive immune cell infiltrating more of a myelosuppressive or immune suppressive immune cell infiltrate.

The reproducibility of IDO synergy with PD-1 is a very common finding. We in fact see it when tumors are IDO-1 positive, we see it in fact when tumors are IDO negative or the IDO activity may be coming from the infiltrating immune cells and from the tumor draining lymph nodes. I think that data set is a fairly robust one.

The question is how those models translate to patients and there as you well know we have to be much more cautious in either over or under interpreting the preclinical data. The approach that Steven has outlined and we are following with the ECHO Program is to not to limit histology selections and the expansion cohorts to only those which have been shown to be PD-1 responsive, or only those which are characterized by cold tumors, or PD-L1 negative status. We tend to look at both of those and then use a more robust translational program on the backend to work through those details. I think that's the strength of the program going forward. So we are not biased based on any preclinical preconceived notions.

I will turn the next question over to Herve.

Herve Hoppenot - *Incyte Corporation - President & CEO*

Concerning the Lilly relationship, you have to remember that the way it works is we will be asked at some point when the program is going to Phase 3 to either obtain or not the co-financing of the Phase 3. Before that, we are literally in a position where we give our opinion and our advice, but we are really not in any kind of decision-making position. I cannot comment on their willingness to go for any of these indications.

As you said, I think we are dealing with a very broad list of possible indications for this mechanism. Lilly, and we are discussing this with them, is looking in multiple places where baricitinib could be applied. The decision to drop diabetic nephropathy recently is something that was not a huge surprise from our side.

Ying Huang - *Bank of America Merrill Lnch - Analyst*

Thanks.

Operator

Our next question today comes from Peter Lawson from SunTrust Robinson Humphrey.

Peter Lawson - *SunTrust Robinson Humphrey - Analyst*

Steven, just with the BMS failure I was wondering if you can walk through the biomarker strategy you have around epacadostat as well as if there is any points of differentiation you have in that biomarker strategy and how you could potentially drive positive outcomes with the strategy?

Steven Stein - *Incyte Corporation - Chief Medical Officer*

Peter hi, it is Steven.

I cannot speak to the specifics of each collaboration and granular details on the biomarker strategy. Just to tell you that it is broad. So it is beyond the immuno- histo- chemistry and staining, and quantitative staining and intensity, looking at other aspects including the genetics of the space and trying to get a comprehensive understanding of where therapies work and why they don't.

Obviously the cut off per se in the first line lung of the one compounded BMS version compound is interesting. Is it 5% or 50% and what that difference means? And then looking at in combination. And then the same for GITR. We have a comprehensive program but it's too early to give you any specifics.

Peter Lawson - *SunTrust Robinson Humphrey - Analyst*

Just a follow-up, is there a chance you can go back and change the cut off rate and manipulate that or you are locked in?

Steven Stein - *Incyte Corporation - Chief Medical Officer*

In terms of the exploratory part of the program, they are hypothesis generating so there is not a question of changing anything. We can look retrospectively across the board.

When you trigger a prospective Phase 3 then you have to prospectively set what you are going to be looking at. Just to be clear our melanoma Phase 3 ECHO-301 does not use any selectivity in terms of PD-L1 staining and that is not really relevant in the melanoma space at the moment. There is no cut off in the Phase 2 programs, we're doing across-the-board hypothesis testing.

Peter Lawson - *SunTrust Robinson Humphrey - Analyst*

Thank you so much.

Operator

Our next question is coming from Ren Benjamin from Raymond James.

Ren Benjamin - *Raymond James & Associates, Inc. - Analyst*

Good morning of thanks for taking the question and congratulations on a great quarter.

I guess one question for Barry, can you give us a sense at all, or any color regarding the split of MF versus PV and you mentioned the 6% prescription increase in the quarter. Is that growth coming from one indication more than the other?

And then one for Steve regarding the IDO studies. Will you be presenting the data based on the trials as a whole or as specific disease cohorts? For example, would you present data from ECHO-202 and ECHO-203 the triple negative breast cancer all at the breast cancer conference at the end of this year but from two different trials?

The final question regarding bladder cancer, can you give any sort of detail or color. Is it muscle invasive, non-muscle invasive, or superficial? Or in combination with BCG?

Barry Flannelly - *Incyte Corporation - General Manager US*

In terms of a breakdown of MF and PV we still have more sales in MF just because the total cohort of patients that we have over time that continue to grow, as I said before, the persistency is good but in terms of growth quarter over quarter it is a greater PV is -- the total number of PV patients growth is outpacing the total growth of MF patients. But they are both still growing quarter over quarter and we think we can actually increase that going forward particularly for the overall survival in MF and now additional data in PV.

Ren Benjamin - *Raymond James & Associates, Inc. - Analyst*

Got it.

Barry Flannelly - *Incyte Corporation - General Manager US*

Steven?

Steven Stein - *Incyte Corporation - Chief Medical Officer*

Ren for your second and third question, in terms of when and where and how to present the IDO data sets it is really a case-by-case discussion with our partners and investigators. To date we have generally presented studies as a whole so if you look at SITC last year, the ECHO-202 experience, and then what's coming up at ESMO this year.

Going forward there may be opportunities around histologies but it's too early to speak to whether, for example you do a single histology at a histology relevant meeting like a breast meeting. Again that's a discussion with our partners and investigators.

In terms of bladder cancer, the current program is directed at metastatic bladder cancer. It is not superficial or non-muscle invasive. The unmet need is in the metastatic setting at the moment. We will going forward look at potential other areas to study but that is where the program is at the moment.

Ren Benjamin - *Raymond James & Associates, Inc. - Analyst*

Got it. Thank you.

Operator

Thank you. Our next question comes from Ian Somaiya from BMO.

Ian Somaiya - *BMO Capital Markets - Analyst*

Thanks and congratulations on a great quarter.

I have two questions first for Herve. Just want to get your sense and your ability -- do you think you have the ability to at this point continue to aggressively fund your growing oncology portfolio as well as take advantage of the options that you have in terms of the baricitinib program? And just some questions related to your financial health do you think you can do both at the same time?

And separately, for Reid, I was just hoping to get your thoughts on some of the early microbiome data that's been published and potential for a combination with the different I/O programs.

Herve Hoppenot - *Incyte Corporation - President & CEO*

On financial health, you can see the numbers yourself. We are in a position where the growth of the top line is giving us a lot of flexibility to invest in our own portfolio and if Lilly would decide to go in an additional indication, which I wish, we would be able to do the co-funding. To give you an idea from last year to this year in terms of co-funding, the Lilly program has gone down because of the cycle in rheumatoid arthritis.

If we were to go in a new indication, there would be no problem to do that. The way we are thinking of our resource allocation is obviously top line growth is what is giving us all of these opportunities. We spoke about ruxolitinib but we have also now hopefully if baricitinib is approved next year there would be a new line of top line growth from the royalties and some of the milestones from Lilly.

We think about portfolio kinetics, how to move the portfolio as fast as possible in the right indication, obviously, based on the scientific understanding we have. We are not limiting our clinical program because of resource constraints at this point.

Based on what you have seen this quarter you can see we have room to maneuver and obviously stay in a positive cash flow from operations. That's important because that is what makes us stable from the financial standpoint and able to do this operation. Of those 3 criteria that we are using and up to now it has been something we have been able to manage very positively. So the answer is yes if there were opportunities to do co-funding with Lilly, we would probably participate assuming the indication is reasonable.

Reid Huber - *Incyte Corporation - Chief Scientific Officer*

And I will take your second question.

Our view of the microbiome data is that they are very interesting. Obviously some of it has come from a close collaborator of ours, Dr. Tom Gajewski, University of Chicago. It is not an area which we have built a particular expertise around. I think we look at the data and how it emerges in the context of our combination possibility much like we look at other platforms such as vaccines or cellular therapeutics.

Some of these may emerge as interesting tools and when they emerge into the clinic, we will always evaluate the science behind them and how we may be able to work together in those sorts of combination strategies. But right now I think it is a little bit early to assign any real strong scientific rationale to specific combination possibilities that may be appropriate for us.

Operator

Our next question is a follow-up for Michael Schmidt from Leerink Partners.

Michael Schmidt - *Leerink Partners - Analyst*

Thanks for taking my follow-up.

I just had one -- so you mentioned the importance of PFS information and assessing the efficacy of epacadostat in combination with checkpoint inhibitors and I just was wondering, number one, looking at the upcoming data at ESMO, could you just remind us of the benchmark of what we would expect for PD-1 alone with the PFS in those patients.

And secondly PD-1 inhibitors historically have been, I believe, most efficacious in providing overall survival as opposed to PFS and I was wondering what your thoughts are in terms of the PFS as a measure that you are considering?

Steven Stein - *Incyte Corporation - Chief Medical Officer*

Michael, it is Steven.

It is one of the three components progression free survival or time to progression. The benchmarks if you look at pembro alone and generalizing, because you have to be very specific about exactly what population you are looking at and then are you enriching for particular biomarker expression, but in general if you look at their label in melanoma you are looking at about six months for pembro alone.

If you look at nivo plus ipi in the same setting you're looking at about 11.5 months for the combination. And then obviously with efficacy you always weigh in the toxicity in terms of getting to your risk balance equation in terms of using therapies.

But those are the numbers we bear in mind in terms of getting there and again I will refer you to the actual presentation. From a tolerability point of view, to date, our data is very encouraging versus other doublets.

You are right, in terms of overall survival, that is ultimately what is most important to patients and often to regulators. You just can't wait in terms of decision-making early on to wait for OS and then they tend not to be randomized studies. So you use surrogates like overall response and progression free which we feel is most important in terms of decision-making.

Michael Schmidt - *Leerink Partners - Analyst*

Great. Thanks for the follow-up.

Operator

Thank you. We've reached the end of our question-and-answer session. I would like to turn the call back over to Herve for closing comments.

Herve Hoppenot - *Incyte Corporation - President & CEO*

Okay. Thank you for your time today and for your questions. I'll just conclude saying that we look forward to providing you with further updates with the Q3 call in early November for now thank you and goodbye.

Operator

Thank you that concludes our teleconference. You may now disconnect and have a wonderful day. We thank you for your participation today.
