

Cue Biopharma Conference Call September 21, 2026

Corporate Speakers

- Agnes Lee; Cue Biopharma; Chief Investor Relations and Communications Officer
- Shao-Lee Lin; Cue Biopharma; President, Chief Executive Officer
- Dominic Borie; Cue Biopharma; Chief Medical Officer, Head of Research and Development

Participants

- Maury Raycroft; Jefferies; Analyst
- Mayank Mamtani; B. Riley Securities; Analyst
- Dev Prasad; Lucid Capital Markets; Analyst

PRESENTATION

Operator^ Good morning everyone. And welcome to the Cue Biopharma Conference Call on the CUE-221 CSU Phase 2 Top Line Results. (Operator Instructions) I would now like to turn the call over to Agnes Lee, Chief Investor Relations and Communications Officer at Cue Biopharma. Please go ahead, Agnes.

Agnes Lee^ Good morning. And thank you for joining us today. Before we begin our formal comments, let me remind you that during today's webcast, we will be making forward-looking statements that represent the company's intentions, expectations or beliefs concerning future events.

These statements represent our views as of this date, are subject to risks and uncertainties and should not be relied upon as representing our views or of any subsequent date in the future. As a reminder, this call is being recorded. And a replay will be made available on the Investors section of Cue Biopharma's website following the conclusion of the event.

With me on the call today are Dr. Shao-Lee Lin, our President and Chief Executive Officer; and Dr. Dominic Borie, our Chief Medical Officer and Head of R&D. Dr. Lin will open the call and then hand off to Dr. Borie to walk through the study data. Dr. Lin will then close our prepared remarks. And we will take questions. With that, I will now turn the call over to Shao-Lee.

Shao-Lee Lin^ Thank you, Agnes. And good morning, everyone. Thank you for joining us. Today we are reporting results from the Phase 2 study of CUE-221 in the chronic spontaneous urticaria, or CSU. We're pleased to share that the study results were positive and that we will be reviewing together those top line clinical results with you today.

The key takeaway is that CUE-221 met its primary and key secondary end points in a manner that reinforces our enthusiasm for the precision engineered unique dual mechanism of action of CUE-221 and its potential to offer clinically meaningful and differentiated benefit in IgE-mediated disease.

Slide 3 summarizes the salient findings from the study. The study demonstrated robust, durable and dose-responsive clinical effects. The primary endpoint of complete resolution of

hives, or HSS7=0 at week 12 was met with the treatment effect further increasing past the final dose at week 16 to its peak at week 22.

The key secondary endpoint of complete response or UAS7=0 was statistically significant in the high dose group, further supporting the clinically meaningful impact of CUE-221. These results were achieved in the context of a favorable safety and tolerability profile.

Of note, for the CUE-221 high dose group, both the complete resolution of hives and the overall complete response, persisted for 12 weeks or full quarter and on the order of five half-lives after the last dose. This was not seen for the lower doses or for omalizumab despite a comparable dose level, highlighting a fundamental clinical difference, consistent with the biological design differences between the two drugs and supporting the concept that CUE-221 has the potential to be a disease-modifying therapy.

This marked difference observed off drug at week 28 relative to omalizumab, provides our first clinical evidence supportive of the CUE-221 engineered dual mechanism of action that includes not only blocking of the IgE-mediated allergic cascade, like omalizumab, but also supports the clinical potential of down regulation of new IgE production unique to the design of CUE-221.

Additional PK and IgE data are of interest. And while not available today, will be the subject of further modeling analyses and future presentations at upcoming medical meetings. Based on this demonstrated potential for differentiated clinical efficacy in CSU and the potential to develop a new tool to target disease modification and the concept of functional cure for IgE-mediated diseases, we are more than excited to now plan both a Phase 2b/3 next study in CSU as well as a Phase 2 study for food allergy. I will now hand the call over to Dominic Borie, our Chief Medical Officer and Head of Research Development. Dominic?

Dominic Borie^ Thank you, Shao-Lee. So let me take a couple of minutes to quickly walk the audience through the science behind CUE-221. First, reminding ourselves that IgE drives allergic disease by engaging two receptors, the high affinity receptor shown here on the left, through which IgE and the allergen will trigger degranulation and allergic symptoms. And the low affinity receptor, also known as CD23, shown on the right which is expressed on the surface of B cells and has an important role too as when bound by IgE, it triggers a natural negative feedback loop that results in reduced IgE synthesis.

On Slide 5, CUE-221 was designed to block IgE binding to the high affinity receptor and in doing so, provides potent IgE neutralization, or IgE blocking. CUE-221 high binding affinity for IgE, shown here on the left, supports a greater potency at preventing IgE-mediated cell degranulation over omalizumab, as shown on the graph on the right.

On Slide 6, on the flip side, CUE-221 was also designed to preserve IgE binding to CD23. And this was achieved through selecting binding sites on IgE that are distant from the IgE region that engages CD23, whereas the binding site on IgE for omalizumab and its analog, RPT904 is the same, as shown on the right and overlaps with the CD23 binding site and therefore does not allow the drug-IgE complexes to bind to CD23.

On Slide 7, as the result of the difference in engineering, increasing concentrations of IgE and complex with CUE-221 can bind to the CD23 receptor, as shown by the orange curve on the

right, whereas no binding at all was measured with IgE complex with omalizumab, as shown by the gray triangles.

On Slide 8, the biological impact of preserved binding to CD23 was demonstrated both at the mRNA and at the protein level where addition of CUE-221 resulted in a significant reduction in IgE synthesis shown by the orange bar, that was not observed for omalizumab, in gray bar, indicating that omalizumab in complex with IgE cannot block new IgE production.

On Slide 9, it was therefore very exciting to test the clinical impact of these fundamental biological differences in the context of a Phase 2, randomized, double-blind, and placebo-controlled study.

An omalizumab arm was included for comparative efficacy and safety, but no formal statistical testing was planned. The study enrolled participants with moderate-to-severe chronic spontaneous urticaria, or CSU, inadequately controlled with H1 anti-histamines. And patients were randomized to either one of CUE-221 dose groups at one, two or four milligrams per kilogram, administered subcutaneously, or to omalizumab at the registered dose or to placebo.

All patients received subcutaneous injections every four weeks. And the primary endpoint was assessed at week 12 and measured the rate of complete resolution of hives or HSS7 score = 0. This endpoint leverages an objective assessment of the number of hives which is carried out by the physician, and achievement requires all clear skin or no hives. The final dose in the study was administered on week 16, for all groups, and after which patients were monitored off drug for 20 weeks through week 36.

Slide 10 shows the subjects' disposition for the trial. Two subjects were randomized but not dosed, one in the 4-milligram per kilogram group, and one in the omalizumab group. The dropout rates are in range with expectations for a clinical trial of that duration, further considering the 5-month observation period off drug. This study was analyzed using a modified intent-to-treat approach. The two subjects who were not dosed were not part of that analysis, but all other subjects were included in the modified intent-to-treat analysis.

On Slide 11, the baseline characteristics were overall well balanced across groups. This is duration range approximately for four to six years across groups, and mean baseline Urticaria Activity Score seven or UAS7, were in the 28-plus point range, indicative of severe disease activity.

On Slide 12, getting into the results, we are excited to report that the study primary endpoint was met. The complete resolution of hives, HSS7=0 at week 12 was significantly higher than the placebo for all dose groups in a dose responsive manner with a high P value of less than 0.005 for the high dose group.

On Slide 13, the key secondary endpoint of complete response or UAS7=0 was also met, and the difference versus placebo was statistically significant for the high-dose group. On Slide 14, another way to visualize the depth of response is to measure the change from baseline in UAS7 score at week 12 which was found to be profound, and statistically significant across all dose groups.

On Slide 15, this slide captures the depth of response at week 12 by presenting the percent reduction from baseline in UAS7 across various mechanisms. Although we note that cross-trial comparisons are for illustrative purposes only, and should not be interpreted as a direct comparison of efficacy, across various modalities, CUE-221 seems to come closest to providing full control of disease activity.

On Slide 16, if you allow me to shift gears now I want to focus on additional exciting findings arising after the week 12 primary endpoint. On the middle graph, across all CUE-221 dose groups, complete resolution of hives continued to increase beyond week 12 and peaked at week 22, which is six weeks after the last injection in a dose responsive manner, more than two-thirds of subjects in the high-dose group responded and achieved complete resolution of hives.

Now if we look at the week 28 results, this is a time point when patients have been off drug for 12 weeks. This corresponds to approximately five half-lives after last dose for both CUE-221 and omalizumab given expected half-life of two to three weeks for these IgE monoclonal antibodies. Let us all note that the 4 mg/kg dose is for a 70-kilo patient, the molar equivalent of the 300-milligram Xolair dose.

At that week 28 timepoint, where 60% of CUE-221 patients are still experiencing complete resolution of hives, only 24% of patients in the omalizumab group are. And this stark contrast can be explained only by pharmacokinetics as residual circulating drug concentrations are likely extremely low to nil for both groups.

Rather, we think that this indicates a fundamental difference in biology and provides clinical data indicating that, for the CUE-221 dose group, there is something different happening which is more than just IgE neutralization. Note that a post-hoc Fisher's exact test was run and found that the difference between CUE-221 and omalizumab was statistically significant.

On Slide 17, to provide further perspective, we are providing here this line graph showing the rate over time of HSS7 for the four mg per kg CUE-221 dose group, shown in orange, versus omalizumab and placebo. After the initial injections, there is a rapid clinical impact and we can then clearly see the curve diverging after the last injection, reaching the maximal difference of week 28, as we previously discussed. This graph clearly shows the sustained high rate of complete hives resolution throughout the 12 weeks that followed the last injection of CUE-221. After week 28, both curves are converging again, trending towards the placebo response in the absence of further administration of drugs beyond the week 16 dose.

On Slide 18, similar to the results seen for HSS7=0, a similar pattern of efficacy was seen with the complete response rates or UAS7=0 which was also sustained for over 12 weeks off drug for the high dose group, again, with the same pattern of marked clinical impact over omalizumab, and supportive of a fundamental difference in biology, that is consistent with the differentiated mechanism of action.

On Slide 19, building on the prior slides, we can start contrasting the features of IgE-targeted therapies. What we think stands out from the week 12 data is that IgE neutralizers, like omalizumab or RPT904 which share the same binding epitopes and therefore the same biology of IgE neutralization, they appear to perform closer to CUE-221 low doses rather than CUE-221 high dose.

Later time point data has not been made publicly available for RAPT but, again, we are extremely pleased with the different efficacy profile seen with CUE-221 over omalizumab at week 28 off drug as it supports fundamental biological differences which may be directly deriving from CUE-221 engineering.

On Slide 20, from a safety standpoint, CUE-221 was well tolerated. There was no treatment-related serious adverse event, or SAE, and no adverse events of anaphylaxis. Treatment-related adverse event of special interest was limited to one case of grade two injection site reaction. There were two instances of treatment-related adverse events leading to study discontinuation, namely one case of worsening urticaria and one instance of Insomnia. As expected, there were no deaths in the study.

On Slide 21, to help place the safety findings in context, and because safety considerations will be part of the benefit-risk analysis when comparing across different mechanisms of action, this slide provides a summary of anticipated side effect profile for different mechanisms investigated currently in CSU.

We believe that CUE-221 dual MOA should not introduce specific mechanism-related safety that will be different for IgE neutralizers, even in the presence of possibly more potent IgE control, and this will be contrasting with the recognized mechanism-related safety findings of other MOAs.

We do recognize that broad interest for the field of allergy and IgE-mediated disease results in additional MOAs, currently tested at earlier stages of clinical development. We believe that although still emerging, CUE-221 potential is already more developed and we look forward to further characterizing CUE-221 safety and efficacy profile for IgE-mediated disease.

On Slide 22, altogether, we are extremely excited to have clinical results in hand that indicate the potential to reach a functional cure for IgE-mediated disease. This did not happen by chance, but may result from the fact that, from the very beginning, CUE-221 was engineered to be different from omalizumab and to not only block the high-affinity receptor but do so while preserving the binding to CD23.

The preserved capacity of CUE-221 IgE complexes to bind CD23 results in reduced IgE synthesis at the mRNA and protein level, something that was not seen with omalizumab. And here, we now have clinical data strongly supportive of fundamental biological differences between CUE-221 and omalizumab. We are eager to characterize the PK/PD relationship and will be presenting these additional results at an upcoming scientific conference.

On Slide 23, I won't read this final slide, but it may be helpful to keep it staying up during the Q&A session. And thank you for your attention. And now I will hand it back to Shao-Lee.

Shao-Lee Lin^ Thank you, Dominic, and thanks to those who have dialed in to be with us today. As you can see, we are very pleased with the potential that CUE-221 has now demonstrated clinically, and which is consistent with its novel precision engineered dual mechanism of action. Based on these exciting positive results, we are working towards the initiation of a Phase 2b/3 study in CSU in addition to our planned Phase 2 study in food allergy.

I would like to thank the Genesis Life Sciences team for designing and executing a robust Phase 2 trial. I would also like to extend our gratitude to all the patients, investigators and study staff who participated in this trial and supported the advancement of CUE-221 for allergic disease.

It has been a privilege to be at Cue over the past five months and witness all the hard work and progress across our portfolio. And I want to thank the entire Cue team for their commitment to our mission to identify and advance transformative therapies, designed to enable functional cures across immunologic diseases. We could not do what we love to do without the support of our Board, investors and the patients we strive to serve. With that, I'll open the call for Q&A.

QUESTIONS AND ANSWERS

Operator^ (Operator Instructions) And our first question coming from the line of Maury Raycroft with Jefferies. Your line is now open.

Maury Raycroft^ Hi good morning congrats on the data update and thanks for taking my questions. For the Phase 2b/3 CSU study, should we expect a longer dosing interval than the every four-week schedule using the current study? And will you also evaluate additional dose levels? Is there anything additional on timing for when the CSU and food allergy studies could start?

Shao-Lee Lin^ Yeah hey good morning Maury. Thanks so much for joining us, and thanks for the question. So yes, I think we wanted to indicate with the IIb part of the Phase 2b/3 that we're interested in understanding a little bit more about the PK IgE and response. And so, we're likely -- we feel like we have a fantastic dose at this point with monthly dosing that's differentiated.

So, we'll likely utilize that as an anchor, but nothing is definitive yet. And then we're going to also explore given the biology that we're seeing of how best to optimize dosing for each indication. And so, for CSU -- or actually for any indication, for instance, we think that we have something where we can think about fixed dosing. We think based on the profile that we're seeing, there's a potential to think about things like an induction and a maintenance regimen where we're fundamentally modifying some disease biology in the background.

We think about this bathtub analogy, sort of not just emptying the bathtub from blocking or neutralizing IgE, but also turning off the spigot relative to the formation of new IgE. And so, one can imagine, if you can continue to turn that off better-and-better over time that you might reach a point where the interval of dosing now instead of induction to achieve that time point, becomes a lower dose and less frequent in terms of the maintenance.

So, all of these things, I think, we're going to explore, interrogate the data carefully and determine what the best sort of approaches to continuing to collect information about this new tool that we have to help patients with IgE-mediated disease. And so that's how we're thinking about it for CSU.

From a timing perspective, obviously we think we have something important for patients, and we'll move as quickly as we can, relative to that. I'd say for food allergy, something completely different.

So, we're -- we think of food allergy as a life-threatening disease. And in the context of thinking about that, in that way, we think that this new tool gives us, as Dominic was saying, the potential to approach this from a functional cure perspective, meaning that we don't want just two-thirds of patients having protection against three peanuts, some of the time, we want to get as close as possible to 100% of people, having 100% protection all the time.

As close to that as one can imagine getting. And so, we'll approach at this, sort of new tool of mechanism, new potential to modify disease in a way that will aggressively pursue that idea. And we don't think about this from a convenience perspective.

I care less in that instance, if it's achievable, whether this is dosed every six months, three months or even weekly, but really about whether or not I can create an entirely different paradigm for ourselves and our kids, and our families who have this life-threatening condition. So, I hope that helps. And thank you for your question.

Maury Raycroft^ Yes, all makes sense. Great perspective there. And one other specific question on the data. You reported change from baseline in UAS7 which appeared fairly similar across the CUE-221 dose groups and omalizumab. How did HSS7 change from baseline compare across the treatment arms? And are you seeing a similar pattern there as well?

Shao-Lee Lin^ Yes. The patterns that we've seen across the endpoints have been very similar which has been comforting to us from a robustness of the signal that we're seeing. So -- and I -- and we chose the change from baseline percent to compare with others because there are some studies that have placebo and some don't, and some have active comparators, and others don't. And this was the best way to have an internal sort of, a given arm to itself comparison within the context of its own study.

Maury Raycroft^ Got it. Makes sense. Thank you for taking my questions.

Shao-Lee Lin^ Thank you so much Maury.

Operator^ Thank you. And our next question in queue coming from the line Mayank Mamtani with B. Riley Securities. Your line is now open.

Mayank Mamtani^ Hi yes good morning team thanks for taking our question and congrats on the spectacular data here. So, on the response building for six weeks after the last dose, which was not a phenomenon for omalizumab, is there anything in the diary data that separates continue deepening in existing responders from maybe new responders converting late? I was obviously interested in that because the neutralize long half-life can only hold an effect, whereas your mechanism maybe can have that depth of response coming in late. If you could talk to that.

Shao-Lee Lin^ Yes Mayank. Thank you for that. So, I think maybe the best place to see that is in the line graph or in the bar chart before that. And I know we're not sort of synchronized in terms of projecting. So, all I have to ask you, the audience, to go to those slides yourselves.

But the bottom line is all of these representations are actually for a complete resolution of -- so resolution of hives what we've shown in the line Graph IV. And so, there's no sort of

deepening of response beyond the zero, right? And so, the percentage just tells you the percentage of individuals that achieve that complete resolution of, in this case, hives, response.

So, we are definitely getting that. And if you look at the what we think is particularly amazing about the line graph is what I'm looking at currently is you start to see the separation by week eight, you see the space between the gray line and the orange line which is XOLAIR[®] versus the 4-milligram group. And that area under the curve is really quite striking.

If you follow gray line which is omalizumab, from the last dose which is week 16, sort of -- and follow your eye down to the week 30 time point and extend that line, you can really see how that continues to degrade towards the placebo line. There was maybe one subject that sort of even off drug at that period of time spontaneously improve which happens in this disease state and certainly happens to small numbers. But you can see how that's degrading towards placebo which you would expect five months off of drug.

What was surprising was the durability of the 4-milligram group off of drug. Again, as Dominic pointed out, sort of same milligram dose level, say, therefore same molarity because they're both monoclonal antibodies, same sort of number of half-lives off of drug, which is like four to five, so the drug itself should really be gone, and yet the effect is still there.

And it tells us that there something fundamentally different in terms of the biological effect that's happening in those outer weeks between omalizumab and 221. And for us, given that 221 was engineered specifically with a second mechanism, our assumption at this point is that that's that second mechanism in terms of synthesis and new IgE that's kicking in.

And one can imagine then, for instance, that over time if you are, in fact, effective against -- again, turning that spigot off that -- and decreasing free IgE over time that, that may actually cross zero at some juncture and modify the way that you approach the dosing of the disease itself as well.

So, you can think about mechanistically things like Total IgE and receptor engagement on the effector cells, or the high-affinity receptor, really downregulating those receptors and making those effector receptors more quiescent over time and then you can think about also turning off the synthesis of new IgE and reaching some sort of steady state that fundamentally is disease modifying for these patients.

And so that's what we're going to explore at a bare minimum, purely clinically all of the scientific hypothesizing aside, the bottom line is that we have a remarkable clinical difference here that we think is meaningful for patients and pointing to the fact that there this molecule is providing a new tool by which we can approach getting more for each of these patient types.

Mayank Mamtani[^] Thank you. That was very thorough. And then I think the IgE data also will help as that becomes available. I was just -- maybe my follow-up was, do you have baseline IgE data that you see that in the table? And I was also asking that in context of CSU, the range is very narrow, in the 100 sort of range. But the food allergy, the baseline IgE can be much higher.

So, if you can maybe comment on how you're thinking of what the induction (inaudible), some of it, I know you commented, but what high dose levels you could go there just because of where you're starting with your IgE levels? And how is the data here, inform how you kind of progress in CSU versus food allergy?

Shao-Lee Lin^ Yes. Thank you for that, Mayank. I mean I think we're very excited to be able to provide specific IgE, PK, PK response data and modeling associated with that. And how we're thinking about that relative to each of these disease states as we said, at a future scientific meeting. And that's not us being coy to be clear. We have the clinical data at this juncture.

We definitely wanted to make good with our promise to provide data by the end of the third quarter which is what we are delighted to be able to do at this time and those data otherwise are just not available at this juncture. But the way I think about this, and obviously it would be a tremendous grand slam to have both of those pieces together today, and tell you -- the other piece of the story.

But the way I think about this, certainly as a clinician and a drug developer is if I had the bioassay data today, I would be asking myself, yes, but is it clinically relevant? What does it really mean clinically? And that's really ultimately the takeaway. And I think that we are incredibly fortunate to have very clear clinical data despite the relatively small numbers. I mean it's a Phase 2 trial. It's not tiny, but it's also not massive. And to get the kind of clarity across endpoints, the kind of statistical significance that we're seeing with these numbers, we're very pleased by, we think it's very clean and very consistent. And it's telling us that there's something different. And we can argue about what that thing might be, but we think the most likely you know Occam's razor and all, we think it's most likely the thing that it was engineered to do. And so, more to come there.

Mayank Mamtani^ Makes a lot of sense. And if I could fire a final question. Are -- ADA formation, anything on tolerability (inaudible), if you could comment because that obviously is relevant as you develop this in subq format. And I know there was one patient in Phase I that had nausea, headache. Just clarify if you've not seen any of that. Thank you for answering my question.

Shao-Lee Lin^ Yes. And I apologize, I didn't quite hear the first part of your question, but I'll go ahead and address the second part which is from a safety perspective, we feel very, very good about the profile. You saw the table with all the zeros all over it. There hasn't been anything. And that subject in the Phase I, I think it stated clearly in the supplemental as well, was not deemed to be related to study drug, if I recall correctly. And --

Mayank Mamtani^ Sorry, the first part was about ADA or any immunogenicity comment.

Shao-Lee Lin^ Yes. I mean like PK, like IgE, ADAs are also seeing that are -- these are require assays to be run. And with typical studies, and this is not specific for this particular study, we'll batch them at the end so that they're all run at the same time should eliminate some variability associated with them and some noise. And so, they typically come after the clinical data. And that's what you're seeing here.

Operator^ Thank you. Our next question in the Q&A queue coming from line Dev Prasad with Lucid Capital Markets. Your line is now open.

Dev Prasad^ Hi congrats on the update and the data and thank you for taking our questions. I have a couple of questions. One is if we look at week 12, the four and two mg per kilogram dose are nearly identical at 54% and 53% for HSS7, but they're substantially different at week 28 at 60% and 31%. How do you interpret that pattern pharmacologically? Does this suggest a four mg per kg across exposure threshold required for durable expansion? Or is it just accumulation? And one question on planned Phase 2b/3 design. Are you aiming to demonstrate superiority over placebo or over omalizumab or both? Thank you.

Shao-Lee Lin^ Yes. No, great question. And maybe I'll take the second question first which is, are we planning to design a head-to-head trial, I think, is what you're asking us. And I think that's to be determined.

I think we had shared previously that we -- based on the Phase 1 data and what we were seeing relative to a single IV dose really having a very prolonged effect on suppressing IgE below limit of quantification that we were especially interested in this molecule and its potential in food allergy because it's such a strictly IgE-mediated disease. It would seem like the perfect place for something like this.

We now feel sort of doubly excited about food allergy as a result of that. We also always stated that CSU is a more complex disease, has lots of potential mechanistic aspects that are involved in the disease state. And so, how much more we would get with better IgE coverage was an unknown, and we would commit to CSU if it had the potential to be differentiated.

I think that what we're seeing with these data, we feel, again, very clean, very consistent dose responses, statically significant and the line graphs really kind of says it all in terms of the difference between those curves, not just at one point, but across the experience of what these patients are achieving and then a durability 12 weeks thereafter.

So, with these data, we're committed to the Phase 2b/3 for CSU. Some of the things, as I sort of shared with more year-over-year as well is in terms of exactly what those dose levels will be and whether we'll run an active comparator in a head-to-head fashion, I think, is TBD as well, and?

Dominic Borie^ Yes, maybe to your -- the first part of your question about the differences or the potential differences between the two mg per kg and the four mg per kg doses. So, you're correct that there is a slight difference at week 22, but that may not be where it's the most relevant to look at it. I think we can agree, for example, we look at Slide 16 that these two dose levels seem to behave somehow similarly at week 12 and -- but maybe with some slight separation at week 22. What we think is really relevant is to look at week 28, after those, and that's where maybe we start unmasking this second mechanism by the fact that we are at the four mg per kg dose, really seem to have done something in excess of the two mg per kg.

Shao-Lee Lin^ And maybe to add on to that, just to reiterate that, that isn't an exposure issue because, again, these are antibodies that are -- have a regular monoclonal anti-body half-life of two to three weeks. And so, we're four to five half-lives out at week 28. We don't expect a lot of drug around. So, it's less about an exposure issue in our minds and more likely a biological threshold of the fact. And we obviously are biased given again the design of the molecule to have one explanation for all of this which is we know we're designed to turn off - - to have the potential to continue to utilize the natural mechanism to dampen new IgE

synthesis. We think as the total IgE complexes are formed as they stabilize as a total IgE complex, we have the benefit not only of sopping up the IgE, preventing it -- from binding to the high affinity receptor, but also utilizing that complex to be a high IgE state, if you will, and dampen new IgE production. And it's not sort of unreasonable to think that that has a threshold associated with it.

So very excited for CSU patients. We think, for food allergy patients, and then frankly, the CSU study being a complex disease and having this kind of effect gives us enthusiasm for other disease as like atopic dermatitis, allergic asthma, et cetera. So, sort of more to come as we continue to explore the biology and think about our study designs moving forward.

Dev Prasad^ Great thank you.

Shao-Lee Lin^ Thank you Dev. Really appreciate it.

Operator^ Thank you. And there appears to be no more questions in the Q&A queue at this time. Ladies and gentlemen, this does conclude today's conference call. Thank you all for your participation. And you may now disconnect.