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<<Dae Gon Ha, Analyst, Stifel>>

All right. Good afternoon everyone. Thanks for joining this next session. My name is Dae Gon Ha. I'm one of the biotech analysts here at Stifel. With me for the next half hour-ish, we have Homology Medicines. And with me on stage, we've got President and CEO, Albert Seymour. So Albert, thanks very much for joining us. This is my first time seeing you after you became the CEO.

<<Albert Seymour, President & Chief Executive Officer>>

Thanks for that.

<<Dae Gon Ha, Analyst, Stifel>>

Maybe if we can just start off with a brief overview of Homology programs that you got going and the catalyst for the next six to twelve months-ish.

<<Albert Seymour, President & Chief Executive Officer>>

Yes, happy to Dae Gon. So, within the catalyst and within the company right now, we're continuing to progress. We have two programs that we're really excited about that are in Phase 1 right now. One is our, the first in vivo gene editing program for PKU, it's HMI-103, again in Phase 1. And then in parallel with that, we have HMI-203, which is a gene therapy approach for Hunter syndrome, taking advantage of our AAVHSCs that can distribute not only peripherally, but crossing the blood brain barrier. And so very eager and excited to move those programs forward. Over the network, anticipating giving a program update on that by the end of the year. And with that, some clarity around guidelines for next year around data release.

And then behind that, we have the third arm of our platform, so gene therapy, gene editing, and then we have a vectorized antibody approach, where we're utilizing our AAVs to basically take advantage of the liver to produce monoclonal antibodies. And that one is in IND-enabling as we speak. We just met with the FDA, and so we're all on track to initiate our GLP tox studies starting early next year.

<<Dae Gon Ha, Analyst, Stifel>>

Okay. Let's start with your PKU program.

<<Albert Seymour, President & Chief Executive Officer>>

Yes.

<<Dae Gon Ha, Analyst, Stifel>>

So, you have an extensive history here first with HMI-102, and now you're pivoting to HMI-103. Can you briefly just remind us what happened with HMI-102 and why the ultimate decision to HMI-103?

<<Albert Seymour, President & Chief Executive Officer>>

Yes, happy to. So when we firsts set up the company back in 2016, we had these new AAVs that had never been in people before. They were discovered naturally in stem cells, but made recombinant and moving into people that had never been done before. So we wanted to move quickly into a disease indication where we could get into the clinic quick. And so we ended up selecting PKU as that lead indication because we felt it really fit those two arms of our platform, go into adults with the initial gene therapy program, and then come in with the editing so that we can integrate into the genome and get through the rapid liver growth with the PKU. And that was the idea initially.

So as we were going through with the gene therapy program, HMI-102, we were able to learn a tremendous amount. Number one, that we could see biologic activity at doses of mid-E13 and higher. And so we were very excited about that. And what I mean by biological activity is we saw phenylalanine come down, we saw tyrosine go up because we're restoring that biochemical pathway. And the only reason way you would see that in a PKU patient is if their liver was actually making the enzyme that was missing in that.

So we were able to then get through the dose escalation part of that, where we tested three doses, two, six and 1E13. And then we moved to expand at two additional dose of six and eight. And over the course of that, again, we were able to see that biological activity, but we also learned a little bit around liver enzyme levels. And so we saw some liver enzyme increases within that trial, learned a lot around how that respond to steroids. And then we could take all that information and in parallel, we had the editing program coming along.

And so with HMI-102, we were able to capture all of those learnings and then build it into our PKU editing as well as our Hunter program for gene therapy. And what I mean by those learnings is we were able to take, understand how steroids respond, or the liver enzymes respond to steroids, but then also assess a little bit more around that immune response. And so what we were able to build in our two most recent based on that experience, is the incorporation of tacrolimus, which is a selective T-cell inhibitor.

And so the PKU editing program has that immune regimen built into it. And so at the time when that program was moving into Phase 1, and HMI-102 was off hold because we were put on hold briefly for the liver transaminitis. We as a company decided because of how we engineered and made HMI-103 for the editing, we put a promoter in that. So we felt with that program we could actually treat the adult, the adolescent, and the peds with

one. And we had the new immune regimen already incorporated into that study. So by pausing the enrollment of HMI-102, it allowed us to focus all of our resources on HMI-103. And that was the reason for that, that particular pivot.

For HMI-102 we're still following the patients that we had dosed as part of that trial. But all current and future enrollment will be in the phe edit program.

<<Dae Gon Ha, Analyst, Stifel>>

Right. So the dose expansion in pheNIX, I guess up until there was that decision to pivot, are you able to share whether or not the LFT elevation, the transaminitis led to a subsequent reduction in, I guess, fee reduction or any of those, I guess, benefits that you would normally expect from gene therapy?

<<Albert Seymour, President & Chief Executive Officer>>

So, one of the things that we – we had presented some of this data back at the end of 2020 from the dose escalation component of that, where we did see that phe elevation, the phe go down and tyrosine go up in that. So we had that information. So we were continuing to see that.

What the LFTs did is it's almost battling against two components. One is we would have to increase the steroids, steroids can be catabolic. And we felt like maybe during that catabolic nature steroids you can increase phe by itself. So you're battling against that. And then the other thing is with the liver enzyme increases we also have seen, and others have seen that even in a non-PKU population, if you have some inflammation in the liver, your phenylalanine levels can increase. And so we are battling against both of those. And so that's one of the things we wanted to get around with the incorporation of the new immune regimen.

<<Dae Gon Ha, Analyst, Stifel>>

So then what is the immune regimen that you are using? And you said it's now broadly applicable to all your programs. Does it also include pediatric as well as adults the same regimen that was applied?

<<Albert Seymour, President & Chief Executive Officer>>

Eventually. And so right now both of our Phase 1 programs are in adults. We're starting in adults for both PKU as well as Hunter. So the regimen is actually, we do a week ahead of time with tacrolimus, and then a day before that we start the high dose steroids, and then we taper the steroids over a 12-week course – no over an eight-week course, and then we have tacrolimus for an additional 12. So the total immune suppression is for 13 weeks, with the new regimen that we have.

So that hits on two fronts. One, focusing more on the T-cell response, and then two, really reducing the overall steroid burden within the PKU population, in the hunter population.

<<Dae Gon Ha, Analyst, Stifel>>

Okay. When you guys presented the pheNIX data towards the end of 2020, there were a couple of questions around what is the bar now, right. The 50% reduction had been used phenylalanine, but then there was also the ACMG guidelines, and then there are also questions around cognitive benefit for these patients. So what is that bar these days for a PKU drug?

<<Dae Gon Ha, Analyst, Stifel>>

Yes. So, the bar is still a meaningful reduction in phenylalanine from baseline. And so we've seen that with Palynziq. The most recent approval in the PKU space with Palynziq was that reduction in phenylalanine. So that's still the registerable endpoint. I think all of us that are in the space as we look at this, we're also looking at those guidelines as well.

So in the United States, the American College of Medical Genetics guideline is you want to get their phe below 360, and that's where you really see the unmet medical need in this population, because on restrictive diet as well as even with some of the therapies, it's very difficult for this patient population to get their phe consistently below that. And that's sort of the target, if you will, from a genetic medicine approach is to do that.

As far as the cognitive, one of the things we're doing now in the Phase 1 is to identify a dose that's safe that can show that clinically meaningful reduction in phenylalanine. We'll assess that first, and then we'll start looking at the impact that consistent and sustained phe reduction could have on the neurocognitive, mainly around executive function.

<<Dae Gon Ha, Analyst, Stifel>>

So presumably not for an approvable endpoint in Phase 3, but a longer term follow-up from a Phase 3 post-approval that you might end up adding onto the label.

<<Albert Seymour, President & Chief Executive Officer>>

Exactly. So, exactly. So, even as you go into your Phase 3 you look at what's the registrable end point that obviously you get that feedback and agreement alignment with the regulatory agencies. And then what is it that you can do to really differentiate that product to help the payers and the patient population as well. We would start to incorporate those in so that we could either get it into the label or utilize it as part of the overall description of the product.

<<Dae Gon Ha, Analyst, Stifel>>

Okay. So one of the concepts that we can fully appreciate about the phe edit or the nuclease-free gene editing is not only impacts adults, but also benefits pediatrics given the cell division. How should we think about the efficacy? You mentioned the common denominator and being AAVHSC vector, but how can one translate your pheNIX experience into a phe edit experience in terms of extrapolate?

<<Albert Seymour, President & Chief Executive Officer>>

Yes. So it's a great question. So one of the things that we can get with the pheNIX experiences, the enzyme that we're making is the same. So it's phenylalanine hydroxylase and the capsid that we're utilizing was the same. So we know with that, that once we got into that midi 13, we were seen biological activity.

We also were able to learn from that a lot of things around vector design and aspects like that. So, a key difference now with the editing component is that what we did was we put in long homology arms and we made this vector all single stranded, whereas the original HMI-102 was self-complementary. And so now taking that into play, with the dosing, the seeing the activity from HMI-102, built that in for HMI-103, we're actually starting our HMI-103 dosing in the midi 13. So, we have that confidence. We don't think we need to start at the very low dose and work our way up into doses where we saw activity.

Secondly, we're able to learn a lot about the immune regimen. And so a lot has gone into that, not only with what we've learned, we've done some preclinical studies evaluating the immune regimen on it. And then also just talking with others in the community that are looking at the same thing, really adding in this selective T-cell in here, whether it's tacrolimus or sirolimus seems to show some benefit with respect to reducing the incidence of the liver transaminitis as seen across almost all the AAV trials, with those regimens. And so all of that combined is what we're looking at with HMI-103.

But I think the main component is showing that activity that we observed with pheNIX with a similar molecule, but it just doesn't integrate and we're starting in the same adult population with HMI-103 as well.

<<Dae Gon Ha, Analyst, Stifel>>

Just quick clarification. I know this is editing program. So you've got the homologous arms for the integration. Does it get expressed if it exists as episome?

<<Albert Seymour, President & Chief Executive Officer>>

It does. We engineered it that way. So when we originally were working on the editing program in early discovery, we started off by trying to stay away from all expression from the episomes. And what we found was that in those cells you would have episomes because we're using AAV to deliver and it takes that single stranded genome turns it into an episome in the cell. And we weren't getting anything off those episomes.

When we integrated, we were getting integration levels around 5% where we see this correction in the phenotype, in the mouse model. So we had that as a baseline. Then what we did was based on the learnings from HMI-102, we looked and we thought all these episomes are going to be there anyway, why not actually put a promoter on so that we can get expression from those episomes in addition to the integration. And that actually allowed us to almost have a dual approach because now where we integrate the expression off that integrated allele is also coming from a liver-specific promoter, not the endogenous promoter. And that liver-specific promoter is more powerful, if you will, than the endogenous.

So overall we're making more PAH in each cell.

<<Dae Gon Ha, Analyst, Stifel>>

So you just mentioned 5% that was the preclinical observation that seems to give you that expression. So when we think about human data, is it really 5% integration that we should be looking about, given that as episome it can still generate protein or PAH, or should people kind of not really get fixated on that 5% number?

<<Albert Seymour, President & Chief Executive Officer>>

So, particularly now that we're going into adults first and we can utilize HMI-103 across the whole population, the main thing is how much PAH does HMI-103 and how much is it able to make in the liver. So that's the main focus of that. The 5%, the integration is what gives it that potential for long-term durability, particularly in the pediatric population. So we'll know that over time. But coming in, having the fact that we're expressing off the episomes is probably the more important aspect of that because you will get a lot of PAH expression off that, particularly in the adult population.

<<Dae Gon Ha, Analyst, Stifel>>

But in the protocol itself, the phe edit protocol, you do not have any biopsies to measure integration.

<<Albert Seymour, President & Chief Executive Officer>>

We don't. We had a lot of discussion around this in the PKU population and the investigators. We just felt that doing that for the PKU population in the adults was an added risk, particularly as we're thinking about blocking the liver inflammation. So we felt by doing a biopsy, we could actually contribute to that versus keeping it away. So in the protocol, we will look at phenylalanine, as well as tyrosine, as the two biomarkers of activity.

<<Dae Gon Ha, Analyst, Stifel>>

Okay. Okay, yes, pheNIX, you did both and then the ratio.

<<Albert Seymour, President & Chief Executive Officer>>

Yes.

<<Dae Gon Ha, Analyst, Stifel>>

Remind us the trial design for phe edit, you mentioned adult first before pediatrics, when would the pediatrics kick in?

<<Albert Seymour, President & Chief Executive Officer>>

So starting in adults we'll do the dose escalation in adults. So it's a three-by-three, open-label, dose-escalation study where we have three individual cohorts across three doses, starting at mid-E13 and then going up. What we'll do is we'll dose three at the first cohort, we'll then evaluate that data, meet with our independent DMC. If everything looks good, then we can go to the second dose. We'll continue on that until we find a dose that we think gives us the efficacy that we're looking for, coupled with the safety that we're looking for. It's at that point in the Phase 1 we'll then go back to the FDA present it as end of Phase 1 type meeting. And then make plans for what is that next step and that would – that's where we would certainly propose to move into the adolescent and the pediatric population as well as additional adults.

<<Dae Gon Ha, Analyst, Stifel>>

How should investors set expectations in terms of your readout? Given that it's a three-by-three? Is it per every DSMC you kind of come out and say we've cleared it and by the way, this is what we saw? Or do you kind of just say, DSMC cleared, but we'll hold off on the data until we get all three cohorts or something in between?

<<Albert Seymour, President & Chief Executive Officer>>

It's probably something in between. I think one of the things that we as a company want to look at, we want to have enough data that's meaningful. Like we don't want to, and so as we go through this and we look at that that may be the first cohort and say there's some meaningful data here. This is what we're – what it looks like, and then here our plans going forward. Because of the confidence that we have at that first starting dose we won't know until we get into the trial and see this, but we do feel very confident that we should see some activity at that first dose. So that could be the first opportunity where we would see some meaningful efficacy and safety data with that first. So – and when we lay out the guidance for next year, that's kind of the framework that we're evaluating it in that we could go out with a meaningful data set maybe on a cohort like that as opposed to all three cohorts before we got to that.

<<Dae Gon Ha, Analyst, Stifel>>

Okay. Can you speak to your level of confidence? I mean, one of the main push-backs we got after your pheNIX update was, how do I make sense of two patients per cohort where the response rate of 50% sounds extremely good, except it's out of two patients?

<<Albert Seymour, President & Chief Executive Officer>>

Yeah, exactly, and that's always the balance that you take, particularly in rare disease. And so with the balance here particularly in a dose escalation where you're trying to identify that first dose and that's where we were with the dose escalation even with pheNIX. So we wanted to balance to have enough patients to give us a sense of activity and safety. But then we want to then expand to increase that end so that we can get a really better estimate of what that that overall response rate is. In the fee edit we're increasing that to three per, now we can balance that as we go along because the protocol does have aspects built into to allow that kind of flexibility. So if we see really great data and two or we get the first two and it's not, we can then go to the – go up without having to get through the full cohort.

<<Dae Gon Ha, Analyst, Stifel>>

Okay. Okay. I lost my train of thought. But the year-end update, we've also wondered what exactly should one expect out of that mentioned status update, but to us that we didn't really know what to make of that language?

<<Albert Seymour, President & Chief Executive Officer>>

Yeah. So I'll give a lot of clarity on that right now. So at the end of the year, what we want to do is really update where we are as a company with the two Phase 1 programs. How many sites do we have? What's the enrollment status look like? That will be the extent of the update coupled with clarity and guidance next year for when we would come out with clinical data readout. So that's what you can expect at the end of the year.

<<Dae Gon Ha, Analyst, Stifel>>

Got it. So year-end update does not necessarily entail clinical data itself but rather sometime next year.

<<Albert Seymour, President & Chief Executive Officer>>

Exactly.

<<Dae Gon Ha, Analyst, Stifel>>

So the question that I forgot to ask was; does pheNIX experience in any way provide you with any enrichment strategies as you enroll the patients at least adults?

<<Albert Seymour, President & Chief Executive Officer>>

What it does is it allowed us when we – when we set out and sort of establish a presence in the PKU population, so pheNIX allowed us to do that. Now one of the things that we did early on before we made the decision to pause enrollment is we were setting up sites for pheEDIT that did not overlap as much with 102 because we didn't want to compete with the two programs at any individual site. Now that we paused, a pheNIX that does allow us to take any patient that was interested in pheNIX and talk to them about moving into pheEDIT, so that does help enrich and bring in more patients into pheEDIT.

<<Dae Gon Ha, Analyst, Stifel>>

Can you speak to the rate of enrollment given that 102 was deprioritize that patients should transition over to 103? BMN 307 being on clinical hold should also in a way help bring more patients. But we've also heard of a private company that's exploring a small molecule in PKU. So how should we think about the overall dynamics of that involvement?

<<Albert Seymour, President & Chief Executive Officer>>

Yeah. So I think one of the things that that we've experienced over the past as we're going through this is some of those headwinds in gene therapy. I think with BioMarins program BMN 307 going on hold, patients had some questions about that. So they were like, what does this mean? How is yours different than BMN 307? We've really overcome that hurdle as we – with the pheEDIT program. I think with pheNIX and pausing that, that also allowed us to take those patients and talk a little bit about pheEDIT and how HMI-103 is different than HMI-102. And I'd say the excitement and encouragement from the patient populations are well investigators, has really grown over the time would allow us to go through that because the BMN 307 hold was completely different than anything that that we've ever seen. It's almost non-comparable.

In terms of PKU generally, I think when you look at the small molecule, this is the transporter inhibitor that they just did raise for this morning, right, GENN. So with that as they go in, they're in healthy volunteers right now, so they're not going to be tapping into the PKU population. And I think when you look at that, a lot of it's going to come down to what's that effect size of that kind of mechanism from a genetic medicines perspective. With HMI-103, we truly are replacing the enzyme that's missing in the liver that we've been able to show in the mouse model can completely normalize at that low doses. So I think the promise and the excitement about that is what's pulling some of these patients into something they could take a onetime administration that can truly let – allow them to liberalize their diet and get their phenol into more measurable – more reasonable levels.

<<Dae Gon Ha, Analyst, Stifel>>

Got it. Moving into 203 quick clarification that also has a year-end update. So same question, that's just a status update and when we can expect data. Will that coincide with the 103 update or are those two separate events?

<<Albert Seymour, President & Chief Executive Officer>>

One, that'll be data driven as we're going along? When we look at 103 versus 203, 203 is an adult Hunter population. It is a much rare population than PKU. There are a lot more PKU patients than that. So as we look at that, that data update, a lot of that's going to be data driven. Ideally you'd want to have them separate, if you will, but it'll be data driven.

<<Dae Gon Ha, Analyst, Stifel>>

So I like the year-end update.

<<Albert Seymour, President & Chief Executive Officer>>

Oh, the year-end update will be at the same time.

<<Dae Gon Ha, Analyst, Stifel>>

Okay. Makes sense. So when Arthur was around, he used to talk a lot about ELAPRASE MPS 2 and how your team has a lot of experience. Can you share with us the experience there? What is lacking? What is the unmet need that you're trying to really address with 203?

<<Albert Seymour, President & Chief Executive Officer>>

Yeah. So this is where, I think, we had a distinct advantage just because several of us, actually our Head of Commercial was the one who launched ELAPRASE back in the early 2000. And what we've learned from that is how patients respond to ELAPRASE and what the unmet medical need. But then also really establish a connection within the investigators in the Hunter syndrome space. What goes unrecognized to some extent is that when you think about Hunter syndrome, everybody, all the patients, 100% of the patients have peripheral disease. So they have joint pain, they have a lot of aspects like that. About 60% will then go on with the more severe that has the CNS component. ELAPRASE treats the peripheral aspect and so now you have some boys that have been on ELAPRASE for more than a decade. And what we're seeing is that as they get older they're continuing to have significant unmet medical needs.

So they're still having joint pain, having trouble standing up. So they can eventually start to have lot of trouble in their mobility, and breathing and aspects like that. So by starting in the adults, that's what we're going to focus on initially is by having a gene therapy approach instead of infusions that have a high exposure and then it goes away and you have these troughs, we'll have this continuous exposure over time. So basically the liver or other cells that are making this can make it in the cell that was transduced, but then it can be taken up by neighboring cells. So we get this pre-clinically, this really broad distribution, that's where we'll start.

The other thing that we've characterized is HMI-203 in preclinical models does cross the blood-brain-barrier. So we're also going to incorporate CSF tap into the 203 studies so that we can evaluate. Do we get enzyme activity on the other side of the brain; blood-brain-barrier. And that will then allow us to make a decision to move into the pediatric population.

<<Dae Gon Ha, Analyst, Stifel>>

Got it. So you would need that substantive amount of data from adults before moving into the pediatrics?

<<Albert Seymour, President & Chief Executive Officer>>

Most likely. We want to make sure that we identify a dose that we now know we're getting across the blood-brain-barrier.

<<Dae Gon Ha, Analyst, Stifel>>

Got it. So in terms of expectation, I guess two-part question on the enzyme level, is there, you mentioned the different trough and peak levels with the ERT. Are you trying to smoothen it out at a normalized ERT level? I mean, not ERT level, but enzyme level? And then second part is given that you're going into adults first, should we be expecting a reversal in disease or more of a stabilization at that stage?

<<Albert Seymour, President & Chief Executive Officer>>

Yeah. It's a really good question. In terms of, I'll start with the enzyme levels. We know within the Hunter population that there's a diagnostic criteria around enzyme activity in the blood. We want from a gene therapy perspective to have it above that diagnostic level. And that means they – that you won't have Hunter syndrome. But there's a big range in what that can be so in the blood. So a person that that doesn't have Hunter syndrome at all could have levels say I'll just arbitrary to use Hunter, but also there might be mild mutations in Hunter or pseudo-Hunter that may have enzyme levels say around 10 and they're also completely normal. So our focus is to get those enzyme levels in above the diagnostic range for Hunter syndrome in the blood.

<<Dae Gon Ha, Analyst, Stifel>>

I see.

<<Albert Seymour, President & Chief Executive Officer>>

And then in terms of the disease perspective, the first study that we're running right now is an Phase 1 dose finding study. And so again, it'll be very similar to pheEDIT. It's the three-by-three dose escalation through three cohorts. The main thing that we're going to be looking at that is safety as well as identifying a dose where we have those enzyme

levels that are above that diagnostic threshold and can we see something in the CSF. And that's what then – then we would utilize that to assess what would that next design look like.

Now we will measure urinary GAGs to see whether or not we stabilize. The thought is to stabilize because all these patients will be on ERT prior to the study, so we want to stabilize. And then we'll also assess with that bettered by distribution and that sustained can we see a reduction? We don't know. But the base would be to stabilize that.

<<Dae Gon Ha, Analyst, Stifel>>

Okay. Would then the approvable endpoint here be ELAPRASE type trial design? Or would you be thinking about differentiation points?

<<Albert Seymour, President & Chief Executive Officer>>

Yeah. So that's something we'll determine during the Phase 1 what and how that data looks like. I think because we're coming in with ELAPRASE, it's almost you can think of it like a – it's similar to ELAPRASE but it's a onetime administration of ELAPRASE. So the design of the trial is actually to have them on ELAPRASE for a period of time and then we're going to measure enzyme levels in the blood at right before they get their next ELAPRASE treatment. If their enzyme levels are high, then we know they don't need the enzyme, the ELAPRASE anymore. So from an approvable end point, there are a couple things that we're considering on there. You can think about a non-inferiority or depending on what the data look like as we come out, there could be a way to differentiate, but time will tell as we get that data.

<<Dae Gon Ha, Analyst, Stifel>>

Okay. Let me pivot to the GTx-mAb, the 104 program. Walk us through why did you pivot to expressing mAb's and maybe why 104?

<<Albert Seymour, President & Chief Executive Officer>>

Yeah. So one another things we're looking at with the whole platform is if you take the editing component there we really – you're going to go in and you're going to correct something in the genome. You want to make sure that that is something that is mutated that causes disease. So it sort of pushes you into an opportunity within rare monogenic disorders. So you want to make sure that it's that – from a gene therapy where you're doing gene replacement, it's very similar to focusing on diseases where you know exactly what the cause is and can you address that cause? Then we as a company, we sat back and we said, well we can utilize these vectors to express proteins.

What could be an opportunity for us to utilize that platform as a third arm to get into diseases maybe that are a little bit larger? Where instead of going after the cause you may go after the symptom of a disease, and so when we had that, we then looked and said,

what antibodies is there a lot of data that shows that it's safe and that you can have patients that have been on it for a long-time with high levels and its safe. And that's when we ended up looking at anti-C5. So we felt that was a good one where we could address an unmet medical need around the sort of the breakthrough hemolysis.

And it's safe enough that we could come in, there's a precedent there. We knew exactly what levels we wanted to get to in order to establish that that clinical phenotype. So that's why we started initially with the anti-C5 and PNH. Secondly, on top of that is that when you look at anti-C5 there's also a broad number of diseases that also compliment blocking C5 can address. So we could do not only PNH, but then you can expand it into others.

<<Dae Gon Ha, Analyst, Stifel>>

Okay. Well with that I guess we will end it there. Albert, thank you very much for your time.

<<Albert Seymour, President & Chief Executive Officer>>

Yeah, thank you Dae Gon.