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EDITED TRANSCRIPT

REGN.OQ - Regeneron Pharmaceuticals Inc at JPMorgan Healthcare Conference

EVENT DATE/TIME: JANUARY 13, 2025 / 10:15PM GMT

OVERVIEW:

Company Summary

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PRESENTATION

Christopher Schott - JPMorgan - Analyst

Good afternoon, everybody. I'm Chris Schott from JPMorgan, and it's my pleasure to be introducing Regeneron today.

From the company, we have Regeneron's Co-founders and Co-Chairs, President, and CEO, Len Schleifer; as well as President and CSO, George Yancopoulos, with a lot of updates on the core business and actually particularly the pipeline, very much looking forward to the presentation today. So we're going to have Len and George make a presentation, and then we'll have a little fireside chat after that.

So with that, over to Len.

Leonard Schleifer - Regeneron Pharmaceuticals Inc - Co-Chairman of the Board, President, Chief Executive Officer, Founder

Thank you. Thank you, Chris, for that very detailed and warm introduction. Let's see if I can get the slides going here. So this is our forward-looking statement slide. And while you're looking at that, I thought it might make sense for me to make a few backward-looking statements.

I, as perhaps the longest-serving CEO in the biotechnology industry and, along with George, part of the longest-serving management team in the biotechnology industry, I've been speaking at this conference for more than three decades.

And while many things have changed, there have been different themes, is it a platform, is it small molecules, is it RNA, is it vaccines, is it biologics. That rotates, it changes. There's one constant. And the constant is investors always want to know, what is that one thing that your company has that should make us want to invest?

And in the early days of Regeneron, I answered that question in sort of an obtuse way. I said, well, it's our highly differentiated science. It's our technology that George and his team are developing. Because if you want to develop breakthrough drugs, you need to spend the time and effort to develop breakthrough technologies and understand the science. And I always felt that it wasn't a matter of if, it was a matter of when and how much it would cost.

I would tell that to investors. I'd tell that to the, I think, five JPMorgan analyst before Chris. And usually, that would get us a slot at next year's conference on Thursday afternoon at 4:00 PM in a small room when the conference ended at 3:00 PM. At any rate -- but we kept giving that answer.

And then fast forward to the 2000s, same question, what is that one thing? And once again, we didn't have one thing. I said, well, all that technology that I told you, George and the gang were busy inventing and discovering and building, well, it's going to deliver this new class of drug, the so-called receptor traps. These things that could -- based on the specificity of receptor, could grab on to a ligand, block it.

And we had a whole slew of them. I didn't know exactly which one was going to make it, but that was it. Nobody was particularly satisfied with that answer either.

When it came to 2010 and I actually told them which one we thought, which trap, the VEGF Trap was going to be the big one, they didn't believe us either, and that, of course, became EYLEA.

Fast forward to the teens and people ask, well, what is that one thing? And once again, what I would say and what George would say, well, it's not one thing. It's the technology we're working on. It's VelocImmune, a special mouse that can create human antibodies, and we didn't know which one was going to be the great one, but we were sure one of them was going to be it. Nobody really believed us.

We tried to convince them eventually by 2016 that there was a drug that was going to be -- actually, we coined the phrase, I think -- I don't know if we're the first to use it, but we've certainly popularized it. There was going to be a pipeline in a single product. It didn't get much traction there either. That turned out to be DUPIXENT.

I will skip over the part of the story where some people did believe us during the pandemic, where we treated some people down in Washington, saved a few lives with REGEN-COV.

But here we are, again, in 2025, lots of technological breakthroughs, more than 10 approved drugs, yet same question, what is that one thing, what is that one thing that why should we invest, what is it that people are missing, so on and so forth. And we have an answer. I am going to tell you, hang on. The answer is, it's the pipeline. That's what people are missing, and that's where we want to spend most of our time today.

So what I'm going to do is I'm going to rip through some of the stuff that normally a company would talk about, our marketed products. We do have four marketed blockbuster products: EYLEA, EYLEA HD, DUPIXENT, and LIBTAYO now. These can drive our near-term market success. But advancing our differentiated pipeline is really, really where we think we're going to make the big hay for the future.

DUPIXENT, just so I don't feel like I haven't -- like I've ignored a drug which has got over 1 million people being treated. It's annualizing, I think, at a rather high rate somewhere in the above $14 billion. It truly was a pipeline in a product. We developed -- we discovered that at Regeneron.

George discovered that with his team, developed it, and commercialize it with Sanofi. And it turns out to be a true pipeline in a product that continues to get even more indications pending and a launch with COPD.

Skipping over to EYLEA. EYLEA net sales in 2024 were about $6 billion, up about 1%. We accomplished some switching to EYLEA HD, perhaps not as much as we would've liked. Our marketing team, I think, is really expert. We know the retinal community very well, but we didn't have all the tools necessary to optimize HD.

And that included: we needed to get the prefilled syringe. Put a check on that. We submitted that to the FDA late last year. We expect to get that approved by middle of the year, we didn't have the Q4 week dosing. Put a check on that. We'll be submitting that to the FDA this quarter. We didn't have the long-term, two-year data. Put a check on that. We'll get that in April. We didn't have RVO, submitting that this quarter.

So we have a lot more tools to optimize HD, but EYLEA continues to be a favorite product among retinal specialists despite the fact that there is quite a bit of competition out there.

LIBTAYO has turned out to be another blockbuster, another $1 billion drug. And we had some exciting data today in advanced cutaneous squamous cell carcinoma that George will get through in a minute.

What I want to do is just quickly run through the pipeline because I said it's the pipeline. I promised everybody on my IR team that I wouldn't say it's the pipeline, dummy, so I won't say that, but I say it to myself. So we remind ourselves it is the pipeline.

Let's just take a quick look. We have eosinophilic COPD. The first and only approved drug for COPD is DUPIXENT. We have a follow-on drug also in COPD, itepekimab, data later this year. We just announced the first and only immunotherapy to work in the adjuvant CSCC setting.

That was an upside surprise despite the fact that Keytruda failed in that setting. We have our drug LAG-3 that's getting some data in metastatic melanoma. We have our myeloma and lymphoma programs, which I think George will convince you that they're potential to be best-in-class.

We're also best-in-class approach to complement-mediated diseases. We think our anticoagulant approach is going to address a very large market opportunity. More obesity data to come later this year, which George will get into, as well as some exciting initial data in food allergy.

All of these are just 25%. These drugs represent only 25% of our drugs that are in clinical development, and we are putting more and more in every year. We expect these drugs to be able to compete in market sizes, the overall market opportunity of over a couple of hundred billion dollars.

So yes, we have four blockbusters. Yes, we have work to do yet with EYLEA and EYLEA HD. Yes, there's still lots of upside to be had from DUPIXENT. But I really believe it is the differentiated pipeline that comes from the differentiated technologies that George and the gang has -- have created and continue to create.

And so I'm going to stop and turn it over to George and ask him to talk to you in more detail about that pipeline.

George Yancopoulos - Regeneron Pharmaceuticals Inc - Co-Chairman of the Board, President, Chief Scientific Officer

Thanks, Len. And just to emphasize again, that's just tip of the iceberg, 10 exciting programs from our pipeline, which is what we have the time to talk about today, but there's a lot more behind that as well.

But as Len said, one of the things that we're proudest of at Regeneron is that we have a long history of being at the bleeding edge of science and technology. And I think what's remarkable is all these years later, I think we believe we are still leading the way in the industry.

And what we do is we start, as Len said, by focusing on the best-in-class technology, that is technological breakthroughs that will yield what we call innovative turnkey platforms that can repeatedly deliver important, new practice-changing medicines as evidenced by delivering over a dozen authorized or approved medicines, the four blockbusters and one of the largest and most innovative pipelines all coming from our own labs or a small number of key collaborations with important partners.

Early on, we were pioneers in creating the field of soluble receptors, as Len mentioned, and that led to EYLEA and other important medicines. We then, as Len mentioned, developed the world's premier human antibody technologies involving our VelocImmune mouse and our Veloci-Bi platforms which delivered, as Len mentioned, DUPIXENT, multiple other important medicines and are still delivering much of the pipeline Len previewed, including one of the world's leading bispecific portfolios.

And over the last years, we've become leaders in high-throughput human DNA sequencing, allowing us to build the world's largest DNA sequence-linked health care database, which is providing invaluable for drug discovery and development and which we believe can help us contribute to revolutionizing the field of health care analytics and management.

While existing platforms continue to generate opportunities, we are already implementing our next generation of technological breakthroughs, which we feel could further transform how certain diseases are treated or even cured, whether it be through silencing pathological genes in the brain, combining antibodies with siRNAs, combining antibodies and bispecifics, or using CRISPR-based gene editing to correct gene diseases.

So today, I'd like to highlight some of these top efforts, most of the ones that Len mentioned, as well as their potential to really shift paradigm -- treatment paradigms for millions of people.

So this year, we anticipate reporting on important milestones from a number of these efforts across diverse therapeutic areas, including, as Len mentioned, in COPD, cancer, obesity, as well as from our anticoagulation programs and other genetic medicines programs.

Let me just start with our next potential advance for COPD. As Len discussed, DUPIXENT was recently approved as the first-ever biologic to treat patients with inadequately controlled eosinophilic COPD. But we haven't stopped there.

Our Regeneron Genetic Center, using our world's leading database, found that interleukin-33 is genetically linked to COPD. And thus, we used our VelocImmune technology to make a potentially best-in-class IL-33 blocker, itepekimab.

While this antibody showed an overall reduction in COPD exacerbations in our Phase 2 trial, post hoc analysis revealed that this reduction was largely driven by a profound 42% reduction in the former smokers' population.

So our Phase 3 trials, which are being done in collaboration with Sanofi, are in former smokers with COPD. It passed an important interim futility analysis, and we expect pivotal results in this program by the second half of this year.

Moving to cancer. Our overall strategy is focused on using the immune system to fight cancer. We have five different classes of immune modulatory agents, all of them discovered and developed by Regeneron. Each class modulates the immune system in a different way, enabling novel combinations that could substantially enhance activity.

And we have clinically validated the first three classes, several showing potentially best-in-class efficacy in their cancer settings that they've been tested in. And we have also early validation of some of the combinatorial opportunities.

I'm going to first talk about clinical data with our checkpoint inhibitors including our PD-1 blocking antibody, LIBTAYO, which as Len mentioned, is already on blockbuster rate, as well as our clinical stage LAG-3 blocking antibody fianlimab, both of which now are demonstrating best-in-class potential. Then I'll discuss two of our CD3 bispecifics.

I remind you, as Len said, Regeneron was again a pioneer in this field by being the first to treat patients with fully human, full-length CD3 bispecifics. And I'll share clinical data suggesting that our CD3 bispecifics have once again best-in-class potential.

And I have time to only briefly discuss our CD28 costimulatory bispecific, which is another class of therapeutics that Regeneron was the first to introduce to the world and which are at earlier stages of clinical development.

This slide highlights that our immuno-oncology efforts were prospectively designed so that the various individual agents could be logically combined to optimize and tailor the right combination for the right tumor, resulting in an incredibly broad and multifaceted portfolio of combinations. And we're showing this growing portfolio of both monotherapies, but also very important combination opportunities, each of which can be targeted to the right cancer.

And to show that we continue to deliver first in this immuno-oncology space with our approaches, with today's announcement, we showed the first positive Phase 3 data for LIBTAYO in the adjuvant cutaneous squamous cell carcinoma setting. This is the first time an immunotherapy has been shown to show benefit in this population.

I remind you that, as Len mentioned, Keytruda failed in this setting, showing once again that even antibodies against the same target do not always have the same efficacy in all settings. In this study, at the first prespecified interim analysis, adjuvant LIBTAYO demonstrated a 68% reduction in recurrence and death, overwhelmingly positive data.

I also remind you that LIBTAYO is the world's leading treatment for non-melanoma skin cancers in the more advanced setting, and this new data in the adjuvant setting should certainly support that position. And we look forward to submitting these results shortly to global regulatory authorities.

Moving on to checkpoint combinations. That was monotherapy now combinations. And moving on to first-line melanoma, which, so far, Regeneron has not been leaders in. Well, ever since the exciting early data with individual checkpoint inhibitors more than a decade ago, particularly in melanoma, it was widely hypothesized that combining two classes of such checkpoint inhibitors might meaningfully enhance anticancer benefit without exacerbating safety issues. I think we can all agree that progress to date has been somewhat disappointing.

But now, our early clinical data in metastatic melanoma suggests that fianlimab plus LIBTAYO might be the first checkpoint inhibitor combination to really deliver on the early hope for such combinations based on our proof-of-concept data, which was consistently replicated across three

independent first-line melanoma cohorts, showing notably higher overall response rates and complete response rates [and] much longer progression-free survival than other PD-1 monotherapies and even in other approved combinations, including the other competing PD-1/LAG-3 combo.

In addition to our ongoing Phase 3 trial in metastatic melanoma, which should read out in the second half of this year, we are also exploring this exciting combination in a variety of other cancer settings with interim Phase 2 data in lung cancer in the first half of this year.

As in the example I just presented, checkpoint inhibitors are providing significant breakthroughs in certain types of cancers. But many other cancers are so-called cold. They do not respond to these inhibitors. For example, prostate cancer.

Regeneron's CD28 costimulatory bispecific platform is designed to produce significant antitumor activity with checkpoint inhibitors in these cold tumors, and we have also shown that they can enhance the activity of CD3 bispecifics.

And indeed, our first-in-human trials of our prostate-specific costimulatory bispecific in combination with our PD-1 antibody showed dramatic activity in late-line prostate cancer patients normally unresponsive to PD-1 blockade with 75% of the late-line patients tested at the highest dose showing rapid and dramatic reductions in PSA, though these responses were complicated by severe immune-mediated adverse events.

And we are working to mitigate these safety concerns in the combination treatment, but we're also prioritizing new combinations with CD3 bispecifics, where the science suggests that we can also enhance efficacy by adding in the CD28 costimulatory bispecifics, but with less immune-mediated adverse concerns. And we'll be updating these exciting innovative programs later this year.

Now moving on to our recent progress with our next class of immunomodulatory agents, the CD3 bispecifics. And I want to talk about Ordspono, our CD20xCD3 bispecific, which was recently approved in Europe for certain lymphomas and for which we anticipate resubmitting our BLA in the first quarter and linvoseltamab, our BCMAxCD3 bispecific myeloma, which we recently resubmitted to the -- for BLA.

Very importantly, and as indicated in the highlighted data boxes in the slide, both of these bispecifics have demonstrated potentially best-in-class data in the late lines for which we are seeking approval. Given these compelling profiles in the late line settings, it gives us the confidence and opportunity, which others are not even considering right now, that is using each of these agents as monotherapies or in very limited and safer combinations in the first-line settings. These approaches have the real potential to be practice-changing in terms of decreasing the substantial treatment and toxicity burden for patients undergoing multitherapy regimens.

Our Ordspono Phase 3 program is efficiently progressing in earlier lines. And as example of what I just said, while competing CD20 bispecifics are evaluating complex combinations in first-line follicular lymphoma, as highlighted on the slide here, our Phase 3 OLYMPIA-1 study is evaluating Ordspono monotherapy directly compared to complex combinations. And recent data from the safety lead-in portion of this confirmatory Phase 3 OLYMPIA-1 trial supports the notion that this could really work as a monotherapy.

We recently presented this data in ASH that shows, as in the highlighted data box, a 100% complete response rate, 12 of the first 12 first-line follicular lymphoma patients achieved complete responses, supporting the excitement and confidence we have about Ordspono monotherapy in this setting.

As a reminder, the standard of care for this setting, rituximab plus multiple chemotherapy agents, has historically achieved complete responses in about 67% of patients. And it's quite a toxic regimen. We look forward to seeing the results of this Phase 3 trial, which offers the first head-to-head evaluation of Ordspono monotherapy compared to this standard of care.

Moving on to our BCMA bispecific, which you'll see has many parallels, in that linvoseltamab is emerging with a differentiated and compelling clinical profile in relapsed, refractory, late-stage multiple myeloma patients. Compared to other BCMA bispecifics, linvoseltamab has almost double the rate of complete responses at similar times of follow-up with a favorable profile in terms of safety, dosing and hospitalization burden.

Just as with Ordspono, these compelling data in late line underpin our confidence to pursue linvoseltamab in earlier lines of therapy as monotherapy or in very limited combinations, which is indicated in this slide.

And furthermore, we're also exploring premalignant conditions, such as smoldering myeloma and even monoclonal gammopathy of unknown -- of undetermined significance, or MGUS, in an attempt to prevent progression to myeloma by eliminating the threat at these early stages.

Moving now to a totally different area, our rapidly advancing anticoagulation efforts, particularly with Factor XI, which involves a two-pronged approach in anticoagulation that offers potential, once again, for improved blood clot prevention and lower bleeding risk.

We believe our approach, supported by genetic data from our Regeneron Genetics Center, has delivered two antibodies with very distinct and unique profiles that may meet very different market needs. Our first antibody targets the catalytic domain of Factor XI, potentially providing improved deficit compared to the standard of care options and intended for patients who need more effective anticoagulation.

On the other hand, there's a very large population that is not taking anticoagulation therapy even though they could benefit from it because they suffer from severe bleeding risk, and our second antibody is designed to provide anti-coagulate benefit but with minimal bleeding risk.

With our recently announced and positive Phase 2 data for both these antibodies in prevention of venous thromboembolism following total knee replacement, we now have clinical data that supports our prior preclinical data supporting our approach.

On the left side of the slide, you see preclinical data comparing our antibodies to competitor antibodies and small molecules for their anticoagulation activity, showing that our catalytic antibody has, by far, the strongest anticoagulation activity while our other A2 antibody demonstrated similar anticoagulation activity to competitors but designed to be potentially safer in terms of bleeding risk.

And on the right side of the slide, our initial Phase 2 clinical data for both the antibodies did, in fact, validate their robust antithrombotic benefits in patients with a catalytic antibody, reducing clotting more strongly, just as expected.

So these data support our approach to advancing both antibodies into broad pivotal programs in multiple indications and patient types with the thinking that these two antibodies can address two sets of different populations or multiple sets of different populations.

Now moving to, once again, a whole different area from anticoagulation -- from cancer to anticoagulation to obesity now, where we have several early programs and have developed an extensive pipeline of preclinical assets.

What we're initially hoping to address are certain issues with GLP-1-based therapy such as semaglutide, which have certainly transformed the management of obesity, but there is increasing awareness that the quality of weight loss from these medicines is suboptimal, where up to 30% to 40% of the weight loss is due to loss in muscle.

Moreover, real-world evidence suggests that there's a lot of patients going off the drugs, as most do within a year according to the real-world analysis. Once they go off the [drug], they rapidly regain the weight entirely as fat. So cycling on and off these treatments could result in profoundly negative consequences in body composition over time.

We already have clinically validated antibodies that human trials have been shown to preserve and grow muscle. And as shown on this slide, our nonhuman primate studies show that when these muscle preservation agents are combined with semaglutide, they can improve the quality of weight loss, resulting in more fat loss while either eliminating muscle loss or even actually increasing muscle [mass].

So we're very excited about combining these muscle preservation approaches with the GLP-1 class of agents, as is being done in our Phase 2 trial, which is fully enrolled, and we look forward to reporting initial data from this trial in the second half of this year.

Regeneron has been focused for a long period of time, many years, on the study of muscle growth factors in the system and genetics as well. And we're now leveraging this expertise and our growing existing capabilities to employ more broadly these source of approaches in the rapidly growing obesity market.

As I said, the first wave of our innovations are focused on improving GLP1-based weight loss by preserving muscle. However, we believe obesity is a very complex disease. There should be a variety of additional types of approaches.

And our next wave of innovations are focused on GLP1-independent mechanisms, and they're very much focused on using muscle growth and increasing muscle metabolism to combat obesity and metabolic disorders. And we aim to move these assets into clinic over the coming years.

As I said before, an important aspect of our portfolio includes opportunities to combine our series of novel assets, as I've already shown you in cancer. Another such combo opportunity involves combining our BCMA bispec initially developed for myeloma cancer patients with DUPIXENT, which, as you've already heard, addresses many allergic diseases, but applying this combination in severe allergy.

It turns out that while DUPIXENT is now approved globally in seven different allergy-related indications, from atopic dermatitis to asthma, the leader in all of these indications, it does not acutely reverse severe allergies themselves. This is because the major driver of severe allergies is high levels of immunoglobulin class known as IgE. I always say the E is for evil.

And while our clinical data shows that DUPIXENT entirely prevents the generation of new IgE-producing plasma cells, it does not affect existing IgE-producing cells. Well, our preclinical data, as well as our findings in the myeloma cancer patients, demonstrated that acute treatment with our BCMA bispecific, which kills antibody-producing plasma cells, can rapidly deplete the allergy-causing IgE.

However, upon stopping the BCMA bispecific, the IgE cells are quickly regenerated. But as I just told you, we have independent clinical data showing that DUPIXENT prevents generation of new IgE-producing cells.

So we thought about putting the two together. These independent realizations led to the hypothesis that we might be able to durably reverse severe allergies with acute short-term treatment with our BCMA bispecific antibody to eliminate the IgE-producing cells, combined with continuous DUPIXENT to prevent their return.

Today, we are sharing on this slide the initial clinical data from the treatment of our first severe food allergy patient, treated acutely with our BCMA bispecific together with ongoing DUPIXENT, which entirely validates the hypothesis, by putting these together, you can demonstrate rapid and profound reduction of about 90% even with the short-term treatment at very low doses in both total and food specific IgE levels.

And so we're very excited about the future applications and developments in this area as a potential way to reverse and cure severe allergies of all sorts, including severe food allergies.

Turning now to our Regeneron Genetics Medicines programs, which include our Regeneron Genetics Center that has built the world's largest DNA sequence-linked health care database, which enables target drug discovery and validation and provides new genetic targets for disease.

Today, we announced a major expansion of these efforts in collaboration with Truveta. Our RGM efforts also include a series of turnkey genetic-based therapeutic platforms that we can apply to these genetic targets.

We either built these platforms in-house or developed them in collaboration with key partners, such as Alnylam or Intellia. And this leaves us uniquely positioned in the entire industry to pick the right genetics-based modality that is siRNA, CRISPR or gene therapy for the particular disease setting and even combine them in some cases with our biologics.

We have already demonstrated several important clinical first with these various genetic medicines-based approaches, from demonstrating the first silencing of a pathological gene in the brain with our partners at Alnylam to progressing the first CRISPR-based therapy to Phase 3 in collaboration with Intellia.

To update you on another important recent advance from our RGM efforts and representing one of the most remarkable successes in the history of the gene therapy field in humans, a onetime gene therapy treatment that enables hearing in children born with profound genetic deafness.

As we present here for the first time on the left side of the slide, 10 of 11 treated ears in previously genetically deaf children had a notable increase in hearing several to the normal hearing range. We look forward on continuing these studies and sharing additional data this year.

And our differentiated approaches in genetic medicines also allow us to combine them not only with each other but to combine them with biologics where we've had our long-standing leadership position. It uniquely positions us to explore unprecedented combinations of genetic medicines with biologics. And one exciting, first-of-its-kind combination is that of an siRNA with an antibody that both target the same protein, in this case, the complement protein C5.

The siRNA is designed to markedly decrease C5 production by the liver, allowing the antibody to more completely and durably block C5 function. This approach could improve treatment in multiple settings where C5 blockers have been approved, and we are testing this innovative combo in generalized myasthenia gravis, geographic atrophy and dry age-related macular degeneration, and paroxysmal nocturnal hemoglobinuria, or PNH, with notable progress and milestones coming this year.

And this slide provides data supporting our confidence in this combination. At last month's ASH conference, we presented data from the lead-in cohort from our ongoing Phase 3 trial in PNH in which we studied our siRNA antibody combo compared to the standard of care ravulizumab.

As shown on the slide, not only did our novel combo decrease the key biomarker of disease activity LDH more than the standard of care, but was the first C5-targeting approach to ever demonstrate lowering this measure of disease activity to the normal rate.

Moreover, when we took the patients treated with the standard of care ravulizumab and whose average resulting biomarkers were left at 1.2 times the upper limit of normal and then switch them to our combo, we were able to rapidly and durably move their measure of disease activity once again to the normal range, another very exciting opportunity.

Beyond these examples, we have a very deep and rich genetic medicines pipeline. I briefly mentioned that with our partners at Alnylam, we, for the first time, demonstrated silencing of a pathological gene in the brain.

Following up on this, we are particularly excited about two programs that follow up on that initial observation, both of which will be entering the clinic this year, that have the opportunity to be disease-modifying for the neurodegenerative diseases that is an siRNA targeting the tau target in Alzheimer's and the synuclein target for Parkinson's.

And of course, over the year, we expect the reporting on many additional key milestones. So as Len said and as I echo, we are incredibly excited about the future and the pipeline, and I've only been able to introduce you to the tip of the iceberg of the pipeline.

But I guess maybe we have a few -- a little time for questions. Thank you.

QUESTIONS AND ANSWERS

Christopher Schott - JPMorgan - Analyst

Well, great. Thanks for the comments. I'm just kick off with a couple here. Maybe just starting on EYLEA HD. Can you just update us latest thinking of how that launch is progressing and just help us a little bit bridge where the 3Q results came in and what we saw from the 4Q pre-announcement today?

Leonard Schleifer - Regeneron Pharmaceuticals Inc - Co-Chairman of the Board, President, Chief Executive Officer, Founder

I'd be happy to do that, Chris, but I would have been happier to talk about the pipeline.

Christopher Schott - JPMorgan - Analyst

I have to get one in on EYLEA first.

Leonard Schleifer - Regeneron Pharmaceuticals Inc - Co-Chairman of the Board, President, Chief Executive Officer, Founder

I think that EYLEA HD is a big advance in our minds over EYLEA in terms of the durability of the result. But I think that it needs a few more arrows in its quiver. We need to get the prefilled syringe, which we submitted at the end of last year. I hope to get that approved by the middle of the year. It needs Q4 week dosing, which we'll be submitting in the first quarter.

It needed some two-year data, which we have a PDUFA date in April, and it needed RVO, which we'll submit this quarter. So there's a lot we can do this year to strengthen that product profile, and we hope to -- that more and more people will take it up.

Christopher Schott - JPMorgan - Analyst

Perfect. Maybe pivoting over to the pipeline. LAG-3, we're getting the melanoma data this year. I guess what do you think has enabled this much higher rate of efficacy that we seem to be seeing relative to the existing agent on the market as you think about the profile here?

George Yancopoulos - Regeneron Pharmaceuticals Inc - Co-Chairman of the Board, President, Chief Scientific Officer

Well, as both Len and I tried to communicate, not every agent against the same target operates the same way. Let me just all remind you that some of the biggest companies in the world tried to make an antibody like DUPIXENT using inferior technology to our VelocImmune technology, and they failed across all of their studies, okay?

We just showed you today the results of an incredible study in adjuvant CSCC, where the leading competitor failed once again. So it's not unusual that antibodies against the same target behave differently. Let me also remind you, nivolumab, the first PD-1, failed in first-line lung cancer whereas Keytruda and, of course, LIBTAYO now has the best data in the field there. So not all agents against the same target are the same.

One thing that Regeneron consistently does is we develop the best-in-class technologies that we've now demonstrated across all of our history and programs to achieve the best-in-class efficacy, and that is such a differentiator. Remember, we did it with our soluble receptor technology, delivering EYLEA.

And we've now done it with our antibody technology, delivering so many best-in-class antibodies where others completely failed. We think the same may be the case in LAG-3 there, but it goes on more to the bispecifics. We believe we've generated the world's first and best bispecific technology, and that's why we're achieving best-in-class data.

So I think, hopefully, you got from my presentation that in many of our programs, there's a lot of competition in the field, but we reproducibly seem to be able, because of this focus on deep underlying technologies, are delivering the best-in-class agents in almost all cases.

Leonard Schleifer - Regeneron Pharmaceuticals Inc - Co-Chairman of the Board, President, Chief Executive Officer, Founder

And I think just to briefly add to that, as George tried to show in his talk, not only do we have the best antibodies, but we have the best repertoire for potential combinations that many other companies don't have. They may have a one-off antibody here or one-off there. You saw a whole panoply of different antibodies that can be combined in unique ways under one roof for Regeneron.

Christopher Schott - JPMorgan - Analyst

All right. And maybe just one last question before we wrap up here. On the Factor XI's range of indications you could pursue, should we think about the company broadly pursuing? Or are there some higher priority ones within those?

George Yancopoulos - Regeneron Pharmaceuticals Inc - Co-Chairman of the Board, President, Chief Scientific Officer

Well, some people believe that if you have an incredibly safe agent that reduce clotting and should be put in the drinking water, okay? There's obviously some people who need much higher levels of coagulation control.

We believe -- once again, as Len said, the beauty of what we do at Regeneron, we can tailor antibodies to exactly what we want. And against the same target, we can have two antibodies that have different profiles addressing different sets of people.

We think there's a major focus, for example, antithrombosis, for example, atrial fibrillation, that's a currently big indication, we think and we imagine and you'll see when we roll out our Phase 3 program, there's many, many settings where you can apply anticoagulation very creatively where certain things have been limiting to date.

But using our profile of antibodies, we may be able to extend that benefit, the right drug for the right patients to get the right results that can help them as they need that help.

Christopher Schott - JPMorgan - Analyst

Versus a one-size fits all --

Leonard Schleifer - Regeneron Pharmaceuticals Inc - Co-Chairman of the Board, President, Chief Executive Officer, Founder

Thank you, Chris. And our hearts do go out to everybody in Southern California, hoping for those fires to go out.

Christopher Schott - JPMorgan - Analyst

Thanks for joining. Thank you.

George Yancopoulos - Regeneron Pharmaceuticals Inc - Co-Chairman of the Board, President, Chief Scientific Officer

Thanks, Chris.
