

Bayer AG – Investor Relations
Finerenone Investor Webinar:
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Welcome

Oliver Maier

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Good afternoon and good morning. It's a great pleasure to be with you today, and I welcome you to our 2021 investor webinar for Pharma. As announced in October at the Crop Science webinar, we are planning a series of these events in an effort to keep you apprised of the key developments and milestones across the group.

Today, we are showcasing one of the most important launch assets from our Pharma pipeline, brand name Kerendia or finerenone. We will be sharing today four things – first, details of the clinical profile, second, the differentiated nature of Kerendia, third, how it fits into the treatment landscape, and, fourth, our current plans for lifecycle management. With me on the webinar today are Christian Rommel, our Head of R&D at Pharma, and Sebastian Guth, Head of Commercial Operations Americas in our Pharmaceutical division.

We will begin the webinar with some prepared remarks from Christian and Sebastian, followed by about, roughly, ball-park 30 minutes of Q&A, so pretty much the same setup as we've had on the last webinar for Crop Science. You'll find instructions for participation in the Q&A in the Zoom chat, and I will remind you of those again later on.

[See disclaimer](#)

As always, before we begin, I would bring your attention to the forward-looking statements included in the materials today and currently on the screen. With that, the floor is yours, Christian.

Presentation

Christian Rommel

Head of R&D, Pharma, Bayer AG

Sebastian Guth

Head of Commercial Operations Americas, Pharma, Bayer AG

Christian Rommel

Thank you, Oliver, for your introduction and welcome, everybody, to today's webinar about our key launch asset, Kerendia. As we outlined at our Capital Markets Day in March, a key task for the new executive team at Pharma is to drive the Pharma top-line during the loss-of-exclusivity phase for Xarelto and Eylea and to return the business to sustainable growth thereafter. Among others, a key building block to achieve this task is Kerendia or finerenone. Kerendia is a nonsteroidal, selective antagonist of the mineralocorticoid receptor, or referred to as MR, which is activated by aldosterone and cortisol and regulates gene transcription.

Kerendia blocks MR overactivation in both epithelial – for example, kidney – and nonepithelial – for example, heart – and blood vessel tissues. Overactivation of the mineralocorticoid receptor – again, MR – is thought to contribute to inflammation and fibrosis. Kerendia has a high potency and selectivity for the mineralocorticoid receptor and has no relevant affinity for androgen, progesterone, oestrogen, and glucocorticoid receptors. The selectivity profile of our molecule is not only important for the mechanism of action but also as it relates to safety.

The first approved indication for Kerendia is chronic kidney disease in patients with type 2 diabetes. This is an under-recognised, chronic and silently progressing, life-threatening condition. Globally, around 160 million patients are affected, and it comes with a high disease burden which shortens life expectancy by 16 years on average compared to those living with neither of the diseases. To help you to put this into context, the prevalence of atrial fibrillation, which is the main indication for Xarelto, is about 45 million patients globally.

Patients with chronic kidney disease, or CKD, and type 2 diabetes have residual cardiorenal morbidity and mortality despite current therapies. The risks of progression towards kidney failure and cardiovascular events increase with severity and stage of CKD. In patients with less advanced CKD, cardiovascular risk is most pertinent, whereas the risk of progression to kidney failure events is higher with more advanced CKD. When we listen to patients who face CKD progression, we often hear the following: 'Diabetes wasn't a big deal to me. The problem was that it killed my kidneys, and I did not know.' This tells us exactly how we can support patients with chronic kidney disease and type 2 diabetes. It is not only about making Kerendia available to patients. It is also about diagnosing the disease to intervene earlier and to educate about the renal and cardiovascular risks these patients are facing.

In fact, these patients face a threefold risk of cardiovascular death, and 50% of CKD patients progress to later stages of the disease which often end up into end-stage renal diseases and the need for dialysis. There is an urgent need for more treatments to protect kidney function and avoid dialysis for as long as possible, as well as to prevent cardiovascular events. The majority of

physicians demand better treatment options beyond current standard of care. Those new options should provide a dual kidney and cardiovascular benefit and have clinical utility across a broad spectrum of patients, including those at earlier stages of the disease, and, in this context, continue to guide our development strategy. Next slide, please.

We executed the largest CKD phase 3 trial programme in patients with type 2 diabetes to date, with more than 13,000 patients enrolled. The aim was to investigate the efficacy and safety of finerenone on top of maximally-tolerated renin-angiotensin system inhibition on kidney and cardiovascular outcomes. The programme included two phase 3 trials, FIDELIO-DKD and FIGARO-DKD, which are complementary in nature due to features such as their near-identical designs and endpoints.

FIDELIO-DKD enrolled late-stage patients with type 2 diabetes and stage three to four CKD and moderately to severely elevated albuminuria. FIGARO-DKD builds on FIDELIO-DKD in that it expands the patient population to those with earlier stages of chronic kidney disease, CKD. Both trials met their primary endpoints. Data were presented at last year's ASN congress and at this year's ESC congress. Both trials are complemented by FIDELITY, an integrated analysis with the aim to pool the complementary studies for more robust estimates of finerenone's efficacy and safety across the spectrum of patients.

The patients' characteristics are depicted in the heat map from the Kidney Disease Improving Global Outcomes organisation overlaid with the eGFR, estimated glomerular filtration rate, and urinary albumin-to-creatinine ratio profiles of patients included in the analysis. As you can see, FIDELITY studied a broad spectrum of patients across the continuum of the disease. Eligible patients with type 2 diabetes and CKD treated with a maximum-tolerated labelled dose of an ACE-inhibitor or an ARB-inhibitor were included in the trial. Patients had to have serum potassium concentrations at or below 4.8 mmol/l at screening. Other key exclusion criteria included clinical diagnosis of symptomatic chronic heart failure with reduced ejection fraction due to a class 1A recommendation of MRAs.

A differentiating feature of the clinical programme for finerenone is the fact that it enrolled more early-stage patients than other contemporary trials in CKD with type 2 diabetes. More than 4,000 patients, including those on drug substance and those on placebo, had moderately increased albuminuria, which is characterised by a UACR of less than 300 mg/g. This number of patients should allow for robust data generation in this particular subgroup.

CREDENCE and DAPA-CKD, two trials that investigated SGLT-2 inhibitors in CKD with type 2 diabetes, enrolled substantially less early-stage patients. The breadth of the patient population that was enrolled in the finerenone trials is further exemplified by the fact that also a high number of late-stage patients were included. These are characterised by severely increased albuminuria with a UACR of greater than 300 mg/g.

Why is it important to have robust data generation in earlier-stage patients with CKD and type 2 diabetes? First, these patients represent the majority of the prevalent patient population. Secondly, positive clinical outcomes including this population should support treatment intervention early in the disease to delay progression and to avoid adverse outcomes for the patients.

Let's now have a look at the FIDELITY data. In this patient population with CKD with moderate-to-severely elevated albuminuria and type 2 diabetes, finerenone led to a 14% reduction of the risk of cardiovascular morbidity and mortality and a 23% reduction of the risk of CKD progression, including a 20% relative risk-reduction in the development of end-stage kidney disease and dialysis. The requirement for dialysis is one of the most dreaded complications of CKD

progression. It is associated with substantial morbidity and costs. A significant reduction of this outcome, which is of great relevance to both patients and payers, is therefore notable.

Importantly, these outcomes were achieved on top of standard-of-care treatment to control blood pressure and HbA1c. All patients were treated with a maximally-tolerated dose of an ACE-inhibitor or ARB. Owing to its mechanism of action, finerenone treatment is expected to increase serum potassium concentration through MR antagonism. In FIDELITY, hyperkalemia was indeed more frequent with finerenone versus placebo. However, the incidence of hyperkalemia-related adverse events with clinical impact was low – in fact, very low. Hyperkalemia-related permanent treatment discontinuation occurred only in about 1% of patients when adjusted for placebo over a median follow-up of three years. This combined analysis of two complementary trials provides robust evidence of both cardiovascular and kidney protection with finerenone versus placebo.

The FIDELITY analysis suggests a strong effect of MR-overactivation in the pathogenesis of both cardiovascular disease and CKD progression in patients with CKD and type 2 diabetes. Cardiovascular and kidney risk can be reduced with finerenone, even in patients with less severe CKD and type 2 diabetes, which highlights the importance of initiating treatment before CKD has progressed.

Since the design of the FIGARO-DKD and FIDELIO-DKD studies, SGLT-2 inhibitors have emerged as a therapeutic option for patients with CKD and type 2 diabetes. In an effort to make results from different studies more comparable, we used data collected in the FIDELIO-DKD trial and a methodology similar to that of CREDENCE, which investigated the use canagliflozin versus placebo in these patients.

The post-hoc analysis of FIDELIO-DKD included patients meeting the CKD inclusion criteria of the CREDENCE trial. In addition, the cardiorenal composite endpoint was equivalent to the CREDENCE primary endpoint. In this analysis, the cardiorenal endpoint risk was significantly reduced by 26% with finerenone versus placebo. After adjusting for history of heart failure of the patient, the risk was reduced by 28%. In CREDENCE, the cardiorenal endpoint risk-reduction was 30% with canagliflozin versus placebo. Thus, both the FIDELIO-DKD and CREDENCE studies demonstrate cardiorenal endpoint risk-reduction of a comparable magnitude when trial differences are still considered.

From a physician's perspective, Kerendia comes with the advantage to build on a known, well-established mode of action – the inhibition of the renin-angiotensin-aldosterone systems or referred to as RAAS. RAAS overactivation is associated with kidney and cardiovascular diseases, which often coexist as a cardiorenal disease. Thus, RAAS inhibition by angiotensin-converting-enzyme-inhibition or by angiotensin-receptor-blockage has become the gold standard for treatment of CKD in patients with type 2 diabetes since decades. Kerendia builds on this mechanism in that it extends the RAAS inhibition to the mineralocorticoid receptor pathway [inaudible].

Although there is a rich history of RAAS inhibition starting some 60 years ago, an innovation gap evolved over the last 25 years with limited clinical progress to address significant residual unmet medical need. Prior attempts to intensify the RAAS blockade have failed. Either there was no clinical benefit obtained or side effects have reached unacceptable levels. Kerendia now opens the door for an extended RAAS inhibition option, and it thus continues the long RAAS-centric treatment history for cardiorenal diseases. This extended RAAS inhibition via mineralocorticoid-receptor-inhibition, on top of ACE and ARBs was not accessible until today.

Steroidal MR-antagonists like spironolactone or eplerenone are not indicated in the US for the treatment of CKD due to their clinical limitations and side-effect profiles.

Kerendia builds on a pathophysiological pathway with known positive treatment outcomes that is well-established among physicians. Against this backdrop, we believe that the clinical profile, the well-established and intensified mode of action, and its familiarity among physicians may put Kerendia in a position to become an established treatment option for CKD and type 2 diabetes on top of existing therapies. Along with this comes the clarity of the US label, which will be explained by my colleague to you now, Sebastian Guth. Sebastian, take it here.

Sebastian Guth

Thank you, Christian, and a very warm welcome also from my side.

Now, as Christian has mentioned, the FDA label largely recognises the clinical benefits that were demonstrated in the FIDELIO-DKD trial. Kerendia in the United States is indicated to reduce the risk of sustained eGFR decline, of end-stage kidney disease, of cardiovascular death, of non-fatal myocardial infarction, and of hospitalisation due to heart failure in adult patients with chronic kidney disease associated with type 2 diabetes.

Also noteworthy is the fact that the US label includes just the risk of hyperkalemia in the 'Warnings and Precautions' section. This is a common characteristic for any RAAS inhibition therapy and is, as such, also well entrenched. A prominent US thought leader recently described the safety profile and label of Kerendia, and I quote, as 'pristine', which, to me, is a good articulation of the label in the United States. Kerendia is safe, efficacious and easy to use as it has minimal unwanted effects and plays well with other drugs making it an ideal foundational treatment for this population of patients.

Now, as you can see on this next slide, we are committed to a global launch of Kerendia. In addition to the United States, where we launched the product in August, we filed in Europe, in China and in other markets. Assuming standard review times, we would assume a potential approval in Europe early next year, and, in China, Kerendia may come to the market already next year, which is much earlier than originally planned.

Now, let's focus on the US. In the United States, we significantly invested to cover both the relevant specialists and tap into a targeted portion of the US primary care market with Kerendia. As our initial launch focus, based on FIDELIO, is on later stage CKD patients, we have an early focus on nephrologists, endocrinologists, select cardiologists and targeted general physicians that largely behave like specialists. As we expand into earlier patient segments over time, we see ourselves continuing to expand the target audience.

To approach today's audience, we built a commercial field organisation that is more than 500 colleagues strong. Now, it is worth mentioning that we received more than 30,000 applications for these new positions. Applicants came from some of the leading players in the cardiorenal field, which allowed us to build an organisation with significant expertise and establish relationships in this therapeutic area right from the get-go. The activities of our medical representatives are complemented by strong digital and medical education engagement, particularly during this time of COVID. We achieved early payer wins and have coverage with some of the key plans. Two of the largest PBMs already cover Kerendia in their commercial formularies, and several national health plans, many regional health plans, and Medicaid plans are also already covering Kerendia. Additionally, access programmes like a co-pay card programme and a free-trial programme are in place to support early access.

Our commitment to Kerendia goes beyond chronic kidney disease in patients with type 2 diabetes. We are building on an extensive clinical programme for Kerendia under the umbrella brand of Fineovate. Just recently, we announced the initiation of the FIND-CKD study, a placebo-controlled phase 3 trial to investigate the efficacy and safety of finerenone on the progression of chronic kidney disease in patients with non-diabetic CKD. The primary objective of the study is to demonstrate superiority of finerenone over placebo in delaying the progression of kidney disease in these patients.

Although diabetes is well-recognised as a leading cause of chronic kidney disease globally, a substantial proportion of the global burden is non-diabetic in origin and attributable to other causes such as hypertension and chronic kidney inflammation. The FIND-CKD study will enrol more than 1,500 patients and we expect a primary study completion for 2025. Already, last year, we initiated the FINEARTS-HF study, a placebo-controlled phase 3 study to evaluate the efficacy and safety of finerenone on morbidity and mortality in patients suffering from symptomatic heart failure with a left ventricular ejection fraction of equal or greater than 40%. The primary objective of the study is to demonstrate superiority of finerenone over placebo in reducing the rate of the composite endpoint of cardiovascular death and total heart failure events.

Blockade of the mineralocorticoid receptor system has been shown to be of benefit in the treatment of heart failure. Considering the reduction in the risk of hospitalisation for heart failure that was demonstrated in FIGARO-DKD, there's probably much more to learn about the potential of finerenone in this setting where only limited treatment options exist today. The trial will enrol more than 5,500 patients and is currently expected to be completed in 2024.

Now, in summary, we believe that Kerendia has the potential to become a foundational cardiorenal disease-modifier with a strong scientific and clinical base. Starting from today with the approved indication of CKD in type 2 diabetes, we believe that the potential for Kerendia goes beyond this indication. It may well expand, as I've alluded to, into heart failure and non-diabetic CKD. Our huge investments in lifecycle management is testimony to our belief. We continue to see significant commercial potential for Kerendia and reiterate our peak sales estimate of more than €1 billion. We hope that you share our excitement about Kerendia and that you will closely follow the developments yet to come. The journey has just begun. Thank you.

With this, I hand it back over to Oliver to open our Q&A session.

Questions & Answers

Oliver Maier

Thank you so much, Sebastian. Thank you, Christian, for your remarks and the presentation. It is very much appreciated, and I think with that we will move to the Q&A. Before we start, as last time, some housekeeping items on the Q&A from my end. If you have a question, please click on the 'raise your hand' icon, and, if your question has been answered or you wish to cancel your request, please click on the 'lower your hand' icon. I see the first hand has gone up, which is great.

When you will be called to ask your question, you first have to unmute yourself by confirming the corresponding prompt that will appear on your screen. If you have joined the conference by telephone, the process is slightly different. Please press the star followed by the 9 on your phone to queue for asking a question, and, when you are being prompted to ask your question, please press the star followed by the 6 to unmute yourself.

Sebastian Bray, Berenberg

Hello. Can you hear me?

Olivier Maier

Yeah, absolutely. Perfect.

Sebastian Bray

Thank you very much for taking the time to have a chat and show us the data from the seminar. I'm wondering. Let's say we get to 2024 and the data on the heart failure with preserved ejection fraction turns out to be promising. How would you think about the relative market size of the chronic kidney failure or the chronic kidney disease – pardon me – which is the core market for this product versus heart failure? Could heart failure be a bigger market than chronic kidney disease? Thank you.

Sebastian Guth

Sebastian, I will take that question, and, first and foremost, obviously, thank you for the question. Now, we have, at this present moment, not communicated a specific commercial potential for Kerendia in either heart failure or non-diabetic chronic kidney disease. As you know, we've communicated peak sales potential of €1 billion which pertains to the indication of chronic kidney disease and type 2 diabetes alone. To be honest, we're not prepared, at this present moment, to guide more specifically to the peak sales potential in our lifecycle management indications. I would prefer to wait for the data to then talk about the peak sales potential at the appropriate time.

Sebastian Bray

That's understood. Just to clarify, the data that you have presented today with the greater than or equal to the €1 billion – greater than, pardon me – does not make any provision for this product being successful in heart failure with preserved ejection fraction. Is that fair?

Sebastian Guth

That is correct. The peak sales potential we have communicated today of greater than €1 billion pertains to the indication of chronic kidney disease and type 2 diabetes alone, and we will update the peak sales potential at the appropriate time predicated upon the results of our lifecycle management studies.

Sebastian Bray

That is very helpful. Thank you.

Peter Verdult, Citi

Just two questions – Seb and Christian, thanks for your time again. Just on the original phase 2 data for finerenone in heart failure, just remind us how you picked that apart to justify moving into phase 3. I just want to get a better sense as to your level of confidence in the data you're using to support that to push that into phase 3. Then, just to play devil's advocate – and I think you've

mentioned this during your pre-prepared remarks – a lot of docs will say, ‘If I’ve got an early-stage kidney disease patient, why would I consider finerenone? I’ve got a patient with lots of co-pays. Why don’t I just give them an SGLT-2 or a GLP-1? I get cardiorenal benefits along with glycemic control and weight loss.’ How are you going to push back against that pushback? Thank you.

Sebastian Guth

Christian, do you want to answer first to the question of heart failure and then I’ll dive in and answer Pete’s second question?

Christian Rommel

Yes, certainly. I am happy to support the second one too, but, first, with both studies – the FIDELIO and FIGARO – we met our predefined primary and secondary endpoints. So let me remind you that, when we look at the composite endpoints of the FIDELITY study and the cardiovascular outcome and the components for hospitalisation for heart failure, cardiovascular deaths and non-fatal MI, Kerendia led to a reduced risk-reduction of 14% of the primary endpoint from heart failure hospitalisation and cardiovascular deaths, while, if you look at the RRR, it was 23% with a component to renal death. Basically, when we had met our top line on the phase 2 studies we decided to go ahead. It was three different endpoints, and we thought that the efficacy data, along with, I think, a stellar safety profile, in particular to potassium elevation mandated us and excited us to go forward. I think the outcome now is FIDELIO and FIGARO on the phase 3 and the composite analysis on FIDELITY. As we mentioned, even when you do the sub-analysis to CREDENCE I think it’s extremely encouraging.

On the second question I’ve quickly missed out, remember that we have a very novel mechanism of action here, which is independent of interference with hyperglycaemia and incident metabolic profile, but I’m sure that Sebastian would love to build on that. Before it gets lost on us, we have an anti-inflammatory and potentially antifibrotic, which is the underlying pathology of chronic kidney disease. Sebastian, maybe you want to take it from here, from the treatment management and the other agents.

Sebastian Guth

Thank you, Christian. Pete, great question. Great to reconnect on top of that. I would say four quick thoughts. The first one: as Christian has demonstrated in the prepared remark, if you simply look at the robustness of the evidence you will note that we’ve studied Kerendia in a substantially larger number of early stage patients than the SGLT2 inhibitors, for example. The robustness and scale of the evidence in and by itself is very significant, and obviously important for physicians that consider the appropriate treatment choice.

The second thought – and again, Christian has spoken to that – is that Kerendia provides mechanistical continuity on top of the well-established RAAS inhibition with ACEi and ARBs inhibitors for the treatment of cardiorenal diseases. In many ways physicians have, for a long time, tried the dual-RAAS therapy, and studies to date have failed. That is changing with the arrival of Kerendia.

Third thought is that Kerendia can be combined with any other standard of care – and Christian has spoken to that – as it works independent and is in fact complementary to other RAAS inhibitors and does not require physicians to adopt diabetes background therapies, which for many of them in daily clinical practice is actually important. In many ways it provides a dual benefit, and that’s

what speaks to physicians. I myself have spent a lot of time out in the field, and I can tell you that it does resonate and it's a very relevant offering that we're bringing forward with Kerendia.

James Quigley, Morgan Stanley

I think I'm unmuted now. Can you hear me? Good stuff. Thanks for taking my questions. A couple of short, sharp ones, really. The heart failure trial and the non-diabetic CKD trial: do you have interim analysis built into those trials, and if so, around about what point? That's question number one.

On question number two, you mentioned that the label is based on the FIDELIO data, so the later-stage trials – patients, sorry. What's the process and the timing for increased – for bringing the data on from FIGARO for the earlier-stage patients, and does that really matter in terms of use, or are physicians already looking at the earlier-stage patients as well? I think I'll leave it there.

Christian Rommel

To the final study, as you know, we are recruiting and we are all in for this. I think unless, Jürgen, you correct me, the primary completion will be Q1 2024. That's our current estimate. The non-diabetic kidney study is also going forward, and there I would need help from Jürgen on the timelines. Jürgen, you may want to look that up.

Oliver Maier

I'll follow up with him on that one, Christian, no problem.

Christian Rommel

Okay, and then the other question was...

Sebastian Guth

The other question I can take. I can – sorry, James, I interrupted you. Go ahead. I think your second question was on the update of – potential update of the label to include the FIGARO data in the United States. Is that a correct understanding?

James Quigley

Yes.

Sebastian Guth

No, absolutely. We've filed the FIGARO data with the FDA. We actually filed on 5 November and are expecting the label to be updated. As I alluded to in my earlier remarks, our initial launch focus is on the FIDELIO population, which is later-stage patients. We're then extending, in our commercial efforts, as we see the label getting updated, and as we continue to gain traction in those later-stage patients. It's a very well-planned out launch and commercial strategy that supports the launch of Kerendia in the United States and beyond.

Florent Cespedes, Société Générale

Good afternoon, gentlemen. Thank you very much for taking my question. Can you hear me? Excellent, thank you very much. A quick question on the [trial find?] CKD: could you elaborate a bit on the challenges of this population versus the non-diabetic CKD patients? It's a quite heterogenous population and the size of the trial looks not extremely big, so I'd just like to have your thoughts on the challenges and why you're so confident to address this population, given the heterogeneity of the population. That's my main point. Thank you.

Christian Rommel

So we acknowledge that the disease, the – basically molecular understanding heterogeneity we'll learn more about as we progress with the trial. In terms of size of the trial, you can imagine that our clinical biostatisticians help planning this and design, and we're confident that the trial of the design that we will have the statistical power to deliver the data for the predefined endpoints. Why are we confident? It's based on our experience in the FIGARO and FIDELIO trials, of course. Please remember that we have a large safety database that helps when you now do the life cycle management and expand into other indications, whether it be earlier patients. We also studied paediatric, as you might have recognised, and now the non-diabetic, so we have a large data set. We know the molecule to the therapy, and that has given us the confidence, and we'll update you as we progress. But I want to acknowledge that the non-diabetic is a more heterogenic disease setting.

Florent Cespedes

And maybe a quick follow-up: I suspect that you have some pre-specified sub-group, sub-populations in the trial that could at some point, depending on the results, give you an indication on what are the patients who are the best responders to the treatment.

Christian Rommel

Yes, that we will look at, but I don't think we want to share much about it right now on this call. But this is our common practice in pursuing those developments.

Florent Cespedes

Excellent. Thank you very much.

Michael Leuchten, UBS

One question for Sebastian and one for Christian. Sebastian, just going back to the launch plan strategy, I think the nephrologist audience is relatively clear, but I think it's going to be a little bit tougher into the end of the maybe the PCPs. With the 500 reps that you referred to, should we expect this to be scaled into the label expansion as just discussed, or is that actually going to be scaled on the readout of the FINEARTS HF study that would also give you heart failure indication, something that the SGLT2s can currently already promote?

A question for Christian: you referred to the potential antifibrotic. When do you think you can demonstrate that?

Sebastian Guth

Michael, a great question; let me address the first one and then Christian will address the second one. To your question, at this present moment, as you highlighted, our initial focus is on nephrologists and on a select group of GPs that in many ways behave somewhat like specialists. We see those as the primary prescribers for Kerendia. As we then extend the population, we cover you can also expect us to continue to extend our sales organisation to cover a greater number of, in particular, primary care physicians. In addition, we are, as I look at our early experiences, encouraged by the response of endocrinologists, and see cardiologists playing a more important role in future strategies. So these are target audiences we will continue to look at and appropriately cover as the launch progresses.

Christian Rommel

Yes. And then to your question of the mechanism of action, it's probably fair to say if you've just come from the science side it's a very strong or valid hypothesis that overreaction on the mineralocorticoid receptor leads to inflammation, reactive oxygen, which is a key mediator and messenger that causes fibrosis. Those underlying inflammation and fibrotic events drive chronic kidney disease and cardiovascular disease progressions. We think that this is part of the, ultimately, mechanism of action, the overaction of the MR signalling system, and the outcome of transcriptional changes. I would refer for now to a good body of evidence in pre-clinical kidney and cardiovascular disease models. Not only there is unbiased, because it's not only generated by ourselves, and in the non-diabetic clinical trial we will look at experimental, exploratory evidence for antifibrotic mechanism of action. There's more data to come, but if I think about what's known and what has been shown in experimental pharmacology.

As we talk to – this is the unmet need, when you think about the unmet need in disease biology. Anti-inflammatory, antifibrotic for this disease, as we then can combine with other therapies.

Sachin Jain, Bank of America

Thanks for taking my questions. I've just got a few, actually, and they're all hopefully quite short. So firstly, US pricing strategy. I think you've priced at a decent premium to SGLT2s, if I'm correct. Can you just talk through the rationale for that, given the data seems similar when you present it equalised for trial compositions? That's the first one.

Secondly, in the introductory comments you mentioned you've got some payers online. I wonder if you could just quantify what percentage of eligible patients are currently covered, and how we should think about that progressing in the next six, 12, 18 months. A similar question for your initial focus on specialists: nephrologists, cardiologists, etc. Do you get a sense – I think you mentioned this in the introductory comments but I missed it – what percentage of patients are managed by specialists versus PCPs, and how important is that PCP expansion?

The last one pulls together the two prior questions. The reason for those questions was I was just trying to get a sense of how you think this product will be sequenced versus SGLT2s, which obviously at the moment have much broader labels with the non-diabetic and heart failure population. Again, similar to a prior question: what's the reality and probability of your payer pricing and specialist strategy relegating this to a second-line agent, post-SGLT2?

Sebastian Guth

Sachin, thank you for the question, or questions, I should say. Let me address the first one, pricing. We've priced Kerendia at a WAC price of \$569.10 per 30-day script, or \$18.97 per day prior to discounts. If you look at the net price, that is aligned with our expectations, and as you know the industry has experienced net price erosion over the last couple of years.

If you reflect – if I reflect on the discussion with payers and give you some colour on that, we clearly see payers recognising the need for diagnosis and treatment of chronic kidney disease associated with type 2 diabetes, and to prevent the progression of end-stage kidney disease and dialysis and cardiovascular complications. In the discussions the payers acknowledge that Kerendia is safe, effective and very easy to use. As you can imagine, we are in active discussions with commercial and Medicare plans at both the national and regional levels. I must say we're really encouraged by the early coverage decisions, and are seeing coverage extending in line with our plans.

You asked specifically whether we can give you guidance on the exact size of the population that is covered today. We're in fact not prepared to provide specific guidance, but what I can tell you is that at this moment both commercial and Medicare access are progressing in line with plans, as I said. We're expecting peak coverage by July 2023, so we see coverage building up to its peak in July 2023, which is in consideration of both commercial processes and Medicare bid cycles.

The next question you had is – was around the role of PCPs and whether or not we cover PCPs already today. I just want to give you a little more colour on that, so at the present moment, as I said before, we cover both nephrologists and primary care physicians that in many ways behave like specialists and take a more active role in managing also later-stage patients. These are typically patients of stage 3, stage 4 disease. That's not an insignificant number of primary care physicians here in the United States, so we are covering as of today already nephrologists, other key specialists, and a fairly significant number of primary care physicians, which we're then extending over time.

To your question of sequencing with SGLT2s, I think as we look at the clinical evidence – and Christian spoke to it – we clearly see that Kerendia is a foundational treatment for patients with chronic kidney disease in type 2 diabetes. If I look at the data at this present moment, just to give you a little bit of colour, about 85% of the patients that have been initiated on Kerendia to date are initiated on Kerendia alone, and not sequenced in any shape or form through, for example, an SGLT2. So that speaks to the robustness of the evidence, and what we're bringing and the importance of what we're bringing to this patient population.

Christian Rommel

Sebastian, may I – you also have preliminary, limited evidence from the FIDELITY trial that, from a safety perspective, we can combine. It's a limited set of data. It's about 7% of patients who had SGLT2 inhibitors at baseline, but from a safety perspective we can combine. And from a mechanistic understanding I do think it's fair to say that SGLT2 inhibitors may require the ACEi and ARBs inhibitors in order to see their full potential, which we think is not the case for finerenone being on the RAAS pathway. You can think of that, going forward, the potential in combinations.

Sachin

Very clear, thank you.

Damien Conover, Morningstar

Can you hear me okay? Super. Thanks for taking the question. I just wanted to follow up on that last question and try to understand how well-positioned Kerendia is versus the SGLT2s. If you look at cross-trial comparisons you could arguably say some of the SGLT2s look like they've performed a little bit better, but in the FIDELIO study there was the allowance of using SGLT2s. I guess the question is how prevalent was the usage of SGLT2s, and, if you were to exclude that, could you talk at all about the strength of Kerendia maybe on a more apples to apples, cross-trial comparison? I get there's a lot of challenges with this question, and statistically it doesn't always work, but what I'm trying to get at is just how strong is Kerendia versus SGLT2s in a more level playing field?

Sebastian Guth

Maybe I'll take the lead and then Christian joins in. I think it's really important to recognise – and we've spoken about this before – that Kerendia is a foundational treatment for patients with chronic kidney disease and type 2 diabetes, and we clearly see that resonating as we engage with physicians across the United States. I've spoken to it earlier, but just a very quick recap. It's really the mechanical continuity on top of well-established RAAS inhibition that makes intuitively a tonne of sense physicians. It's the fact it can be well-combined with any standard of care, as it works independent and complementary to other RAAS inhibitors. It's the fact that provides specific treatment and does not interfere with any diabetes medication and does not require physicians to adjust and play around with their diabetes background therapy, which again, in daily clinical practice is obviously very important to them.

We see that clearly resonating with the physician audiences here across the United States, and if you then look at the data in combination with SGLT2s for those interested in doing so, as Christian has alluded to, we do have a pretty robust 7% of patients of such a large trial, which in absolute terms is actually a pretty substantial data set. We do have a substantial data set, and Christian can speak to that best. That gives us a clear indication of both safety and early indicators, also, on the efficacy of a combination if physicians choose to go there.

Christian Rommel

Thanks, Sebastian. Two or three points to add. One is that in our study we enrolled patients at an earlier stage compared to any of these SGLT2 inhibitors. There are some trial differences, of course. When we did what you suggested, and you may back to – after the meeting you can look again at slide 12 of our presentation, of the CREDENCE-like post hoc analysis, what we concluded and analysed was that the data are rather comparable more than they are different, yet they have very different mechanism of actions. By the robustness of the data of FIDELIO and FIGARO understanding the patient population and earlier-stage patients, and then this analysis of CREDENCE-like analysis, I think that we feel strongly, again, like Sebastian says, that we're referring to finerenone as a foundation therapy for heart and kidney disease.

Sebastian Guth

And Christian – or Damien – one aspect that we haven't spelt out but that is important to recognise is that when you look at the results of FIDELITY you see that the cardiovascular benefits of Kerendia that were demonstrated in FIDELITY were consistent, regardless of the baseline use of SGLT2s, which again is a strong signal that all patients should be on Kerendia, as we have alluded to earlier.

Damien Conover

Great, thank you.

Closing Remarks

Oliver Maier

Thank you, Damien. I think that concludes our programme for today. Thank you so much, Sebastian. Thank you so much, Christian, for taking the time today. I would also like to thank the team behind the scenes and everybody involved making this happen. It always seems flawless but it's a lot of work behind it, so we hope you enjoyed this event. We would appreciate, obviously, your feedback on the format, as we intend to keep that format also going forward. We'd like to improve where we can at any point in time.

I'd like to thank all of you for your continued interest, and hope you stay safe. Thank you so much. Talk to you guys soon. If there are any follow-ups please don't hesitate to get in touch with me or with Jürgen. Thank you, guys. Stay safe.

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