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Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

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PRESENTATION

Jennifer Davis - Pfizer Inc. - IR

Good evening. Thank you for joining us for Pfizer's analyst and investor briefing here at ACR. I'm Jennifer Davis from the Pfizer Investor Relations team, and I'd like to welcome those of you here with us in San Francisco and also those participating by webcast and phone.

This evening's meeting will include presentations by Dr. Ken Verburg, Senior Vice President and Development Head for Pain Therapeutics, and Dr. Ethan Weiner, Senior Vice President and Development Head for Information Therapeutics.

Their presentations will be focused on three of our pipeline candidates -- Tanezumab, Esreboxetine, and CP-690,550, including data that has been presented here at ACR.

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

Following our prepared remarks, Mark White, Senior Director and Group Leader for Pain Therapeutics Commercial Development, will be available, along with Ken and Ethan to answer questions.

I'd like to remind you that we will make forward-looking statements during tonight's presentation, and our actual results may differ from those forward-looking statements. I would refer you to our SEC filings for additional information. Now those are available on Pfizer.com under the Investors section.

With that, I'd like to turn it over to Ken Verburg.

Dr. Ken Verburg - Pfizer Inc. - Development Head for Pain Therapeutics

Thanks. My name is Ken Verburg. As Jen mentioned, I'm the Development Head for Pain Therapeutics at Pfizer. I'd like to thank you all for attending the session tonight.

My presentation ostensibly is targeted to describe the results that were presented this week on the Esreboxetine and Tanezumab studies and to provide a little bit of context around those clinical development programs.

After Dr. Wiener presents on the JAK-inhibitor, I'll be happy to answer any questions you might have about that program or pain therapeutics at Pfizer in general.

Before I get into those two programs, I thought it important that we take a step back and evaluate a little bit about the potential of the pain therapeutic markets. Any risk reward trade-off that you make in terms of development projects, of course, is very dependent on your assessment of the market and the market potential going forward.

So one of the things that characterizes the pain market is that it affects a tremendous number of patients, and that is only supposed to increase over the next several years, and, in fact, here on the bar chart, you can see that the prevalence of pain across various conditions could affect up to 250 million patients by the year 2011. This really represents a steady growth in patients as a reflection of age, as reflecting the aging population.

Another thing that really characterizes this market is that when you go out and do market research on health care providers, physicians, etc., you find that they are very dissatisfied and displeased with their current therapeutic options.

I have pulled a quote out from a physician out of some of the market research that we have done, and it reads as follows. Currently, medications have very low efficacy -- only one in five patients are treated successfully. And if you have this type of efficacy in another field of medicine, it would not be accepted. We accept it because we have nothing better.

One of the things that characterizes the pain therapeutic market is that there is a lot of individual therapies that are available to health care providers and/or reformulations or different formulations of the same product.

Now in 2007 this was about a \$35 billion market and is projected to grow over the next three to five years to about \$45 billion, and a lot of that growth is anticipated to occur as a result, as I mentioned previously, of the expanding population, but also the development work that's going on within these classes of drugs. There are, in fact, if you distill down all the individual therapies that are available to physicians, they actually come down and distill down to very few therapeutic classes. And I have listed the predominant classes here under acute musculoskeletal pain or [no-susceptive] pain and neuropathic pain.

Most of these classes of drugs have been around for tens of years, if not hundreds of years, and in fact, in the case of NSAIDs and opioids, you can actually trace their use back to natural products, willow bark and the opium poppy seed used first described by ancient Greeks.

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

There is a lot of development work, as I have mentioned, that goes on. Most of that development work is within these existing therapeutic classes. But when you take a step back step back, and several surveys now have come back with the same types of findings, patients are also dissatisfied with their therapies, and in fact, greater than 50% of patients report that these therapies are really sub-optimal in relieving their pain.

So I think the message here is there is not much headspace in these existing therapeutic classes, even though there is a lot of development work. And it's our view at Pfizer that really only way to move on this market and grow this market is to take a step aside and go after innovative drug targets that produce innovative medicines, that deliver substantially on the efficacy and safety over the existing therapeutic classes.

It's perhaps of certain benefit that the potential for the therapeutic market in pain is matched by really an explosion in the understanding of the biology and the pathophysiology behind pain. And, in fact, it's really that understanding has led to the idea that a number of therapeutic targets are potentially advantageous in the treatment of pain, and I have listed just some of the targets down here below.

So it is really our view of the potential therapeutic targets, the novelty that those targets may represent in terms of clinical benefit, that really is the confidence that -- provides the confidence that we have to really fund and invest in pain, both in research and in development at Pfizer.

And that confidence we believe is actually matched to a great degree by competency. We've spent a long time -- a number of years now in studying pain, and we hope to build upon those learnings both by internal expertise, as well as our reach externally, to partner with external collaborators in a fashion that puts us in a first mover advantage for whatever targets in this larger rate break out to be of some significance in terms of therapeutic potential.

So the pain therapeutic development portfolio, as we currently have it, is shown on this slide. In addition to Lyrica and Celebrex, we have a total of 11 compounds currently in clinical testing with another four compounds in preclinical testing, heat up really to enter into the clinic in the next 12 to 24 months.

For the most part, these compounds represent novel science. And, in fact, nine of the 11 are new molecular entities targeted against novel drug targets for the treatment of pain.

I think with that backdrop, let's go into Tanezumab and review the current status, as well as the emerging data.

First, nerve growth factor is a key mediator in pain, and that's becoming ever so increasingly clear as more and more investigations report out. One of the pieces of evidence that I like to point to that suggests that it is a key mediator in pain actually comes from studies in what is exogenous recombinant NGF was administered to patients in the attempt to correct for diabetic neuropathy. One of the key findings in those studies, even though it did not help necessarily reverse or stop the progression of the neuropathy itself, was that patients reported back to the investigators significant and long lasting pain.

Tanezumab is a humanized IgG2 subtype monoclonal antibody that has high specificity and affinity for nerve growth factor. It has a mutation in the constant region of the antibody which prevents or limits antibody dependent cell mediated toxicity, as well as complement activation, things that you may see with other antibodies therapies. It's shown efficacy in multiple preclinical models of pain spanning anywhere from experimental models of cancer pain through visceral pain and acute pain, and we've demonstrated clinical efficacy in osteoarthritis pain.

We have been administering the drug as a slow IV push, essentially a five-minute intravenous infusion. We're projecting a low dose that is approximately 10 mg or less as a fixed dose given every eight weeks. So 10 mg given approximately at six times per year.

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

The Phase III intravenous program is keyed up and ready to go, and we're on plan to start yet this year, again in osteoarthritis. And the program objectives are pretty simple. We want to move quickly with an approval with osteoarthritis pain using the intravenous route of administration, and we project a BLA submission for this particular program in 2011/2012 time period and to follow that initial submission with a submission looking at subcutaneous administration of Tanezumab for osteoarthritis pain and then broadening out that indication structure to other conditions of chronic pain, again using the subcutaneous formulation.

So neurotrophic factors, or neurotrophins, are substances that control neuronal maturation and survival during the development of functional contacts or neuronal circuits. So typically this occurs in the developing fetus, as well as several weeks postnatally.

But in the adult, neurotrophic factors have other effects on neurons. They up regulate sensitivity, nerve transmitter synthesis and release. They have been shown to protect from various insults such as metabolic, chemical, viral or physical, and they induce or increase sprouting or regeneration.

Nerve growth factor is one of a family of neurotrophins. On this slide I have outlined the agents that belong this family, as well as their respective receptors.

So if we start over here on the left, nerve growth factor, its receptor is trkA. trkA is located predominantly on C fiber neurons that mediate pain and temperature sensitivity.

If we go over here to the far right, NT3 is another neurotrophin. Its receptor is trkC. This receptor is located predominantly on A-beta fibers that mediate proprioception or balance and a vibration sensation.

And then down in the middle is NT4/5. It's per common receptor is trkB, and it is located on slowly adapting mechano receptor neurons, A-delta fibers to some degree, and it mediates light touch.

Now brain-derived neurotrophic factor up here at the top is a neurotrophin found predominantly in the central nervous system, and it seems to also have a role in pain processing, but within the CNS.

Note that NGF is pretty selective for the trkA receptor, and this is distinct from some of the other neurotrophins that are a little bit promiscuous in terms of their ability to bind to various receptors.

Note that NT3 and NT4 can both bind trkA, in addition to trkB and trkC, respectively.

So the message here is that NGF is just but one of several nerve growth factors, and that they may have and in some cases do have redundant activities.

So how does NGF cause pain? It seems based on the literature to date that it occurs by three different mechanisms.

First, NGF sensitizes TRPV1 ion channels. This channel mediates noxious heat or acidity within the extracellular space. This is also known as the capsaicin receptor or the hot chili pepper receptor. And once NGF binds to the trkA receptor, a cascade of sort of second messengers occur, which results in the phosphorylation of the TRPV1 channel, which in turn leads to the increased transport of the channel from the endoplasm to the cell's surface, but also increases the channel activity for any channels that are already on the surface. So the increase in activity here is the result of an increased number of TRPV1 channels, as well as heightened activity of the channels overall.

The second wave that NGF can cause pain is by activation of mast cells. Mast cells are fairly frequently found around and in the skin around sensory neurons, and NGF again by binding the trkA receptor results in a degranulation of mast cells and a dumping of a host of different mediators that can either directly cause pain, for example, prostaglandin, bradykinin or sensitize the neuron to other [no-susceptive] or agents that can cause pain.

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

And the final way by which NGF can result in pain is by binding to the trkA receptor. That complex is internalized and carried up by a retrograde fashion up to the cell body of the neuron located in the dorsal root ganglia. At that point, they can turn on gene print transcription and result really in a host of different mediators being produced such as substance P and CGRP, as well as brain-derived neurotrophic factor, which again it can be transported to the terminus of the neuron released and then activated ascending neuronal tracks that process pain.

There also appears to be an increase in sodium and calcium channels, which can result in increased activation of the neuron by generating additional action potentials.

So I want to make a couple points on this slide. First, anti-NGF therapy does not affect acute pain sensation or neuronal survival in animals. So I want to tackle the first one first -- does not affect acute pain sensations.

Over here on the left, you see some data in a double-panel here that was from a study done in rats. And these rats were normal, for all intents and purposes as normal as a rat can be. And what you see here are rats treated with [vehicle] or the rat version, if you will, of Tanezumab over a period of several weeks.

During the course of this time, the rats were challenged with either a heat pain stimulus or a mechanical pain stimulus. And the upshot of this study was that anti-NGF therapy is not intrinsically hypoalgesic, nor is it analgesic. It will not prevent someone from feeling acute pain. So acute pain processing, which we all recognize as being very important for safely withdrawing from an injurious insult, is still intact, at least in animal studies.

Over here on the right in this electro micrograph photograph, what you see here are -- it is really a histopathological type of look at CGRP, and in this study CGRP is used as a signal of peripheral neuronal density, if you will, in rat skin. And I've shown you four panels here, comparing first animals treated just with vehicle, and then over here on the right, animals treated with NGF. And the takeaway message here is, that if you look at either naive animals or animals with an experimentally induced osteosarcoma, or that is cancer of the bone, you can see that NGF therapy does not affect the intensity, if you will, of the fluorescence that you're seeing, and that's really sort of a signal that CGRP and the neurons that CGRP is located in are unaffected by NGF therapy.

Now up here on the top, these animals had a profound pain response with respect to NGF therapy. So at levels of NGF that can reduce metastatic bone pain, the level of morphine, or perhaps even beyond, you can see that there is no effect on neuronal survival in the skin.

The other study that I want to direct your attention to was a 20-week study -- 26-week study, excuse me, that we conducted as a prelude to opening the IND and clinical investigation of Tanezumab. This was done in adult cynomolgus monkeys, and they were dosed with either 0, 1, 10 or 30 milligram per kilogram per week of Tanezumab. This represents an excess of 10 to 300-fold higher concentrations of Tanezumab than what we would administer in the clinic, given every week, not every other month, which we are intending to go forward with in the clinic.

The study was a toxicology study, and there were no target organ toxicities identified at these doses over 26 weeks of administration. And importantly, with extensive evaluation of the nervous system, there were also no structural toxicities noted.

So this slide summarizes the status of the clinical development program for Tanezumab as it currently sits. So in terms of exposure, we dosed now well over 600 patients with at least one dose of Tanezumab, and of these 250 have received six months or greater of treatment.

The osteoarthritis program is complete. We'll talk a little bit more about study 1008, the Phase IIb study in just a minute. We also just recently completed a one-year open label study in which patients were treated for up to one year with a dose of 50 micrograms per kilogram of Tanezumab given every eight weeks. We're really quite pleased with the data that we have seen

Oct. 28, 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

both in terms of efficacy and the durability of efficacy that we have seen with this dose, as well as the safety, and we intend to disclose those results sometime next year at the first chance that we get.

In terms of other Phase II studies that we're conducting, we have recently completed a study in chronic low back pain. Again, we've seen the results, we're very encouraged by the results, and we anticipate submitting an abstract to the American Pain Society meetings that will occur in early spring next year to report those results.

We have completed enrollment in a neuropathic pain study specifically in patients with Post-Herpetic Neuralgia. We have a study on going in visceral pain in interstitial cystitis, and we plan to start a trial in endometriosis, as well as prostatitis in the fourth quarter of this year and first quarter of 2009, respectively.

Finally, we are intending to study the effects of Tanezumab in patients with metastatic bone pain. That trial will also kick off first quarter of next year.

In addition to the intravenous administration program, we're also vigorously pursuing subcutaneous relative administration as I had mentioned previously. We will initiate an intravenous subcutaneous bioequivalence study in the fourth quarter of this year, again as our first clinical study in that respect.

Finally, just to comment about the product development that is ongoing with the product. We have developed a ready-to-use liquid formulation of the product that is stable with refrigeration, that is it does not necessitate freezing conditions, and we're also pursuing single-dose syringe administration as well.

So study 1008 was presented this week, in fact, just this afternoon, at ACR. As I mentioned, this was a Phase IIb trial with Tanezumab. It was a randomized double-blind controlled trial, placebo controlled, evaluating five different doses of Tanezumab as outlined here with roughly 75 patients enrolled in each of the treatment arms. Each dose or placebo was administered in a blinded fashion twice during the course of the study, once at baseline following a randomization, and then again a second dose was administered eight weeks later at day 56. The patients were followed for efficacy for 16 weeks to day 112 and were followed for 26 weeks or day 182 for safety due to the long half-life of the antibody.

To be eligible to enter the trial, patients were required to have osteoarthritis of the knee as defined by ACR criteria. They had to have a certain level of pain, typically 4 on a scale of zero to 10, and they had to either be one of the following -- NSAID failures, patients that refused to take NSAID treatment, or were candidates for more invasive therapy. That is intra-articular corticosteroids or total knee replacement.

So the Western Ontario McMaster University Osteoarthritis Index, known by that acronym WOMAC, is a validated and well-recognized efficacy measure or efficacy instrument that is used to evaluate osteoarthritis patients. The index, by the way, I should mention is made up of three different components or subscales. The components are pain, physical function and joint stiffness.

These were secondary efficacy measures in studies 1008, but the reason that I am focusing on the WOMAC is that these will be the primary efficacy measures that will be used in the Phase III program per our agreement at end of Phase II meeting with the FDA. In fact, the three primary end points will be WOMAC pain, WOMAC function and a global assessment of the patient.

So I'm going to show you the WOMAC pain scale, as well as the WOMAC function scale in my presentation tonight.

So the patients will evaluate their pain on a visual analog scale ranging from zero to 100 millimeter. The scale is just anchored at one end by no pain that is zero and then 100 millimeter by a worst pain imaginable. And the patient simply puts a mark on the scale as to where they believe that their pain is over the course of the trial.

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

Note that patients entered the trial here with baseline scores between 62 and 69. So these patients had moderate to severe pain based on the visual analog scale.

So note that all doses of Tanezumab as compared to placebo resulted in a reduction in pain as recorded by the patients through the course of the trial. It's clear that, as you escalated the dose, the magnitude of the pain response, as well as the duration of the response, over the eight-week dosing interval was increased. And the dose response relationship was fairly well ordered. There was really only one place where there was somewhat of a dose inversion, and that is it is at the 25 mg microgram per kilogram dose as compared to the 50 microgram per kilogram dose. It's almost like those two doses were completely reversed.

Note also that we studied both 100 and 200 microgram per kilogram, and the doses were fairly similar with respect to the WOMAC pain scale. So our assessment is that 100 microgram per kilogram, which equates to 10 mg on a fixed dose. So ignoring bodyweight and just sort of dosing it at a fixed rate, we believe that that is the full therapeutic dose, and that will be the top dose that we carry in to the Phase III program for osteoarthritis anyway. Now that may not be the top dose for other conditions, but it will be for osteoarthritis.

Okay, here's the WOMAC function subscale. Again, same scale used. Patients entered with moderate to severe dysfunction, and your first impression here is that this is almost a mirror image of a result on the pain subscale. And to some degree, this is a verification or a validation, if you will, that the response, the mean responses in the WOMAC pain scale were clinically meaningful. That is if those responses were meaningful to the patient, we would expect that in other aspects of their function or their global well-being, you would see an improvement, and in fact, on the function scale, it is almost identical to the results that we saw on the pain scale.

Note that the 200 microgram per kilogram dose again did not achieve any significantly greater effect than did the 100, so once again, our conclusion from here is that the 100 microgram per kilogram dose is the fully therapeutic dose for OA.

Another way to gauge clinical meaningfulness of mean responses in pain trials is to look at the responder rates. Now you will hear from Dr. Weiner in a minute that in the area of rheumatoid arthritis, they use what they call the ACR20/50/70 responder rates to gauge clinical meaningfulness of their responses.

In the pain field, we typically rely on 30% responder rates and 50% responder rates, predominantly based on the work of [John Ferrara] and others that established that these reductions in pain are clinically meaningful to patients based on their correlations to improvements in global well-being. And, in fact, 30% response rates represents a clinically meaningful response. A 50% response rate represents a clinically significant response rate or a significant improvement in pain.

That's typically where we stop. We don't typically in pain go after or assess higher responder rates. And the reason for that is we usually with traditional therapeutic agents don't really see much there.

So I could have selected any response rate over the 50% responder rates to sort of give you a sense of the Tanezumab effect. And I just arbitrarily chose 90% with the idea that what's meaningful to patients is not that their pain is half gone, they want their pain completely gone. And 90% is about as far up on that scale as you can possibly get.

So I want to make a couple points on this slide. First, that all doses of Tanezumab resulted in statistically significant higher response rates, where they are used 30%, 50% or 90% as you are cut off as compared to placebo.

Secondly, note that in the terms of a 30% response rate, the delta from placebo was also quite significant. In other words, across the doses of Tanezumab, you see that the response rates, patients achieving a 30% response rate ranged from 70 to 90% of the patients that took Tanezumab as compared to less than 40% of the placebo-treated patients.

You see similarly with using a 50% cut-off, you see that the responders ranged from 50% up to 70%, suggesting or reporting that their pain had been reduced by 50% or more, and finally over here at the 90% mark, you can see that patients ranging --

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

that the percentage of patients range from about 15% up to as high as 30% who claimed that their pain had been reduced by 90% or greater.

Now these results were taken in week 12, so they were taken right in the middle of that second dosing interval. If I had selected week 16, so right at the top level, these results really would not had been too different.

The only thing that you would have noticed is that with the lower doses where the durability of effect is not quite as significant as the higher doses over this entire dosing interval, you would have seen these numbers come down some.

Note again, that as we've seen in all other views of the data, 200 microgram per kilogram does not offer any significant benefit over 100 microgram per kilogram.

So to put those results in context a little bit and to give you a sense of why we're quite excited about the efficacy profile of Tanezumab, I went back and extracted out pain responder rates out of a NSAID Phase III program in osteoarthritis pain that we did several years ago. And I have masked the identity of the NSAID, but if you're fairly clever and you know that the studies were done with Pfizer, you might be able to guess what that NSAID was. Maybe.

Now this is traditionally what we see, so this is not atypical at all. And, in fact, it's really not atypical of what we see if you step to other therapeutic agents in different settings of pain, whether it be neuropathic pain, whether it be an acute pain or fibromyalgia.

In terms of a 30% response rate, you see that the results are statistically significant. You can see about 50% of the patients did achieve this response rate. But the delta between the active treatments and placebo is much less.

You see the same pattern occurring with a 50% response rate, and as I've mentioned previously, we tend to totally ignore response rates beyond that because most of the responders don't report levels of pain relief in that type of category, and the data usually are not particularly enlightening.

So I'll stepped into the safety results now from study 1008. First of all, I want to make sure that you appreciate that this was a 16-week study. Okay? That's a little bit different than you are used to seeing in chronic pain trials. We typically go 12 weeks, three months, particularly in placebo-controlled trials. That's about as far as we can go, and thankfully, that fulfills pretty much a regulatory criteria as well.

So we have taken this trial out a little bit longer, and it will probably be indicative of types of results that you will see with Tanezumab in Phase III as well in terms of duration of trials.

We're quite pleased, actually, and encouraged by the safety and tolerability data that we've seen with Tanezumab to date. So this is a summary of the safety data from sort of the 10,000 foot view where I'm just showing you the incidence or the percent of patients reporting either adverse events who withdrew due to adverse events or who had serious adverse events in study 1008.

Note that there were more patients who experienced or reported adverse events in all the Tanezumab treatment arms as compared to placebo. If you look real hard, you can perhaps see a dose response relationship, not a very well-characterized one, and if you took the opposite view, I don't think I could convince you otherwise.

Also, you see similarly that the withdrawal due to adverse events also occurred more frequently across the Tanezumab groups, and if anything, the dose response relationship here was even less well-defined than it was in terms of adverse events.

And for serious adverse events, the incidence across all of the treatment arms, including placebo, were remarkably low and not different from each other.

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

So zeroing in on adverse events a little bit more in a little bit more detail, headache, upper respiratory tract infection, paresthesias, hypoesthesias, and arthralgias or joint pain, were the most frequently reported adverse events for patients treated with Tanezumab.

I will have a little more to say about these latter three events in just a few minutes.

Infusion site reactions or pain or experiences of burning sensations upon Tanezumab infusion were rare. Okay? So that IV bolus push over five minutes was well tolerated by the vast majority of patients, which is a good thing.

The incidence of abnormal neurological exams in all the Tanezumab treatment groups was similar to placebo. There were no effects on heart rate, blood pressure, electrocardiograms or clinical laboratory values with Tanezumab patients as compared to placebo, and finally and importantly, there was no evidence of anti-Tanezumab antibodies seen in patients treated with Tanezumab. So as best we can tell to this point, Tanezumab is not immunogenic. Okay?

In terms of withdrawals due to adverse events, only arthralgia or worsening diabetes led to the withdrawal in more than one patient across the treatment arms. I spelled these out here. Arthralgia occurred in two patients, one treated with 100 and the other treated with 200 microgram per kilogram doses.

For worsening diabetes, there were three patients, one treated with 10 microgram per kilogram, the other 150, and the other one 200 microgram per kilogram. And one patient discontinued the study due to abnormal cutaneous sensations. That is a report of hyperesthesia. So keep that in mind as we get into that topic a little bit more.

Finally, in terms of serious adverse events, only seven patients in total experienced a serious adverse event in this study. That included one patient treated with placebo with chest pain, a case of cellulitis and appendicitis, and a patient treated with 10 microgram per kilogram Tanezumab, a case of breast cancer and bacterial arthritis in patients treated with 50 microgram per kilogram Tanezumab, and finally, one case of syncope and one case of lumbar spine stenosis in patients treated with 200 microgram per kilogram. Really nothing of major -- of any concern here actually in terms of serious adverse events, and the patterns that we've seen with Tanezumab, all of these were considered unrelated to study medication by the investigator.

Okay. One thing that we have seen in the pre-Phase III program with Tanezumab are adverse events related to abnormal sensation. And typically these adverse events code up or classify in the following manner. Either to what we call allodynia, which is really a pain sensation that can be actually quite severe and debilitating, to stimuli that you and I would normally think to be totally innocuous. That is to clothing or to touch.

The second category would be dysesthesias, and I like to typically think of this as a sunburn or a hot sensation experienced by patients.

The third category is paresthesias. This is a tingling, prickling or pins and needles sensation. So this is the feeling after your hand or foot has gone to asleep, that type of sensation. And the final category is hyperesthesia and also hypoesthesia. So this is either a higher or lower sensitivity to touch, pain or other sensory stimuli.

Okay, the incidence of these abnormal adverse events related to abnormal sensation in the study 1008 are shown on this slide. It's a fairly busy slide. There's a lot of numbers on it, so let me walk you through it.

Focus your attention way over here to the right. These are all doses of Tanezumab combined. And also, what you're looking at are all of these various adverse events in these four categories here, combined up for any adverse event related to one of these sensations.

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

So you can see that the incidence here was 10%. One in ten patients reported back these types of sensations. These sensations, by the way, are usually reported by patients to occur on the feet and the legs and the hands and arms, so the extremities. There are some cases, though, where patients will report these sensations either on their face or on their trunk.

So overall again, 10%, this compares to about 3% of patients reporting touch sensations or adverse events related to these sensations taking placebo. If you break this category down into its subcomponents, you can see that for the most part these were classified as either hyperesthesias, that is increased sensitivity to stimuli, or paresthesias, the pins and needles sensation. That's predominantly what patients report back to us in terms of the types of sensations that they're feeling.

Note that hypoesthesia is every bit as common as hyperesthesia. So in many cases, you can think of either activating the peripheral central nervous system, and in this case you can think of it as a deactivating system, and there seems to be sort of a balance in what patients report.

So a couple comments about the clinical characteristics of these adverse events. First, they occur early. They occur in the one to two-week period after the first infusion. Okay? They are transient. They usually in the vast majority of patients go away in about one to two -weeks. And upon second infusion do not occur again.

So they seem to be located around the time of the first infusion in a minority of patients. They are typically mild to moderate in terms of their severity. As I mentioned previously, only one patient withdrew from this study because of an adverse event related to these effects.

There are normal or minor changes on neurological exams. So on neurological exams, they will go through and basically evoke various sensations from the patients, either by pinprick or by vibratory sensation, and for the most part, those exams are normal.

So all of this leads up to the conclusion at this is not really consistent with a structural damage to our peripheral neurons in any stretch of the imagination. There is some perturbation that we hypothesize that's going on between the sensory neuron population as a result of the withdrawal of NGF's stimuli from these neurons and/or a resetting of the homeostasis within NGF-sensitive neurons themselves.

It could be a completely different effect as well, but our two hypotheses are sort of going in that direction at the present time.

Okay, to summarize, we are really encouraged by what we've seeing with Tanezumab in the Phase II program, and we believe that this compound has therapeutic potential in a number of chronic pain conditions. We're zeroing in on fixed low doses. We do not see the need to dose this on a per-kilogram body weight basis. So we will be studying fixed doses of Tanezumab in Phase III administered six times per year, and we believe that will help with the dosing schedule and the ease of administration to patients.

We're encouraged by the safety and tolerability profile that we've seen to date. We have seen dose-related transient, abnormal, cutaneous or peripheral sensations. They're not treatment limiting at this point in time. And the long-term effects on sensory neuronal function, as well as sympathetic autonomic function, will be investigated pretty significantly in Phase III. We're going to do this in a number of different ways.

First, we're going to bring in a large safety database in terms of our registration package. Secondly, we're going to implement neurological impairment scores in the trial. That is we're going to systematically, in addition to collecting adverse events, go out and ask patients to fill out impairment scores in terms of their neurological function.

There will be routine neurological exams that are conducted by physicians, clinical investigators in the trial. And in a separate trial, we will be evaluating nerve conduction velocity, autonomic function testing, as well as cutaneous peripheral nerve density to convince ourselves that we are not over a long-term administration causing any structural damage to either autonomic or sensory peripheral nerves.

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

In terms of upcoming milestones, I have mentioned a few of these. We intend to initiate Phase III in the osteoarthritis program this year with intravenous administration. We will begin the subcutaneous program by a small bioequivalent study comparing subcutaneous administration to intravenous. We will have a readout of various Phase II studies throughout the course of 2009. We plan to disclose the chronic low back pain results sometime during 2009 as well, as well as to begin our assessment of subcutaneous administration in osteoarthritis later in 2009.

Okay. That's really all I want to say about Tanezumab, and now let me take just a couple of minutes to walk you through Esreboxetine for the management of fibromyalgia.

As most of you probably are well aware, fibromyalgia is really a syndrome that is characterized by widespread soft tissue or muscular pain. And in particular, there is either spontaneous pain or there is a significant pain response invoked by pressure at certain discrete locations on the body. They're outlying here with the orange circles, and these are so-called tender points.

The syndrome was also characterized by an overwhelming majority of patients reporting that they are constantly fatigued, that they have disturbed sleep. Many patients report back anxiety or depression-like symptoms if not outright diagnosis of such. They also are characterized by some comorbidity, particularly around irritable bowel syndrome, Raynaud's disease, headache and paresthesias.

In terms of prevalence, it's estimated that it affects about 3 to 6 million people in the US. But the diagnosis rate is much lower. Confirmed diagnosis may be on the order of about 1 million patients with diagnosis. It's a syndrome or a disease that affects women more commonly than men and particularly women over the age of 50. And it's clear this is a difficult patient -- a difficult condition for patients, both in terms of getting the correct diagnosis, as well as once they are diagnosed, getting the correct treatment.

So the clinical development program for Esreboxetine really is placed against evaluating the activity and the efficacy of this compound across all of the domains that patients are affected by in this particular disease ranging from pain through fatigue, sleep, depression symptoms, as well as difficulty concentrating. And, in particular, we're going to focus on fatigue and in cognitive impairment, which we believe represents a significant unmet medical need when you compare that to existing treatments that have been registered for fibromyalgia or are still in development for such.

In terms of cognitive impairment, we currently have a Phase II trial ongoing to evaluate the effects of Esreboxetine on cognitive impairment versus placebo in patients with fibromyalgia, and if that trial is successful, we will then drive on that aspect in Phase III.

Now typically if you go to focus groups with fibromyalgia patients, you'll hear them throw around the term fibro fog. So this is really their code word for cognitive impairment. And when you sort of drill down on what does that mean to patients in a little more detail, it primarily comes back to you in three different ways.

First, they have a problem with short-term or episodic memory. They have a problem with vocabulary and verbal fluency, and they generally have a difficulty with sort of overall mental slowness. And this is particularly evident when they are under stress. And it just so happens that the trial that is in the literature with racemic reboxetine, or Edronax, has shown that at least the racemic combination can improve cognitive function along those particular aspects in patients with major depressive disorder. And that's unlike the results that we're seeing with paroxetine and selected serotonin inhibitor, reuptake inhibitor, in which no benefit was seen on cognitive impairment.

So we're going to take that story a little bit further and see how it plays out with Esreboxetine, as well as characterize the compound with respect to fatigue.

As I mentioned, Esreboxetine is a selective norepinephrine reuptake inhibitor. It's the more selective stereoisomer of Edronax, racemic reboxetine. The racemic reboxetine has been approved in depression in over 48 countries outside the US, so it's been

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

around for awhile. Esreboxetine represents a different pharmacologic approach than Lyrica. We will get into that story a little bit more in a minute. It is a true once-a-day product, and it's currently in Phase III development for fibromyalgia.

And in terms of its norepinephrine reuptake selectivity versus serotonin, it is very, very selective for norepinephrine reuptake, much more so than racemic reboxetine, which in turn is much more selective for norepinephrine versus serotonin reuptake than is milnacipran, duloxetine, the so-called SNRIs, which are then in turn much more selective than fluoxetine, which basically a selective serotonin reuptake inhibitor.

So one of the characteristics or deficits that is pretty clear that occurs in fibromyalgia patients is, if they have a defect in central pain processing, and to be more specific, they are overprocessing pain in the central nervous system, in the spinal cord and through the cortex, and if you read the literature carefully, this is sometimes characterized as central sensitization.

So Esreboxetine, by inhibiting norepinephrine reuptake, basically prolongs the activity of norepinephrine-containing neurons. Because the reuptake of norepinephrine back into the neuron is one of the major ways by which the action of this neurotransmitter is inactivated.

Now it just so happens that one of the descending projections, a populations of neurons from the brain stem down through the dorsal horn of the spinal cord contains norepinephrine. And these neurons are inhibitory in action with regard to their effects on pain. In other words, signals that come in from the periphery are transmitted in an ascending manner up to the brain and then processed and recognized as pain by individuals. But there is a descending neuron that can inhibit that activity and by increasing the activity of norepinephrine in this particular area here, when essentially what you're doing is increasing the inhibitory signals that tamp down pain. This is sort of the punitive area or location where Esreboxetine or other norepinephrine reuptake inhibitors is postulated to work in terms of their analgesia effects.

In other areas of the CNS, there are also norepinephrine-containing neurons. In general, when these neurons are activated, they have an excitatory or activating activity. So you can kind of easily take a step back and think about, well, that pharmacology matches quite a bit what we think about these agents in terms of reducing fatigue, as well as improving cognitive function. So those are the postulated mechanisms behind Esreboxetine.

For the Phase II study, study 1034, the results of the study were presented at ACR on Sunday. This was a Phase II study. It was a randomized controlled trial, placebo-controlled, and in patients receiving Esreboxetine, they began treatment at 2 milligrams once daily for a two-week period. And thereafter, they were forced those titrated at 2 milligrams increments per day at two-week intervals up to a total daily dose of 8 milligrams for the final two weeks of the eight-week study.

Now up to or over 60% of the patients actually were titrated up to the particular 8 milligrams, and only 10% of the patients were not titrated beyond 2 milligrams once daily. If the patients couldn't tolerate these doses, they were allowed a one-step dose titration.

And the reason we use this particular design is it's very efficient in the Phase II space to get information about the dose response relationship. It does have a particular weakness, and that is that you don't spend a lot of time on any given dose. So if the magnitude of your response is tending to grow over the course of time at any given dose, you may now see that play out to its full efficacy.

So to be eligible for this trial, patients had to have ACR-defined fibromyalgia. They had to have a score of greater than 40 on 100 millimeter pain visual analog scale and a mean score of 4 greater on a categorical zero to 4, a numerical pain rating scale.

So this slide summarizes the result on pain and the results on improvement in function in study 1034.

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

First, in terms of pain, this was measured on a zero to 10 point scale. Zero being no pain, 10 being worse pain. Patients came into the trial reporting their baseline scores of about 6.8 in both groups, and again, that represents moderate to severe pain on this particular scale.

We saw a significant reduction in pain with Esreboxetine treatment at the end of eight weeks, about a 0.6 point reduction. In most respects this is very comparable to what we have seen with Lyrica and what you can see in the literature with other treatments intended for the treatment of fibromyalgia.

Improvement in function, in terms of the fixed instrument that was used, this was again on a scale of zero to 100. Baseline scores were in the 60s as patients came in. Patients had a significantly greater improvement in their function when given Esreboxetine as compared with placebo-treated patients. In fact, the response was nearly double. Basically an 8-point difference is clinically meaningful on this scale based on our analysis of the Lyrica program. So on average, patients had a clinically meaningful response in terms of improvement in function.

Improvement in fatigue and the patient global impression of their condition are shown on this slide. Again, patients reported a significant improvement in their fatigue symptoms when given Esreboxetine as compared to placebo. In fact, the response was nearly doubled in Esreboxetine-treated patients. Note that again that these patients are significantly impacted by fatigue based on their baseline scores.

Finally, the patient global impression of change also was quite encouraging. Significantly more patients reported that their condition had been much improved or very much improved when they received Esreboxetine as compared to placebo, and even if you subdivide that out into very much improved, you can see again a very impressive difference between Esreboxetine and placebo-treated patients.

This slide outlines the time course of pain relief that was observed in the trial and also gives you a little bit of an idea about the dose response relationship. Recall that during the first two weeks, patients predominantly were on 2 milligrams of Esreboxetine daily, were titrated up to 4 milligrams during weeks three and four, four, up to 6 milligrams during weeks five and six, and up to 8 milligrams during weeks seven and eight. What you see is with increasing doses, the pain response gets greater. In fact, at doses between 4 and 8 milligrams, you're seeing statistically significant differences in magnitude of pain relief as compared to placebo-treated patients.

Just as I went through with you with the Tanezumab program, the responder rates that were observed in this trial lend themselves to some analysis of the clinically meaningfulness of the mean pain responses. Again, you can see that a 30% response rate here again was characterized as clinically important, was achieved by Esreboxetine-treated patients, more frequently than placebo-treated patients, and the same response occurred if you use the 50% response rate as your gauge of clinically meaningful response.

Now how does this compare to what we have seen previously? Well, in the Lyrica program, when you go back and pool across the data from all the studies that we did, we see a 50% response rate with Lyrica of 23% versus a placebo response of 15%. So you can see that Esreboxetine compares pretty favorably to our Lyrica database, which admittedly we have a lot more experience and a lot more exposure than we do with Esreboxetine at the current time. But anyway, the results are fairly encouraging.

I will very quickly step through a summary of the safety. Again, same format as what you saw with Tanezumab, adverse events, withdrawals and serious adverse events. You can see again that patients receiving Esreboxetine reported adverse events more frequently than those receiving placebo. Not unexpected. Withdrawal rates also were more frequent in Esreboxetine-treated patients as compared to placebo-treated patients. Overall, though, this withdrawal due to adverse event rate is quite low, and actually we're quite pleased to see this. These agents tend to be fairly active in terms of their profile in patients as we will get to in a minute.

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

Finally, there were no differences in serious adverse events between the treatment groups. And, in fact, there was one serious adverse event in each treatment arm. A myocardial infarction that occurred in Esreboxetine-treated patients after the trial had concluded, but within a 30-day window by which we monitor serious adverse events. And one case of a pelvic fracture in a placebo-treated patient.

This lists the most common adverse events that we are seeing. I want to draw your attention to the first four. So You can see that constipation, insomnia, dry mouth and headache were all more frequent in Esreboxetine-treated patients than they were placebo. Again, not unexpected based on what we know about the pharmacology of these particular agents.

Nausea was reported also more frequently in the Esreboxetine treatment group. But overall we're quite pleased actually with the incidence of nausea that we have seen in the trial as compared to other agents in the literature.

Dizziness also, while more frequent in Esreboxetine treatment group than placebo, was actually quite encouraging as well.

The reports of fatigue actually followed what we saw in terms of the efficacy assessment. That is fatigue actually was reported as an adverse event more frequently in patients receiving placebo.

Finally, this slide summarizes the effects on blood pressure and heartrate. Effects on blood pressure, either systolic or diastolic pressure, were seen in Esreboxetine-treated patients through the course of the trial. Again, no changes in placebo-treated patients for either assessment as well.

We did see an increase of about 8 beats per minute in terms of sitting heart rate. This seems be fairly comparable to what's been reported for Edronax. Again, this is at 8 milligrams once daily. What we don't really have a good appreciation of is how significant and robust the dose response relationship is. So we will see how that plays out in Phase III as we study doses ranging through 4 to 10 milligrams once daily.

So to sum up, based on study 1034, we believe that Esreboxetine has a significant therapeutic potential in fibromyalgia. We're basing that on the results that we have seen in pain function and fatigue and our assessment behind the pharmacologic rationale that the agent may provide benefit in cognition. Based on its pharmacologic mechanism, it does have the potential for study as either adjunctive therapy or combination therapy with Lyrica. And it has also the potential to be differentiated from mixed reuptake inhibitors that, of course, also inhibit serotonin. Particularly serotonin reuptake inhibition starts to result in some sexual dysfunction, as well as some GI intolerance.

And we may be able to, during the course of the trial, optimize the benefit risk for certain phenotypic subgroups within the fibromyalgia population. It's been a lot of work and a lot of work ongoing about looking at the subgroups or the components of the fibromyalgia syndrome, and basically evaluating patients based on what is the predominant symptomology that the patients are reporting.

Now I think where that leads us is the fibromyalgia syndrome in aggregate may not be what we target several years down the road. We may be looking at more targeted therapy to particular subgroups based on the phenotypic expression of the syndrome itself.

We have encouraging safety data to date. And again, we will be looking at differentiation from mixed reuptake inhibitors in Phase III. And we will also be looking very carefully at the mechanism-based safety and tolerability in Phase III, particularly with respect to cardiovascular safety, i.e. effects on blood pressure, heart rate, as well as orthostatic hypertension, palpitations, etc., and also carefully evaluating the anticholinergic effect -- constipation, urinary retention, dry mouth, etc.

Well, I've mentioned some of the upcoming milestones. We will have a readout of the first Phase III trial. In late 2009 is when we are expecting to have the results in hand. We will be initiating other Phase II trials next year to move that program along. And again, we're projecting an NDA submission again somewhere in the 2011/2012 time period for this particular program.

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

Okay. So with that, I will turn the podium over to Dr. Weiner, who will take you through the CP-690,550 program.

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

Good evening. I'm Ethan Weiner, the development area head for inflammation at Pfizer, and I like to talk to you about some of the data that we presented at this year's ACR meeting regarding CP690,550, our oral JAK inhibitor.

I'll begin by reviewed why we feel inhibition of the Janus kinases is an important new approach to rheumatoid arthritis and then orient you as to where the data we presented at this meeting fit into our overall development plan. I will then summarize some results from our two presentations and one poster at ACR. Following my presentation, all of us on the panel will be happy to answer any questions that you might have.

Let's first take a look at the current unmet need in the RA market. The last decade has seen many advances in the treatment of rheumatoid arthritis with the introduction of anti-TNF therapy and other biologic response modifiers. These agents have generally shown a degree of efficacy that has not been observed with the older oral agents.

However, as large molecules, they are subject to limitations which include injectable administration and high treatment cost, and hence, there is suboptimal access to them in the US and especially in many other parts of the world.

There has been no new oral small molecule treatment introduced for RA in over a decade, since the introduction of Leflunomide. Both Leflunomide and methotrexate are generally more effective than the previous generation of DMARDs before that, or disease-modifying anti-rheumatic drugs such as gold or penicillamine or asulphadine. However, like the older treatments, Leflunomide and methotrexate do not have clear targets or mechanisms of action. None of these DMARDs, oral DMARDs agents overall, alone or in combination, are as effective as the biologic immune response modifiers that target specific aspects of the immune system, especially when the latter are combined with methotrexate.

Both physicians and patients, therefore, need new therapies which combine the benefits of small molecule treatments with the effectiveness of targeted mechanism of action.

We believe that CP690,550, if successfully developed, will be well received in the RA marketplace, with the potential to combine the advantages of small molecules, rapid kinetics, ease and convenience of administration; with those of biologics, targeted modulation of the immune system. It has the potential to be the first new oral DMARD to be introduced in the market in over 10 years. CP690,550 could be an exciting and important new development for the RA treatment armamentarium, as well as for other inflammatory or autoimmune conditions.

As you will see from Study 1025, the efficacy of CP690,550, which I will refer to as CP for the rest of this presentation, maybe get a little bit of time back that way, generally improved with higher doses. And based on the data we've seen to this point, we believe that adverse events are manageable and can be well-characterized in a suitably designed Phase III program.

Let's turn now to the role of Janus kinase inhibition in the treatment of rheumatoid arthritis. Blocking JAK 1 and 3 activity interferes with the function of a series of receptors which depend on these kinases to initiate downstream events when the receptor is occupied by certain cytokines. JAK, when phosphorylated -- when a cytokine binds to the receptor, JAK gets phosphorylated and activates STAT, which then immediate downstream effects, switching on and off various effector genes. Blocking JAK, therefore, blocks the activity of STAT. Because JAK is an integral part, both JAK 1 and 3, an integral part of the receptor for a whole host of inflammatory cytokines, blocking its activity produces multiple downstream effects at all of these different receptors.

Inhibition of these multiple downstream cytokine-mediated effects operate in several areas to reduce the disease process in rheumatoid arthritis. And hence, via inhibiting the activity of these cytokines, there is an effect on both B cells and T cells, as

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

well as monocytes and NK cells. Interference with either B cell or T cell activity has already shown efficacy in rheumatoid arthritis and is the basis of biologic response modifiers currently marketed, such as Rituximab or [Abitasef], respectively. CP also inhibits another JAK isoform, JAK 2, about 20- to 30-fold less potently than JAKs 1 and 3. JAK 2 is necessary for generation of white and red blood cells and may be expected to have effects on their production at higher doses.

Before turning to the data presented at this ACR meeting, let me briefly review the program and the data we presented at the ACR meeting two years ago. In 2006 we presented data on Study 1019 at that ACR meeting. This was our initial proof of concept study in RA, and there are several things to note about this study relative to the results we presented this year. Study 1019 was of six weeks duration only versus current longer studies. Patients were methotrexate or TNF inhibitor failures and were on no background DMARDs during the study, including the background methotrexate.

Doses evaluated in Study 1019 were 5, 15 and 30 milligrams twice a day, which were higher than the doses studied subsequently, ranged higher, subsequent doses of 1, 3, 5, 10 and 15 twice a day.

Before reviewing the results of Study 1019, I would like to briefly review the ACR 20, 50 and 70 scores that we and most of the industry and regulators use as our main efficacy end point. Most of you are probably familiar with it, but for those of you who are not, the ACR score represents the proportion of patients who respond to the predetermined criteria. These predetermined criteria are comprised of a composite primarily of clinical findings -- How does a patient feel? How does a physician think the patient is doing? -- and one laboratory parameter measuring inflammation, either CRP or ESR.

ACR 20 was originally designed to distinguish the older DMARDs from placebo. Individual studies varied tremendously for a host of factors, but as a very crude rule of thumb, older DMARDs produce ACR 20 changes of about 10 to 20 points higher than placebo, and newer biologic response modifiers average 30 or more points higher than placebo. Likewise, a 20-point increase in ACR 50, for those that have 50% response in these parameters, is reflective of the type of efficacy that newer biologic response modifiers produce.

I think most of you are probably familiar with the efficacy results from Study 1019, which showed both rapid onset of efficacy and fairly robust results relative to placebo with onset of activity as early as one-week. ACR 20 responses at the various doses ranged from about 41% to 52% above placebo at six weeks. Several safety findings were also observed in that study. As expected, there were dose-related decreases in mean hemoglobin, a measure of red blood cells, and neutrophils. Moderate to severe neutropenia, or less than 1500 white cells per cubic millimeter, and was not associated with infections over the six-week period. Dose-related increases in total cholesterol, LDL and HDL cholesterol, were observed as well as small increases in mean serum creatinine in all the CP treatment arms. Individual increases in serum creatinine were generally a very small magnitude with few values exceeding normal limits. Values of red cells, white cells, lipids and creatinine generally normalized during the six weeks of follow-up off-study drug.

Due to the rapid and robust efficacy observed with what promised to be a manageable safety profile, we felt this molecule had great potential in RA and therefore embarked on a larger program.

Since the completion of Study 1019, and that's colored in that little 2006 ACR presentation there, we've launched an extensive global Phase II program, key elements of which are six-month studies both on background of methotrexate -- and again, this is what was presented this year -- and monotherapy as well. These studies will inform the dosing regimen for Phase III. In addition, subjects who complete any of these studies get enrolled into open label extension studies to gather additional safety data and confirm durability of efficacy. Subjects who do not complete treatment are followed up off-drug to ascertain long-term sequelae of treatment with CP. Thus, even in advance of Phase III, we will be accumulating both long-term safety data and efficacy data to augment the results generated in the Phase III program.

Our plan has been to study CP both as monotherapy, i.e., the only DMARD in a patient's regimen, and in combination with methotrexate. Methotrexate is often combined with other agents in rheumatologic practice in order to provide additional therapeutic options. At this ACR meeting, we presented the results of the 12-week interim of the six-month background

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

methotrexate Study 1025 and also the results to date of Study 1024, an open-label extension Study for subjects completing 1025, 1035 and 1019.

Finally, we presented results of a drug-drug interaction study. The data from this study showed that levels of CP and methotrexate are not significantly affected by one another, making dosage adjustments of either medication in the presence of the other unlikely to be necessary.

I will now take you through the data that has been presented at this ACR meeting. Unlike Study 1019, which included primarily methotrexate failures, Study 1025 enrolled subjects who had failed to respond completely to methotrexate. They did, however, remain on their stable background dose of methotrexate throughout the study in order to evaluate both the safety and efficacy of using these two treatments together. So we were really studying CP plus methotrexate versus methotrexate alone in this study, not CP monotherapy versus placebo as in 1019. Perhaps as a result of being incomplete responders to methotrexate versus total DMARD failures, disease activity at baseline was somewhat lower in Study 1025 versus Study 1019. Subjects in Study 1025 were randomized equally to a range of doses of CP, including 1, 3, 5, 10, 15 milligrams twice a day, 20 milligrams once a day or placebo, for 24 weeks. The primary efficacy end point, however, was ACR response at 12 weeks, which is presented here.

Durability of response is assessed at the end of the study at 24 weeks, and safety is assessed throughout. It is also important to note that subject who failed to achieve an adequate response at 12 weeks to placebo or the lower doses of CP, or 20 milligrams once a day, were automatically advanced to 5 milligrams twice a day for the second half of the study. This advancement was blinded to Pfizer investigators and subjects. Roughly 70 subjects per treatment arm were enrolled for a total of 509 study subjects.

This slide shows the ACR 20, 50 and 70 responses at 12 weeks. As you can see, all the responses, or all the doses, except 1 milligram twice a day, produced a statistically significant ACR 20 response relative to placebo. The ACR response for placebo itself was somewhat higher than for 1019 -- 37% at 12 weeks as opposed to 29% in 1019 at six weeks. All doses above 3 milligrams appeared to be efficacious.

Taking other time points besides 12 weeks into account, a dose response emerges with a plateau of the dose response curve between 10 and 15 milligrams twice a day. Modeling suggests that 5 milligrams twice a day is roughly the ED 70, or 70% of the theoretical maximum effect, and 10 milligrams BID is roughly the ED 80, or it's 80% of the theoretical maximum.

This slide shows time to onset. As in Study 1019, statistically significant differentiation from placebo at all doses but 1 milligram is apparent at the first visit; in this case, at two weeks.

So, to summarize the efficacy, all the doses except 1 milligram twice a day showed a significantly better response than placebo, confirming the efficacy from the earlier 1019 Study. Efficacy generally improved with higher doses, and maximal effects were generally observed at 15 twice a day. Onset of efficacy was as early as two weeks, peaked at eight weeks and was maintained for all 12 weeks, as you can see here.

So let's turn to safety. One key concern with any immune suppressive agent is infection. As you can see here there were five serious infections among 509 study subjects, consistent with the roughly four per 100 patient year incidents observed throughout our RA database of about 900 patients, approximately 250 patient years of treatment overall. This rate is similar to the rates reported in the literature for RA in general of about four to six per 100 patient years. No clear dose response was observed, and we have not seen a correlation of low white counts with any of these serious infections. We will need longer-term follow-up in larger numbers to assure ourselves that this risk is manageable, but these results are so far encouraging that we will in fact achieve that.

As expected, there are dose-related decreases in hemoglobin and absolute neutrophil count relative to placebo hemoglobin received primarily at the higher doses, bearing in mind that placebo itself has a change of minus 0.35. We see that there. And

Oct. 28, 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

-- sorry -- and for absolute neutrophil count, again, we see changes at the higher doses; and for hemoglobin, we see it at all doses -- well, just, actually, the higher ones as well, relative to placebo. These are actually increased somewhat relative to placebo.

Importantly, however, severe anemia and neutropenia were infrequent at any of the doses. Severe anemia was observed a little bit more frequently than for placebo at the higher doses. Only three subjects had absolute neutrophil counts less than 1000, and you can see two on a high dose, one on a low dose, and no potentially life-threatening cases of neutropenia were observed.

We also observed dose-related elevations at liver enzymes at the higher doses. These transaminases, which are a laboratory measurement of possible liver injury, could have been due to the addition of CP to methotrexate, which is known to have liver effects. The elevations in liver enzymes appear to be a problem largely limited to the higher doses. And no subject met Hy's rule for life-threatening drug-induced liver injury.

As we saw in Study 1019 as well, there were elevations in both LDL, or bad cholesterol, as well as HDL, the so-called good cholesterol, which again occurred largely in a dose-related manner that could be observed at all doses. The reason for the lipid changes is currently not well understood. These changes are of a level that should be controllable with statins or other interventions, if necessary. And since HDL and LDL elevations are balanced, they should in theory not lead to any increased cardiovascular events. However, we will be carefully watching for and adjudicating such events throughout our Phase III program.

So, to summarize the safety, serious infections were few and not dose-responsive. Dose-related changes in hemoglobin and neutrophils were seen, but incidences of severe or life-threatening anemia and neutropenia were low. No subject was withdrawn for protocol-specified hemoglobin or neutrophil levels.

Small increases in serum creatinine over baseline were seen in all dose groups, including placebo, and no increases were progressive over time compared to placebo. Dose-dependent increases in LDL and HDL and total cholesterol were observed, but increases appeared to plateau by week six.

Increased incidence of potentially significant ALT increases were observed at 15 milligram BID dose, the highest dose. Based on the data at this point, we believe that these safety issues are manageable and can be well-characterized in a suitably design Phase III program.

Our overall conclusions from Study 1025, therefore, was that efficacy and safety profile justifying progression to Phase III was observed. These conclusions will need to be confirmed by the 24-week data from the same study as well as what we learned from the 12-week data from Study 1035, the large Phase IIb monotherapy study.

As I said earlier in my presentation, we are starting to accrue longer-term safety and efficacy data, even in Phase II. We will continue to accrue such data throughout the development program by use of similar open-label studies in Phase III.

I will now take you through the results of Study 1024, which we presented at this meeting as well.

This study accepted patient who has participated either in Study 1019, 1025 or 1035. At the time of this data cut in February, only a handful of subjects had entered from Study 1035, a monotherapy study, and are not discussed further. All patients entered into this study could continue methotrexate or whatever other DMARDs, other than potent immunosuppressants or biologics, they had been put on since the completion of Studies and 1025, respectively. All study subjects were given 5 milligrams twice a day of CP in addition to their other therapy. Dose reductions were allowed for adverse events, but only two subjects had to have their dose permanently reduced.

An interim analysis was conducted of all safety data for all 129 patients enrolled as of the 22nd of February of this year. Comparisons in laboratory data and disease activity score were made at one and six months. As of the cut-off, 40 patients had completed six months of treatment in the extension, and their laboratory parameters and DAS were compared between months

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

one and six. DAS was used instead of ACR because the ACR score is based on comparison to baseline, and these subjects all had multiple baselines to compare to. And at the time of the analysis, the 1025 baselines were still blinded.

Of the full complement of 129 patients, there were 160 adverse events after they were enrolled into the follow-up study. Most were mild or moderate with only three severe adverse events. Among the 160 adverse events, there were 32 infections, but none were characterized as severe, and there were no serious infections at the time of the data cut, although several occurred thereafter.

At six months, serum creatinine ranged from 0.38 to 1.58 milligrams per deciliter. 28 patients were actually below the normal range of 0.57 to 1.06, and 21 were above. The highest serum creatinine reported was 1.58 milligrams per deciliter in a patient with a UTI and abdominal pain.

Neutrophils ranged from 1.25 to 15 -- 46 patients above the upper limit of normal and four patients were below the lower normal range. The lowest reported neutrophil count was 1.2 in one patient.

Hemoglobin ranged from 9.1 to 16.5 grams per deciliter, with only four patients having one hemoglobin reported below 10 while on study drug. LDL and HDL cholesterol levels remained generally within the normal range.

Overall, therefore, these key lab parameters remained stable after six months of treatment, mostly within a normal range, with few significant outliers.

One way of assessing disease activity was the DAS for this study, rather than the ACR. As you will recall, ACR measures improvement from baseline, but these study subjects had multiple baselines, i.e., when they first entered their blinded study, when they finished the blinded study, when they started open-label treatment. We therefore compared disease activity at various time points as measured by the DAS. DAS is a mathematical formula-based composite end point based on tenderness and swelling of 28 joints plus either CRP or ESR.

What is important to assess in terms of durability of treatment is the score at one month into this open-label study compared to the score at six months. Looking at two different versions of the DAS28 score, either based on CRP, as in this line, or ESRs in the next slide, durability of effect can be observed between one month and six months, regardless of which study the patients originally came from. And again, same with the ESR-calculated DAS.

So far, the open-label extension has been encouraging and has demonstrated sustained efficacy. The dose used 5 milligrams BID, was sufficient to retain control of the underlying disease state and was generally well-tolerated. DAS28 was similar in all patients at six months, regardless of prior study experience and mean laboratory values remained within normal limits at six months. Importantly, no patient required discontinuation because of changes in laboratory values.

I will now briefly summarize the poster we presented, looking at the pharmacokinetics of CP and methotrexate together. Study 1013 was a two-week study to estimate the effects of methotrexate on the pharmacokinetics of CP, and vice versa, to guide any dosing adjustments of either that could be potentially be necessary when the two agents are combined. This is important information for practitioners, ultimately, because methotrexate is commonly used to treat patients with RA, and doctors need to understand how to safely use CP with methotrexate.

12 RA patients on stable methotrexate doses of 15 to 25 milligrams per week were enrolled. They were dosed with 30 milligrams of CP every 12 hours for nine doses. Co-administration of the two agents was well-tolerated, and neither drug had a sufficient effect on the pharmacokinetics of the other to warrant dose adjustments. Results of this study are intended to be combined with the safety and tolerability observed in the background methotrexate studies, such as 1025, to informed dosing regimens in Phase III.

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

Since you last saw our presentation at the 2006 ACR, we have been assiduously studying CP in rheumatoid arthritis. We are pleased to share some of our recent results with you and feel confident that these data will support starting a Phase III program in the near future.

So, to summarize, RA is a disease area with significant unmet medical need. There have been few advances with DMARDs in more than a decade, and large molecules are subject to a number of limitations, including injectable administration, and high treatment costs. Both physicians and patients need new therapeutic options that combine the benefits of small-molecule oral treatments with the effectiveness of targeted biologic -- targeted mechanism of action seen with biologics.

We believe that CP690,550, if successfully developed, has the potential to combine the advantages of small molecules with those of a biologic. As I've mentioned, CP has a novel and well-characterized mechanism of action and has demonstrated promising profile in clinical studies to date. In Study 1025, efficacy generally improved with higher doses and oral administration at 15 milligrams twice a day resulted in maximal effects.

Additionally, we believe that the adverse events seen in study 1025 are manageable and can be well characterized in a suitably designed Phase III program. A robust clinical program for CP is being executed, and we believe the efficacy and safety profiles seen to date justifies progressing into Phase III. We anticipate moving into Phase III during the first half of 2009 and we'll keep you apprised of our progress.

I will now turn the meeting back to Jennifer Davis, who will open up the floor for questions. Thank you.

Jennifer Davis - Pfizer Inc. - IR

Thank you, Ethan. We will now take questions both from the audience here in San Francisco and from those on the webcast. For those who are on the webcast, you should see a box that you can type a question into. And we'll go back and forth between the room and the questions we see coming in online. For those here in the room, I just ask that you raise your hand; we can bring a microphone to you. And if you could start with your name and your firm, that would be great.

And then finally, just given the depth of experience and expertise our panelists have regarding Tanezumab, Esreboxetine and CP690,550, I would ask that you keep your questions really focused on those three candidates.

So we'll take our first question here in the room.

QUESTIONS AND ANSWERS

Brian Abrahams - Oppenheimer & Co. - Analyst

Brian Abrahams from Oppenheimer & Co. A question for Dr. Wiener. Can you talk about the geographic distribution of patients enrolled in Study 1025 and whether or not that had impact on the response rates?

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

Yes, actually a really good question. So patients from that study were from North America, Eastern Europe and Latin America, primarily. And in that study, as I recall, we did not really see a big difference -- actually, in North America I think we saw a bigger placebo response than the other two regions, to some degree, which was the opposite of what we had seen in 1019, where I believe placebo response was much smaller in North America than other parts of the world. So we have not seen consistent a pattern as far as geography is concerned.

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

Seamus Fernandez - *Leerink Swann - Analyst*

Seamus Fernandez from Leerink Swann. Just a couple of questions. One, can you -- this is for Dr. Wiener on 550. Just wondering if you can give us a little bit more clarity on the last observation carried forward question versus the ITT analysis. Can you give us some data on actually what an ITT analysis would have shown with this data? And then, separately, can you also give us a little bit more clarity on how many patients are actually required at this point for an FDA approval of CP -- of 550 so that we can get a better sense of the timeline?

I think, as far as we are concerned on this one, I think Ken gave us timelines for both those products, but we haven't gotten a timeline for 550.

Dr. Ethan Weiner - *Pfizer Inc. - Development Head for Inflammation*

Okay, so a few questions. So, for 1025, to clarify, the analysis done was an intention-to-treat analysis. And that just means that anybody that gets randomized -- they go through their baseline visit, is included in the analysis. What we presented was last observation carried forward data. So that means a patient makes it out to week 12. That's the data that we use at week 12. If they drop out for any reason, say at week six or something, the results at week six are carried forward.

We also did not present BLCF, or baseline carried forward, which is a more conservative analysis. So if a patient drops out at week six, rather than their week six result, where they may have improved, we actually take their baseline result, which of course, by definition, is and ACR of zero; right? Because they are compared to baseline -- and carry that forward. Actually, the results for that looked very similar.

What we have not done is to take patients who dropped -- any patient who drops out, and automatically call them a treatment failure. I suspect that analysis would probably look pretty similar to the BLCF one, but we haven't actually done that.

But whether something is a last observation or baseline carried forward is independent of whether it's intent to treat or not.

Your second question was about time line and how many patients we need. So we are currently planning our Phase III program now, and we plan to vet this with regulatory authorities in the US, Europe and Japan pretty much at the same time so we can have a global simultaneous filing. But what the requirements will be will largely come out of that discussion, I think. So it's premature to say anything, other than we anticipate that, of the 2010 to 2012 large cohort that Pfizer hopes to file in those years, this will be among them. But I, at this point, couldn't really pinpoint where on that time frame it would end up.

Jason Kantor - *RBC Capital Markets - Analyst*

Jason Kantor from RBC. On the CP compound as well, as you're looking to Phase III, what do you think the right dose is? What do you think the right program to manage all these various AEs are, in terms of -- do you reduce dose, do you take people off, how often do you have to monitor patients? What do you think is the appropriate way to handle these patients?

Dr. Ethan Weiner - *Pfizer Inc. - Development Head for Inflammation*

Well, there's a bunch of questions inherent in that. So, in terms of what we think the best dose is -- again, we are still accruing data from 1035. We still have the final data from 1025 to look at. And, we have discussions with the regulators to actually determine what the doses that we bring into Phase III are going to be.

So we haven't totally locked in on those yet. Some of the information in terms of dosing up or down or adding or taking out other medications will probably be answered by the fact that patients from our large parallel group studies will all be feeding

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

into, again, many more open-label studies where we'll probably have more flexibility of dosing than we currently do in our open-label studies.

As far as safety goes, one of our strategies will be to adjudicate for key events that we are interested in and actually pool, make the entire Phase III program one sort of giant outcome study for all those adjudicated events. And I think, with the numbers that we probably will have in this program, which, again, have not exactly calculated yet, but it should be pretty large -- I think that will give us a pretty good handle on some of these things, such as infection, whether or not the lipid changes in fact do have any cardiovascular events associated with them, things of that nature.

Jennifer Davis - Pfizer Inc. - IR

I've just got one from the webcast here I'll go to real quick. [Chris Schott] says -- just looking out, what additional indications for the JAK 3 product are you most enthusiastic about?

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

Well, I'm enthusiastic about all of them, I guess. But the ones that we currently are pursuing in Phase II, at least, are psoriasis, both oral and topical; inflammatory bowel disease, both ulcerative colitis and Crohn's disease; and, of course, solid organ transplant, which is the most advanced program behind RA.

Rachel McMinn - Cowen and Company - Analyst

Rachel McMinn from Cowen. I wanted to just follow up on the cardiovascular commentary. In Phase III, will you actually have people monitoring for lipids and then recommend that they start statins to see how that works, or will you just leave it up to the practice of the individual physicians?

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

I don't know that we have spent much detail -- but I will actually defer that question to [Sam Zwillick], who is our clinical lead for the program in rheumatoid arthritis.

Sam Zwillick - Pfizer Inc.

So, the problem with blanket recommendations that individuals' risk factors differ at baseline and standards of care are not uniform throughout the world, so we have not planned to dictate the response. But the data on lipids are never blinded from the investigators, and they are encouraged to deal with them appropriately as per local standards of care and the individual patients' risk factors.

We do have evidence from both our transplant and our RA program that, for example, appropriate therapy to lower lipid agents is efficacious when it has been initiated, but we have not dictated when to do so.

Tim Anderson - Sanford Bernstein - Analyst

Tim Anderson of Sanford Bernstein. How do you make sure that your JAK 3 inhibitor doesn't end up like another Leflunomide? Because you're entering into a category where you've got a whole bunch of nonbiological oral DMARDs out there. You talked about how these older agents have ACR 20s above placebo that kind of range from 10 to 20. Your ACR 20s, out of your study

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

1025, was right around 20. And, you also have this treat paradigm, as well, where you're using biologics earlier in therapy to try to prevent joint erosion.

So, coincident with that question, is more specifically, what would the Phase III development program look like? I would anticipate that you might run something like radiographic studies right out of the gates, for example, and have that by the time you submit the package.

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

Certainly, radiographic studies will be part of our Phase III program. I think, in terms of the efficacy, there's a range between 1019 and 1025. And, actually, even the ACR 50s, though, on 1025 look better than, I think, what you see with some of the older oral DMARDs, although -- and again, there's a wide range even of what one sees with biologic response modifiers. Even there, some of the studies have gone -- the deltas are as low as 20. So in terms of what the ultimate efficacy picture looks like, I think we'll have a better idea at the end of Phase II, when we collect data from 1035, and certainly a much better idea at the end of Phase III.

But what we are depending on is a degree of efficacy that is better than, I think, one -- more a degree of efficacy that one generally associates physicians and patients with the effects that you get from a biologic, rather than the effects that you get from some of the older oral agents. And again, radiographic studies will certainly be part of our program.

Tim Anderson - Sanford Bernstein - Analyst

And, if I could ask one other question on that. You talked about LFTs, but did you have any bilirubin elevations at all in that program? And then, moving on to your NGF, you showed -- I guess you said you basically had 26-week animal tox on that program. But I would imagine you have to run substantially longer animal tox studies to look at [neurologic] degeneration and stuff like that. Is that correct?

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

So as far as bilirubin elevations, I do not think we had any. Just check -- Sam?

Sam Zwillick - Pfizer Inc.

The highest was 1.4 or 1.5 nothing (inaudible question - microphone inaccessible) 2 or 2X, which is (inaudible - microphone inaccessible).

Jennifer Davis - Pfizer Inc. - IR

Can you repeat that, just for the webcast?

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

We just asked Sam Zwillick -- the highest that we had was about 1.4 or 1.5, which is very minimally elevated.

Tim Anderson - Sanford Bernstein - Analyst

Is this an [ALP] (inaudible - microphone inaccessible) elevation?

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

Sam Zwillick - Pfizer Inc.

For that, I would have to go back to the database to look at.

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

If we have that, I can't pull it out of my memory.

Tim Anderson - Sanford Bernstein - Analyst

(Inaudible question - microphone inaccessible) animal studies?

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

Yes, so we completed what's necessary in terms of toxicology exposures for NGF. In other words, we won't go beyond the 26 weeks. Basically, as you know, we've got coverage, and we'll take it through on a clinic.

The only toxicology study that we will run is specific to embryo fetal tox. So we'll conduct that study, and that will be sufficient for registration.

Tom Russo - Robert W. Baird - Analyst

This is Tom Russo with Baird. Two quick questions. Can you talk about the temporal pattern in the study with the elevated liver enzymes and the neutrophils counts, what that looked like over time?

And also, secondly, I've heard a couple of comments about the high cost of current therapies and I was wondering -- if your efficacy is commensurate with biologics, do you envision a discounted price point? Or would you look at to price at parity?

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

Why don't you do that one first, and I will do the second one.

Unidentified Company Representative

So, from a price standpoint, it really is very early for us to be making decisions like that. We're just starting to see the profile come out of Phase II, and we will certainly want to see the Phase III data before we make those types of decisions. But we do think we will come forward with a very strong value proposition that payors will be interested in and be willing to reimburse the product.

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

So, as far as the lab abnormalities go, we generally -- and actually, if we could have the third from the last slide, I think? Let's see if this deck is put together similar to what I had.

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

Jennifer Davis - Pfizer Inc. - IR

Here, let me grab the --

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

But generally, the pattern is that things seem to stabilize after several weeks. And, again, in our long-term follow-up study for six months, all of those parameters tended to be pretty flat. So this is just an example with neutrophils, for instance, where you see decreases early on. But, by the end of -- by about six weeks or so, they are already starting to stabilize at that point.

I think the LSTs probably showed generally similar pattern to that.

Bert Hazlett - BMO Capital Markets - Analyst

It's Bert Hazlett from BMO Capital Markets. On the nerve growth factor, the data from EULAR, I believe, indicated some peripheral edema, 0% at the low dose, but up to 11% at the higher dose. Could you comment on that and characterize it, please?

Dr. Ken Verburg - Pfizer Inc. - Development Head for Pain Therapeutics

Yes. I can't speak about the precise percentages; but, yes, your recollection is right. Peripheral edema was evident in the trial. That is not atypical, again, of the population of that study. Typically, those patients [need] or the patients are over 60 with some degree of cardiovascular morbidity.

I don't know that we ascribe any potential mechanism related to NGF to that particular finding at this point. We're going to look at that more carefully and see whether or not the temporal relationship that we've seen plays out in Phase III.

Bert Hazlett - BMO Capital Markets - Analyst

And on Esreboxetine, could you comment on the percentage of patients that actually reduced dose? I believe that was something that was included in the trial. And the primary reason for the dose reduction?

Dr. Ken Verburg - Pfizer Inc. - Development Head for Pain Therapeutics

I gave you the numbers that -- about 63% or so of the patients achieved the 8 milligram dose, and 10% of the patients stayed at about 2 milligrams. So I think, if you do the arithmetic there, the remainder of the patients either achieved one of those middle doses or down-titrated from the 8 milligrams.

Unidentified Audience Member

(Inaudible question - microphone inaccessible).

Dr. Ken Verburg - Pfizer Inc. - Development Head for Pain Therapeutics

Well, typically we left that to the discretion of the investigator. It would basically be their clinical judgment as to whether or not the patient was tolerating the dose in a reasonable fashion. So I don't have that categorized neatly for you, but it was left to the investigator's discretion, based on feedback from the patients.

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

Jennifer Davis - Pfizer Inc. - IR

Another question from the webcast, Chris Schott, JP Morgan.

For Tanezumab, how do you think about the market opportunity for an injectable product, given that the existing treatments are oral?

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

Sure. Well, one of the things that's really clear in the research we've been doing is, there is a large population of the moderate to severe pain patients that still aren't receiving adequate pain relief. The numbers we've seen in the US and the big five in Europe is about 10 million osteoarthritis patients that are not receiving -- that are in that moderate to severe category. And of those, about 25% are in the severe category.

So we do think there's still quite a bit of unmet need there, given the available therapies that there are. When you get more to that severe category, then you have to start going to the strong opioids, which have, certainly, a lot of their own issues.

The IV portion -- the one thing to remember is, it's not a typical infusion like you would see with the RA biologics; it's a five-minute push. So we do think that will be something that will not be that difficult, but we are also quickly following on with a subcutaneous, and we think that will be, certainly, a way to open up the market further and allow patients then to self-inject at home.

Jerry Wheeler - - Analyst

[Jerry Wheeler] from the (inaudible) Fund. I have a question on CP. For the patients that became either neutropenic or anemic, did you dose-reduce or discontinue the drug? And then, what was the time course for the count to normalize?

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

Well, in the long-term extension study we did not discontinue any patients for lab abnormalities. And in 1025, I don't recall anyone -- I don't recall any discontinuations for lab abnormalities, either. But, Sam, I don't know if --

Sam Zwillick - Pfizer Inc.

For 1025 there was no dose reduction in Capital allowed, and there were retest criteria, and there were protocol-mediated withdrawal criteria. The protocol required withdrawal for confirmed neutrophil counts less than 500. We never got close to that.

We did not do a formal analysis of the time to recovery in the study. Investigators were required by protocol to follow up abnormalities for recovery, and they did. But in the 1019 study, we saw a rapid recovery of changes from baseline in the six-week follow-up off of drug. And I wouldn't expect that analysis would show anything differently; we just haven't tried to track those data down [to see] Dr. X did a second count at 10 days and Dr. Y at 13 days, and try to pull that together yet. We haven't done that.

Jennifer Davis - Pfizer Inc. - IR

We have one more on the webcast here. Back for Ethan -- would Phase III be a monotherapy or a combination with methotrexate, and do you think it would be either once-a-day or twice-a-day dosing?

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

Well, I'm not ready to comment on dosing yet. But our program will be looking at a combination therapy and monotherapy.

Unidentified Audience Member

On CP again, did lipid levels decrease once you stopped the compound? And then can you characterize the patients that might have been on other biologics, TNF therapies? Did you note any differences either in safety or efficacy with those patients with this compound?

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

Okay, so the first question -- lipid levels did go down within several weeks at most. We know that from 1019, because we brought everybody back after six weeks. As far as any differences among patients that had been on TNFs, there were probably not enough of them to really tell. I think we had something like 7% or 8% of the patients, I think, went into 1019 were TNF failures, and an even lower percentage from 1025.

So there just aren't the numbers to make those conclusions at this point.

Tim Anderson - Sanford Bernstein - Analyst

Tim Anderson at Sanford Bernstein. On Tanezumab, the name nerve growth factor implies that that actually has functions above and beyond just pain modulation. And I'm wondering, if you knock out NGF, if there's any theoretical toxicities that you need to monitor, such as any sort of impact on the central nervous system or anything like that? And if so, how do you monitor those in Phase III, in terms of duration, and what sort of study do you have to do?

Dr. Ken Verburg - Pfizer Inc. - Development Head for Pain Therapeutics

Yes; we are focused on a couple theoretical safety concerns around blocking of NGF. And it really involves where we believe that NGF, at least in some course of development or in the adult, plays a functional role. That seems to be located or predominantly in the C-fiber peripheral neurons, as well as C-fiber peripheral neural as well as sympathetic neurons.

And it's very hard to identify structural damage to neurons, particularly sensory neurons. And hence, the nerve conduction velocity study is a physiologic measure of, say, damage, particularly, though, to highly myelinated fibers. So that population may not exhibit any toxicity. But then we are going beyond that by doing some autonomic function testing as well as looking at peripheral sensory nerve density in the skin with long-term treatment.

We think that those three avenues will teach us a lot about the theoretical aspects of long-term nerve growth factor treatment.

One aspect that we can't control but we are very mindful of is patients that suffer from injury. If you think about it, people involved in auto accidents, et cetera, that have peripheral trauma likely have peripheral nerve damage. And those are things you can't predict and you can't, a priori, study. But we'll be very curious to see, as to the prognosis of those patients and whether or not we're able to detect any changes in sort of their recovery and in recovery, say, of peripheral sensory function.

The way to model that, by the way -- we can pharmacologically induce peripheral sensory neuropathies by a high-dose [gas-based] -- and we have been thinking about, actually, trying to conduct a clinical pharmacology study to evaluate that.

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

In terms of CNS function, we are of the view that, unlike, say, antibodies that are targeting and useful in Alzheimer's therapy, there is no transporter for NGF out of the brain. And obviously, the antibody is not getting into the brain. So we feel pretty confident that we are not going to disturb any equilibrium there. And, at least going in, we will be monitoring aspects of, say, mental cognitive function. But, a priori, we are not going to put anything specific in place.

We have done some cognitive function testing in the Phase II trial and (inaudible - microphone inaccessible).

Tim Anderson - Sanford Bernstein - Analyst

And I have a couple of other questions, which is -- for the subcutaneous, is all you need to do the is a BE study to get the subcutaneous formulation approved, once you get the IV approved?

Dr. Ken Verburg - Pfizer Inc. - Development Head for Pain Therapeutics

Oh, I wish. Let me tell you how that might go. So we've done a bridging study looking at IV and sub-cu administration in non-human primates, in cynomolgous monkeys. What we've found so far is really very encouraging, and that is that the administration of the drug does not cause any local irritation or particular pain.

And, actually, the bioequivalence is fairly similar to intravenous. So that's a good thing; that means that we are not going to be administering the compound at a lot higher dose or a lot more frequently than maybe what we do intravenous.

Now, that has to hold in the clinic. So that's question number one, is -- does that bioequivalency equilibrium hold when you extrapolate from non-human primates into humans. So you want to get that established first.

But then we are going to have to go a step further. We are actually going to have to take a study and evaluate the efficacy of subcutaneous administration in an osteoarthritis population. Now, we are not going to repeat the entire intravenous program in terms of efficacy and safety, but we are hoping and we'll need to do some bridging into the osteoarthritis population to make sure that we are drawing basically appropriate conclusions. So it will be a lot smaller, but it won't be (inaudible) larger than one study.

Tim Anderson - Sanford Bernstein - Analyst

Last question, which is -- the Phase III population for NGF -- is that in osteoarthritis? And it seems like that's the lead pain state you're studying. But it seems like the bar -- it seems like you could have a lower bar for approval if you studied something like cancer pain.

Dr. Ken Verburg - Pfizer Inc. - Development Head for Pain Therapeutics

Yes, I agree. I wish that -- so we -- just by way of background, we inherited this program from Rinat Neurosciences when we acquired Rinat a couple of years ago. It just so happens that Rinat had staked out a clinical development strategy that was based on moving in osteoarthritis quickly.

When we inherited the program, we realized that the compound had a lot more potential than just osteoarthritis, and basically so did Rinat. But with limited resources, they had to start somewhere. So we inherited a program where osteoarthritis [is] further ahead, and we've taken a look at that and decided that there's significant opportunity and the results are sufficiently robust that we are going to go forward and take that time advantage with OA and evaluate the other clinical conditions as they come in.

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

If we had started it from scratch, we might have chosen a little bit different pathway than what we have.

Jennifer Davis - Pfizer Inc. - IR

One on the webcast from Marko Kozul at Jefferies. Follow-up question on CP. Was the observed neutropenia transient and reversible? And, can you please remind us what the percentage of patients treated with the 5 milligrams dose experienced the side effect?

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

So it was both transient -- it's certainly reversible, and in most patients it was transient. And I think, actually, we have [stable] -- so he wanted to know for 5 milligrams?

Jennifer Davis - Pfizer Inc. - IR

Yes, 5 milligrams.

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

So the mean change in the absolute neutrophil count at 5 milligrams was -- between baseline and week 12 was minus 0.82 compared with placebo, which was minus 0.35. The only change that was actually statistically significantly different was 15 milligrams twice a day.

Jennifer Davis - Pfizer Inc. - IR

Thank you. Any more questions?

Seamus Fernandez - Leerink Swann - Analyst

Seamus Fernandez from Leerink Swann. Just a couple of questions on Tanezumab. The patients in the study were quite severe. Just wondering if there's any risk that you're masking a patient that might need to go to joint surgery, and if there would be any issues or outcomes that you would be monitoring to make sure that that's not a potential issue. And then, separately, the percentage of patients in the OA population that would be going to opioids in this patient population, is that something where you see that as an attractive opportunity where you could kind of short-circuit the use of opioids with this product?

Dr. Ken Verburg - Pfizer Inc. - Development Head for Pain Therapeutics

I'll start out and let Mark comment as well on the question of the opioids. So one of the clinical outcomes that you can study, if you were, say, concerned about total lack of sensitivity to pain and whether, because of that, the patient is able to run a marathon and thereby, you know, do more damage to their joint? There is a condition called Charcot's joint, where it's basically denervation, or nerve giant sort of damage. We'll be looking very carefully at that.

Based on what we see -- and also, by the way, we will be doing complete radiographic assessments in one of the trials. In fact, one of our larger trials will be taking baseline x-rays as well as -- and serial x-rays during the course of the study to basically get a radiographic handle on that. I think, in general, we'll have a real good feel for anything that's going on structurally related to the patient increased activity, and also monitoring what I'll call basically a neurogenic joint, where that joint gets completely

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

(inaudible - microphone inaccessible) nonsensitive. Again, based on the profile of the compound that we've seen preclinically, we think that it's probably pretty rare that we are going to see any effects like that over the long run.

I think the second aspect touches on some of the clinical development strategy. And yes, one strategy would be -- is to think very carefully about how Tanezumab fits within the current treatment paradigm for treating osteoarthritis. One place where patients have really no alternatives is when they exhaust their ability or the efficacy for nonsteroidal therapy, and they are basically left with some pretty poor treatment choices, one being intraarticular corticosteroids, the other one being opioid therapy or total joint.

So we see that as a place to begin to sort of look at the benefit-risk of Tanezumab in comparison to treatment alternatives. I think that's one place that we would think about very carefully, because one of the, shall we say, misconceptions, if you will, is that opioids are fairly useful in osteoarthritis patients. They are not any more efficacious than nonsteroidals. Many more patients can't tolerate them. And, in fact, if you go back and look at how many strong opioids are actually given to osteoarthritis patients, it's very, very small. And there's a good reason for that.

So I think there's some significant upside there. I happen to be one of the individuals, though, that think that there's utility, perhaps, of this drug in osteoarthritis patients across the moderate to severe spectrum. So it's really anybody's, say, other than can maintain their pain relief on acetaminophen, I think, it's in (inaudible) that population. I think Tanezumab may have a role. Mark?

Mark White - Pfizer Inc. - Senior Director & Group Leader for Pain Therapeutics Commercial Development

No; I think you covered it quite well. I think the misperception out there may be that a lot of people do use strong opioids for osteoarthritis and get relief, and it seems like that's not the case, as it might be in some other pain conditions. So there does seem to be this fairly severe, moderate to severe population that isn't receiving the relief they need. And a lot of them, or at least a fair number, go on to joint replacement. But even in the interim, as they are progressing to that place, Tanezumab could be a nice option for them if the profile continues to look the way it has so far.

Jennifer Davis - Pfizer Inc. - IR

We have one more question, I think, for Ethan from the webcast. For CP, which solid organs are priorities for you in transplant? Is renal being looked at, and do you have any time lines for data on those?

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

Renal is being looked at. It's in Phase II, at this point. And it's really too early in Phase II to comment when it will transition to Phase III. It's likely -- usually, in solid organ transplant, the thing that you look at after kidney is liver. But we have not started to do that yet.

Jennifer Davis - Pfizer Inc. - IR

I think we've got time for one more question here in the room.

Thomas Wei - Piper Jaffray - Analyst

Thomas Wei from Piper Jaffray. I just wanted to clarify a comment that was made about the neutropenia and the [NP] anemia, the more severe cases that we're seeing, grade 3/grade 4. It sounds like basically there was no intervention allowed of those

Oct. 28. 2008 / 9:00PM, PFE - Pfizer Analyst and Investor Meeting

patients. They were just monitored. And basically, should we think that they continue to be at their grade 3/grade 4 level for the rest of the study unless they happen to spontaneously resolve?

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

Obviously, the investigators -- there were, first of all, protocol mandated discontinuation rules for levels that got sufficiently severe. But those rules never had to actually be triggered. Then the other major recourse is for the investigator to discontinue the patient for an adverse event. So in other words, if the neutrophil count goes down -- and not for the protocol-mandated discontinuation level, but the investigator is uncomfortable, then they would discontinue the patient.

I do not recall us having any discontinuations for laboratory abnormality.

The other thing that the investigators do is they monitor the patients. They repeat abnormal values and then monitor them over time. Again, if a study subject were to continue to showed a decline, they would get discontinued from the study. If they stabilized or improved, remained at an acceptable level, then they would stay in the study. And I think, generally, that was the pattern so that patients would dip and then go back up. and obviously, in Phase III, we will acquire more experience with that. But, in other words, we have not had large number of patients have to discontinue for laboratory abnormalities.

Thomas Wei - Piper Jaffray - Analyst

And do use envision than those are the sorts of rules that you would employ in a Phase III trial?

Dr. Ethan Weiner - Pfizer Inc. - Development Head for Inflammation

Yes, the rules in Phase III will be similar. And then the next obvious question is, well, once it's out in market, what will the rules be? And that we really need to determine by looking at Phase III data in terms of whether any kind of monitoring is necessary at all, or not.

Jennifer Davis - Pfizer Inc. - IR

Okay. Thank you. I would like to thank you all for attending tonight, and we would invite you to join us just across the hall for a reception where the speakers will be available for additional Q&A. Thanks.

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