Filed by Aduro Biotech, Inc. Pursuant to Rule 425 Under the Securities Act of 1933 And Deemed Filed Pursuant to Rule 14a-12 Under the Securities Exchange Act of 1934

Subject Company: Chinook Therapeutics U.S., Inc. Commission File No. 001-37345



communications simplified.

Chinook Therapeutics

Aduro Biotech and Chinook Therapeutics Definitive Merger Agreement

June 2, 2020

CORPORATEPARTICIPANTS

Noopur Liffick, Vice President, Investor Relations & Corporate Communications, Aduro Biotech

Stephen Isaacs, Ph.D., Chairman, President & CEO, Aduro Biotech

Eric Dobmeier, President & CEO, Chinook Therapeutics

Tom Frohlich, Chief Business Officer, Chinook Therapeutics

Andrew King, DVM, Ph.D., Head of Renal Discovery and Translational Medicine, Chinook Therapeutics

Andrea van Elsas, Ph.D., Chief Scientific Officer, Aduro Biotech

Dimitry S.A. Nuyten, M.D., Ph.D., Chief Medical Officer, Aduro Biotech

William G. Kachioff, Interim Chief Financial Officer, Aduro Biotech

C O N F E R E N C E C A L L P A R T I C I P A N T S

Joe Pantginis, H.C. Wainwright

John Newman, Canaccord Genuity

Matt Phipps, William Blair

Chris Shibutani, Cowen & Co

PRESENTATION

Operator

Good morning everyone, and thank you for joining us today.

At this time, all participants are in listen-only mode. Following Management's remarks, we will hold a brief question-and-answer session, and at that time, the lines will be open for you. If anyone would like to ask a question during this time, please press star, followed by the number one on your telephone keypad. As a reminder, this conference is being recorded.

I would like to turn the call over to Ms. Noopur Liffick, Vice President of Investor Relations and Corporate Communications at Aduro Biotech. Please go ahead.

Noopur Liffick

Thank you. Good morning.

Joining me on today's call are Stephen Isaacs, Chairman, President & CEO of Aduro Biotech; Bill Kachioff, Interim Chief Financial Officer of Aduro Biotech; Dimitry Nuyten, Chief Medical Officer of Aduro Biotech; Andrea van Elsas, Chief Scientific Officer of Aduro Biotech; Eric Dobmeier, President & CEO of Chinook Therapeutics; Tom Frohlich, Chief Business Officer of Chinook Therapeutics; and Andrew King, Head of Renal Discovery and Translational Medicine of Chinook Therapeutics

The recording and the slides presented during the call will be available on the Investor section of Aduro's website and the News section of Chinook's website for 90 days.

Before we start, I would like to remind you that today's call will include forward-looking statements based on current expectations. Such statements represent Management's judgment and intention as of today and involve assumptions, risks and uncertainties. Aduro and Chinook undertake no obligation to update or revise any forward-looking statements. Please refer to Aduro's filings with the SEC, which are available from the SEC or on the Aduro website for information concerning the risk factors that could affect the Company.

I will now turn the call over to Steve Isaacs, Chairman, President and CEO of Aduro.

Dr. Stephen Isaacs

Thanks, Noopur, and good morning everyone.

Today, we announced the proposed merger of Aduro Biotech and Chinook Therapeutics.

I'm pleased to have the opportunity to discuss this transformative news with you.

I'll start by providing details surrounding Aduro's process to identify strategic alternatives and the rationale for entering into this transaction. I'll then hand it over to Eric Dobmeier, President and CEO of Chinook, who will provide an introduction to Chinook Therapeutics, including its technology, lead programs and future plans for the merged company.

Let's quickly recap what brought us here today.

In January 2019, Aduro underwent a strategic reset as well as a subsequent corporate restructuring in January 2020 to reduce operating expenses and extend our cash position into 2023. In the second quarter of 2019, Aduro initiated a process of evaluating strategic alternatives for the company. Throughout the process, we remained dedicated to identifying an option that we believe could provide value to our stockholders.

As part of this evaluation process, we looked at potential business combinations with numerous companies. With recent increased focus on our investigational anti-APRIL antibody, BION-1301 for IgA nephropathy, Aduro's Board of Directors and Executive Team began evaluating companies focused on renal disease for potential synergies. Ultimately, Aduro's Board of Directors determined to merge with Chinook Therapeutics, which we believe offers a compelling opportunity to build a leading kidney disease Company.

Aduro stockholders should be excited about this transaction for a number of reasons. Chinook's pipeline is led by atrasentan, a Phase 3-ready program in IgA nephropathy and includes a number of investigational precision medicines in development for other rare, severe chronic kidney diseases. These programs, in conjunction with BION-1301, will provide the combined company with a focused pipeline in kidney disease. We believe Chinook's seasoned Management Team will be instrumental in driving the development of these programs.

2

Speaking of BION-1301, we're enthusiastic about the progress we're making with the program, and earlier this morning, we announced that data from nonclinical studies and Parts 1 and 2 of our ongoing Phase 1 study are being presented as posters as part of the 57th Fully Virtual Congress of ERA-EDTA, the European Renal Association-European Dialysis and Transplant Association.

Results presented are from placebo-controlled arms of the Phase 1 study that evaluated BION-1301 in single and multiple ascending dose cohorts in healthy volunteers. BION-1301 was well-tolerated, with no serious adverse events, treatment discontinuations or events meeting stopping criteria. Non-neutralizing ADAs occurred in less than 10% of subjects with no correlation to dose. PK was relatively dose-proportional with an estimated half-life of 33 days, suggesting the potential for monthly doses. The data demonstrated corresponding changes in free APRIL, with over 90% target engagement achieved with a single 450 milligram dose. BION-1301 dose-dependently and durably reduced IgA and IgM levels, and to a lesser extent IgG. Importantly, IgG values remained in normal ranges with no increase in infections.

In addition, no tox findings were reported in nonclinical toxicology studies of BION-1301 evaluating IV administration for up to six months and subcutaneous administration for up to one month.

Next steps for the BION-1301 program are to complete enrollment of adult patients with IgA nephropathy and to initiate an optional Open-Label Extension study. Separately, we will determine the subcutaneous bioavailability of a new high concentration formulation to support subcutaneous administration for long-term dosing.

We look forward to discussing the BION-1301 data in greater depth during our scheduled conference call and webcast on Monday, June 8th at 1:00 pm Pacific with Dr. Jonathan Barratt, the Mayer Professor of Renal Medicine at University of Leicester.

In addition to closing the merger with Chinook, we remain focused on exploring strategic alternatives for our legacy programs including outside of kidney disease, including our STING agonist program in collaboration with Novartis, our cGAS-STING inhibitor program in collaboration with Lilly, and our out-licensed anti-CD27 program with Merck, as well as deprioritized programs such as our anti-SIRP α and anti-CTLA-4 antibodies. Prior to the closing of the proposed merger, Aduro stockholders will be issued contingent value rights representing the right to receive certain cash payments from proceeds received by Aduro, if any, related to our non-renal assets for a period of ten years following closing.

With respect to the Management Team for the new combined company, I will step down as CEO and leave the Board upon close of the transaction. The combined company will be led by Chinook's Executive Team, including Eric Dobmeier, President and CEO; Tom Frohlich, Chief Business Officer; Andrew King, Head of Renal Discovery and Translational Medicine; Alan Glicklich, Chief Medical Officer; and Renata Oballa, Vice President of Chemistry and Site Head for Chinook's Vancouver, British Columbia site.

Aduro's Executive Team and Board of Directors unanimously supported this transaction and see great potential for Chinook to build an industry-leading kidney disease company.

Finally, I would like to extend my sincere thanks to Aduro's employees for remaining diligently focused on our programs and to our stockholders, partners and Board of Directors who have supported us all along the way.

With that, I would now like to turn the call over to Eric.

Eric Dobmeier

Thanks Steve, and good morning everyone.

We are thrilled with the opportunity to combine Chinook's technology, pipeline and Leadership Team with Aduro's BION-1301 program and its financial and operational resources. In my remarks, I'll discuss Chinook's rationale for this deal, the opportunity and unmet need in kidney disease, particularly IgA nephropathy, and the new Company strategy, pipeline and Management Team.

Chinook was launched in 2019 with a mission to create a leading company in the kidney disease space. The Company was built around a group of experienced and talented scientists from Inception Sciences, a drug discovery unit incubated within Versant Ventures. Versant and Apple Tree Partners initially seeded Chinook, and they were joined by Samsara Biocapital in our Series A financing. During 2019 and into 2020, we made great progress advancing our pipeline, building our research and development teams, and in-licensing atrasentan from AbbVie.

We evaluated many strategic options for continuing to grow Chinook, and we believe this proposed merger with Aduro is an attractive path to advance our kidney disease pipeline. At the time of closing, we expect the combined company to have three programs already in, or advancing towards, clinical trials, a strong preclinical pipeline and a cash position of approximately \$200 million.

Next, I'll share why we believe the time is now for kidney disease drug development.

As many of you know, kidney disease has been a tough area for drug development over the past several decades, with a huge unmet medical need and few therapeutic options. There are no approved drugs for many types of chronic kidney diseases. Patients are often treated with blood pressure lowering medications, steroids and other off-label therapies, while watching and waiting as kidney function declines. In the United States alone, we're spending over \$100 billion on kidney disease, with much of it spent on dialysis and transplant when a patient's kidneys have already failed.

However, several new kidney disease drugs have been recently approved, including tolvaptan for polycystic kidney disease and canagliflozin, an SGLT2-inhibitor, for diabetic nephropathy. Dynamics are changing in the kidney space that have opened up exciting opportunities for drug development.

First, there are novel genetic targets, translational platforms and patient stratification approaches increasingly being utilized in kidney disease. Similar to other therapeutic areas like oncology and rare disease, researchers can now target causal mutations, employ new preclinical models and utilize biomarkers to design targeted therapies and run studies in more homogenous patient populations. For example, at Chinook, we're utilizing single-cell RNA sequencing, kidney organoids, mesangial cell systems and other novel approaches in our research efforts. We're also utilizing biomarkers for patient selection strategies in our planned clinical trial designs.

Second, and importantly, the FDA has indicated a willingness to grant accelerated approval to drugs based on their observed effects on surrogate endpoints, which we believe enables more rapid and efficient drug development. For example, proteinuria, or elevated levels of protein in the urine, and estimated glomerular filtration rate, or eGFR, a measure of kidney function, are both endpoints that the FDA has indicated may be acceptable for approval in primary glomerular diseases. These diseases include IgA nephropathy, or IgAN, which is the lead indication for Chinook's atrasentan and Aduro's BION-1301 programs. We believe using surrogate endpoints such as proteinuria and eGFR will allow us to design more efficient clinical trials.

Following the consummation of the proposed merger, the combined company's pipeline will contain multiple product candidates in both early and late stages of development.

4

Chinook's lead program is atrasentan, an investigational selective endothelin receptor antagonist or ERA. We in-licensed atrasentan from AbbVie in late 2019, and plan to move it into a Phase 3 clinical trial for IgAN in early 2021. Atrasentan was studied by AbbVie in multiple Phase 2 and Phase 3 studies for diabetic nephropathy, including the more than 5,000 patient SONAR trial, where it showed promising activity. Although AbbVie discontinued the SONAR trial early for strategic reasons before an interim analysis, clinical investigators closed out the study per protocol. When the study was ultimately un-blinded, analyzed and published in Lancet in 2019, it demonstrated that the trial achieved a p-value of 0.029 on its primary endpoint of a composite of hard kidney outcomes.

Our initial interest in atrasentan stemmed from our research team's recognition of the key role of endothelin pathway activation in a variety of proteinuric kidney diseases. Thus, we are particularly excited by the strong and consistent reductions in proteinuria, as well as the impact on eGFR, observed in SONAR. The FDA has indicated that proteinuria reduction may be an acceptable endpoint for seeking accelerated approval in IgAN, and that reduction of eGFR decline may serve as a confirmatory endpoint to support full approval. We have had positive interactions with the FDA regarding our planned Phase 3 trial, the design of which we intend to describe in more detail in the coming months.

We are also planning to initiate a Phase 2 basket trial of atrasentan designed to enroll open-label cohorts of patients with IgAN who would otherwise be ineligible for the Phase 3 trial, as well as patients with other primary glomerular diseases. We anticipate that this Phase 2 study will be a signal-seeking trial that can inform our life cycle management plans for atrasentan and generate key data readouts we can present at medical conferences while our Phase 3 trial is ongoing.

In addition to atrasentan, we are enthusiastic about adding Aduro's BION-1301 program to our pipeline. We believe APRIL plays a key role in the underlying immunologic cause of IgAN, and therefore believe blocking APRIL with BION-1301 is a potential disease-modifying approach. Preclinical data with BION-1301 in non-human primates showed significant reductions in serum IgA levels, and we look forward to the two posters Aduro is presenting later this week at the ERA-EDTA virtual congress featuring data from parts 1 and 2 of the Phase 1 study of BION-1301 in healthy volunteers, as well as data from long-term nonclinical studies.

We also look forward to exploring synergies and efficiencies that may be realized by running the atrasentan and BION-1301 clinical programs simultaneously. We believe the proposed mechanisms of action of atrasentan and BION-1301 are complementary and could potentially treat a broad spectrum of patients with IgAN and other primary glomerular diseases. In addition, we're interested in the possibility of exploring a combination study of atrasentan and BION-1301 as part of the life cycle management of both product candidates in the future.

Beyond the atrasentan and BION-1301 programs, we are developing a preclinical pipeline of investigational precision medicines for rare, severe chronic kidney diseases globally. Our most advanced preclinical program is CHK-336, which we're developing for an ultra-orphan kidney disease, and is currently in IND-enabling studies. We also have research and discovery efforts underway in other diseases, including polycystic kidney disease and other primary glomerular diseases.

Next, I'd like to speak about IgAN, which is the lead indication for both atrasentan and BION-1301. IgAN is the most common primary glomerular disease, with a prevalence of approximately 140,000 patients in the U.S., approximately 100,000 in the EU5 and an even larger population of patients in Asia, where the disease is more prevalent.

IgAN has no approved therapies and few effective treatment options. At diagnosis, patients present with a variety of symptoms ranging from asymptomatic microscopic hematuria, or blood in their urine, to high levels of proteinuria and rapidly progressive kidney function decline. When patients are initially diagnosed with IgAN, they're generally placed on blood pressure lowering medications called ACE inhibitors and ARBs, and their kidney function is monitored. If they continue to progress, a variety of off-label therapies may be tried, including immunosuppressive drugs such as steroids. Ultimately, many IgAN patients continue to progress, and up to 40% of the IgAN population develop end-stage renal disease within 20 years, requiring dialysis or transplantation.

Thus, there is a strong need for better treatment options, and atrasentan and BION-1301 target the disease in different ways. These therapies have the potential to delay progression, improve symptoms and provide a better quality of life for people living with IgAN.

Moving forward, Chinook will be run by a seasoned Management Team with substantial drug development and leadership expertise.

As CEO, I bring 20 years of experience in the biotechnology space, including more than 15 years at Seattle Genetics, which I joined in 2002 when it was a small, newly-public company and helped it grow to more than 1,200 employees and more than a \$10 billion market capitalization. At Seattle Genetics, I held titles of increasing responsibility in business, operations and corporate strategy, including the last six years as Chief Operating Officer.

Tom Frohlich is Chinook's Chief Business Officer, with 20 years of experience in commercial and business development roles at Merck, Janssen and Arbutus.

Andrew King is our Head of Renal Discovery and Translational Medicine, with 15 years of research and translational experience and deep knowledge of the kidney disease space from his time at AbbVie and Ardelyx.

Alan Glicklich is our Chief Medical Officer with 20 years of drug development experience in both big pharma and biotechnology settings, including most recently at Arena and Bird Rock Bio.

Finally, Renata Oballa is our Vice President of Chemistry and site head of our Vancouver-based research group, with more than 20 years of experience in research and drug development from Merck and Inception Sciences.

In addition to our strong Management Team, Chinook will have a Board comprised of talented and experienced professionals who will help guide us as a public company. Jerel Davis from Versant Ventures and Srini Akkaraju from Samsara Biocapital will be joining the Board, representing two of Chinook's largest investors. In addition, William M. Greenman, President and Chief Executive Officer of Cerus Corporation, and Ross Haghighat, Founder, Chairman and Managing Partner of Triton Systems, Inc., are both continuing as members of the previous Aduro Board. We expect there to be two additional independent directors. I will also be joining the Board and look forward to working closely with these directors to advance Chinook Therapeutics as a public company.

Looking forward, I'd like to describe our vision for the future of Chinook. Since its founding in 2019, Chinook has been on a steep trajectory of growth and advancement of its pipeline. 2020 is a year of execution, with start-up activities underway for the Phase 3 and Phase 2 basket trials of atrasentan, IgAN patient treatment with BION-1301 imminent and IND-enabling activities ongoing for CHK-336. We plan to have four clinical trials ongoing across our three lead programs in 2021. We will also continue to apply our guiding R&D principles to advance our preclinical pipeline, and will evaluate partnering opportunities in several areas. Ultimately, our goal is to build a leading company in the kidney disease space with a strong, diverse clinical pipeline and active research and discovery efforts.

I'll now hand the call over to Tom Frohlich, Chinook's Chief Business Officer, to discuss some specifics of the proposed merger.

Tom Frohlich

Thanks Eric, and thanks to everyone for being on the call today.

ViaVid has made considerable efforts to provide an accurate transcription. There may be material errors, omissions, or inaccuracies in the reporting of the substance of the conference call. This transcript is being made available for information purposes only. 1-888-562-0262 1-604-929-1352 <u>www.viavid.com</u>

6

I'd like to briefly summarize the rationale and details of the merger.

By combining Aduro and Chinook, we intend to create a leading company in the kidney disease space, with a multi-product clinical pipeline, strong research capabilities and financial resources. Between the combined capital of the companies, and an additional \$25 million funding commitment from Chinook's existing investors, the new Company will be in a strong financial position to execute on our goals with approximately \$200 million in cash, cash equivalents and marketable securities expected at closing.

Pursuant to the merger agreement, and as described in more detail in our press release and 8-K filing, Aduro will acquire all of the outstanding capital stock of Chinook in exchange for the issuance of newly issued shares of Aduro common stock. Immediately following the completion of the merger, Aduro's then current equity-holders will own approximately 50% of Aduro's capital stock and the former Chinook equity-holders will own approximately 50% of Aduro's capital stock and based on expected net cash balances and before the \$25 million financing from Chinook's existing investors.

Each of Versant Ventures, Apple Tree Partners, Samsara BioCapital, AbbVie, Inc., Morningside Venture (VI) Investments Limited, Morningside Foundation and Ultimate Keen Limited, as well as the directors and certain officers of both companies, representing, in the aggregate, approximately 85.0% of the outstanding stock of Chinook and approximately 22.4% of the outstanding stock of Aduro, have signed support agreements committing to vote in favor of the transaction and lock-up agreements restricting transfers of Aduro's capital stock for 180 days post-closing.

We are targeting the transaction to close in the second half of 2020, pending stockholder approval. The combined company will operate under the Chinook name and its common stock will trade on Nasdaq under the new ticker symbol "KDNY". The combined company will be headquartered out of Chinook's existing facilities in Vancouver, British Columbia and Seattle, Washington.

In summary, we believe this combination represents a terrific opportunity. Our Board and major stockholders support this transaction and see great potential for Chinook, its team, pipeline and technology to bring a transformational approach to kidney disease drug development and help patients.

With that, I'll turn it back over to Eric for some closing remarks.

Eric Dobmeier

Thanks, Tom.

In closing, we believe the combination of Aduro and Chinook represents a strong value proposition for multiple reasons:

First, kidney disease is an underserved market with tremendous unmet medical need, which we believe is on the cusp of being transformed by regulatory authority acceptance of surrogate endpoints and a new focus on genetic drivers of disease, biomarkers and patient stratification tools.

Second, we believe the new company presents a compelling opportunity with multiple clinical-stage product candidates under evaluation for rare, severe chronic kidney diseases.

Third, Chinook will be run by a seasoned Management Team and Board with many years of successful drug development and operational experience.

And lastly, we'll be well-capitalized to advance our pipeline and seek opportunities for strategic partnerships and long-term value generation.

ViaVid has made considerable efforts to provide an accurate transcription. There may be material errors, omissions, or inaccuracies in the reporting of the substance of the conference call. This transcript is being made available for information purposes only. 1-888-562-0262 1-604-929-1352 <u>www.viavid.com</u>

7

I encourage you to view our website for more information, including slides that were developed to complement this discussion. We look forward to keeping you updated on our progress in the coming months.

At this point, we'll open the line for Q&A. Operator, please open the call for questions.

Operator

At this time I would like to remind everyone, in order to ask a question, press star then the number one on your telephone keypad.

Your first question comes from the line of Joe Pantginis from H.C. Wainwright. Your line is open.

Joe Pantginis

Hi everybody. Good morning. Hope you're all well and welcome to the Chinook Team. I have two questions, if you don't mind. First, maybe one for Eric. Just wanted to get your views on how Chinook views the initial 1301 data here. And then the second part of that is, you talked about the potential complementarity with atrasentan and I was just wondering if you can give a little more detail as to the complementary aspects.

Eric Dobmeier

Absolutely. Thanks for the question. So we're really enthusiastic about adding BION-1301 to our pipeline. We think targeting APRIL is a really promising way to address the underlying cause of IgAN, and we're encouraged by the data that's been announced this morning and think that there's room in IgAN where there's no approved therapies for multiple approaches to address the unmet medical need. We think addressing the underlying mechanism of action by targeting APRIL is promising and we also think atrasentan's mechanism of action, which is more of a hemodynamic antifibrotic and antiinflammatory mechanism, combined together are both interesting ways to approach the disease. So we think they're very complementary.

I think I'll turn it over to Andrew King to talk a little bit more in detail about the data and our view of the data that was presented, and also his thoughts on how the two drugs could combine. Andrew?

Dr. Andrew King

Yes. Thanks Eric.

Again, very enthusiastic about the potential for 1301. We find a strong biological rationale supporting the disease modifying potential of APRIL neutralization in IgA nephropathy by targeting key mechanisms that underlie the pathogenic IgA immune complex formation very promising. And the Phase 1 healthy volunteer data is very encouraging, supporting the pharmacodynamic mechanism of action of 1301.

Again, we believe that multiple complementary mechanisms are ultimately going to be required to fully address the large unmet need in IgA patients, and the 1301 MoA is very complementary to atrasentan, which targets a key molecular pathway activated in the kidney in response to IgA immune complex deposition that contribute to proteinuria inflammation, fibrosis and kidney function loss.

Joe Pantginis

That's really helpful, thank you. And hopefully, my next question doesn't get too much into the weeds here as we're just getting used to the story now. But I was just curious as to when you look at the differentiated properties of atrasentan how that might compare to a dual receptor approach such as sparsentan that Retrophin is developing here and has already shown effects on proteinuria, etc. for FSGS.

8

Eric Dobmeier

Yes, good question. So, sparsentan as you mentioned is a dual ARB-ETA inhibitor, while atrasentan's highly specific to ETA. So, we think there's some advantages to targeting ETA specifically with atrasentan. For one, physicians can leave their patients on their currently optimized doses of ACEs or ARBs and can adjust those doses as necessary and independently from atrasentan, whereas with sparsentan, physicians have to take their patients off their current therapies and then put them on sparsentan fresh.

There's also I think some risks with sparsentan that they may be hitting ARBs hard and ETA less hard and they may have hypotension in some of their patients, especially the younger and healthier patients which is largely what the IgAN population is. We have a huge safety database for atrasentan and it's been demonstrated in SONAR and other trials to reduce proteinuria, to reduce decline in EGFR, and there's less data thus far on sparsentan in terms of their safety profile.

So we obviously think it hitting ETA and IgAN and other chronic kidney diseases is an important mechanism to supplement ACEs and ARBs but we think the atrasentan approach has some advantages over sparsentan.

Joe Pantginis

Thank you for that. And then my last question is very quick, hopefully, for Steve. Steve, I know you've put a lot of work into this transaction and you've looked at a lot of options. It certainly looks promising. I was just curious with regard to the support for your shareholders for this transaction.

Stephen Isaacs

Yes, I think there's pretty broad support. We've been working hard to look at ways to provide value to our stockholders and we do share the sentiment that now is the time for kidney drug development. We like the Chinook folks a lot. I think you've just heard about the opportunity for synergies between 1301 and atrasentan, and just leveraging the synergies of two organizations, the new Management Team, and especially the near-term catalysts that atrasentan offers in terms of the Phase 3 that'll be starting in early 2021.

So I do expect broad support for the deal.

Joe Pantginis

Great. Thank you guys.

Stephen Isaacs

Thanks Joe.

Operator

Your next question comes from the line of John Newman from Canaccord. Your line is open.

John Newman

Hi there. Good morning. Thanks for taking my question. Just a question for the new team at Chinook. Just curious if you plan to advance both BION-1301 and atrasentan forward in parallel, or if you would look to push your asset through Phase 3 first and then potentially push forward on 1301? Thank you.

Eric Dobmeier

Yes, thanks for the question. We intend to advance both programs in parallel. As I mentioned, actually we have a planned Phase 3 for atrasentan and IgA nephropathy that we are already executing on getting started and hope to have patients' on study by early next year. We're also doing the Phase 2 basket trial of atrasentan I mentioned in other primary glomerular disease that wouldn't be eligible for the Phase 3. And then, in parallel with that, as you know, BION-1301 is on the cusp of going into IgAN patients in the current trial and if the data's supportive, we would plan to move forward with that program into Phase 2 after that.

So, we're not staging this and we're not staging one behind the other, we think with their complementary mechanisms of action and potentially different patient populations who could benefit from different approaches, we're going to move them forward both in parallel. And there's a huge unmet need in IgAN with no approved drugs. It's an orphan disease but it's not particularly rare and it's under diagnosed and we think if there were available therapies for patients that actually a lot more patients would seek treatment. And there's an opportunity for both drugs to be significant commercial opportunities.

John Newman

Great, thank you very much.

Operator

Your next question comes from the line of Matt Phipps from William Blair. Your line is open.

Matt Phipps, your line is open.

Matt Phipps

Yes. Can you hear me? Hello?

Stephen Isaacs

Yes, we hear you.

Matt Phipps

Okay.

Stephen Isaacs

Now we can.

Matt Phipps

Okay. Sorry. Taking a drug that showed some effect in diabetic nephropathy across multiple endpoints and translating it to IgANs, they have different drivers of the kidney damage but obviously you are ultimately ending up in glomerular damage. So just confidence in being able to translate the benefit from one indication to the next there. And then any risk in the IgAN population with respect to side effects from ETA in addition, such as any peripheral edema or anything that are different than diabetic nephropathy?

Eric Dobmeier

Yes, great question, Matt. I'll start and then I'll hand it over to Andrew to talk a little bit more about the SONAR data in detail. So, in terms of your second question about safety, the main side effect of atrasentan in SONAR and other trials was fluid retention, which can be a problem in diabetic kidney disease patients who tend to be older and have other health problems, like cardiovascular issues or high blood pressure. So that was something that was monitored and actually managed quite well in the SONAR trial. In IgA nephropathy, the patient population is generally much younger and healthier than in diabetic nephropathy. So we think that the fluid retention is not likely to be as much of an issue in that population, so we're optimistic about our ability to go into that patient population from a safety perspective.

In terms of your question about translation of the effects we saw in DKD to IgA, I'll ask Andrew to speak to that. He actually worked on this program with AbbVie for a number of years so is really familiar with it.

Dr. Andrew King

Thanks, Eric. Yes, we do believe there will be strong translation of the anti-proteinuric effects from DKD to IgA given the conserved mechanism of action to reduce intra-glomerular pressure and reduce proteinuria, which is a key driver of both diseases. There is preclinical proof of concept for selective endothil and A-antagonism in spontaneous mouse model of IgA nephropathy and there's also a small respiratory clinical study performed with sitaxsentan, a selective endothil and A-antagonism in a small cohort of chronic proteinuric CKD patients, half of which had biopsy proven IgA nephropathy and we saw a very similar magnitude of proteinuria reduction with the selective ETA antagonist, sitaxsentan, in that patient population as was observed consistently with atrasentan in DKD.

So we do anticipate translation of the anti-proteinuric effect across the diseases.

Matt Phipps

Great, thanks. And then another question on BION-1301. The biggest thing I've tried to bait and be interested in hearing the Chinook Team's opinion is how the—again, we see the drops in IgA nephropathy levels—IgA antibody levels in healthy volunteers pretty robustly now. How does that drop in healthy volunteer IgA levels translate to trying to reduce the galactose-deficient IgA that's really the pathogenic driver of IgAN in these patients? Is it—how do you know that just reducing everything's going to actually reduce those one specific type of antibody?

Matt, I think that's probably a question—I mean, Andrew could give his thoughts, but I think it's probably a question that the Aduro Team could probably better answer than (multiple speakers)

Stephen Isaacs

Yes. Right, I would agree. So we Andrea van Elsas, our CSO, on the phone and he spent several years thinking about this. So Andrea, can you take that?

Dr. Andrea van Elsas

Sure. Hi Matt. Good morning. Basically, we have some evidence from other studies where, for instance, lymph node cells were cultured, tonsillar lymph nodes showing that APRIL regulates galactose-deficient IgA1 production in these tonsillar cultures. There's also data that shows in larger cohorts that circulating levels of APRIL are associated or correlated not just with IgA but also with galactose-deficient IgA1 levels. And then, finally, I think we're eagerly awaiting the data from not just the healthy volunteers where we hope to also be able to show some—at least to have some reliable data quantifying galactose-deficient IgA1 because you and I also produce that and that's certainly going to be true in these healthy volunteers we've studied. But moreover, we'll have, hopefully, patient data to specifically answer that question. I don't think there is—beyond that there is any real data that hence is banking on the manipulation of APRIL levels in a decent fashion.

So we haven't seen that data yet but I would say all the cues we get from other studies, including epidemiology, serology study, tonsillar cultures suggest exactly that.

Matt Phipps

Thanks Andrea. I didn't know you'd be on the call. Good to hear from you too. And one last question, if I may, and obviously this is not kidney related. The initial data that jumped out right away to me is the drop in IgM levels. I mean, 70%, 80%. Do you guys think about BION-1301 in other diseases, I mean, cold agglutinin disease. I realize this is not kind of in the wheelhouse here but that's a pretty robust drop in IgM.

Eric Dobmeier

Andrea, do you want to take that or ...

Dr. Andrea van Elsas

Sure. Steve, do you want me to take ...

Stephen Isaacs

Yes, go ahead.

Dr. Andrea van Elsas

Let me start and then Dimitry can take over. I think it's pretty remarkable. What's really remarkable is that the efficacy towards—and potency and efficacy towards IgA actually is reflected almost exactly towards IgM levels, and certainly that makes you think of other potential indications in these areas that are currently unmet. I think what's also remarkable is at least both in the nonclinical and clinical first healthy volunteer data, we do see this window appearing in terms of regulation of IgA and IgM versus more controlled and less efficacious regulation of IgG, which might just provide us a good window.

I think that's what stands out for me and I know what you're saying. I don't think we have a concrete answer for you yet how that will translate to potential other indications but I'm sure the team going forward will be starting to look at that.

Dimitry?

Dr. Dimitry S.A. Nuyten

Yes. Thanks, Andrea. I actually have nothing to add. I mean, we'll go into more details about the exact data and (inaudible) there are potential other indications that could be studied with this profile and we're very encouraged about the data. And to be continued.

Matt Phipps

All right. Thanks for taking my questions.

Eric Dobmeier

Thanks Matt.

Operator

Again, if you would like to ask a question, please press star, and the number one on your telephone keypad.

Your next question comes from the line of Chris Shibutani from Cowen. Your line is open.

Hello, Chris, your line is open.

Chris Shibutani

Apologies, I was on mute. A question about atrasentan in terms of when we will see some data. I think you've shared that you're going to begin trial in 2021. And also I guess, it's been an asset in clinical development for a number of years going back to AbbVie five years ago. Can you remind us what the IP profile and timeline is for that?

Eric Dobmeier

Yes. Yes, sure, no problem Chris. Good question. So, in terms of timelines, as we said, we're looking to start the Phase 3 by early 2021 with atrasentan, IgA nephropathy and soon after that plan to start a Phase 2 basket trial in other populations of IgA and other primary glomerular diseases. We expect we could have some data from the basket trial starting in 2022 from that program, and then after that, we would have the proteinuria data readout from atrasentan. And that's a relatively rapid endpoint, the proteinuria endpoint. It does allow us to do an efficient trial in the Phase 3 with atrasentan and IgA nephropathy.

So, stay tuned. We'll be giving more details on the trial design and our timelines later in the year, but it is a relatively streamlined clinical pathway for atrasentan.

In terms of IP, I think I'll turn that over to Tom Frohlich, our Chief Business Officer, to answer that question.

Tom Frohlich

Thanks Eric. Yes, it's a great question. The atrasentan composition of matter patent actually expired in 2015. It is a drug that AbbVie has spent some time on. But we feel we have quite a robust IP strategy going forward. But first we've developed an IP strategy with actually three layers of protection. The first layer really revolves around orphan exclusivity, which in the U.S. would give us seven to seven and a half years depending on a pediatric plan and then 10 to 12 years in Europe.

The second layer is based around some formulation patents. There was some oxidation issues in the original formulation of atrasentan which actually caused some stability issues, and then AbbVie going into Phase 3 actually reformulated with what we believe is quite a unique formulation, and we believe that that's quite a robust layer of protection. That runs a few years out further into the mid 2030s, with an ability for patent extension.

And then the third layer really focuses on method of treatment. The day after we closed the transaction in late 2019, we filed a method of treatment for atrasentan.

So we believe between those three layers we have quite a good exclusivity period for the product beyond commercial launch.

Chris Shibutani

Great. And then I'm just trying to piece together exactly what shareholders of Aduro will have on hand. If I take a look at where we were at the last closing price, market cap roughly \$270 million. It looks as if you comment that the Company closing as a combined entity around \$200 million in cash sometime during the second half, which in theory could be as soon as a month but second half to be fair. I guess we had been projecting with a burn of Aduro as an independent company having that cash go down to about \$160 million or so by the end of the year. So some interval there. You're also expecting to have some CVRs that Aduro shareholders will have, and I read the press release correctly it looks as if the timelines will be out until 10 years. But can you provide us a little bit more concretely what you expect the key milestone event will be? How should we think about valuing the CVRs and are there certain triggers that we should be aware of that might be a little bit nearer term, let's say between now and the end of the year or now and the end of 2021? Just for Aduro shareholders, how should they think about the value and the opportunity with these CVRs...

Stephen Isaacs

Yes, okay.

Chris Shibutani

... (multiple speakers) for Chinook?

Stephen Isaacs

Right. So, Eric, I don't know if you want to comment on milestones. We can obviously take the CVR question.

Eric Dobmeier

Yes, why don't you do the CVR question first and then I'll talk about milestones.

Stephen Isaacs

Okay. All right. So, yes, we put a CVR program in place for the Aduro shareholders and this covers the non-renal assets and the non-BION-1301 assets. So our Interim CFO, Bill Kachioff, will give you some of the detail there. Bill, do you want to cover that?

William G. Kachioff

Sure. So, the CVR holders will have the right to receive cash payments in the event of several events happening with the non-renal assets. That would include any consideration paid to Aduro as a result of disposition of any of those assets. Also, license revenues, such as the stream that's due to Aduro from Merck for the development of CD27. And then also any proceeds resulting from Aduro's equity ownership in any entity that we create to hold these assets, including the proceeds from any disposition of that equity. We'll be filing the CVR agreement with our 8-K, which is coming soon. I'd encourage you to take a look at that. There's a lot of details with respect to how payments are calculated and those are in the agreement and easier for you to take a look.

Eric Dobmeier

And with respect to milestones for the combined company, as I mentioned in my earlier remarks, we plan to have three programs in at least four ongoing trials next year. We plan to get the Phase 3 and Phase 2 trials of atrasentan going early in 2021, and then we will have continued data readouts from BION-1301 as we enroll IgAN patients into that trial. And then we also have a preclinical program called CHK-336. It's for an undisclosed ultra-orphan kidney disease. We'll be talking more about that program later in the year. And that's an IND candidate in 2021 as well.

So across the pipeline we think we'll have a good amount of news flow from multiple clinical programs that should be getting underway, that are underway already or will be getting underway next year.

Chris Shibutani

And then lastly, I know you guys have been private. Can you share with us what your expected cash is at the end of the—say, for instance, the most recent quarterly reporting period would have been from March and what your anticipated 2020 burn was, as Chinook?

Tom Frohlich

As Chinook?

Chris Shibutani

Yes. As a private entity, what was your cash at the end of the period and anticipated 2020 burn?

Tom Frohlich

So what we're disclosing is that at closing we expect to have \$10 million in cash and cash equivalent at closing. We haven't given guidance yet for burn rate for Chinook or for the combined company, but our plan is to have about \$200 million in cash in the combined company when we close and we think that's a substantial amount of capital to advance the pipeline and accomplish our goals over the next couple of years.

Chris Shibutani

Great. Thank you.

Operator

There are no further questions at this time. Ms. Liffick, I'd like to turn the conference back to you for additional or closing remarks.

15

Noopur Liffick

Great. Thank you Operator, and thank you to everybody for joining us this morning.

Operator

Ladies and gentlemen, thank you for participating on today's webcast and conference call. This call was recorded and will be available shortly for replay in the Investors section of Aduro's website and the News section of Chinook's website. This concludes our call. Have a great day.

16