UNITED STATES
SECURITIES AND EXCHANGE COMMISSION

Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d) of
The Securities Exchange Act of 1934

Date of Report (Date of earliest event reported)

August 16, 2006

VIVUS, INC.

(Exact name of registrant as specified in its charter)

Delaware	**000-23490**	**94-3136179**
(State or other jurisdiction of incorporation)	(Commission File Number)	(IRS Employer Identification No.)

1172 CASTRO STREET
MOUNTAIN VIEW, CA 94040
(Address of principal executive offices, including zip code)

(650) 934-5200
(Registrant's telephone number, including area code)

N/A
(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (see General Instruction A.2. below):

o Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

o Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

o Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

o Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Item 8.01. Other Events.

On August 16, 2006, VIVUS, Inc. conducted a Qnexa program update conference call. A copy of the transcript of the conference call is attached hereto as Exhibit 99.1.

The information in this Form 8-K and the exhibit attached hereto shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or incorporated by reference into any of the Registrant's filings under the Securities Act of 1933, as amended, or the Exchange Act, except as shall be expressly set forth by specific reference in any such filing.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits.

Exhibit No.	Description
99.1	Transcript of VIVUS, Inc. Qnexa Program Update Conference Call on August 16, 2006, 10:00 a.m. EDT

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

VIVUS, INC.

By:/s/ Timothy E. Morris
Timothy E. Morris
Vice President and Chief Financial Officer

Date: **August 18, 2006**

EXHIBIT INDEX

Exhibit No.	Description
99.1	Transcript of VIVUS, Inc. Qnexa Program Update Conference Call on August 16, 2006, 10:00 a.m. EDT

Exhibit 99.1

VIVUS.COM

Moderator: Timothy Morris
August 16, 2006
9:00 am CT

Operator: Welcome to VIVUS Incorporated's Qnexa Program Update conference call. Joining the call from VIVUS are Lee Wilson, President and Chief Executive Officer, Dr. Thomas Najarian, Principal Scientist and Inventor of Qnexa, Peter Tam, Senior Vice President of Product and Corporate Development, and Dr. Wesley Day, Vice President of Clinical Development.

Also joining the call, from Duke University Medical Center is Dr. Kishore Gadde, Principal Investigator for the phase 2 Qnexa study.

At this time all participants are on a listen-only mode. Certain statements and comments made during the course of this conference call, including responses to questions, are forward-looking within the meaning of the Private Securities Litigation Reform Act of 1995.

These statements may be identified by the use of forward-looking words such as "anticipate," "expect," "project," "believe," "forecast," "estimated" and "intend," among others. These forward-looking statements are based on VIVUS's current expectations and actual results could differ materially.

There are a number of factors that could cause actual events to differ materially from those indicated by such forward-looking statements.

These factors include but are not limited to substantial competition, uncertainties of patent protection and litigation, uncertainties of government or third-party payer reimbursement, reliance on sole source suppliers, limited sales and marketing efforts and dependence upon third-party risks related to the development of innovative products, and risks related to failure to obtain FDA clearances or approvals, and noncompliance with FDA regulations.

As with any pharmaceutical under development, there are significant risks in the development, regulatory approval and commercialization of new products. Head-to-head clinical trials of newproducts being developed by VIVUS, and approved products or other products in development, have not been completed.

Comparisons made to other products and the results of their studies are based on published data. And while we believe the data and comparisons to be correct, there can be no assurance that the published results are in fact accurate or that a direct head-to-head comparison in a clinical trial format would not result in a different conclusion.

There are no guarantees that future clinical studies discussed in this call will be completed or successful, that any product will receive regulatory approval for any indication or prove to be commercially successful.

VIVUS does not undertake an obligation to update or revise any forward-looking statement. Investors should read the risk factors set forth in VIVUS's Form 10-K for the year ended December 31, 2005 and periodic reports filed with the Securities and Exchange Commission.

Following the speakers' prepared remarks we will hold a Q&A session. To ask a question, please press star followed by 1 on your touchtone phone. If anyone has difficulty hearing the conference, please press star 0 for operator assistance.

I will now turn the conference call over to Mr. Lee Wilson, President and CEO. Please go ahead, sir.

Lee Wilson: Good morning and thank you for joining us. With us today on the call is Dr. Kishore Gadde. Dr. Gadde is Director of Clinical Obesity Trials Program at Duke University Medical Center and is considered an expert in designing and conducting clinical trials for obesity.

Dr. Gadde was the principal investigator for the Qnexa phase 2 trial and today will discuss the design and results of his study.

I know many of you have tried to contact Dr. Gadde with questions about the study and the data. And at the end of today's prepared remarks, you will have an opportunity to get answers to those questions.

This is your opportunity, and I call it a "rare opportunity," to have time with Dr. Gadde and to Dr. Najarian.

Also joining me today - joining us today is Dr. Thomas Najarian. Dr. Najarian is the Principal Scientist for VIVUS's obesity program. Dr. Najarian is a board-certified specialist in internal medicine, a graduate of both MIT and Harvard Medical School, and a former faculty member at Harvard Medical School.

He was the former Medical Director and Vice President at Interneuron Pharmaceuticals, now Indevus, a company whose primary focus was on the treatment of obesity and obesity-related illnesses.

Dr. Najarian has authored many scientific publications in such journals as the New England Journal of Medicine, The Lancet, Technology Review and Circulation.

He is the holder of several U.S. and international patents. Most importantly, Dr. Najarian has 25 years' experience in treating obesity. Dr. Najarian discovered Qnexa and licensed it to VIVUS in late 2001.

Dr. Najarian has successfully used this treatment in over 6000 patients and is here with us today to share his clinical experience with you. And he is also available to answer questions as well.

Peter Tam is also here with me today. Peter, as you know, is our Vice President of Corporate - Product and Corporate Development. Peter is an internal product champion for Qnexa and will be available to answer your questions on current status of the project and future development plans for Qnexa.

With that, we will turn the call over to Dr. Gadde. I would say when we open the call for questions, that I will - because we're in several locations here, I will direct the questions to the individual - the appropriate individual person.

And with that, I'll turn it over to Dr. Gadde. Thank you.

Kishore Gadde: Good morning. First of all I apologize, some of you have repeatedly tried to contact me and - well I'm a very busy clinician. I have about 300, 400 patients

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who are in clinical trials. And I also have research responsibilities and teaching responsibilities.

Just looking at yesterday, I - was about 8:30 or 9 o'clock by the time I went home. And so with this kind of workload, it's really difficult for me to get to all these email messages.

So this is a great opportunity that we have today to go over this study one more time. And since I was the principal investigator of the study, I'll be happy to answer all the methodological questions that you have, as well as any other questions.

With that background, let's get to the details of this study. This was a randomized double-blind controlled study with four treatment groups. We enrolled 200 subjects.

And there were four treatments and the treatments were topiramate, phentermine, placebo and Qnexa, the combination of topiramate and phentermine. The study duration was 24 weeks and after randomization we had a 4-week dose titration period. I'll get to that in just a minute.

As with all the other - all the obesity trials, the way they actually need to be done as far as NIH and the FDA guidelines are, all of these subjects are provided brief diet and lifestyle counseling at every visit. That includes the placebo subjects, the topiramate subjects, phentermine subjects and all the subjects in the study.

They were seen at baseline. That's the visit at which they are randomized to one of the treatments. And we saw them two weeks later just to make sure that

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they're tolerating the dose titration okay. Then we saw them at week 4 and from that point we saw them every four weeks until week 24.

The dose titration in this study is something that is a unique design and - well it's - VIVUS has decided that it's - you know, it's best not discussed in detail at this point. So we can get to that in the Q&A.

The primary endpoint in the study is the body weight - change in body weight over the 24-week period. And as with all the other obesity trials, secondary efficacy endpoints are looking at changes in blood pressure, waist circumference and changes in lipids.

And for safety, because we have phentermine, which is a sympathomimetic drug, we obtained an ECG at the beginning and at the end. Now, you know, there's always going to be concerns about any combination involving phentermine.

Just to give you the background, in the fen-phen combination you heard about the problems with the heart valve - the valvulopathy, mostly aortic and mitral regurgitation. And it's never been really shown in any scientific study that phentermine by itself had caused any of these problems.

But regardless, just to reassure ourselves that there are no concerns with this, we decided to use very highly sensitive M-mode 2D echo with color Doppler — the same thing that has been used in the various studies that examined the valvulopathy with fen-phen. So we did that at the beginning and at the end of the study.

I'm sorry, I should be saying "Next slide, please." Okay. We are now at Slide 5. The inclusion criteria in the study - we took subjects from age 18 to 60 years. They had a BMI of 30 to 50 kilograms.

Regarding the BMI, you need to understand that obesity drugs are recommended for use by the NIH and by the FDA in people that have a BMI of at least 30. Or in the presence of health risks such as diabetes or high blood pressure or dyslipidemia.

In those instances you can actually use these drugs for people with BMI of 27 and higher. So that - those are the criteria. In this study, because this - - the primary endpoint in the study is weight change, we decided that we should take patients with a BMI of at least 30.

And this is not any different from the RIO trial - the RIO-North America trial with 3500 subjects, which was published just a few months ago. So our criteria actually are very similar to the RIO trials of rimonabant.

In terms of exclusion criteria, we listed those there - that's on Slide number 6 - obesity of unknown endocrine origin, serious unstable illness, significant hepatic or renal disease, history of renal calculi, which is kidney stones - because topiramate has been known to have about 1% or so incidence of kidney stones, which actually - we had only one subject in the study with a kidney stone. And that turned out to be a patient who was on phentermine.

And we did not take patients with type 1 or type 2 diabetes, which is usually the case unless you're actually doing a trial with obese diabetic subjects.

We also excluded patients with thyroid disease. Hyperthyroidism is an exclusion - is a contrary indication for phentermine use and so is glaucoma, which is a contrary indication for phentermine use.

Next slide, please. And going to Slide number 7, looking at the other exclusion, history of pulmonary hypertension, it's - that's a very rare condition which affects about 2 in 1 million. So we really didn't expect to see anyone with that.

Any past history of valvular heart disease - so we actually screened out some patients who had questionable valve disease to begin with. I mean for example, we had someone who took fen-phen about ten years ago and never had an echocardiogram done.

We actually found that this person had valvular disease. And we had to exclude that person and arrange for that person to be followed up clinically.

And we excluded the patients who were taking other weight loss drugs, anyone with current major psychiatric disorder - when I say this though that - unlike with the trials done with some of the very emerging medications, we actually took patients who were stable on antidepressants.

In the rimonabant trials they excluded anyone who was currently taking antidepressants. In fact, if you have access to the product label that's approved by the European Union for rimonabant, it says under "warnings" that it should not be used in patients who are currently taking antidepressants.

This is not the case here. We actually took patients who are on antidepressants except that if they're on Wellbutrin. Or if we knew that they're on an

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antidepressant which really has significant effect on body weight such as the drugs that would actually cause weight gain such as Paxil.

We kind of made the decision to see if they had any weight gain since they were on the antidepressant. But we did this study in a very realistic fashion. We excluded patients with alcohol or drug abuse and anyone who is allergic to topiramate or phentermine.

Next slide, please. Looking at the characteristics of the study participants at baseline, we had 50 people in each group. And we had a total of 41 men in this study.

For those of you that are not entirely familiar with obesity clinical trials and the prevalence of obesity, the prevalence of obesity is not all that different in the (community sample) between men and women.

The prevalence of overweight right now is a little bit higher for men. But over time, you know, they will become obese and the numbers are going to be actually equal.

But it always happens like this. Whenever we advertise for a clinical trial, it's women who call in great numbers and women enroll.

And typically when you look at any obesity pharmacological clinical trial, you find that about 85%, 90% of these study participants are women, that - - probably because the weight gain bothers women much more so than it does men. And also there's society pressures.

But we actually made sure that we studied some men as well and that is the only stratification that we did. So at the time of randomization - we were blind, by the way.

You need to understand that until we completed the study and we had the data entered and the database was locked and at that point we could make no further changes, that's when we'd go and get the key to the randomization, like who was receiving what treatment. Until that point, we did not know what people were receiving.

But we had about ten men approximately in each of the treatment groups. And we had a 50 - so we had like 40 women and 10 men in each treatment group.

Looking at the age, this is very typical. A typical participant in any obesity clinical trial is a 40-year-old woman. And that's what you see here. There's no difference between the groups.

And looking at the baseline body weight, 105, 106, 107 kilograms, that would translate to about 220 pounds or so. That gives them a BMI of about 38.5, which is really very similar to the BMI of 38 in the RIO-North America study.

That's the most recent study that was released. That was back in - I think it was in February the study was published in JAMA with 3500 subjects. And the BMI in that study was exactly the same — about 38.

And race distribution was the same. There is no significant differences with regard to how people were assigned to different treatments.

Next slide, please. This is a really very impressive finding in this study, that we haven't had many patients drop out of this study. And I've heard that that

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was one of the questions that came from the analysts looking at the high retention rate — well why was it so high? I'll get to that as actually we look at the efficacy data. That easily explains why so.

Now looking at the reasons for early withdrawal, we only had - I think we had a total of 41 patients that dropped out, of which only four subjects, that is 8%, dropped out due to side effects with the Qnexa treatment - with the combination.

And of these, only one dropped out due to an adverse event. The patient complained of feeling cold all the time - a little chilly.

And as far as topiramate, we had three patients drop out with topiramate. One had memory problems, one had tingling sensations and one complained of swollen feet.

With phentermine one complained of rapid heartbeat, one had increase in headaches and we had one person who actually lost a significant amount of weight — about 35, 40 pounds — but had a kidney stone.

And we actually thought that we had a hunch - we thought that maybe this patient was receiving either topiramate or Qnexa. But when you broke the blind, we found out this patient was on phentermine.

Now it's interesting, as you see with placebo we had three patients drop out due to the side effects. So we had more patients drop out complaining of side effects with placebo. One had mood problems.

And I actually distinctly remember this patient said she really enjoyed shopping before. Now she would go to Nordstrom and stand there and think,

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"Oh, I'd suddenly start crying and become very tearful," wondering why she wasn't happy.

And we were really kind of concerned. "Is this patient getting topiramate or something?" Well when we broke the blind we found out that this patient was on placebo. And we had two patients on placebo complain of memory problems.

Now when you think about it, why patients with placebo could have side effects, it's very simple really, that whether we take a medicine or not, we go through changes in how we feel. We - sometimes our concentration is not good.

So if you are actually administering a drug in an open-label fashion, you would automatically assume that, yeah, this problem is related to a drug. But that's the beauty of the placebo-controlled trial, that when you do it you find out that even with placebo, some patients will have problems.

And a true side effect is something that occurs far more frequently than it would occur with placebo treatment.

Next slide, please. The next slide, Slide number 10, shows the main finding of this study. The main finding of this study is that the combination Qnexa - with Qnexa - by the way, this is the intent-to-treat analysis, including all the 200 subjects in this analysis with the last observation carry-forward.

By the way, this is the most conservative analysis. For those of you that are not completely familiar with the ITT LOCF analysis, what we do here is that supposing that you have a patient who has lost about 4 pounds at week 8 and

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decided to drop out of the study. Or had something come up, had to move to a different region, had to relocate and couldn't be in the study.

We would then make a - make the assumption that if this subject were to stay in the study, would not have lost any further weight. And so we put that 4 pound weight loss at the last visit in that particular study.

And looking at the other treatments, with topiramate you see a weight loss of about 14 pounds, with phentermine you see about 12 pounds and with placebo you see 4.8 pounds.

So that is a really highly significant finding. It's the kind of finding where - see, with some analysis that we do, we find that you have to do sophisticated analysis to really see the differences between the groups.

Sometimes you don't find - you don't get a very highly significant P value in certain analysis. Then you go and do a mixed models analysis, which has been - you find actually difference.

If you look at the rimonabant studies you find that there was a higher treatment effect when they did the mixed model analysis rather than the LOCF analysis. This is particularly the case in the RIO-Lipids trial, which was published in the New England Journal of Medicine.

So we actually went with the most conservative analysis here. A difference as large as this - now by the way, we've done the modified intent-treat analysis also, which is where you take only those subjects who took at least one dose of the medicine and had at least one post-baseline visit.

And then we also did the completer analysis. As a backup I also looked at various linear regression and mixed models. But no matter what we did, the difference as large as this would be just so obvious.

It's the kind of thing where let's say that you walk into a room and you have four groups of people sitting there. You can tell that one group looks very different than the other three groups. So it's a kind of thing where a statistician would be thrilled to have these kind of data.

Next slide, please. Slide number 11 pretty much says the same thing in a table format. In the intent-to-treat last observation carry-forward analysis, with Qnexa treatment you have a 25 pound weight loss, topiramate - 14 pounds, phentermine - 11.6 and placebo - 4.8 pounds. And when you look at the completers, it's really not all that different.

You know, in studies where you have a very high dropout, what you find is that people often present only the completer analysis because the completer analysis typically in studies where you have a lot of dropouts, inflates the treatment effect because you're only looking at the people who are doing well and have chosen to remain in the treatment.

Here - you don't really find a big difference here because we haven't really had many dropouts.

Next slide, please. Slide number 12 is a bar graph showing - that's a categorical analysis. This is like doing - looking at the proportions of subjects who have lost 5%, 10% and 15% weight and looking at the differences between the treatment.

Now looking at 5% weight loss, 82% of patients — 41 out of 50 — with Qnexa had lost 5% or more. Half of the subjects with topiramate lost 5%, 38% with phentermine and 14% with placebo.

So we actually provided some ancillary treatment. We did not completely neglect placebo. If we did that, you know, the study would be criticized for not really doing enough. So we had to do it to make the study look decent to the scientific world.

Now looking at 10% weight loss, half of the subjects lost more than 10% with Qnexa treatment, 16% with topiramate, 14% with phentermine and 8% with placebo.

Now an analysis which is almost never done and presented in most studies is a 15% weight loss because the weight loss here is really highly, highly impressive in this study.

So looking at those who lost 15%, ten subjects or 20% of the sample lost more than 15% with Qnexa. Nobody in the placebo group lost 15%. We had two subjects with phentermine and two with topiramate and ten with Qnexa.

Now this is in line with what I actually saw. I have actually seen all these patients. I mean that's the reason I said we have a compact program. We - two dieticians, who also worked - acted as study coordinators, followed these patients.

And I remember the time when they would send - they would have so much enthusiasm they would send emails to me saying, “Hey, I got somebody who lost 68. I got the star.” And my other coordinator would compete with her and say, “Look, I got a 74. I got the real star.”

And so this went on for months and months. So we had a number of subjects here who had lost 50, 60 pounds. And they would be walking around - because about 1/3 of the subjects that entered the study were employees of the university or medical center, we would see them in the hallways.

And they would go around telling all their friends that, “Look, you know, this is - I used to wear these pants,” and, you know, “I lost 5 inches in my waist.” So this was a study that we will remember for a long time.

Next slide, please. When you have weight loss, you also want to see reduction in abdominal obesity, which is measured as the waist circumference.

In the past, people would look at waist-to-hip ratios. But according to the NIH guidelines, waist circumference is actually the measure that correlates best with the risk for diabetes and coronary heart disease and various other problems.

In fact, in the World Health Organization-funded huge international study recently, what they found was that it’s not really the BMI that is the predictor of health risks such as diabetes and coronary heart disease. But it is really the abdominal obesity that has the best correlation with all the major health problems.

With Qnexa, as you see there, you have almost 13 centimeters, which comes to about 5 inches reduction in waist circumference. So if you have - if your jeans size is about, let’s say 42, now at the end of the study you’re wearing a 37 size jeans. Imagine how big that difference is.

Compared with placebo, you have a 6 centimeter - twice as much reduction in waist circumference with Qnexa. Just to give you a comparison, with rimonabant - with, you know, Accomplia - that’s currently being reviewed by the FDA. And actually it’s now available in some European (Union) countries.

In the four studies - four RIO studies - phase 3 studies with rimonabant, the average reduction in waist circumference is 4 centimeters relative to placebo. Here we have about 6.2 centimeters relative to placebo. So this is about one of the best results that you could expect in regard to waist circumference with any pharmacologically assisted weight loss.

Next slide, please. Next slide shows you the most commonly reported adverse events basically that occurred in any group, any - with any treatment, at an incidence greater than 10%.

Well the first couple of side effects that we listed there - actually the first three side effects — decreased appetite, increased satiety and altered taste — one could argue that they’re not really side effects. They’re desirable effects.

But, you know, what we do in clinical trials is when people come back, every visit we ask them typically, “Since you began the study, have you experienced any changes in the way that you feel? Or do you have any side effects?” We ask an open-ended question and let the patient report to us what they see as a change.

And people say, “Well I’m not hungry anymore,” which is the decreased appetite. And increased satiety, typically they say, “I sit down to eat. Before, I had to eat the whole pie. Now I have a little bite and I feel full. I don’t have to complete the plate anymore.” That is the satiety.

And the altered taste, some people say they have - said they had a metallic taste, particularly to carbonated beverages or chocolate. They say, “Well I don’t really have that taste anymore that I used to have. So that’s good for me. I don’t eat those foods as much.”

But in clinical trials, regardless of whether we think it is attributable to a treatment, we just go ahead and mark that side effect has occurred.

So decreased appetite was seen more often with Qnexa treatment. Forty percent of them clearly reported decreased appetite compared with 8% with placebo.

So you can think of this as a mechanism by which this drug is actually working for them is by decreasing appetite and increasing the satiety. And there’s also some alteration of taste that you see there. Sixteen percent of the sample reported that.

Paresthesia - for those of you that are not familiar with the word “paresthesia,” it’s a medical jargon for tingling sensations. These are probably driven by topiramate. In all the topiramate studies paresthesia has been the most commonly reported side effect.

These are typically presented by patients as sort of tingling sensations. They would last for a few seconds. They would feel them at the tip of their fingers or the bottom of their earlobe or the tip of their nose.

As long as you explain to them at the beginning of the study — in fact, we have this in the consent that they could experience this, particularly in the titration phase of the first four weeks — they're fine with it.

We don't have - I mean looking at the dropouts, we only had one person that dropped out citing the tingling sensations as a reason. And that was with topiramate-alone treatment.

So this is - people report this. But sometimes they actually report this with amusement rather than distress. That was seen more frequently with Qnexa treatment - 38% - and not surprisingly, 32% with topiramate, and not so with phentermine or placebo.

Increased urinary frequency - again, this was a very mild side effect. And it might have been related to the fact that people - we ask people to drink plenty of water — at least eight glasses of water a day. And that's something that we do anyway as part of the diet plan. And so that might have been the reason but it occurred a little bit more often with Qnexa treatment.

Headache - not much different - not much difference between the groups. Phentermine had a little bit more.

Memory problems - this is an interesting finding, that 12% of them had memory problems with placebo, 8% with Qnexa and none with phentermine and 4% with topiramate. These were generally very mild.

People say, "I'm not sure but I think, you know, I missed the directions a couple of days ago. I'm not sure. I might have been preoccupied with something." But we don't really try to make attributions about it. We just go ahead and whenever we're in doubt, we just go ahead and list it as an adverse event.

Not surprisingly, insomnia was more common in the placebo group. We've seen that in some other studies as well. So Qnexa doesn't seem to cause insomnia.

Next slide, please. The highlights of this study were that Qnexa treatment led to approximately 11% weight loss in the ITT analysis — this is the most conservative analysis; 10.7% to be precise — whereas topiramate alone had 6.3% weight loss, phentermine - 4.6% and placebo - 2.1% weight loss.

Now looking at the proportions of patients losing at least 10%, half of the patients lost 10% with Qnexa treatment compared to only 8% with placebo and approximately 15% with topiramate and phentermine.

Now how do these data compare with what we know about the other drugs? One way of finding - because studies, you know, use slightly different methodology. Each study is different.

But if you look at the studies of one year duration using very similar criteria, taking data from phase 3 studies and looking at what is the weight loss relative to placebo, some people present the data as weight loss in kilograms. It's easier if we look at the percent change because the baseline BMI may be slightly different in each study - with each trial.

So looking at the placebo subtracted weight loss, that is how much more the drug - weight loss is there with the drug relative to placebo, it's 8.6% at six months. So 12 weeks with Qnexa.

Accomplia, rimonabant, had four RIO trials done - four phase 3 trials. And in the four trials, in RIO-Europe the weight loss was 4.7 kilograms relative to

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placebo, in RIO-Lipids it's 5.4 kilograms and RIO-North America it's 4.7 kilograms.

And the RIO-Diabetes trial, which has been presented but the paper is not published yet, it's 3.9 kilograms. It averages out to about 4.7 kilograms or about 4.8% at one year.

Now with Meridia, which is sibutramine, there was - there has been a recent meta analysis of one-year studies. And the weight loss relative to placebo is 4.6%.

Xenical, orlistat, seems to be about the weakest of all these weight loss drugs that we have. Relative to placebo the weight loss is only about 2.9% at one year.

Last slide. Slide number 17, the last slide of my presentation, gives you a summary - the highlights of this trial that we conducted.

Fifty percent of patients lost 10% - at least 10% body weight in 24 weeks with Qnexa treatment. And 92% of patients on Qnexa completed the study. That speaks about the tolerability of this treatment.

And we have not had any severe or serious adverse events on Qnexa. We had one patient who had a kidney stone. That patient actually received phentermine. That was the only severe adverse event that we had in the study and that was not on the Qnexa.

At this point what we see here is that my sense is that a difference as large as this kind of treatment effect, I'm very confident that it's going to be replicated

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in studies of larger sample and for longer duration. I have no doubts at all about it.

The - you know, the - regarding safety, the adverse events, if there are any adverse events that would occur - let's say 1 in 1000 patients - and they could be significant, that's the kind of information that we could never get from a study like this because we had 50 people in each group.

So a study like this would not pick up a rare, serious side effect. If there is something, you know, lingering there, something hidden there, we would not know from this study.

And those kind of things would be better assessed in phase 3 studies, which would typically - the FDA requires phase 3 studies to be of one year duration. And you need to have at least 1500 subjects on the active treatment.

And typically, sponsors would run trials - you know, two or three or four phase 3 trials with about, you know, 2000 to 3000 patients on the active drug and about 2000 or so on placebo. So that's - those would be the next set of trials.

So with this, I will stop here and I will turn the call back to Leland Wilson.

Leland Wilson: Thank you, Dr. Gadde. The purpose of Dr. Gadde's study was to meet the FDA phase 2 clinical requirements for safety and efficacy. And second, to confirm in a double-blind randomized placebo-controlled trial the results previously seen by Dr. Najarian in his clinical practice.

Dr. Gadde's results in the phase 2 study, in fact did closely match and therefore confirm the observational results seen by Dr. Najarian in his practice.

I now would like to turn the call over to Dr. Najarian for a few comments before we open it to questions.

Thomas Najarian: Thank you, Lee. I worked with VIVUS and Dr. Gadde to help design this trial, basically to mimic what I've been doing in my own practice for the last seven years and exclusively obesity for the last five years. And I have treated now over 6000 patients with the combination in similar doses to what were done in this trial.

I have a few patients - one as long as seven years - but many, many patients on treatment for as long as four or five years. The clinical experience that I get in my practice is comparable to the results seen at Duke and the Qnexa phase.

I'm not a genius at getting people to lose weight because I've been trying to do it for 25 years. And without good medication, as much encouragement as you might give a patient with diet and exercise and various diets, they don't work well long term.

If you look at the slope on the Qnexa results, you'll see that it's pretty steady down from about months 2 to 6. And in my own experience, that will continue in patients who need to lose that much weight, right up to month 12.

And then after that you continue to lose weight — maybe not at quite the severe of slope as you see in that study. But that's rather unusual for any medical treatment of obesity.

I have - if people need to lose as much weight as 20%, 30%, they'll continue to lose weight even two to three years. And I can keep them stable on the weight loss if they've reached a good weight with lower doses of the same combination.

I've also seen in my practice, and as you might expect but not necessarily true - for example, Meridia, although you get weight loss, you don't have lower blood pressure. You don't have improvement of risk factors with cigarette smoking and you have early death with cigarette smoking but you get weight loss.

But normally you'd expect - with good weight loss you'd expect reductions in other complications related to obesity. And in my practice I have seen improvements in a number of areas, in addition to what's shown on the slide. And these are all obesity-related problems.

And that would include the obvious cholesterol improvements, improvement in HDL, lower LDL, lower total cholesterol, lower triglycerides, reduction in blood pressure to the point where patients can get off of blood pressure medications, improvement in insulin sensitivity to the point where patients who are taking both insulin and several oral diabetic agents including, for example, Byetta - able to get off all medications and have normal blood sugars on the program and keep the sugars normal.

In addition to that, you see improvement in coronary artery disease symptoms, angina, improved exercise tolerance. There's presumably a reduced risk of stroke because you lower blood pressure.

I've seen patients with congestive heart failure in my practice who take this treatment, have improvements in ejection fraction. This is not completely

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unseen because you've seen some of these results if patients have various risk factors who have had gastric bypass surgery, some of these improvements you also see.

You also see improvement in fatty liver disease, which is the major cause of cirrhosis, polycystic ovary disease and infertility, sleep apnea, fatigue because they're not - they're sleeping better at night. They're not so sleepy during the day and they don't have as much fatigue during the day.

Asthma is weight related. You see improvements in that. Even psoriasis, which is partly weight related but there may seem to be some separate effects on topiramate in psoriasis.

And also there's improvement in other risks, in particular mood. A number of people have asked me about depression. Topamax alone in higher doses can sometimes aggravate depression. We had depressed patients in this study. They were not excluded in the Qnexa trial and there was no sign of decreased mood.

In fact, a lot of people with obesity, and I'm sure Kishore has the same experience of treating patients, actually have pretty mood. They feel that their condition is hopeless. They often feel distressed because they can't control eating. There's not much they can do to help themselves.

And once they're taking something that actually helps them control eating for the first time, in most cases their mood is much, much better. They have an improved lifestyle and able to exercise when they weren't able to before because their arthritis is better.

So I'll end my comments there.

Lee Wilson: Okay. Now we're getting into the real important part of the - today's program. And that is open for questions. And so please, I encourage a very rousing round of questions. So let's go.

Operator: If you would like to ask a question at this time, please press star then the number 1 on your telephone keypad. We will pause for just a moment to compile the Q&A roster.

Your first question comes from Michael Tong with Wachovia Securities.

Michael Tong: Hi. Just a quick question. Can you disclose the doses of the different treatment arms?

Lee Wilson: Michael - - Lee here. And I'll take that question. No, we're not going to disclose the doses. That's part of our strategy to prevent generic competition going forward. And so we'll not disclose that at this point. We will, obviously at some point.

Michael Tong: And if I can just follow up on that, I mean with respect to that being a generic defense strategy, what patent or idea say, do you have around the doses that you're administering right now?

Lee Wilson: Okay. I hear two questions in there. You know, one regards the generic strategy in general. And part of that, obviously is the patent situation. And let me just kind of go over a little bit the strategy, if you will, against generic competition.

You know, our pedigree at VIVUS comes out of ALZA Corporation, which made its life taking drugs which were generic and bringing them to the market

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in very successful fashion. And so Ditropan is a very good example of that - of many others that we've had.

But at ALZA they were able to develop a very clear strategy for how to prevent the generic competition. And I'll just take a second here to run down a few of these things.

The first key to this is that you have to offer patients a superior product over the generic combinations themselves. And in the case of Qnexa, we offer both a delayed release and a sustained release component to this drug delivery technology, which will offer us optimum safety and efficacy results for the program.

In addition, the QD dosing is a very convenient approach to it versus what - the complexity of taking the two individual drugs, particularly if you consider that there is a four-week dose escalation or dose titration portion of the study when people go on to begin with.

So it becomes very complicated. With the Qnexa it's a simple card, which is one tablet per day during the four-week dose escalation program. And so it's a very simple, easy way for them to get to their final dose.

The second one is that the doses in Qnexa are different than all doses, including those in the dose titration - are different from what is available generically. And pharmacy laws, many of you know, prohibit the substitution of products at different doses. So that's a second part of the defense against generics here.

The third is that we do have a patent, as you all know. It's a very strong patent. It has both composition and matter and methods of use components to

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it. And any use of this combination of phentermine and topiramate is directly in violation of our patent.

The fourth one is that the indication for Qnexa will be for the long-term treatment of obesity. And as many of you know, pharmacy benefits management groups actually have adopted a principle of not reimbursing for products that are not on-label for - obviously for a risk management basis from their standpoint.

So that's not universal but most, if not - most of the PBMs are in that category. So this will be the - Qnexa will be indicated for the long-term treatment of obesity. Neither of the components are.

And then the fifth and last one is one that we're working on - is that phentermine is currently a scheduled product. We believe that Qnexa will not be a scheduled product.

So all those things go into the strategy for prevention of generic competition. And maybe I'll ask Peter to talk a little bit about our patent situation. Peter?

Peter Tam: Yeah. Thanks, Lee. The patent situation is that, you know, when we first licensed the technology from Dr. Najarian, we went through a thorough review of the patents.

We are very comfortable with where we are with regard to our patent position, our ability to practice the combination. And that's why we're moving the program full speed ahead forward. And we'll be entering phase 3 sometime next year.

Lee Wilson: Okay. Next question, please.

Operator: Your next question comes from the line of Ken Trbovich with RBC Capital Markets.

Ken Trbovich: Thanks. I guess the question's really directed more to clinical aspects for those patients when they come off treatment. Do the rebound weight gains parallel what you see in placebo-treated patients as well as those on Qnexa?

And what does this tell us with regard to how these patients need to be managed longer term?

Lee Wilson: Well I hear two components to that, Ken. One is based on Qnexa. What is the characteristics after patients - weight characteristics after patients go off the drug?

And I think maybe Dr. Gadde can chime in at a point here and what he sees with other medications as well. But first, Dr. Najarian, what's your experience when patients go off of Qnexa and weight loss drugs in general?

Thomas Najarian: Ken, good question. Obesity is a chronic disease, just like diabetes, hyper tension. And you're not going to be able to take insulin for one month, get your sugar nominal, go off insulin and expect your diabetes to be controlled.

Appetite is a very potent mechanism. There are well over 20, perhaps as many as 50 different mechanisms in the human body that make you eat. And it's very hard to control that.

And typically when patients are on my program, they often say, "Oh, this is simple, all you have to do is just not eat," when they go on the medication and

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they're easily able to control their eating and lose weight. And many of them think they can just do this on their own.

Invariably if they go off the medication, they come back a year later having gained back half the weight that they lost. So I believe you need to treat this chronically.

And even with gastric bypass where you bypass - essentially remove 95% of the stomach and bypass 1/2 or 2/3 of the intestines, the maximum weight loss they get with gastric bypass is 12 to 18 months after the surgery. There's the weight gain of 1/3 to 1/2 on average over the next ten years. Some people gain back all of it.

So this is going to be a long-term treatment. And in my practice, if the patients stay with the treatment and then get to goal weight and go to maintenance treatment where I typically see them every three to five months and they're on lower doses of medications, with occasionally bumping them up to full dose medication, they can maintain their weight loss for a long term.

The only exceptions in my experience, if someone gained weight because of a single episode, for example a pregnancy, and they were normally thin or they had an injury and they couldn't exercise, some of those people can keep the weight off on their - with willpower, proper eating, diet and exercise for long periods of time. But that's very, very rare.

Lee Wilson: Dr. Gadde, do you have a comment?

Kishore Gadde: No. I completely agree with Dr. Najarian. He's explained the clinical experience quite well.

What we typically see is that when a treatment has been very effective in assisting patients to lose weight and maintain the weight loss, that treatment probably needs to be continued for a quite long period because in the absence of continued intervention, whether the treatment is pharmacologically driven or whether it's a lifestyle change such as using diet or exercise as the principal intervention, or even behavioral therapy, what you find is that when you stop the intervention completely, especially after six months or one year, people begin to regain at least some of the weight.

And that - we have not done this in this study. We haven't followed the patients at the end of study. But I would not be very surprised if we found out that some of them have regained some of the weight at least because that is something that we would expect with any treatment when it is stopped.

Lee Wilson: Okay. A number of investors have sent in questions so I'll chime in here on occasion with a couple of questions that investors have posed.

Dr. Gadde, you had a very low dropout rate in your study. Was that because you did some - gave some special incentives to patients to remain on the study or is it because of the drug?

Kishore Gadde: Well, you know, the biggest reason that you see dropout in obesity clinical trials is because they're not losing weight. That has been always the reason.

So typically, you know, obesity clinical trials, you have a greater number of dropouts in the placebo group because placebo is generally not as effective as a good active intervention. So sometimes it's recorded as a lack of efficacy when they clearly tell us, "Okay, I'm dropping out of the study because it's not working for me."

But other patients, they don't want to tell us directly on our face. So they come and say, "Well I have a lot going in my life. I have stress and my car is not working and my dog is sick." They drop out. But we do know that that's because the treatment is not working for them.

The reason that - my guess is that the reason - I mean just talking to our study coordinators and the dieticians, the main reason — it's actually quite obvious if you look at Slide number 12 — is that 82% of the subjects had lost at least 5% with Qnexa.

So when the treatment is being that effective, people remain in the study. And that's probably the main reason for the very high retention.

Keep in mind that high retention is a good thing in clinical trials. It actually reduces the burden of having to do unusual complex analysis trying to figure out what would have happened if the patient had stayed in the study. But I think the tremendous efficacy that we've seen with this treatment is probably the reason for it.

Lee Wilson: Okay. May I open it up for the next question, please?

Operator: Your next question comes from the line of Ian Sanderson of Cowen & Company.

Ian Sanderson: Good morning and thanks for doing this call. Maybe Dr. Gadde could comment a little bit more on the paresthesia side effect. And maybe speculate on why he thinks J&J terminated their study of topiramate alone, which I believe was for safety issues.

Yet, their drug seems to show - topiramate alone in this study showed a lower rate of paresthesia. And is the difference here maybe in the titration schedule or something else?

Secondly, you alluded to some titration issues that you might elaborate on. I don't know if there's any further elaboration you can provide.

And third, if you could maybe speculate on how the placebo response in this trial compared to that of the placebo arm in the RIO trials.

Lee Wilson: All right. Let's take them one at a time. Dr. Gadde, do you want to comment on the first part of it, please? And then we'll take it from there.

Kishore Gadde: Sure. Paresthesia or tingling sensations has been actually the most frequent side effect of topiramate in all the trials really.

In the trials in epilepsy and migraine prophylaxis, obesity and obese patients with diabetes, that has been the most frequently reported side effect. But then again, even in those trials we hadn't seen any - we hadn't seen a very high number of dropouts that were attributed to that particular side effect.

You know, I can only comment on the announcements made by J&J in regard to the reason why they terminated the program because that's publicly available information. It's not because of paresthesia. It's because of CNS side effects.

They said that the drug had unacceptable central nervous system side effects. They didn't elaborate on it but central nervous side effects that have been seen with topiramate in their trials are memory problems, concentration problems and some mood changes.

So, you know, one could speculate that those might have been the reason. And so that's - the paresthesias are not really that problematic. It's the kind of thing that people would experience in the first month, particularly in the first - particularly in the second week or third week during the dose titration, say.

It's not something that is there with them all day. It's something that they would feel for a few seconds and would go away. Sometimes people just describe to us as if they were kind of amused, that they're - you know, "This is funny. I had this."

You know, they could be - in a way to describe it, they are like mild sort of electric shock-like sensations. They're not as strong but they're like sensations that would be a bit, you know, unusual. You don't experience them.

But they go away typically - you know, we actually went back and looked at - did a detailed observation of when these side effects occurred. And in most cases have they gone - we rarely have seen anything - any such complaints beyond the first month. So we haven't - that has not been a major issue with this drug combination.

Now about the placebo response, the placebo response that we have of 4 - - about 5 pounds over six months, that is, you know, pretty much similar to what has been seen in pharmacologically assisted - pharmacological trials in obesity.

The placebo response - I can give you that in a minute. In the RIO trials in - just looking at the RIO-North America trial, which is the largest trial, at one year follow-up in the ITT LOCF analysis, those that received rimonabant 20

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milligrams had a mean weight loss of 6.3 kilograms. And placebo group had lost 1.6 kilograms.

So we are up there. So this is a trial which is very prototypical. And we actually - we were looking at the placebo response rates the other day in sibutramine. And even the dexfenfluramine, the drug that's been withdrawn, it's about the same.

At one year typically if you don't do very rigorous, intensive lifestyle therapy, you're going to see about 2% weight loss at one year with the placebo arm, which is - it's not really the placebo. That's the sort of low-key dietary intervention that you do. That's what you can expect with that.

In the orlistat trials, because orlistat is not a very effective treatment, they really had to - if you want to show 7%, 8% weight loss at one year with orlistat, which is Xenical, you really have to provide a rigorous ancillary lifestyle therapy.

Then you end up with - if you get 7% weight loss with Xenical, you're going to get about, you know, 4-1/2% or 5% weight loss with placebo because the intensive - because of the intensive lifestyle counseling there.

But in clinical trials where you are testing a treatment that you believe is quite effective and you really want to determine in a phase 2 trial particularly, the strength of the signal, it's best to keep the noise low.

So you keep the placebo intervention, the ancillary intervention relatively clean and you get about 2% weight loss. That's exactly what we had in this study.

Lee Wilson: Okay. Can I turn it - ask Dr. Najarian to comment first on paresthesia and then the J&J results? And I think probably importantly, why Qnexa has apparently a better therapeutic window?

Thomas Najarian: Yeah. Thanks, Lee. I actually totally concur with what Kishore just said about the paresthesias. They're usually seen in the first month as you're escalating the dose. They're very mild.

That is not the reason why the trials at J&J were discontinued and why their development discontinued. It was the mental slowing, as you said - as Dr. Gadde said. And that's really the big problem.

The reason why we see less here is because there is some complementary reduction of side effects of each drug, the phentermine and Topamax, versus the other.

You have - phentermine and other stimulants have been shown in some clinical trials to improve functioning in patients who have some reason to have mental slowing. And that's why the military often uses stimulants in patients that are awake for long periods of time, to keep them awake.

So we have less of that I believe, with the combination than you get at the same doses as J&J - at similar doses you would get less using the phentermine.

Paresthesia is a tingling of the hands and feet. It's nothing serious. If you tell the patients at the beginning - which I tell all my patients, "You might get this." It's usually transient. It goes away after you've been on that dose for a few days.

It's not serious. It's not something considered serious medically and it's a tingling of the hands and feet or other parts of the body that typically comes for a few minutes. If you drink a little water it goes away.

But the mental slowing is definitely better with the combination. I have patients who could not tolerate phentermine for some of the same side effects that people dropped out in this trial, including nervousness or anxiety or palpitations. They couldn't tolerate Topamax alone at a given dose because of mental slowing or just feeling tired.

You get the two of them together, that same patient that could not tolerate phentermine alone at a particular dose, could not tolerate Topamax alone at a particular dose, could tolerate the combination very well. And that's why this product works so well.

Lee Wilson: Okay. Let's take the next question, please.

Operator: Your next question comes from Ilya Kravets with Rodman & Renshaw.

Ilya Kravets: Hi. Thank you for taking the question. I had one question for Dr. Gadde and one for Dr. Najarian.

Dr. Gadde, can you maybe tell us about the dosing - frequency dosing regimen in the phase 2? And can you tell us a little bit about this four-week titration run-in period?

And then for Dr. Najarian, you mentioned that for some patients, after they've lost a great portion of the desired weight, after a year or two you put them on a maintenance dose.

Can you please tell us a little bit more about that? How do you titrate them down and what those, you know, dosing regimens may look like?

Lee Wilson: Okay. Ilya, as you know, we're not going to release the doses here. So what we would comment on as far as the titration is concerned, it is a four-week escalated dose titration of both phentermine and topiramate.

Ask Dr. Gadde to comment on why do we do dose escalation with these compounds and why is that necessary?

Kishore Gadde: Well why the dose titration necessary, it's simply because of topiramate. Topiramate has side effects which are more common in the first month. So we gradually have to increase the dose of that particular drug.

We didn't test different doses in the study. We tested one dose combination in the study but we gradually got them to the highest dose that we used in the study. And if patients could not tolerate the high dose, we could back off on the dose.

But, you know, because these doses - formulations we thought about, we discussed these and arrived at these based on a lot of clinical experience that Dr. Najarian had. We knew beforehand what's going to be tolerated well and we really didn't have to back off.

In fact, there's only one patient that I could think of where we had to reduce the dose a little bit for tolerability reasons. We got them to the dose that we wanted to use in this study.

Lee Wilson: Okay. Dr. Najarian, do you have a comment there, too?

Thomas Najarian: Yeah. On the phentermine, it's also helpful to titrate that because if you were giving phentermine alone, people do better as far as tolerability if you start at lower doses going higher doses. Same with Topamax.

As far as the maintenance, I go directly from a full treatment to maintenance, cutting the dose of both drugs. And patients can actually stop these drugs abruptly without any problems at all.

I've had to stop - in rare cases I've had to stop because of an allergic reaction or something like that, like a rash. But you can stop these medications. Patients are doing it on their own a lot.

I will stop them abruptly prior to the patients going through surgery, only because if they need surgery for something like an appendix or some other problem, if you - losing weight, you will not recover well from a surgical wound. You need to eat nutritionally to be able to repair tissue from surgery.

So I stop the meds abruptly all the time. There is no withdrawal side effects, which is why I believe this will be an unscheduled product.

And I go abruptly from a weight loss to weight maintenance, telling patients, "Don't be afraid. You're going to be a little more hungry at the lower doses. But you need to eat a little bit more than you're eating now in order to maintain your weight and not keep losing."

And so I don't have to titrate down. I go direct from a full dose to a maintenance dose.

Ilya Kravets: And then maybe just two follow-up questions (unintelligible) Dr. Najarian. What is the longest that you've had patients on chronic therapy? And maybe the number of patients.

And then for Dr. Gadde, you said that you were trying to get patients to this highest optimal dose. And then am I correct in understanding that out of the 50 patients, only 1 couldn't reach that dose?

Lee Wilson: Okay. Dr. Najarian, length of time on studying the number of patients that you have, et cetera.

Thomas Najarian: The very first patient that I treated, who used to weigh 450 pounds, the last I heard is still at about 250. He's a man of 6 (feet), 4 (inches). He's been on seven years.

He was the first one I gave because he had an epilepsy disorder. So I switched him from his current epilepsy drug to topiramate. He had previously been on phentermine and Redux.

When Redux was withdrawn he remained on phentermine alone. He did very, very well and he's continued to maintain a weight of around 250, which for him is actually not very heavy. He's a big guy.

But really I only had a limited practice up until 2001 when I went full time into obesity. And I was still working at Interneuron Pharmaceuticals, another company, so that I was not practicing medicine back seven years ago except for a few hours a week.

Now my practice has escalated exponentially. So I obviously have a lot more people who have been on treatment for a year than I do for people who've been on five years.

But I have a substantial number of people who have been on treatment for four or five years. And if they continue to see me and maintain on the treatment, they can maintain their weight loss.

Lee Wilson: Okay. I think I'll take the next question here. Let me just see. One of the questions that has been posed by investors is, Dr. Gadde, you have a reputation of getting excellent results in your clinical trial program.

Do you have something special about how you do these clinical trials that gets excellent results? Do you incentivize them to stick with a very severe diet and exercise program? And does that have any impact on the clinical results?

Kishore Gadde: This is a randomized double-blind clinical trial. And there's really not much we can do to influence the results of a randomized double-blind clinical trial because as the trial is going on and until everyone has completed the trial, until we have entered the data and reviewed the data, audited the data and locked the database, we will not have - we do not have access to the treatment assignments in a double-blind randomized clinical trial.

So there's no way that an investigator can influence the outcome of a randomized clinical trial. Now that could be the case if we were to present results of an open-label treatment. Then you could just - like the kind of advertisements that you see for these nutritional supplements.

They say that somebody took the fat buster and somebody lost 55 pounds. But they don't tell us what other treatments they received. They were working six hours a day in a gym.

But we didn't do anything - we did not provide an intensive ancillary treatment because if that were to be the case, you would have seen maybe a 10 pound to 15 pound weight loss with placebo. We only saw - we saw less than 5 pounds weight loss with placebo.

We - I mean the things that we might have done well is that we - you know, there are - we had - there are some places outside. These are freestanding businesses that enroll patients. And what they do is they have a database and they take patients who have been in several clinical trials.

We have not enrolled those kind of patients. We enroll patients that were relatively clean, that we would get from newspaper and flyer advertisements, people who have not recently received any other treatment.

So we didn't take patients who had lost 50 pounds in the last six months because we knew that there's really nothing else to prove. Even if they're a very good treatment, they wouldn't be losing further weight.

So we carefully take patients in our trials because it's not entirely a business-oriented operation. We want to do well. We want to be able to publish in good journals and we want to practice good science.

Lee Wilson: Okay. Thank you. Next question, please.

Operator: Your next question comes from the line of Ian Sanderson with Cowen & Company.

Ian Sanderson: Thanks very much for taking this follow-up. A couple of questions on just the (unintelligible). Lee, you mentioned it'll have both an immediate release and a sustained release component. Actually, could you talk a little about what the formulation technology is and where the formulation efforts currently stand?

And then on the issue of scheduling, is it simply the lower (dose of) phentermine that gives confidence (unintelligible) scheduled compound?

Lee Wilson: Okay. I'll have Peter take the first question. That's regarding the formulation technology and where we stand with formulation. And I'll ask Dr. Najarian to comment on the scheduling program.

So Peter?

Peter Tam: Yeah. The formulation - we have a few prototypes that are ready to go. That's moving along well from a formulation development standpoint.

There's not a whole lot of complexity in this program. Existing proprietary technologies are out there. And this can be done provided that you know what the end goal is.

And as you said, as Lee said, the technology does provide both components of delayed and sustained release to help further improve on the safety profile of this combination product.

Lee Wilson: Okay. And Dr. Najarian?

Thomas Najarian: There are several issues on scheduling. I mentioned briefly that these two medications work very well together because they tend to cancel out each other's bad side effects.

And that includes the fact that you don't get - if you take high doses of phentermine - so dose is part of it. Lower doses, things are less likely to be as habit forming and also how fast it gets into your system.

But the main effect is you don't get the buzz that some people would get with phentermine alone because Topamax mutes it. So you don't have that sort of high feeling, which some people actually find unpleasant, especially if they have a history of an anxiety disorder.

In addition to that, because - so part of it is reduction of the feeling that you would have from phentermine alone. That's probably the main effect. The other is the lower doses.

And what you have to prove to de-schedule something is that the combination - that you can't easily extract the medication that's the potentially habit-forming medication from the combination.

And in addition to that, you show that when you stop the medications abruptly, there are no physical or psychological signs of withdrawal, which I can tell you from my experience I don't see. And we'll need to prove that in a randomized trial, which we will do, hopefully.

Lee Wilson: Okay. Next question, please.

Operator: Your next question comes from Len Yaffe with Stoc*Doc Partners.

Len Yaffe: Thank you very much. And thank you a lot, Lee for hosting the conference call. I had two questions - one for Dr. Gadde. It seems that obesity is at the forefront of most of major pharmaceutical companies' research efforts. I haven't seen it this high a priority at any time before.

And I was wondering if you could comment if there are any products in the pipelines in clinical studies that you either have been involved with or have heard about that perhaps also show promise for the treatment of obesity?

And then a question for Lee. I know that you're not disclosing the composition of the two respective drugs in Qnexa.

But I'm assuming that at some point between now and 2010 when you get approval, either that composition will become known or will have to be presented at some medical meeting for doctors to understand and accept it. And was just wondering if that would be also consistent with your expectations.

And then wouldn't that be an issue as it relates to potential generic competition? Or are you just trying to cut down any lead time that other companies may have? Thank you very much.

Lee Wilson: Okay. I'll let Dr. Gadde comment on the first part of that. Obesity is a priority in almost - most of the major pharmaceutical companies. And what do you see coming and how does this compare with what - the data that you were able to see on Qnexa?

Kishore Gadde: Well there are - yeah, I do agree that obesity is now an area of really high significance for most of the pharmaceutical companies.

Obesity and related metabolic disorders is the area that most companies are exploring. There are a number of compounds that are in development. There are probably about 150 or so compounds that are in development.

But what's been happening is that a lot of compounds are specifically targeted for a certain receptor or a certain neuropeptide. And you see interesting results using animal models but when you get these drugs into the clinical stages and you get into phase 2, sometimes you see a small signal.

Let me give you the example of Axokine, which was developed and they failed a couple of years ago. Regeneron Pharmaceuticals had this thing that they originally developed for Lou Gehrig's disease, which is ALS. And they saw some weight loss. It might have been related to the disease itself.

But, you know, there is some evidence in animal models that it is a compound that is somewhat similar to leptin. It has some features very similar to leptin. And they did a phase 2 trial. There was some signal that it could cause more weight loss than placebo. This is a drug that had to be given as subcutaneous injection every day like insulin.

But when they did the phase 3 trial, it was really eagerly expected what was going to happen. What they found was that at one year with the - with daily injections of Axokine, the weight loss was 6 pounds. And with placebo it's about 2-1/2 pounds.

Well it was highly statistically significant with about 2000 patients. But it was clinically meaningless because I mean who's going to take an injection every day to lose about 6 pounds when you can just go to the gym and lose a lot more.

So that is actually what we've been finding. There are lot of other drugs. GSK made an announcement about failure of pTK agonist. And so that's been the story.

And with the exception of rimonabant, which is awaiting final approval from the FDA and it is currently approved in the EU countries, there is nothing out there that is ready to come out - I mean as far as I know. I haven't seen any announcements by companies that they've filed NDAs for any other drugs.

Len Yaffe: As a follow-up, could you quickly comment on why leptin seems not to be as desirous a target as it once was?

Kishore Gadde: Well leptin - well it is still being developed in a different form, as far as I know. Leptin - originally it was tested as an injectable form. Again, it's the same thing with Leptin. You have to give it as a subcutaneous injection.

Overall, it's tolerated well. There's some swelling at the injection site in some patients. But the weight loss itself was not really that great. With the highest dose you had a little bit of weight loss that was statistically significant (unintelligible) dummy injections.

But especially when you are giving a treatment in the form of an injection, people's expectations are going to be very different. It's kind of like, I mean would you, you know, take a pill that gives you 20 pound weight loss or would you take injections - daily injections that would give you about 5 or 6 pounds weight loss in one year.

So that has been - there's been some talk about Amgen developing leptin in a tablet form. But again, you know, if they're good results then we would have seen by now. I do not know the inside story.

Lee Wilson: Okay. And Len, if I could ask Dr. Najarian to comment on why we're seeing such - potentially why we're seeing such positive results on Qnexa?

Thomas Najarian: Thanks, Lee. I - Kishore kind of touched on this. There are so many receptors that make you eat, that when you block just one of them, the - these studies - for example, a product that blocks one like leptin. I think these products eventually might be useful in combination - multiple drug combinations.

But blocking just one receptor will usually not result in significant weight loss because we have mechanisms to override that and make you eat from all kinds of others.

And one - I think one reason why this combination works so well is because phentermine probably blocks appetite through three or four mechanisms. And Topamax maybe as many as four or five additional mechanisms. And they're not the same mechanisms.

The fact that you hit so many different reasons why people eat or receptors, is maybe part of the reasons why the results are so good with this combination. And I doubt that any single drug that hits only one receptor is likely to get significant weight loss.

In the future, you're going to need something either with combinations of drugs or a drug that hits more than one receptor.

Lee Wilson: Okay. Next question, please.

Operator: Your next question comes from the line of Holly Tippitt with ThinkEquity.

Holly Tippitt: Hi. Just one question. Given that the phentermine label is limited to short-term use, how do you see this affecting your being able to move into longer-term studies like a one-year phase 3 study or eventually to affect your label?

Lee Wilson: Okay. Well I mean - I'll take a piece of it and we'll ask Peter to comment and Dr. Najarian as well. But the - obviously the purpose of doing the phase 3 studies is to get the labeling for long-term treatment of obesity. So we feel comfortable that that'll move forward.

Having said that, maybe Peter, you could comment where we stand with the FDA on what the requirements are for phase 3 trials.

Peter Tam: Yeah. We've had extensive discussions with the FDA in terms of the work that we need to do in order to enter into a phase 3 program. Essentially, those are preclinical toxicology studies. These are standard tox studies that the FDA is looking for.

We are - we've begun most of those studies. Actually, we've begun all of those studies. And by - I would say the end of this year, we'll have completed all the necessary toxicology studies for us to enter into phase 3.

Lee Wilson: And how about the length of time on treatment and numbers of patients, et cetera, that were being directed because we do have questions from investors that they have seen requirements for other companies which are quite a bit higher than what - the requirements that we've been given by the agency.

Peter Tam: Yes. You know, there are couple of factors here. One is that we're using low doses of these two approved agents. That's one.

And we've had this discussion with the FDA from the very beginning, since our IND discussion with FDA, with regard to the program and the duration. And the FDA has given us very clear guidance as to why we need to have 1500 patients on active medication, treated over a one-year period.

So we figure that including placebo, including a certain percentage of patients dropping out from this study, we'll probably need to enroll in the neighborhood of 3000 patients for the entire program over a one-year period in terms of treatment duration.

Lee Wilson: Okay. Dr. Najarian?

Thomas Najarian: This is partly to answer that question but also to go back to Mr. Yaffe's. I don't think we answered his about generic competition. There can be no generic of this product until at least 2019 in any case.

But the phentermine is currently indicated for just short-term use. It will be approved I hope, in this combination for long-term use. So anybody prescribing it separately, it would be an off-label use. The patient would have to pay for it themselves.

Topiramate will not be approved by itself as an obesity treatment. So again, if someone were to write separate prescriptions and try to mimic this in some sort of semi-generic type of product, they wouldn't be the same dose as in this product.

In addition to that, because it's a non-approved use of topiramate alone, pharmacy benefit managers typically will not cover that. This product should be covered.

Lee Wilson: Okay. Next question, please.

Operator: Your next question comes from Michael Tong with Wachovia Securities.

Michael Tong: Hi. Thanks for taking the follow-up. Actually, a follow-up on the phase 3 protocol. In the phase 2 studies you had a four-to-one ratio of women to men. Now obviously it's a fairly small population. But was there any observable difference between the responses between the two genders?

And if I heard you correctly, Lee - Peter, you talked about enrolling 3000 patients to get data on 1500. For your phase 3 program are you doing one phase 3 or two separate studies?

And then finally for Dr. Gadde, how long did it take you to enroll the 200 patients for the phase 2 studies?

Lee Wilson: Okay. Let's start out with the gender response question. Dr. Gadde, did you see any differences between what happens with men and women in your study?

Kishore Gadde: No, we have not. Men lost just about as much weight as the women did.

Lee Wilson: Okay. The second part of that is phase 3. Peter, do you want to take that?

Peter Tam: Could you repeat the question again? Sorry.

Michael Tong: Yeah. I just heard you say, you know, in order to get data on 1500 patients, you need to enroll 3000. Now is that accounting for the dropout? And do you need two phase 3 studies or just one?

And then just while I'm on the microphone here, do you expect to stay at a four-to-one ratio between women and men in phase 3 or you'll try to get a more balanced population?

Peter Tam: I think in terms of - I'll answer your last question first. The enrollment will be dictated by supply and demand.

We're going to probably - likely to see more women coming in. And we're not going to make any special attempt, nor has any request made by the FDA for us to try to recruit more male patients. So we're going to, you know, let supply and demand dictate the enrollment rate for the program.

And with regard to the number of phase 3 studies, our intention is to conduct three separate phase 3 studies looking at, you know, primarily weight loss but perhaps in a slightly different population to further elucidate the additional benefits of Qnexa on some of the metabolic morbidities.

Lee Wilson: Okay. And, thanks Peter. And I would say that yeah, we're still working with the FDA on finalizing the phase three program. So that's open for discussion with them as well.

And maybe we could ask Dr. Gadde to comment on how easy it was to enroll patients and what he would project for the ease of enrollment of phase three trials, since he has experience in that area as well.

Kishore Gadde: We were a single center for this trial. And we actually took upon an ambitious project of enrolling all the 200 subjects. And it took us eight months to enroll.

So that's 25 subjects each month. And we are, you know, I currently have a very large study that's funded by the National Institute of Diabetes and Digestive Diseases, and that's kind of the typical enrollment.

We can enroll at our site. And we've seen the same enrollment at the sites of my colleagues, who also do a lot of - conduct a lot of clinical trials.

It's not difficult for a site to enroll one to two patients on each business day. So you can - if we have two or three trials, we can enroll ten subjects each month. We just will have to put more coordinators on that study.

And so for phase three studies, we have been involved with phase three studies with other drugs. So let's say that we have to enroll, you know, 2000 subjects for one phase three trial.

And if I were to be running that clinical trial, I will probably have about 50 sites. Anywhere from, like, you know, 30 to 35 to 65 depending on how fast you want to enroll.

And with that many sites, you can actually enroll, if it is the straight obesity trial. We are not talking about obesity with associated diabetes.

So if weight loss is the primary outcome measure, if you're taking patients who have a BMI of 30 and obesity as the primary problem, for - to enroll - you can actually enroll 2000 subjects at several sites in about five, six months. It's not a problem at all. It can be even sooner as well.

Lee Wilson: Okay. (Michael), I think there was one component that we didn't answer in your question, and that is how do we get to the 3000 patients?

Well, the requirement is 1500 patients on active treatment. The majority of those will be on the high dose. We will have a maintenance dose, which is a half dose in the phase three clinical program and a placebo group as well.

So you have 1500 on active treatment, then you'll have your placebo arm as well. And that's how we get to 3000 patients approximately if you include dropout rates.

(Michael): Great. Thank you.

Lee Wilson: Okay. Now, next question, please.

Operator: Your next question comes from Ken Trbovich with RBC Capital Markets.

Ken Trbovich: Lee, just quickly a couple of questions with regard to phase three design. You mentioned the titration phase in the phase two. Given that you're going to be moving to once a day, does that affect at all the titration?

In other words, were the doses being changed from mornings to afternoon dose? How might that change the titration phase in the phase three?

Then specifically within the phase three program, are you able or required to - are you able to exclude diabetics or are you required to specifically study them in a separate study?

Lee Wilson: Okay. The first part of it, I'll take a shot at it, and Peter can chime in as well.

In the phase three program, obviously going to a once a day therapy will be a - make it simpler for the patients. And there is involved in this technology, as

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you know, a delay component and sustained release component of the technology as well.

So it will mimic as close as we possibly can the blood plasma levels that we were able to achieve in the Duke study.

And so that's the program there. So it'll be a much simplified titration program for the phase three program.

And, let's see, the other one - I think - does that answer that question, Ken?

Ken Trbovich: Well, yeah. I guess I just wanted some clarification on the titration phase then in the phase two where the dose is the same in the morning and the afternoon.

Lee Wilson: Yes. What - in all of our studies, the ratio of Phentermine to Topiramate has remained stable. The only thing that changes is that you increase the amount of drug on a weekly basis until you get to you end result.

Okay, so the ratio is the same, and it'll be the same in the phase three clinical program, both for the maintenance dose and for the final dose.

So the ratios are the same. The art form here is in when the drugs are released and in addition to the sustained release component.

So we're working hard to give the patients everything they want in order to get the exact same results that we got in the phase two study.

And with that, I heard Dr. Gadde comment on the predictability of this phase two results going into phase three. But since we had a number of questions from investors about, you know, that this study was a single-center study and I

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think it's worth maybe a couple other comments, Dr. Gadde, that - what is your view about the predictability of this going into phase three programs?

Kishore Gadde: Well, I'll repeat the comments that I made before. And I'm quite confident that since the difference - the treatment effect is really large here. You are seeing a difference of about 8%, 8-1/2% between the active treatment. It's 8.7% relative to placebo.

That is a huge difference. And a difference as large as that, I mean, it would be really surprising to me if that could not be replicated. I think that would be very easy to replicate that finding.

So as far as the efficacy is concerned, I have no doubt that these findings will be replicated.

The only thing that I would say I don't know for sure is about whether there are any side effects that are rare but are significant. Let's say the kind of side effects that would occur in let's say one in 200 people or one in 500 or one in 1000.

Those are the kind of side effects that a study like this would not give us the required information. You would need studies with a few thousand patients to know the rare side effects.

Lee Wilson: Okay. Any more questions please.

Ken Trobovich: Lee, just on the diabetes, I apologize.

Lee Wilson: I'm sorry, Ken. I left that. Yeah. The answer to the diabetes question; obviously we're very interested in diabetic patients. I think Dr. Najarian

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commented that he has been able to see in many of his patients that were obese diabetics, that they've been able to come off their diabetes medicine.

We're working with the FDA now to design the - a trial with diabetic patients in mind. And so we'll - as we go forward, we'll have more clarity about that. But clearly that's a very important part of this.

The whole concept of metabolic syndrome, those that have high blood pressure, high lipid issues, cholesterol, etcetera, we're very - going to be very aggressive in looking at those, because in Dr. Najarian's clinical practice, and he can comment on this, he has found excellent results in the ability to eliminate many of the medications that a patient is on that has metabolic syndrome. So, Dr. Najarian, would you comment?

Thomas Najarian: Yeah. And I've encouraged Vivus to do these studies because there is such profound results.

If you look at patients with hypertension, in my practice I get a better blood pressure response with this treatment than you can get from any single blood pressure medication.

For example, a patient with a blood pressure of 200 over 120 would normally require more than one medication to get them to a normal range. I can often get someone with even that bad a blood pressure to be normal after a few months on this treatment.

And it isn't just the weight loss. I think Topamax has a hypotensive effect; that is lowering blood pressure. Partly because it's a carbonic anhydrase inhibitor and acts like a diuretic a little bit.

And also, patients with elevated lipids, you can see better cholesterol numbers that any single statin in my patients. You can see better reductions in diabetes except if you use high-dose insulin, you force someone to be hypoglycemic, which you don't want to do anyway.

But then in the oral agent, you can see much better results in blood sugar than any single oral agent, and comparable results to insulin except that the patients are actually a lot better because insulin over a long term tends to cause weight gain as well.

Lee Wilson: Okay.

Kishore Gadde: Lee, can I add something to what Dr. Najarian has just mentioned?

Lee Wilson: Absolutely.

Kishore Gadde: Lee, in the trial I think we didn't have a slide there showing changes in systolic and diastolic blood pressure. But let me just give you the numbers there for the attendees or the participants in the meeting.

With the Qnexa, we saw a seven millimeter reduction in systolic blood pressure compared to a one millimeter reduction with placebo. That's a placebo subtracted difference of six millimeters.

And just recently, I had the opportunity to review what has been the effect seen with the approved anti-hypertensives. I actually went and looked at the product labels of all the approved anti-hypertensives, and typically with the - in an anti-hypertensive such as Norvasc, the relative to placebo they give about six to ten millimeters greater reduction in systolic.

And this is actually almost there. And in fact, even with diastolic, we've seen a four millimeter reduction with Qnexa compared to a 1.5 increase with placebo.

That's a, you know, relative to placebo, it's about a, you know, five, 5-1/2 millimeter difference. And if you compare this with the Rimonabant trials, in four Rimonabant trials, with regard to reduction in systolic blood pressure, it ranges from a reduction of 0.2 to 2.3.

So barely there was a two millimeter reduction; one to two millimeter reduction with Rimonabant. And we've seen a seven millimeter reduction with - of systolic with the combination.

And we actually saw pretty decent reduction of Topiramate by itself too; it's 5.7 millimeters.

Lee Wilson: Okay. Dr. Najarian, would you comment as well?

Thomas Najarian: Thanks, Kishore, for pointing that out. And I want to add to that that when you test a drug like Norvasc for hypertension, you're testing patients who actually have hypertension; maybe 160/90 or above.

In this study, because we were selecting to be healthy obese people, the actual baseline systolic blood pressure was 117, and the baseline diastolic was 78. Those are normal readings, and to show a seven millimeter drop from a normal reading is pretty remarkable.

Again, if you had tested people that had hypertension like I see in my practice all the time, you would have seen even bigger numbers. But thanks for pointing that out.

Kishore Gadde: Well, Lee, this is Kishore again. I really appreciate the comment that's made by Dr. Najarian, because this is really the most important point to consider; that if you want to demonstrate a larger effect on risk factors such as blood pressure or changes in lipids, you really need to enroll patients who have abnormal values at baseline.

That's, you know, been something that we've found in all clinical trials. So even with Rimonabant, the differences in metabolic risk factors have only been really found in people who had these problems at baseline.

So this is a big effect that we saw. That these are patients who are normal tensive; they had normal blood pressure at baseline. Yet we saw this nice reduction in systolic and diastolic.

Lee Wilson: Okay. Next question, please.

Operator: Your next question comes from Marc Robins with the Robins Group.

Marc Robins: Thank you. I have to apologize. I had to duck out for a moment. I know that you chatted a little bit about lack of sleep or loss of sleep. Is there anything else that you can add or review?

Lee Wilson: On insomnia?

Marc Robins: Yes, please.

Lee Wilson: Okay. Well, the slide - you want to put it back up for me please. And Dr. Gadde, would you comment on any issues that you may have seen regarding insomnia in this clinical trial?

Kishore Gadde: If the slide is back up, actually I might have not described it very well. What we saw was that there was no significant insomnia with the Qnexa. It's only 2%. That would mean one subject complained of insomnia.

We saw the highest rate of insomnia was actually with placebo, 12%. So insomnia; we actually expected some degree of insomnia with Phentermine, because that is there seen in the package insert for this drug.

And it's a sympatoneumetic drug. It's a stimulant, so we expected to see a greater incidence of insomnia with Phentermine. Topiramate typically the instance is not quite as high. We didn't expect to see it as much.

So we thought that actually, if anything in the case of Qnexa, Topiramate, which could be a bit sedating could actually offset any risk of insomnia with - associated with Phentermine. It kind of looked as we expected. We didn't see much insomnia with Qnexa.

Marc Robins: Thank you very much.

Lee Wilson: Okay, Marc. Next question please.

Operator: Your next question comes from Ilya Kravets with Rodman & Renshaw.

Lee Wilson: Are you there, Ilya?

Ilya Kravets: Yes. Sorry about that. Dr. Gadde, some of the comments you made earlier about possible, you know, long term adverse events and that the only way we see them would be the larger trials.

Now, we do have long term trials for Topiramate and for Phentermine indicated for short term use.

We see the synergistic effect on the efficacy side. Would you expect anything like that to happen on the adverse event side? I mean, at this point, they seem pretty safe, especially given that they've been around for such a long time and have been used in such a large number of people.

But would you - could you speculate on any possible reason that the combination of these drugs over a long term could pose some sort of a increase in the adverse event?

Kishore Gadde: Alright. Lee, I'm just going to go ahead and answer the question.

Lee Wilson: Yes. Go ahead. I'm sorry.

Kishore Gadde: The - - I tend to be a very cautious person. I tend to be ultra-conservative. So whenever I have results of a study, you know, media, they keep calling and say, you know, in fact I remember talking to a New York Times reporter once who kept saying, you know, why don't you make recommendation that you like this; it is working.

You know, I typically say that, you know, based on one study, I'm not going to recommend any treatment. Typically, you know, we wait until we have data obtained, results obtained in a large trial, particularly with regard to safety, simply because it's a general statement that I made that with any treatment, with any active intervention, you - if there are any side effects, which would only occur in one in 1000 patients, you don't know until you've studied maybe, you know, 2000 or 3000 patients that kind of risk.

So it's a general cautious statement that I made. But you know, based on the results that we've seen and just the experience of personally interacting with patients, I don't really suspect that we're going to have any major issues.

Let me also talk about the echocardiogram results. Actually, it's not part of the slides. We meant to include those in the slides.

We did not see any significant changes on the echocardiogram with any of the treatments; any of the four treatments. We have obtained probably, well, we got echoes on anyone who had been exposed to the treatment for at least four weeks.

So pretty much we had echoes on everyone repeated at study - the end of the study. And we did not see anything.

I think that would have been the one. When we began the study, we had to deal with our insurance review board; just the name of Phentermine, although, you know, Phentermine by itself had never been implicated in causing this, if you read the product insert for Phentermine, it states that the association could not be ruled out simply because if I'm a clinician and I pick up the phone and I call the FDA or I fill out the Med Watch program and I say I had a patient who took Phentermine, and I found aortic regurgitation, the FDA would then record that as an event, even though the clinician for sure did not know that it was Phentermine that actually caused it.

This patient might have had this problem from childhood. So the only way that you can find something like this is in a systematic study where you look at something before and after treatment.

And there is no scientific study that showed that Phentermine had been associated. Regardless, the fact that you mentioned Phentermine as being a part of Fen-Phen, which was the thing that scared many people there, the IRB — insurance review board — and the hospital liability department, which is the risk management, they said, well, you've got to get this done.

And actually hired Dr. (Jawlos) and another cardiologist who had actually published a lot of studies looking at Fen-Phen. And we looked at - we used a very highly sensitive echo with color Doppler. And we didn't find anything.

So as far as that, we were really - we were comforted by the fact that we didn't see valve (unintelligible), because that would have been - if we found something occurring at a high frequency, I think that would have been very discouraging. Probably would have been the end of the story.

But we didn't. So I'm confident at this point that the efficacy will be replicated. And I don't see anything at this point, from my end, although (Tom Najarian) can probably tell us a lot more, because I don't - I mean, apart from the clinical trial, I have not treated many patients with this combination.

And (Tom) has tremendous experience not just number of patients, but patients he has followed for many, many years. And if he - he mentioned that he hadn't seen any major problems. If only he saw - he had been seeing cumulative benefits that the, you know, people remained - people kept the weight off for long periods and their cardiac conditions got better.

Those are all so - so you're going to find about any potential, rare adverse event in a long term study, but you will also find the improvement over time. So in a six-month study, you're not going to see the effects of translational

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weight loss into overall health benefits. You're going to find that in one year and two year trials.

Lee Wilson: Great. Thank you. Okay. Thanks. Any other questions, please? We're running up against the timeline here. So let's cut it off at this point.

I think we've had a nice, rousing discussion here, particularly as we got into the question part of it. And I appreciate it.

I really want to thank particularly Dr. Gadde and Dr. Najarian for taking time out of their very busy schedules to make themselves available to all of us to answer these questions.

I hope that our investors out there have been able to gain some degree of comfort around Qnexa. It - you've heard me say that everything that we know about Qnexa is transparent out to you right now.

The data speaks for itself. We're very confident in the product because of its tremendous experience with both Phentermine and Topiramate in the marketplace as well, Topiramate being recently approved by the FDA and obviously the tremendous review that Phentermine went through during the Fen-Phen error.

So we're comfortable with the side effect profile, that what we have seen both in Dr. Gadde's study and Dr. (Najarian's) experience. And obviously the efficacy is nothing short of wonderful. And we feel very confident that that is real. And we actually feel that there are a lot of good reasons why - that that is the case.

Dr. Najarian talked around the issues and Dr. Gadde as well, that the fact that you need to block a number of receptors and it's theorized that both Phentermine and Topiramate each block a number of these hormones or whatever that cause us to either to eat or to not be satisfied.

So there's some good rationale for the mechanisms, and that helps us get comfortable with the kind of efficacy that we're seeing in this drug.

I would remind everybody that Dr. Gadde will be presenting in a scientific forum the results of this study. And that will happen on October 22 at the (NAASO) meetings in Boston.

And so we would encourage anyone to attend if that's possible. And so with that, I would just like to thank everybody for their participation. Some wonderful questions today. And thanks again Dr. Gadde for taking the time out of your busy schedule.

Kishore Gadde: Thank you.

Operator: Thank you. This does conclude the Qnexa program update conference call. Thank you for your participation. You may now disconnect.

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