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Biogen Inc.

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William Pickering Analyst

Welcome, everyone. My name is Will Pickering. I'm Bernstein's analyst for U.S. Biotech. Very pleased to have Biogen here today joined by Priya Singhal, Head of Development.

Priya, thank you so much for coming.

Priya Singhal Executive

Thank you for having me.

William Pickering Analyst

In many ways, I think that Biogen needs no introduction. It's a long well-known company in our industry, but -- maybe you could take a few moments to speak to the journey that you've been on to create a new Biogen, how your approach to R&D is evolving?

Priya Singhal Executive

Yes, we've been very excited to create the new Biogen. And I'm really pleased because I think we are well on our way. And I'll start with the fact that from a commercial perspective, the Alzheimer's launch, we are seeing some inflection points in the LEQEMBI launch. This is important because we just got approval for subcutaneous maintenance, which we think is a very important option for patients. And we've also begun our rolling submission for the subcutaneous initiation.

That means patients, hopefully, once we have the outcome next year for subcutaneous initiation and we already have it for maintenance, patients could actually have an autoinjector that they take at home. And I think it provides optionality because they can still choose to come to an

infusion center, and we know there are many different segments of patients as well as health care providers.

The other important catalyst in the field that we've seen is that blood-based biomarkers have, as you know, FDA approved the first one, the IVDR, but there are many available today. And I think what it does for the field is it provides the option of being tested. And that has many downstream positive consequences because it can free up neurology chairs, it can free up the reason to have a CSF or a PET. And actually, we've seen data that the number of blood-based biomarker test has gone up exponentially. And there has been an increase in the positive outcomes from CSF and PET, and we think those could be related.

And so it's important.

The other piece from a commercial perspective, as you know, we've launched several drugs in the last few years, all first-in-class, so it's really been a pioneering effort. But I think what we're really pleased about is we're seeing year-on-year revenue growth from our growth products more than offset the decline from our MS product. And this is important because I think the growth products are where we are really focused and it really is looking encouraging at the moment.

The R&D piece that you asked is very dear to my heart, of course, and it's very important because we embarked upon a journey to curate, evaluate the total pipeline, starting from research and going into development. And what we have today is a late-stage high scientific conviction set of programs that we expect registrational data as early as next year. So it's been a very important move because we've gone from low value, low probability of success to high-value, high probability of success. And the reason that we have been able to make that change is we are advancing programs into Phase III and late stage based on a derisking POC data set and that is important. And we are treating internal assets and external innovation the same.

So you've seen us advance litifilimab. You've seen us advance dapi, both for lupus into Phase III, very exciting potential build of a lupus franchise here. But equally, we broadened and acquired HI-Bio. And with that, we have already initiated 3 Phase III trials for felzartamab in the rare nephrology space. And we are evaluating it for several other indications, including a Phase II MVI indication that we think we can start next year.

So it's a very exciting time. We also did more business development with the Stoke Therapeutics. You saw us have an alliance with Stoke on zorevunersen, which we think could be really meaningful and disease-modifying for patients and children with Dravet syndrome. And here, we have seen very exciting data specifically on the Vineland Adaptive Scale. And we -- I have already started our Phase III trial with Stoke and dosed patients.

We also are building our pre-POC pipeline, and I'd like to focus on that next because here, we have some very important readouts coming up next year. We have BIIB080, we have BIIB122, we have BIIB091, as well as we have just announced that we've transitioned an internal asset, which is an IRAK4 degrader. This is coming from our collaboration with C4. So again, a great example of collaboration but starting early. And this is a very important -- IRAK4 is a very important node in the inflammatory pathway.

And we think it could be very meaningful once we get data from our first in-human. We're looking to dose our first patient actually in few weeks now. We think we could get important insights that could bring us into considering several autoimmune conditions, including lupus.

So really, what we have is we are building depth and breadth across our key franchises like Alzheimer's lupus. And with immunology, we're really going broad. We are looking at many aspects in research where we could enhance tissue delivery, but also look at different immunological pathways that could have applications in different system organ classes. So exciting times and looking forward to the next wave of potentially registrational readouts next year.

William Pickering Analyst

Excellent. Well, maybe we can start digging in on LEQEMBI, and congrats on the recent subcu maintenance approval. Maybe talk about why that approval is important and sort of how it derisks the path to the subcu induction approval next year?

Priya Singhal Executive

Yes. it's really important because -- and I'll start with the fact that LEQEMBI is an anti-amyloid therapy, really, we believe the only one with a dual mechanism of action. Because not only does it target aggregated amyloid plaque, but it also targets soluble amyloid species. And with that, we were able to make the case, and we have now in our U.S. label, the need to treat or the option to treat intravenous maintenance, right?

And we've shown data out to 4 years very recently at AAIC, 3 years before, recently 4 years. And what we see is that patients who continue to get LEQEMBI, either once monthly or intravenous or biweekly, they actually differentiate from patients who are being followed in ADNI and other cohorts. So we see that they do tend to decline, but they don't decline as much. And this is very meaningful at that early stage of disease. And so what we did was we were very systematic about it.

And this is, of course, in collaboration and partnership with our partner, Eisai, we submitted for subcutaneous maintenance at a first step and we were able to get approval for that. So this is very exciting because now patients actually have the optionality to transition from intravenous to subcutaneous autoinjector. As I said, it could be at home, it could be in the clinic, and it can transition from intravenous. So you could be on intravenous and then transition to subcutaneous.

We've already shown data on bioequivalence on the 500-milligram, the initiation dose, and that is what we have started our filing. And we -- this is a rolling submission. So -- and it's an sNDA -- sBLA to our subcutaneous filing, maintenance filing, we expect to hear sometime mid next year. And if that comes through, then it will really be exciting because I think it gives the patients and HCPs an opportunity to start at home.

William Pickering Analyst

Great. Great. You also have a tau ASO in Phase II with the CELIA study, which I believe is going to read out in the middle of next year. Maybe could you talk about what you're hoping to see in that study and more broadly, how you see tau fitting into the treatment landscape?

Priya Singhal Executive

Yes. So as we know for Alzheimer's disease, 2 proteins are really pathognomonic and they are thought to be interconnected actually. And we know that tau is important, especially as symptoms initiate right before that. So this is an important area of query, scientific query. And I think that we ourselves have shown that extra cell -- targeting extracellular tau is actually does

not lead to any clinical benefit and even does not impact the tangle, and we now know the tangles to be intracellular.

So the way we've approached this is we've approached this through a modality of antisense oligonucleotide. And that is what BIIB080 is. It's an ASO, it's delivered intrathecally. We did a very small Phase Ib trial. And what we observed in that trial, a small number of patients, but it was an important observation that we were actually able to reduce tau by 50% to 60%.

And we were able to see an impact on tau tangles, which is what tau PET shows, but also tau fluid biomarkers and emerging signs of some clinical benefit. And we have designed the CELIA trial as the important next stage in this development where we can really have the scientific query be answered on if you reduce all form -- all 6 isoforms of tau, what is the impact on tau, both fluid and PET biomarkers, but also is there an impact on clinical benefit. And that is what we hope to see. Now we are testing 3 doses and 2 frequencies. So we have the quarterly but we also have the 6 monthly intrathecal delivery, and that's important.

And we'll see a readout mid next year. So we'll see what the trial shows us.

William Pickering Analyst

Great. Looking forward to it. Any interest in working on a subcu version for that? And maybe speak to what's the feedback from the trial in terms of intrathecal dosing?

Priya Singhal Executive

Yes. The feedback from the trial maybe start there first, was very encouraging because actually, the trial was enrolled quite rapidly. And so we are excited about that aspect. I think that we are definitely looking at tissue delivery mechanisms and more efficient ways of tissue delivery early in research and latent research. So we're looking at shuttles, we're looking at other forms, other modalities.

So it's a very important priority area that we are focusing on.

William Pickering Analyst

Yes. Great. Great. And then for the GLP-1 trial in Alzheimer's. I mean there's some debate on how -- which way that cuts for amyloid targeted therapies.

Maybe you could share any thoughts there?

Priya Singhal Executive

Yes. It's an important area. And I would say that maybe just to back up GLP-1s, I think that the reason -- there were some failures in Phase II. These were different programs. And now this is, of course, Novo Nordisk with EVOKE and EVOKE+ with semaglutide.

And I think what's important here is the hypothesis is that addressing neuro inflammatory pathways could have an impact in Alzheimer's. The hypothesis is based on meta analysis that was debenture of all causes and then further refined and it showed some benefit in the meta-analysis. I think that regardless of the outcome, it is a very important area. Number one, GLP-1s are really being widely prescribed. The fact that these trials are being run for early Alzheimer's disease gives and creates more awareness.

And one of the areas that we have been really thinking forward towards is how does this market expand. And I think market expands with competitors. This is a very large space with newer

patient populations as presymptomatic populations, but also with newer ways in which you could tackle other pathways that may be relevant. So I think that's number one that we think it will be -- it's positive because it's already creating some increased awareness.

Second thing is, I think that we'll see what the data shows. But in their trial, they actually used anti-amyloid as a backbone. And we think that is important and is relevant and is the right approach because we don't expect that the GLP-1 will directly target amyloid. And so even if this is positive, we think this could go into a space of combination therapy where tackling the anti-amyloid -- tackling the amyloid will be important in addition to potentially any benefit that we might see. So we think net-net, it's a positive.

Of course, we have to wait to see the data. And so we look forward to the data.

William Pickering Analyst

Great. Great. And then another trial that there's a lot of interest in is Lilly's TRAILBLAZER-ALZ 3 and sort of what that could mean for your AHEAD 3-45. Maybe if we could just sort of start with key differences in those trials. Why you design your trial the way that you did?

And then if you want to share any comments on what investors should read through when we see those results?

Priya Singhal Executive

Yes, sure. So I think just stepping back, as the world has been focused on longevity, we think that Alzheimer's disease, both early and presymptomatic stages will be really important. In fact, we think presymptomatic is going to be a very large opportunity. It is -- with this sort of data and landscape in mind on how we think this will evolve, that we embarked upon the AHEAD 3-45 trial with Eisai as our collaborators. And we're really trying to address the landmark questions that are relevant for the presymptomatic, preclinical stage of Alzheimer's disease.

Number one, which -- and this is 2 trials, AHEAD 3 and AHEAD 45. AHEAD 3 is aiming to answer the question that if you have 40 centiloids of amyloid in your brain, can we prevent further accumulation because what we know well is that it's a cascade. So amyloid continues to aggregate, then you have tau and then you have symptoms. And so that's the first question. That's AHEAD 3 that has a biomarker endpoint which is further accumulation of amyloid.

So it's amyloid PET.

AHEAD 45 is tackling a slightly different question, which is, okay, you've now accumulated amyloid beyond the 40 centiloids. So you may be at 60, 70, and this can vary at what point patients will have cognitive decline, but can we prevent symptom onset and cognitive decline. So that is how we've designed the 2 trials. That second trial, the 45 is the larger of the two. It is a more important question.

I think kind of to answer because of the way patients are being diagnosed today, and this could change in the future. And that has a primary endpoint that is a sensitive preclinical Alzheimer's disease composite endpoint. It's the PAC5, but it also measures amyloid and biomarker as a secondary endpoint. And I think both are going to be really, really important.

Now with this in mind, when we screen patients, we use amyloid PET, we'll be looking at that at certain frequency. We also incorporated blood-based biomarkers to reduce screen failures and all of these efforts were really successful. And I think what's important here is to be able to

answer those two questions because I think you can think of the presymptomatic population as being stratified. And as we get deeper into blood-based biomarkers, we may have more amyloid leveling biomarkers. We're not there yet but we could have those.

And that would be a very important aspect, and we've already tried to incorporate some of the early aspects of this.

Now the way that our competitor has gone after it is they are looking at can they slow down the progression to the next stage of Alzheimer's. But with that in mind, I think their inclusion criteria ended up including patients who are CDR global score different from sum of boxes. So GS, CDR GS 0.5 and some 1. And that's a big chunk of patients. That means they are already sort of symptomatic.

So I would wait to see, and I'm curious to see what they read out and whether they do a subgroup analysis that answers the true presymptomatic population. So we're looking forward to our AHEAD 3-45 trial. We think that's the trial, and those are the answers that communities and patients and regulators will be looking for.

William Pickering Analyst

Great. Maybe we can move over to SMA, and you've got a couple of sort of efforts ongoing there with the high dose and also with salanersen. So maybe if we could just sort of start with the unmet need you see in SMA today and how that maps to Biogen's strategy there?

Priya Singhal Executive

Yes. I think this is an area of deep pride and success at Biogen, and we believe it's going to be a very important franchise that can endure. And the reason for that is we launched SPINRAZA and had approval in the '16, '17 -- 2016, '17 time frame. It was the first disease-modifying therapy. It is still really, really important today.

But despite the advent of small molecule as well as gene therapy, we know there's a residual high unmet need. This is clear. And there are many important reasons for that. So we have continued to build out what we think is going to be the next important step. And with that, the DEVOTE study for SPINRAZA high dose.

And the reason for that was we went with the right dose we thought was at the time, but we believe based on our pharmacology metrics -- pharmacometrics modeling that a higher dose was possible. And when we did the DEVOTE trial and the pivotal Part B trial where we had symptomatic children with SPINRAZA naive, we saw an important drop in neurofilament and then we saw that translate into motor milestones. And we compare this to a matched control from the standard dose. And in the Part C, we actually transition patients from standard to high dose.

So we believe this was a very important trial. We filed for this. And actually, we were in review. And yesterday, we got a complete response letter. The important aspect here to note is that the complete response letter is not focused on any deficiencies in the clinical data package.

It is actually related to CMC Module 3 and to technical specifications. And the FDA offered us options in their CRL letter to actually resolve them. And the good news here is that we believe we have the data readily available to submit -- resubmit. So this will be a resubmission and we believe that will happen really soon in weeks. And so I'll move on, but I think that the high dose is attempting to really address the high unmet need, outstanding unmet need.

Now I'm going to bridge that because we haven't stopped there. We have a follow-on new molecular entity, also an antisense oligonucleotide, which preclinically we engineered and built with Ionis with higher potency, durability and stability with a potential once year dosing. And we recently actually declared proof of concept based on interim Phase Ib data because we were looking at 2 doses. Now what's important to recognize here is our patient cohort with both the doses, the 40 milligrams and the 80 milligrams for salanersen, as it's called, is really important because we chose in this trial to really go with a high bar. We chose previously treated patients, and then we dose them with one dose of salanersen because it's a once-a-year antisense oligonucleotide.

And we look -- we were looking for neurofilament light chains because we know that, that's to be a reliable biomarker.

But interestingly, we saw really exciting clinical anecdotal data from patients. And I'll just quote one, we have a few, but I'll quote one, which is a child, a 5-year-old, who received gene therapy above the age of 1 could not fit still the age of 5 and then actually received one dose of salanersen and was sitting few months after. So very exciting. We have some several other [vignettes] very similar. And so now we're laser-focused on initiating our Phase III trial.

And I think we'll be able to initiate it in the first part of 2026.

William Pickering Analyst

Excellent. Well, I could ask you many more questions on both of those, but I want to make sure that we have time for lupus and also for felza so maybe we could move over there. In lupus, you have dapi and litifilimab. You gave us a very thorough review of those just a few weeks back. But maybe before we get into the details of each drug, if you want to start by just sort of framing the opportunity in lupus that you see.

Priya Singhal Executive

Yes. I think it's a very critical question. I mean if you step back and think about it, lupus is a very heterogeneous disease. If you saw our webinar, you heard us there saying and patients spoke at the webinar as well. And patients tell each other, my lupus is not your lupus.

That tends to go towards undertreatment and underdiagnosis. And actually, we have a statistic, which is only about 20% of lupus patients are treated with one of the two biologics that are in the marketplace. And while lupus is recognized as an entity and has been for many, many years, there are only two biologics that are available today. And dapi, which is one of our products, and we had a positive Phase III trial, it's the third ever positive Phase III trial in lupus, which is hard to imagine because it's a really important high unmet need, also happens to be underserved populations. So it's a very critical high unmet need.

So that, I think, is the big opportunity. We think this will need to be shaped. But we think that bringing therapies that matter to patients and to -- from our market research to KMEs and to providers will make the difference. So we really believe that this is a very untapped market and bringing the right therapies will matter.

Now we are going after it in two different ways. We have dapi, of course, we're in our second Phase III trial. And then we have litifilimab, which is tackling the type 1 interferon signature and really anti-BDCA2, but we are tackling both CLE and SLE. CLE cutaneous lupus, so we have 3 Phase III trials that are ongoing. And we are not stopping there.

We have a Phase I trial with lupus nephritis with felza. So two late-stage assets, one early-stage asset. And as I mentioned, we just transitioned IRAK4 and lupus is very much within our indication selection possibility for IRAK4 as well. So we're looking at breadth and depth, and we're looking at different mechanisms of action. And this is really based on our conviction that this is a very important market.

Now obviously, it's a large market. We need to expand it and so we're not just waiting for data. We're taking a very different approach. With MS, we have a lot of experience in autoimmunity. We also have a lot of experience that you can have multiple products all of whom could have a significant share.

And we don't think this will be a winner take all. So we are already preparing for our potential lupus readouts, SLE potentially end of next year in '26, and then hopefully, a cadence thereafter of CLE and dapi in years to follow. So we are also working with our medical team, our providers and building the education because these are all different mechanisms of action. And we think lupus may be in the future ripe for sequential combination therapy. So we're really thinking about a lot of these things.

William Pickering Analyst

Great. And I believe your next Phase III for that will be litifilimab next year in SLE?

Priya Singhal Executive

Exactly. We have two Phase IIIs, and we are doing everything we can, and we're excited because we're getting to the close of our recruitment.

William Pickering Analyst

How would you frame what you're hoping to see in those data sets?

Priya Singhal Executive

Well, those data sets, what's exciting about litifilimab is that we published in the New England Journal of Medicine. We published our LILAC study, and we had two parts in that study. We had where we were going for patients who had manifestations of systemic lupus with fever, joint involvement and all of that, and then we have cutaneous lupus. And so really success in all of it, and we think this will be a package. SRI-4 is our primary endpoint for lupus and then the CLASI for our CLE, but we have a lot of secondary endpoints.

So it will be a very robust. Look, with dapi, we're looking at BICLA. And we are also looking at steroid sparing, which today has made its way into the guidelines because patients have a lot of steroids on board, and we are -- and severe flare reduction.

William Pickering Analyst

And for litifilimab, I believe you're testing both the low and the high dose. What is the rationale for that?

Priya Singhal Executive

Yes. So we had one dose in LILAC, but we did a lot of modeling, and we believe that while the low dose will work we want to see if a high dose could work better. We're taking our lessons across drug development and trying to do it all so that we have the right answers at the time of approval.

William Pickering Analyst

Great. And then for your dapi, the second Phase III trial, I believe you've said '27 to '28 for that one?

Priya Singhal Executive

That's right. That's right.

William Pickering Analyst

And I noticed that you upsized that trial compared to the first Phase III. What were some of the considerations that went into that?

Priya Singhal Executive

Yes. I mean, we're very excited about the data that we had from our first Phase III. And I think this is just making sure that we leave no stone unturned from having the right-sized safety database. It is a new molecule, and so we want to have that.

William Pickering Analyst

Great. And it's an interesting market because on the one hand, as you said, there's only 2 approved drugs. There's limited uptake of those. On the other hand, it's fairly crowded in terms of the development landscape. And so fast forward a few years, hopefully, at least one of your drugs is on the market, and perhaps we'll see some competitors on the market as well.

What would you expect to be the key differentiators for Biogen and lupus?

Priya Singhal Executive

Yes. I'm glad you brought up the point about being crowded competitive landscape. It's an area where it's hard to recruit. And I'm really proud of what the teams are doing because we've managed to do a really spectacular job despite COVID and all the geopolitical issues and the competitive landscape. And I think that bodes well.

That's helpful, right? It bodes well. The other piece I think that's important to remember here is that this is not a winner takes all. So we think the market will expand. We think many therapies will be needed, and we think it will be like MS eventually, where there will be different mechanisms of action where patients may respond to one, not respond to the other.

For example, with dapi, we recently shared data on fatigue. And what we've heard from patients is that you're going after all these other endpoints, but what I can really deal with is fatigue. And we showed that we can really reduce that.

So now it's another question of getting all of these things discussed with regulators and getting it into labels, but I think these are very, very much differentiated. With SLE and CLE for litifilimab, they're going after type 1 interferon and anti-BDCA2. And we think that, for example, with CLE, we think it's really critical. It's a huge market. No real therapies out there for 70 years.

And steroids that are the foundation of all these lupus manifestations are now in the guidelines as being steroid like you have to get your patients off. So the patients when you ask them, they are looking to get off steroids. KME's prescribers are looking to get their patients off but they need an alternative that can match it on the endpoints and the treatment. And I think that will be a differentiator.

William Pickering Analyst

Great. Maybe we'll move to felza and you're pursuing several different indications here, AMR, IgAN, PMN, lupus nephritis -- in one sense, they're all rare kidney but they're also different in terms of unmet need, competition, so on. And so how do you think about the role that felza could play in those? And maybe your relative excitement across the 3 -- the 4 at this point?

Priya Singhal Executive

Yes. Actually, more than 4 because MVI that we'll start next year. So it's very exciting. And we're looking at several other indications. So I think felza is really exciting space.

And the way we've integrated the company, very exciting, really exciting team that's leading that forward and we are excited because we started all 3 Phase IIIs. That was a goal for '25 and we were done with initiation. So it's very exciting.

Now let me start with the fact that we believe that all these are 3 are relevant indications where we've shown proof of concept. So it goes back to my overarching principle that we don't take things into Phase III until we show proof of concept. So we've derisked them to a great degree. I'll start with AMR, where we could get data as early as 2027. And that's going to be exciting because today, if you think about patients and if you think about a societal burden, 75% of patients lose their kidney, they end up getting a rejection due to late AMR.

And that is exactly where we saw transformational efficacy in a small trial, but really transformational efficacy. And we believe that this is going to be really important and is driven by the anti-CD38 sort of auto antibody play, and we think that's going to be really relevant. So I think that from an AMR perspective, it's very exciting. We think that it's going to be important because the CD38 that's expressed on plasma cells, but we also think natural killer cells have a very important role to play, which may have been the reason some other products have failed because they weren't really targeting the key cell types that are relevant for this very important condition.

Moving on to IgAN, you're right, it's very crowded. But I think what the beauty of IgAN and the [MOA] here is, it's this [for] hit hypothesis, where we think it's galactose-deficient IgA that's relevant for the mechanism. It's the autoantibodies to that, they form the immune complexes and they get settled in the kidney. And what we saw that was interesting to us, even when we did the diligence and of course, the Hi-Bio team is published on this. What we saw is that the proteinuria, the IgA reduction coincides with the proteinuria and impact on eGFR.

And we saw the durability 18 months after the 9 dose 5-month regimen was dosed. Now it was a small trial, right, because many doses were tried, but we see that there's stabilization versus placebo. And that's very important because there's a potential based on market research that we've done, there's quite a significant potential on patients being able to have a nonchronic therapy. And so we think that's going to be relevant while maintaining the efficacy relevant to the APRIL and the BAFFs. So we -- of course, it's an area of deep continuous education and all of that, and that is why we have really stepped up on our premarket approval, education and efforts on immunology.

And that encompasses rare kidney but also lupus and all of that.

William Pickering Analyst

Great. Well, you preempted my question on AMR and why CD38 is a better mechanism. But maybe I'll move over to IgAN. And you get some questions around, well, the UPCR stays depressed, but then it looks like the GFR is starting to trend lower a little bit. What gives you kind of the confidence that it's the UPCR that, that signal is what you should rely on.

And strategically, you're comfortable going forward with this dosing holiday strategy?

Priya Singhal Executive

Yes. I think the first and foremost is, I'll just say the main thing is that we saw the reduction in IgA and that coincided with the reduction in proteinuria and eGFR. But the other piece here is that post dosing, we saw the stabilization. So the stabilization gives us quite a bit of confidence. We always have the opportunity to explore other paradigms.

But I think that this was a significant time of drug and that was quite compelling. And we've also done a lot of market research, and that is the response that we also get from those angles.

William Pickering Analyst

Great. And then the PMS. We haven't talked about that one yet.

Priya Singhal Executive

Very important. I mean I think what we've seen is that the refractory patients, the relapsed patients do really well. And now the way we've designed our Phase III is that we think that the high-risk patients with the high levels of circulating PLA to our antibodies, but also patients who don't have that can potentially benefit. So we are expanding that base. We also know that more than 1/3 of the patients today do not respond to the anti-CD20.

So we think this is a very large opportunity, 36,000 patients in the U.S. and 1/3 of those do not respond to anti-CD20s. So that is the way we've designed the trial, and we have confidence that we're going to see this movement and response in this broader population. So we are really going after the broader population, not just the refractory and the relapsed population. Also treatment-naive, high-risk population.

William Pickering Analyst

Got it. And then IRAK4, you've made -- you've mentioned this a few times. Maybe let's just give it this kind of center stage if you want to talk about that program and what the development plan would be for that?

Priya Singhal Executive

Yes. Right now, I think we're very excited. IND is accepted. We're about to dose our first patient. So we are very excited.

We're moving very fast. The first in-human trial, which will be normal healthy volunteers will give us the right data set to really expand. And I think that Biogen as it looks at expanding into immunology is looking, and we're doing with felzartamab, you will see us considering the portfolio in a pill more and more. And IRAK4, I think, will peak center stage as we kind of contemplate that. But more than contemplate because we've already started building out what a potential Phase II program could look like.

And that's the beauty, I think, of targeting such a critical node in the inflammatory pathway like IRAK4, that it gives us the opportunity. So we'll have the good problem of thinking of choices.

And we may choose more than one like we have with felzartamab.

William Pickering Analyst

Great. Well, we look forward to learning more on that one. And we've covered a lot of different programs today. Would you like to maybe share some concluding thoughts on how all of these fit together into the kind of the coherent Biogen R&D strategy?

Priya Singhal Executive

Yes. First, thanks so much, Will, for great questions here. I think stepping back Biogen is at a very different point in many, many ways. One is our new launches are outpacing the decline of the MS portfolio, which is obviously a question and -- so we think we are addressing that really well. The Alzheimer's franchise continuing to grow and we believe we have a differentiated asset, we think we're in the early stages.

We are continuing to build a lot of depth in our lupus franchise. And as I spoke to, we have multiple shots and multiple mechanisms of action that we're targeting. Felzartamab is bringing our immunology, rare nephrology into the sort of forefront and we are already enrolled in all the Phase III trials. And we continue to build on our expertise in neurology. You saw that with salanersen but also with Zorevunersen that has the potential to read out in 2027 in Dravet syndrome.

So very exciting. And we have just changed the shape, nature, look and confidence in the aggregate profile of the pipeline today, but also the individual programs. So any one of the individual programs, we believe is very high value, potentially $1 billion or more. But if more than one comes to fruition, obviously, that exponentially increases the value of the pipeline. And we remain focused and disciplined on augmenting the pipeline with external and innovation -- internal innovation with the research, reinvigoration and reimagination of open innovation.

So we're really tackling it from all sides, while being very, very careful and disciplined about cost. So I think I'm really excited about what we've been able to do and what the future could bring.

William Pickering Analyst

Wonderful. Thank you so much for coming.

Priya Singhal Executive