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**Start of Transcript**

Bill Ketelbey: Thank you and welcome to all of you on this Actinogen Medical conference call. I'm Bill Ketelbey, as you heard, CEO of Actinogen Medical and we're joined today on the call by Dr Geoff Brooke, Chairman of Actinogen Medical and our three clinical advisory board members, Professor Craig Ritchie from Edinburgh University, Professor Colin Masters from Melbourne University and Professor Jeff Cummings from the Cleveland Clinic in the US. For those of you aware of time zones, this call is live and so a special note of thanks to Craig Ritchie; it's currently 1:30 in the morning in the UK, so thank you Craig for your commitment and for joining us.

We only have 45 minutes and I want to give you, our audience, as much time as possible to ask questions, so I'll start off with a short summary of where the business is and the very positive announcement that we had in the last week, the announcement on the interim analysis and the capital raise and then ask each of the speaker to say a few words of introduction and open up to questions. I've uploaded the investor presentation to our website and to the ASX and you're welcome to reference that. Obviously we don't have time to be able to go through the presentation in any detail at all, but it is there for your reference and if a question does come up, it may be referenced in answering any of the questions.

So, Xanamem is our lead asset, as you're well aware and we began the development of Xanamem some three-and-a-half years ago. The drug has always intrigued me, whether it's the science or the immense opportunity that sits behind Xanamem and I'm pleased to say that we've progressed with the development, progressed really well with the development and as we've progressed and as we've received results from the various studies over the years, the drug has only continued to prove itself to all of us. It's a first in class oral brain penetrant drug, so that is a key feature; it gets into the brain.

Most drugs don't get into the brain. This one was specifically designed to get across the blood brain barrier in concentrations adequate to inhibit the key enzyme that we're trying to inhibit, the 11β-HSD1 enzyme. This enzyme is concentrated particularly in hippocampus and frontal cortex of the brain, areas that are very impacted, primarily impacted by Alzheimer's disease. Our hypothesis is that by inhibiting that production of cortisol in the brain, we can positively impact Alzheimer's disease.

Our drug is clearly differentiated from the many drugs under development for Alzheimer's disease. We are only too well aware of the reports of drugs that have failed in recent times; many of them related to amyloid clearance or prevention, some of them related to tau, others related to 5-HT6, so there have been a number of failures. But significantly, Xanamem, our drug, has a mechanism of action that is substantially differentiated from any of those other three drugs. We have progressed well with the development to the point that we've dosed over 100, well over 100 patients with Xanamem now and the safety profile continues to look very promising.

All of the studies that we've undertaken to date, the drug has behaved exactly as expected, exactly as predicted from early studies, including our animal work, whether that's safety or whether it's the pharmacokinetics, the absorption, the metabolism of the drug, it appears to behave exactly as expected. So we confidently went forward to start XanADu, our Phase II trial in Alzheimer's disease and we began that pretty much exactly a year ago today; we got our first patient in mid-May last year.

But a significant feature of Xanamem is the mechanism of action through the inhibition of cortisol opens up the potential for an array of other indications. So Alzheimer's, while it's our key target for development at the moment, is just one of the indications, the potential indications for this intriguing drug and I'll speak to that a little bit further on. So we are in an extremely exciting time, particularly following the announcements over the last two weeks and the positive development that we've achieved to date.

Talking of the announcements over the last two weeks, the interim analysis was the first announcement to come out. Now the interim analysis, we announced the intention to undertake it in November last year, it was undertaken on the first 50 evaluable patients, although an addition 37 patient safety data was also included in that analysis. The data was both efficacy and safety data evaluated to ensure the integrity of the trial, both now and going forward. We ensured that the data was evaluated and analysed by an independent data safety monitoring board, so the company, the researchers, the patients, nobody saw the un-blinded data.

The only group, the body, to see the un-blinded data, was the DSMB, the Data Safety Monitoring Board and they recommended, having reviewed the un-blinded data, they recommended continuation without modification, which to us was a very pleasing outcome because it reflects a confidence the safety of the drug and the trial's design and the assumptions we had made in designing the trial and in the statistical model that we'd used to develop the study. So, very pleasing outcome and it gives us confidence to move forward and it supports the broader development of the drug. Importantly and significantly, no treatment related side effects were identified during that interim analysis, during the analysis of that data, so a very pleasing and encouraging outcome from the interim analysis.

On the same day, so this was Wednesday last week as we announced the recommendation of the interim analysis, we also announced a very significantly institutional investment in Actinogen, so these two announcements came out together. There are a number of particularly significant features around this investment and the fact that it was concurrent with the interim analysis. Significance in who invested in us, so the investment was made by BVF, Biotechnology Value Fund from the USA, a leading specialist biotech investment fund; Platinum Asset management from Australia; and Australian Ethical Investment, another Australian institutional investor, all three of them highly respected, conservative institutional investors.

What this investment reflected was a maturing of the business. We are now a business that institutions are investing in. We've moved beyond the retail investors of earlier days and we now have our register dominated at the top end by institutional investors. In fact, once the full placement is complete and you would have seen by the announcement that we have Tranche 1, Tranche 2 and an SPP, once that is all complete, the expectation is that the top four shareholders for Actinogen would reflect the three institutions: BVF, Platinum and Australian Ethical and Edinburgh University. Now to me, that is a very solid, very profound register and reflects the quality of investors that we're after.

But other significance about the investment, that BVF is and American based fund, but has a history of backing Australian biotech winners and none more so than Viralytics. For those of you who are aware, Viralytics has just been acquired by MSD, Merck Sharp and Dohme, the huge US-based pharmaceutical multinational, for $500 million and BVF was a very substantial investor in Viralytics. The timing of the investment, as I've said as well, also very significant, that it was concurrent with the interim analysis announcement and it reflects the due diligence that was undertaken by BVF. Ultimately, I guess the quote that came from Mark Lampert, the founder of BVF, that he was impressed by the calibre and dedication of the management team, board and scientific advisers.

Just speaking to that, during the due diligence undertaken by BVF, they asked obviously multiple questions. We had dozens of phone calls with them, but we obviously also put them in contact with our advisers, including members of our advisory board on the call now and other scientific advisers involved in the development and discovery of Xanamem. They came back with a very deep impression of the quality of the scientific advisers underpinning the development of Xanamem.

The size of the investment and the premium of the investment, very significant reflection on the investment that was made: the size of the investment, $15 million, across the three institutions, of which BVF is taking $10.5 million and they will have 19.9% ownership of Actinogen, a very substantial investment. The investment was done at a premium, 13.4% premium to the share price, the VWAP at the point in time, again a very substantial reflection of the quality and the confidence that BVF had in Actinogen. The use of the funds is also significant and I will come to that in a few minutes and all of that together points to a substantial validation of Actinogen and Xanamem's potential and recognises and endorses the strategy that we are undertaking in the development of Xanamem.

Now I would make the point that this funding goes on top of the funding that we raised in November last year. I have had investors email me and phone me and say, but I thought you said you had all the funding you needed. Yes, we do. We raised the capital in November last year to fully fund the Alzheimer's trial. But developing any drug requires more than just one trial and this investment by BVF, Platinum and Aussie Ethical, goes a long way to supporting these important additional studies that we're undertaking.

Speaking of those additional studies, we want to enhance and strengthen our dataset beyond just the Alzheimer's work. We want to add substantial value to our asset and by undertaking this work, that's exactly what we'll do and we'll value add to our discussions with big pharma and as I'll speak in a few minutes, we're going to BIO International to meet with big pharma next week. Importantly, these studies follow feedback that we've received in our conversations with the biotech community in recent years, where they've indicated there are data points or data studies or information additional that they would like. So target occupancy study and higher dose study, to optimise the dosing of the study, new indications will be looked at and a range of toxicology studies, as required by regulators in any drug development program, so all of these studies will round out our dataset beautifully.

I've already alluded to the fact that my head of business development and myself will be at BIO International next week, in fact, in Boston. It's the largest, most prestigious biotech convention and we'll be showcasing Xanamem and Actinogen there and presenting to the meeting and meeting with big pharma. We attended the same meeting last year and at that time had one patient on trial. We're now going to the meeting being able to say we have at least 101 patients on trial. So very substantial difference to the message we had last year.

Speaking of patients on the trial, we've made very significant progress with XanADu. We have 101 patients on the trial, but I'm led to believe another two patients are being enrolled as we speak, so by the time I get to BIO, I'm expecting to have upwards of perhaps 105 patients on the trial. This trial very significantly is on track to finish enrolment in the fourth quarter of this year and readout data from the trial within 12 months. So the next 12 months for us is hugely significant, both with the new studies, as we've alluded, so the significant investment that we've received, but most importantly, to the readout from the XanADu trial.

So I've gone on a little bit longer than I wanted to, but I think, I hope, I'm sure it's given you a real reflection of the exciting time that we're in now and the very significant 12 months that we have ahead of us. But let me, before we go on to open the lines, ask Geoff Brooke, the Chairman for Actinogen Medical, to say a few words before handing over to the advisory board members. Geoff.

Geoff Brooke: Very good, thanks Bill. It's Geoff Brooke here, some of you I know, some of you I don't. I'm the Chairman, I've been Chairman for just over a year, although I've known about Actinogen's technology and drug product for some time and I'm very pleased to be now Chairman and think that in the past 14 months, since I've been Chairman, we've really achieved some significant changes, positive changes for the Company. I'd like to thank everybody for joining this call, especially obviously our overseas participants, whether they be investors and shareholders or obviously our advisory board members that have called in from overseas, Craig, Professor Ritchie and Professor Cummings. Also thanks to Professor Masters from Melbourne.

It's obviously a very exciting time for the Company. We've been enrolling patients, as Bill said, for 12 months. Just out of investing and probably 40 or 50 early stage biotech companies in my 30 years in this industry, this is one of the few ones that I've had that has actually enrolled patients pretty much on schedule. Some of you may be aware that really is quite unique, so that speaks a lot for what we're trying to do.

It's a very exciting time, as Bill mentioned, because we had our interim analysis performed by the DSMB and we're really pleased, we're thrilled actually, with the result from that. Then obviously the third thing I wanted to mention was the placement. I hope you can appreciate how significant it is for us to get the three institutions invested in the Company. This Company, until last week, was a 100% private investor owned company and it's now transformed into a company that's about to close enrolment on a significant global clinical trial in Alzheimer's with absolutely premier institutional investors as the lead shareholders now. So I hope you do appreciate how significant this last week or so has been for us.

I hope everybody gets as much information and value from this call as they are looking for and please, once we've finished with our advisory board members introducing themselves, I would encourage you to ask as many questions as you would like. Thanks, Bill.

Bill Ketelbey: Cheers, thanks Geoff. I'd now like to hand over to the advisory board members to say a few words. These three professors are world leaders in Alzheimer's research and it's been an absolute pleasure and privilege to have them on our advisory board from day one. I'd first like to hand over to Professor Craig Ritchie over in Edinburgh. Professor Ritchie is a professor of psychiatry of the aged at Edinburgh University and chairman of the Xanamem clinical advisory board. He's chair of the Scottish Dementia Research Consortium and director of the Centre of Dementia Prevention at Edinburgh University. Craig, a few words from you please.

Craig Ritchie: Thanks very much Bill and thanks to everyone for joining on the call and welcome as well. I think one of the key things for me and one of the excitements I've had obviously working with Actinogen over the last few years, has been…

Bill Ketelbey: I'm sorry Craig, your line - are you able to get a slightly clearer line? Could you perhaps speak up just a little bit? Thanks.

Craig Ritchie: Oh sorry, is that any better?

Bill Ketelbey: Thank you.

Craig Ritchie: Okay, well I'll be brief then. All I was saying was that there is a huge excitement I think in the field around numerous developments that are taking place in the Alzheimer's space. I think one of the things over the last five or 10 years has been a recognition that there are probably a multitude of different targets which are going to be effective in Alzheimer's disease. We traditionally talked about amyloid and we talked about tau, but I think there is now clear recognition that throughout the course of the disease, other mechanisms, other pathologies are playing a critical part and I think that's really where there's a lot of excitement around cortisol and cortisol metabolism. I think to have a drug like Xanamem which clearly gets into the brain, has an incredibly potent pharmaco-dynamic effect, is really exciting [unclear].

Bill Ketelbey: Sorry Craig, I think we are having a line problem with you. It dropped out, Craig. I think for expediency, let me hand over to Colin Masters to say a word or two. Colin Masters is the laureate professor of dementia research and head of neurodegeneration division of the Florey Institute at the University of Melbourne. Colin, could you say a few words?

Colin Masters: With pleasure. Thanks Bill, it's a great honour to be part of your clinical advisory board. What excited me most about the concept is that here we are applying a new technology, a new pathway for disease modification. The science, the pre-clinical science that underlies inhibiting this enzyme in the brain which makes the brain cortisol is totally novel and new and never been tried before, but the science that underpins this is looking better and better as the years go by. So I'm very pleased to be associated and of course we look forward to getting some very interesting results as this Phase II study rolls along. Thanks.

Bill Ketelbey: Thank you. Thank you, Colin. Now over to Jeff Cummings in the USA. Jeff is the chair of the Neurological Institute at the Cleveland Clinic and professor of medicine in neurology at the Cleveland Clinic College of Medicine. Jeff, over to you.

Jeffrey Cummings: Yes, thank you Bill. I'll just say that I consult with a lot of companies and when I look at a drug, I'm looking for some very specific characteristics: is the biology well defined, which it is here; is the population right, which they moved this population or we moved this population back towards an earlier group of cognitively impaired patients; is there a biomarker so that you can see whether the drug is working from a biological point of view, does it get into the brain, we've emphasised that and are the dosing strategies reasonable. So as I look at the profile of Xanamem, I'm very impressed that with by putting our collective wisdom together, we've been able to come up with a program that is advancing well and is based on very solid drug development and biological principles.

Bill Ketelbey: Thank you. Thank you, Jeff, appreciated. Craig Ritchie, are you back online? Are we able to get a comment from you or still having technical problem? No, unfortunately silence.

Okay, well let me now open up, go back to the operator to open up the lines for questions so that we can get the Q&A from the audience. So back to the operator, thank you.

Operator: Thank you. Ladies and gentlemen, we will now begin the question and answer session. If you wish to ask a question, please press star/one on your telephone and wait for your name to be announced. If you wish to cancel your request, please press the pound or hash key. Your first question comes from the line of Derek Jellinek from Morgans. Please go ahead.

Derek Jellinek: (Morgans, Analyst) Great, thanks guys, thanks for taking the questions, I appreciate the call as well. Just from me, I guess I was wondering, Bill, if you can kind of walk us through or any of the clinical advisory board, the DSMB obviously had pre-specified assessments to go through. The un-blinded the data both on safety and efficacy, obviously everyone else outside them, it remains blinded, but can you share with us any information outside of there's no treatment-related SAEs, that's great, but anything on the efficacy side, specifically on the primary endpoints, ADAS-Cog and ADCOM? Anything as far as, because they have the baseline numbers, they have the 12-week data for the 50 patients, anything you can share on either front would be really helpful.

Bill Ketelbey: Okay, well let me start by saying we obviously have the blinded data, we don’t have the un-blinded data, so we are unaware of the outcome from that. The DSMB evaluated, while they evaluated the data, they provided - the recommendation they provided was, as we said, the recommendation to continue the trial without modification. Now the reason we have blinded the study and the reason we've been very diligent and rigid about maintaining the blind in the study is to ensure the ongoing integrity of the trial. The end points in Alzheimer's clearly are subjective end points and anything leaking from the blinding of the trial has the potential to obviously undermine the potential future patient coming into the trial.

So the DSMB recommendation to us to continue the trial without modification is the total content of our feedback on both the efficacy and safety. But what we can take from that is a sense of confidence that there is no treatment-related adverse events, there is no safety concern, there is no recommendation from the DSMB to make any changes either related to safety or patient numbers or patient profiles. So we take from that that the drug is safe in this patient

population and appropriately safe in this patient population and that the assumptions we made in the design of the trial appear to be correct.

We recognise that it was 50 patients, it is early on in the trial and there is a limit to the likely efficacy information that could be evaluated from that. But within those limitations, I think we've got a very positive sense that the trial is appropriately designed, going in the right direction and we look forward to the outcome from the full dataset in a year from now, in 12 months from now.

Derek Jellinek: (Morgans, Analyst) No, I do appreciate that detailed explanation, thanks, but I do realise the blind is maintained for integrity of the trial. But maybe you can answer this, perhaps: of the 50 patients, are the patient characteristics well balanced? I really wanted to know really of those, how many are on concomitant drugs, like the cholinesterase inhibitors or how many were treatment naïve?

Bill Ketelbey: Sure. That number, I don't have that detail to hand, but all of them, the patient profile, is classically early Alzheimer's, so MMSE 20 to 26, on current therapy, which is usually a cholinesterase inhibitor, who have now begun to plateau or decline on the effect from the cholinesterase inhibitor, so very classic, early Alzheimer's patient population and our trial is then offered on top of that current therapy. Now the assumption has to be that there will be a very reasonable, equal distribution across both the placebo and the active group for patients on cholinesterase inhibitors, but obviously I don't have that information at this point in time. We will clearly report that after the end of the trial.

Derek Jellinek: (Morgans, Analyst) Right, okay, so you don't have the information on MMSE specifically?

Bill Ketelbey: Yes, the MMSE as we have it now seems to sit at about 22.5. Now that was me just eyeballing the data, the blinded data, as I've seen it, so about 22.5.

Derek Jellinek: (Morgans, Analyst) No, that's good, because it does sit within the 20/24 mild dementia, which is great to see.

Bill Ketelbey: Yes, oh yes, no, those parameters, 20 to 26, are defined in the entry criteria to the trial.

Derek Jellinek: (Morgans, Analyst) Right okay and sorry, if I may, another one from me, obviously you have no treatment related serious adverse events, but maybe you can talk to were there any AEs at all and were they in line with expectations and were any doses missed, any patients withdrawn or any dropouts?

Bill Ketelbey: A few patients did drop out for various reasons, none withdrawn and clearly - well not clearly, none absolutely have been withdrawn because of side effects, no, that hasn't happened. The side effects that have been reported to us are the standard sort of side effects that one gets across all trials: headaches, a bit of nausea, nothing that in any way concerns us or alarms us and as we heard from the DSMB, nothing that concerns them in any way to indicate a modification to the trial was necessary.

Derek Jellinek: (Morgans, Analyst) I'm sorry, getting back to you said some patients did drop out, are those of the 50, I'm assuming, patients, how many did drop out and why?

Bill Ketelbey: It would be three or four. There were various reasons: there was one patient who was moving interstate; there was one patient who half way through the trial decided the impost on them and the caregiver was too great, so they elected not to continue. They're the sort of standard reasons people drop out of trials. We've built, into our 174 patient total, we've built a 10% dropout. We need 156 for statistics, evaluable patients, we've built a 10% dropout into that cohort which is a very standard, expected dropout rate in this type of trial.

Derek Jellinek: (Morgans, Analyst) Right and sorry, back to my first question was about the dose reductions, any one missed a qd dose?

Bill Ketelbey: Off hand, that information I don't have to hand. Knowing trials and knowing patients, clearly it will happen, but that is built into the protocol and will be reported as appropriate in the final case report form.

Derek Jellinek: (Morgans, Analyst) Sure. Sorry, I only have one more, sorry guys. Can you just remind us again, Bill, the powering of the trial?

Bill Ketelbey: So it's 80%, so 0.05. It is appropriately powered. We've had the biostatisticians work on the trial and ensure that this will end up an appropriately powered dataset and an appropriate outcome at the end. I'm sorry I think we probably need to move on to other questioners, because we are 10 after the hour at the moment.

Operator: The next question comes from the line of Mark Pachacz from Bioshares. Please go ahead.

Mark Pachacz: (Bioshares, Analyst) Hi Bill, thanks for your time everyone. This is a question for Jeff. Jeff, you talked about the dosing regime being or the dosing strategy being reasonable. I just wanted to, from a publication on this drug, the recommendation was that a dose of 20 mg a day is what would be effective even though you're trialling 10, so maybe do you think the current dose is a little bit low and are there plans for subsequent trials at a higher dose?

Bill Ketelbey: Perhaps Mark, was that - I just need to clarify which Geoff that was…

Mark Pachacz: (Bioshares, Analyst) That was for Jeff in the US.

Bill Ketelbey: Oh Jeff in the US, go ahead, otherwise I'll certainly add in my commentary there.

Jeffrey Cummings: Sure, be glad to hear from you Bill. Mark, dosing is one of the biggest challenges in these trials. The thing that gives me some confidence here is that the dose in the CSF is ninefold higher than the IC50. So even if - of course you're measuring in the lumbar space, not in the brain space, but ninefold should give us an adequate margin to know that we're achieving the IC50 or above in the brain. So I think the 10 mg dose, as it has played out in the pharmacologic measures in the CSF, gives you a fair amount of confidence that the dosing strategy is fine. You always would like to explore a higher dose range and also to find a dose response curve, but I would say that 10 mg dose is not in jeopardy of under-dosing.

Mark Pachacz: (Bioshares, Analyst) That's very helpful. Bill, can you elaborate perhaps on just with this funding, what other trials you might be doing and whether you will be exploring the higher doses?

Bill Ketelbey: Sure and the answer is yes, we will be exploring higher doses. So we're doing a target occupancy study and a higher dose safety study really to optimise the dose. So while we have modelled and Jeff has already alluded to the fact that 10 mg appears to be a correct dose, an appropriate dose, obviously it may not be the optimal dose and to ensure that we have a comprehensive dataset in time to initiate Phase III, we are undertaking safety - we will undertake safety studies at higher doses along with the target occupancy to ensure that we can optimise the dose of Xanamem.

Then additional to that, so that's one set of new studies, additional to that is a cohort of toxicology studies, as required by any regulatory authority for any drug development. We're moving into Phase III, so that's the second lot. The third lot will be the additional indication that I alluded to. Now because of the mechanism of action of Xanamem through inhibiting the 11β-HSD1 and cortisol production, it opens up the potential for use in a number of areas and one of the more intriguing and compelling proposals that was put forward to us by Craig Ritchie and the bigger endocrinology team, Brian Walker, in Edinburgh, looking at diabetes cognitive impairment.

Now we haven't yet firmed up exactly which indication to go for because the reality is there are a number of competing opportunities, but certainly we're undertaking the appropriate investigation, in fact we're undertaking a full systematic review of the literature to identify the best new indication to target as our second indication for Xanamem.

Mark Pachacz: (Bioshares, Analyst) Right. So can you talk about timing of that? Will some of these trials, maybe all of them, start in the second half of this year?

Bill Ketelbey: One of them has already begun. The target occupancy study is requiring a number of different steps and the first step is being undertaken by Sydney University. That is complete. The second step will be undertaken down in Melbourne and that is underway right now. It will go on to - the trial will include human dosing and that will initiate later this year. But all of these studies that I've alluded to, we've obviously had them in planning and we expect, we plan, we hope, to initiate them rapidly.

Mark Pachacz: (Bioshares, Analyst) Great, all right, that's very helpful. Thanks Bill. Thanks Jeff.

Operator: Your next question comes from the line of Matthijs Smith from Canaccord. Please go ahead.

Bill Ketelbey: Hello Matt? Matthijs? No.

Operator: Your next question comes from the line of John Hester from Bell Potter. Please go ahead.

John Hester: (Bell Potter, Analyst) Good morning and good evening to the folks in the US. I have a question for Professor Cummings actually. Professor Cummings, I would like to just get your view on where you see this drug sitting relative to various drugs that were tried for the treatment of β-amyloid, going back over the last decade or so. I'd like to just get the assessment of your confidence level as to how we stand today relative to what you perhaps thought of those other drugs, when they were at a similar stage.

Jeffrey Cummings: Yes, thanks, difficult question to answer briefly. I don’t think the β-amyloid hypothesis is by any means disproven, but I think the widening our repertoire of agents to try to treat Alzheimer's disease is very important and using the biology to drive that is, I think, key. A lot of the drugs have not been shots on goal, they've been shots in the dark because they didn’t really have target engagement defined or appropriate dosing defined and in some cases, as you've heard, even blood brain barrier penetration defined.

So my view is that this represents, in this kind of trial, this is a cognitive enhancing trial, of a drug that may have both cognitive enhancing and disease modification properties, but you can't chill both of those in the same trial, so the attempt here is to find a dose that will give us cognitive enhancement, that we can see in a short period of time and then to explore disease modification later. We have been successful in cognitive enhancing trials. The five drugs that are approved are all cognitive enhancers and so we know more about how to do that, we know more about how to measure it.

The measures that we're using are specifically sensitive to this phase of the disease. The ADCOM's analysis that we're using was developed specifically for mild Alzheimer's disease, so I think this is a well validated target, not validated by therapy, that's what we're trying to do, but validated from biological studies, both at the animal level and at the human autopsy level. This pathway, I think this is a very strong approach.

John Hester: (Bell Potter, Analyst) Well thank you very much and perhaps same question to Geoff Brooke.

Bill Ketelbey: Can you just repeat the question?

John Hester: (Bell Potter, Analyst) Yeah, sure. I was just trying to get an assessment of the relative merits of Xanamem versus the drugs, the various drugs that have been tried over the last decade for β-amyloid and so yes, Geoff, please.

Geoff Brooke: Yeah, sure, thanks for the question. Look I mean there are complex answers to your question and simple answers to your question, so maybe I'll stick to the simple ones because I like to think along simple lines. Having been a clinician and having prescribed, in the old days you did everything on a pad, a prescription pad, having written umpteen prescriptions for patients, I can tell you it's a lot easier to have a small molecule, small mass, in other words, 10, 20-odd milligrams in a plastic bottle in a medicine cabinet that you take one or two doses of a day to treat a disease, than to have a potentially more complicated type of treatment regime, such as an antibody infusion.

So I really think that is significant. I have direct experience with somebody, my wife actually, who has been through a whole course of different types of treatment. Unfortunately she's had all sorts of treatment for the condition that she's got and I can tell you, her preference by miles is just taking one table a day. So I think that is really significant and this is a chemical that is not inexpensive to manufacture now because we're only making relatively small amounts, but by the time we hopefully get to volume, it will cost - the cost comparison to other forms of treatment, like proteins and antibodies and the like, let alone the cost of administration, will be significant.

So that means it will be cheaper for the patient, it will be cheaper for the insurance companies and for the government payers and it will be far easier for the patient, particularly a patient who might forget where their car keys are. So it can be next to their tube of toothpaste, which presumably they probably won't forget to brush their teeth in the morning and I know that sounds incredibly, in some ways, silly, but I can tell you from having been a practising physician and prescribing things for patients, it would be much, much - we think it will be much easier. So I think that's probably in my mind, other than the complicated response of the pathophysiology that we're chasing and the pharmacology, that's a massive thing, advantage, from my point of view.

John Hester: (Bell Potter, Analyst) Good, thank you very much.

Bill Ketelbey: Thanks. I recognise that we've gone way over time. We have a number of written questions that have come in as well. Geoff, is there one that you want?

Geoff Brooke: Sure.

Bill Ketelbey: So we've had a written question, if I might hand over to Geoff, he can speak to it.

Geoff Brooke: Yes, we have two questions and it's more to do with the placement and the first question is if the share price remains below $0.05 during the offer period, I think that refers to the share purchase plan that we have in place, where investors are being offered to purchase up to $15,000 each at $0.05 to add $2 million to the $15 million that we've already secured, presumably - so the question is: the share price remains $0.05 during the share purchase plan, presumably no funds will be raised by the retail issue.

Look that remains to be seen. We think that some should still by the shares. Obviously $2 million, if a group went on to buy $2 million on market, it would push the share price way up, given that our daily turnover is less than $100,000. So the ability to buy such a large amount of money at $0.05 actually is an advantage. There's no commission, so you save on the commission as well, plus you know what price you would be getting, whereas if you sat in the market and everyone bought $15,000 on market, the price would go way higher, we feel, presumably.

The second question is: will the shortfall of $2 million have any noticeable impact on operations? In other words, if we don't get the SPP, will it have any noticeable impact on our cash flow? No, it won't. The extra $2 million would build strength in the Company, adds to our ability to do some of these additional studies that Bill mentioned and also builds in a little extra padding on contingency. So we don’t think it will have any serious impact.

Bill Ketelbey: Thanks. Thanks Geoff. I appreciate there are a number of you still on the line wanting to ask questions and we certainly have got other written questions here that have come here, but considering that it is getting on for 50, 55 minutes that we've been online, I think we do need to wrap it up. So what I'd suggest is we will respond, if you can email your question, the link is on the website and on the announcement about the conference call, please email us and we will certainly answer your questions for you. But I feel we really do need to wrap up at this point in time and what I'd like to do at this time is find out if there are any final quick comments from any of our advisory board members. Let's reverse the order this time. Jeff, do you have a final comment before we close off?

Jeffrey Cummings: No, I think we've covered this very thoroughly, Bill, so no final comments.

Bill Ketelbey: Great, thank you. Colin, a comment from you?

Colin Masters: That I'm still very enthusiastic because from our clinical studies in normal individuals or people who are not on treatment, their plasma cortisols are increased in Alzheimer's disease and for the first time we now have a drug that is directed at the brain that can lower this biomarker and it's going to be very interesting to see what effect that has on cognition.

Bill Ketelbey: Thank you, Colin. Craig, at 2:30 at the moment, a final comment from you.

Craig Ritchie: Well two comments, number one an apology for the dreadful phone line from the UK, so I apologise for that. No, I just think that it's really exciting times leading up to quarter three 2019 when we get top line results. I think this is a really exciting target clearly, very biologically well-known target in terms of cortisol metabolism in a field where we really are welcoming these targets away from just β-amyloid and tau. So I think we're set there for a very exciting next 12 months as we finish off the trial and then really looking forward to these results and getting on to the next study.

Bill Ketelbey: Thank you. Thank you, Craig. Geoff, Geoff Brooke, no final comment? No. Okay, so then a few final words from me. I trust this call has provided you with a sense of confidence and excitement that we have in the business and you've heard from the advisory board members their hopes and expectations that this new technology, that Xanamem, that our trial presents. As you've heard, we're well set, incredibly well set for the next 12 months ahead of us with the interim analysis done, the capital raise completed by very substantial institutional investors, providing the capital necessary to round out our dataset in the lead up to Phase III, so a very exciting time for us.

This time next year will be really a watershed for us. We'll be reading out on the XanADu trial on the Alzheimer's trial. We by then should have feedback and results, if not then, soon after, for some of the other studies that we're initiating and all in all, this time next year we will have a very resounding message to be able to give to the market about cortisol and cortisol inhibition in Alzheimer's disease and the future of our business moving forward.

So thank you very much indeed for your involvement. We eagerly await the results in the next 12 months and if Xanamem can be shown to be an effective treatment for Alzheimer's, as you've heard, it truly will represent a globally significant breakthrough in managing this disease. So in closing, a very significant thank you to all of you for your support, thank you to the advisory board for being online and thank you for all of you in helping move Xanamem development forward, a drug, an incredibly important drug in development and a drug that may well prove to be incredibly important in the treatment of Alzheimer's disease going forward.

I look forward to speaking to you in 12 months' time when we have the results. So to all of you and to the advisory board, thank you and good bye.

**End of Transcript**