

FINAL TRANSCRIPT

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PFE - Pfizer Conference Call to Review Phase 2 data for CP-690,550 at EULAR

Event Date/Time: Jun. 12. 2009 / 5:00PM GMT



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PRESENTATION

Jennifer Davis - Pfizer - IR

Good afternoon. I'm Jennifer Davis from the Pfizer Investor Relations team and I'd like to welcome you to our conference call and webcast to review the data that has been presented here at the EULAR conference for our JAK-3 inhibitor, CP-690,550. With me today are Dr. Bernie Zeiher, Vice President in Inflammation Disease area development lead, Mark White, Senior Director Information in Immunology Disease commercial development lead along with several other members of the medical and commercial teams that support CP-690,550 development.

The format of our event would be a presentation about one hour in length in which Bernie will review key data presented during EULAR. This presentation we're reviewing is now available on Pfizer.com under the investor section of the website. After our prepared remarks, we'll open it up for Q&A regarding this compound and data both from the webcast and via the phone lines.



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Before we begin I'd like to remind you that we will make forward-looking statements during this presentation and our actual results may differ materially from those forward-looking statements. Please refer to our SEC filings for additional information. Those filings are available on Pfizer.com in the investor section of the website.

With that, I'll turn it over to Bernie Zeiher. Bernie?

Bernie Zeiher - Pfizer - VP, Inflammation Disease

Thanks, Jen. My name is Dr. Bernie Zeiher, and I'm the Inflammation Disease area lead here at Pfizer within the specialty business unit. The purpose of today's presentation is to give you an update on CP-690,550 our JAK inhibitor program. Before I do that I'd like to briefly review the compounds mechanism of action to provide an overview of the Phase II program. From there, I'll discuss new data that were or will be presented this week at EULAR focusing on results from three key studies, a 12 week interim analysis of a six week monotherapy study, a 12 week Japanese study on background methotrexate and an open label extension study. I'll then conclude with an overview of our ongoing Phase III program in RA.

Now let's look at this compound and its unique mechanism of action. PP690550 is a potentially exciting new development in the treatment of RA and could be the first new oral disease modifying andromatic drug or DMARD to be introduced to the market in more than 10 years. Based on the results we've seen to date from our Phase II program, we believe CP-690,550 may combine the traditional benefits of a small molecule therapeutic, in other words rapid absorption and elimination, keys and convenience of administration with certain advantage to the biologic, such as targeted modulation of the immune system. In addition to potentially being an exciting and important new development that could expand the treatment options available to patients with RA, we believe that CP-690,550 could have applicability in other inflammatory and autoimmune conditions.

PP690550 is an orally bioavailable highly selective inhibitor of the Janice kinases. In terms of its behavior as a drug candidate, it has very well behaved pharmacokinetics including dose proportional it and a relatively short half life of two to five hours. It is primarily excreted via urinary excretion with 30% of the drug being unchanged. Its metabolism is via 3A45 and it has limited metabolism via 2-C19. Preliminary efficacy has been demonstrated in rheumatoid arthritis, psoriasis and renal allograft transplantation.

The Janice kinases are a family of enzymes including JAK-1, 2, 3, and TIK-2. The figure on the left of this slide depicts a typical gamma chain cytokine receptor. Janice kinases are signaling molecules that are recruited to the cytoplasmic tail of gamma chain cytokine receptors as well as receptors nor a number of different cytokines. Janice kinases always signal in combination with themselves or other Janice kinases. For example, the gamma chain cytokine receptors always signal through JAK-1 and JAK-3 and thus inhibition of JAK-1 and/or JAK-3 will attenuate signaling of aisle two, four, seven, nine, 15 and 21. Other receptors such as the aisle six receptor signal via Janice kinase and they signal via JAK-1 and JAK-2.

At the cellular level we know that CP-690,550 inhibits JAK-3 and JAK-1 and is functionally selective over JAK 2. What this should mean is that CP-690,550 should attenuate signaling of the gamma chain cytokines as well as a number of other cytokines including interferon alpha, beta and gamma as well as the aisle six family of cytokines. This may account for its broad potential to treat a variety of autoimmune and immunologic disorders. The functional selectivity over JAK 2 at doses utilized in our clinical development program should translate to less inhibition of receptors exclusively using JAK 2 such as arithopoietan. Currently CP-690,550 is being evaluated in a number of inflammatory and immunologic diseases. As we'll discuss in a bit, it is in Phase 3-4 rheumatoid arthritis but we also have ongoing Phase II programs for psoriasis and renal transplantation and we have earlier stage programs in Crohn's disease, ulcerative colitis and dry eye using a topical formation. Across the entire development program, including both patients and healthy volunteers, more than 1800 individuals have been exposed to CP-690,550 in Phase I and Phase II studies. We also have substantial experience with longer term therapy particularly from patients in our renal transplant program some of whom have been treated with this candidate for more than three years.



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I'd now like to give you an overview of the RA program in particular the Phase II program. The Phase II program that Pfizer undertook in RA which to our knowledge is the largest program of its kind conducted in this setting began with an initial six week proof of concept study known as Study 1019. The encouraging efficacy and safety results we saw from this initial monotherapy study triggered the expansion of our program in RA and this has in turn led to encouraging data I'll be discussing today. This included Study 1025, a 24 week study on background methotrexate for which we've previously presented the 12 week interim results at the American College of Rheumatology meeting last year. Data from two additional studies are being presented this week at EULAR. Today, we presented a 12 week interim analysis of Study 1035 which is the six-month Phase 2b monotherapy study in RA patients who have not previously responded to DMARD, and tomorrow we'll be presenting Study 1039, a 12 week Phase 2b Japanese study in patients on background methotrexate.

All patients who were enrolled in studies 1019, 1025 and 1035 as well as our future Phase III studies will have the option of enrolling in Study 1024, our long term open label study. Interim results from Study 1024 were presented earlier today at the conference. Throughout this program, we've explored a broad dose range including doses from 1 to 30 milligrams dosed twice per day and our Phase II RA program has enrolled more than a thousand patients. In our long term extension studies we have more than 100 RA patients who have been treated with CP-690,550 for more than a year. This extensive patient experience has positioned us well to move forward into Phase III.

This slide provides a visual representation of how CP-690,550 program has developed over the years and the timing of some of our presentations. The original study, Study 1019 was completed and presented at the ACR meeting in 2006. As I mentioned, Study 1019 subsequently led to study 1025 a background methotrexate study. The 12 week interim analysis of this study was presented at the 2008 meeting of the American College of Rheumatology. We've now also completed the study 1035 and 1039, results of which will be presented here at EULAR and we'll be providing the update at the open label extension study 1024.

You'll note we have presented the 12 week primary end point for both Study 1025 and 1035. We plan to submit the final study results going out to the full 24 week at ACR in 2009. I would now like to review some of the data that has been presented at EULAR. First I'll discuss the 12 week interim analysis of our six-month monotherapy study which is Study 1035. This will be followed by Study 1039, the 12 week Japanese methotrexate study and then I'll finish with an update of Study 1024, our open label extension study.

Before reviewing these results I'd like to review the ACR response criteria, ACR20/50 and 70 for anyone who may be less familiar with these endpoints. The ACR 20, 50 and 70 response criteria are commonly used in rheumatoid arthritis studies and are accepted by regulatory authorities for demonstration of efficacy in this disease. ACR response is a composite end point in which patients are classified as either responders or non-responders using certain criteria. First to be a 20, 50 or 70 responder patients have to improve by 20, 50 or 70% on their tender joint and swollen joint counts. In addition, they have to respond on at least three of five other variables by 20, 50 or 70%. These five variables include the patient global assessment, physician global assessment, patient pain visual analog score, the health assessment questionnaire disability index, and either the C-reactive protein or erythrocyte sedimentation rate.

Now let me move on to Study 1035. Study 1035 is six-month monotherapy study in patients who inadequately responded prior to DMARD or disease modifying therapies for rheumatoid arthritis. These patients again were not on background methotrexate. In other words it was a monotherapy study. Patients were randomized to one of seven treatment groups: Placebo, one, three, five, 10, or 15 milligrams BID of CP-690,550 or adalimumab 40 milligrams subcutaneously every other week. Importantly, adalimumab was not included in this study as a direct comparator, it was simply an active control in this study.

Patients had to have active rheumatoid arthritis in order to enter the study including more than six swollen and tender joints and either a CRP of greater than seven or an elevated erythrocyte sedimentation rate. Patients were then treated for 24 weeks in the primary efficacy end point was the ACR 20 responder rate at week 12. After 12 weeks if patients failed to demonstrate a 20% response, and were in the groups of placebo, 1, or 3 milligrams BID, they were automatically transitioned to 5 milligrams BID because we know from previous work that 5 milligrams BID was effective in rheumatoid arthritis and neither the patients, investigators nor Pfizer were unblinded to this transition.



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Regardless of their response, patients on adilumumab at the end of the 12 week period were automatically switched to 5 milligrams BID for the remainder of the study. What we've presented today at EULAR is the 12 week data which will be summarized and presented. This study has been completed and as mentioned earlier we plan to submit the full six-month data at a future scientific meeting.

This slide depicts the disposition of patients through week 12 in Study 1035. The discontinuation rate for patients on placebo, adilumumab and 1 milligram BID was somewhat higher than what was observed in the three, five, 10 and 15-milligram treatment groups. Overall relatively few patients discontinued due to adverse events that were thought to be potentially related to study drug.

This figure depicts the ACR 20, 50 and 70% response rates at week 12 using the non-responder. Given this more stringent analysis, patients who do not complete 12 weeks of treatment are considered non-responders regardless of what sort of response they may have achieved prior to dropping out. The non-responder imputation is the most accepted approach by the FDA. A statistically significant difference relative to placebo was demonstrated in the primary end point of ACR 20 for that three, five, 10 and 15-milligram BID dose groups. The placebo ACR20 response was 24% while it was 72% of the highest two doses of CP-690,550. Similarly, if you look at the ACR50 response rates, again, the three, five, 10 and 15 milligrams were statistically significantly better than the placebo group. The placebo ACR50 response rate was 10% while it was 46 to 51% at the two highest doses. When looking at the highest response criteria, the ACR70 response, the 5, 10 and 15-milligram dose groups were superior to placebo. The placebo ACR70 response was 4% while it was 25% at the two highest doses. Somewhat surprisingly, adilumumab was not statistically significant as compared to placebo. The reason for this lack of significance is unknown; however it is important to note several things.

First, this is a monotherapy study. Adilumumab like many biologics tends to perform better on background methotrexate as opposed to when it's dosed as a monotherapy. Second the dose utilized was 40 milligrams every other week. This is the label dose but the adilumumab label does indicate that a higher dose of 40 milligrams every week maybe made when dosed as monotherapy. Finally, the patient population in this study tended to some somewhat lower disease activity as compared to previously studied monotherapy patients with adilumumab.

This slide depicts the time course of ACR20 response. As you can see the response of CP-690,550 occurs relatively quickly. At the earlier time point assessed which was at two weeks we saw a statistically significant separation from placebo for the three, five, 10, and 15-milligram dose groups. The health assessment questionnaire, or HAQ is a commonly used patient reported outcome measure which is also recognized by regulatory authorities as a measure of physical function in RA. Lower measures on the HAQ disability index reflect improved function with a change of 0.22 representing a clinically meaningful benefit. At end point statistically significant and clinically meaningful reductions in the HAQ disability index were noted with the 5, 10, and 15-milligram BID dose groups as compared to placebo.

This slide depicts the most common treatment in adverse events from baseline to week 12 in Study 1035. The most common adverse events were urinary tract infections, headaches, bronchitis, diarrhea, chest pain, and it should be of note that this was chest discomfort and not cardiac chest pain, dizziness and rash. There were no major differences across treatment groups relative to our placebo or active control at adilumumab. All in all, these results were fairly typical for this patient population.

With respect to serious adverse events, several other serious adverse events did occur during the 12 week period, in particular one serious infectious complication occurred. One patient developed pneumococcal pneumonia in the highest 15-milligram BID dose group. This patient also had sepsis and meningitis but was successfully treated.

Based on the mechanism of action several laboratory values have been closely monitored throughout the program including neutrophil counts, hemoglobin, cholesterol and transaminases. No increase in the proportion of patients having transaminase elevations was noted as compared to control groups. With respect to neutrophil counts as depicted on this slide, dose dependent reductions in mean absolute neutrophil count were noted at week 12; however there were no cases of severe neutropenia and here were no discontinuations due to neutropenia.



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Hemoglobin is of interest as reduction of hemoglobin would suggest inhibition of (inaudible) signaling which exclusively signals via JAK-2. Change in hemoglobin levels from baseline to week 12 is depicted on this slide. As you can see there was a small decrease in hemoglobin for the placebo arm which is likely attributable to the phlebotomy associated with this clinical study. No dose was associated with significant reduction in hemoglobin and in fact the 5-milligram BID dose group had a significant difference which was an increase in hemoglobin relative to placebo. No patients discontinued drug due to anemia.

Consistent with previous results we have shown in studies 1019 and 1025, we saw dose dependent increase in both HDL and LDL cholesterol in patients treated with CP-690,550. As you can see there were mean increases of HDL on the order of 10 to 12-milligram per deciliter and increases of LDL up to 25 to 29 milligrams per deciliter. We have ongoing clinical and pre-clinical investigations designed to further understand the mechanism of this elevation and its potential clinical significance.

So in summary the time of this 12 week interim analysis, Study 1035 has demonstrated that CP-690,550 dosed at 3 to 15 milligrams BID was superior to placebo on ACR20 response rate which was the primary end point. In addition these doses demonstrate superiority on ACR50 response rates at week 12. These responses were seen rapidly with separation from placebo in ACR20 response at the earliest time point of two weeks. At doses of 5 to 15 milligrams there was superiority on ACR70 response rates as well as on the HAQ disability index. From a safety perspective there were no significant decreases in hemoglobin, there was however significant dose dependent decreases in mean neutrophil count but no severe neutropenia or discontinuation due to neutropenia. Consistent with prior studies we saw increases in mean LDL and HDL. No dose response was seen for infections and no opportunistic infections were seen.

Now I'd like to discuss the results of Study 1039. Study 1039 was a Phase II, 12 week Japanese study on background methotrexate with a primary end point of ACR20 response rate. This study was designed and conducted so that we could collect safety and efficacy data from Japanese and RA patients so as to enable us to include Japan in our Phase III program. In this study patients were randomized to one of five treatment groups. One, three, five, or 10 milligrams of CP-690,550 dosed BID or placebo. Patients had to be on background methotrexate at a dose of at least six milligrams per week for at least six weeks prior to baseline. Again as in our other studies patients had to have six tender and six swollen joints plus either a CRP of greater than or equal to 7 milligrams per liter or an abnormal erythrocyte sedimentation rate.

This slide depicts patient disposition for Study 1039 after 12 weeks of treatment. The discontinuation rate was lowest in the 1 milligram BID dose group and was similar across the placebo three, five, and 10-milligram BID groups. Relatively few patients discontinued due to adverse events. The discontinuations due to adverse events were somewhat higher in the five and 10-milligram BID groups although the sample sizes in this study were relatively small. Efficacy results from this study demonstrated a statistically significant improvement in the primary end point of ACR20 response at the one, three, five, and 10-milligram doses. Although this study is fairly small, the ACR20 responses were quite impressive with ACR20 responses above 80% at the highest doses while the placebo response rate was 14%. We also saw statistically significant differences versus placebo for the 3, 5 and 10-milligram dose groups on ACR50 and for the 5 and 10-milligram dose groups on ACR70 response.

The time course of ACR20 response is depicted on this slide. As you can see the onset of action or separation from placebo occurs quite early. In this study we capture data as early as one week following the first dose of study drug, while the three milligram dose group lagged a bit, the one, five and 10-milligram doses separated from placebo as early as one week. By two weeks all dose groups separated from placebo and then continued to show statistically significant differences from placebo throughout the treatment period.

Like Study 1035, significant improvements in the HAQ disability index were noted with CP-690,550. As compared to placebo, all dose groups demonstrated clinically and statistically significant reductions in the HAQ disability index at week 12. The most common adverse events occurring in at least three patients across all treatment groups were classified as infections or infestations using a measure of codeine. Nasal pharyngitis was the most common adverse event although the actual incidents of nasal pharyngitis was not different between the placebo and CP-690,550 treatment groups. ALT and AST outpatients were recorded as adverse events, more commonly in the CP-690,550 5-milligram BID treatment group.



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This table depicts the proportion of people exceeding various thresholds for elevation of AFT and ALT. In the five milligram dose group, two out of 27 patients or 8% had elevations above three times the upper limit of normal and one patient in a 10-milligram BID dose group had a similar elevation. No patients in the placebo group exceeded this threshold. Importantly, no patients with transaminase elevations, (inaudible) and bilirubin or other signs of liver injury.

This slide depicts adverse events that lead to discontinuation from treatment. We saw a case of osteoarthritis, femur fracture, cardiac failure and dyspnea. Of note the patient with cardiac failure had a previous history of cardiac disease and the case of dyspnea was never explained. As noted previously we had some discontinuations due to AFT and ALT decreases in the 3, 5 and 10-milligram dose groups. No serious infections or opportunistic infections occurred.

Mean absolute neutrophil counts were lower in the CP-690,550 treatment groups at week 12 but this only achieved statistical significance in the 10-milligram BID dose group. There were no cases of severe neutropenia on CP-690,550. You may have noted that on the previous slide one patient discontinued due to a low white blood cell count; however this patient's neutrophil count was above 1.5.

Relative to placebo, no significant difference in hemoglobin changed from baseline was noted with CP-690,550 treatment groups. Consistent with what we have seen in prior studies there were dose dependent increases in HDL and LDL. As mentioned previously we have ongoing investigations to understand the mechanism of this elevation. So in summary study 1039 demonstrated that all CP-690,550 doses were efficacious versus placebo in the primary end point of ACR20 response. For ACR50 response, all doses except the 1 milligram BID dose achieved statistical significance at week 12 and for ACR70 response, the 5 and 10-milligram BID doses achieved statistical significance at week 12. Importantly, these differences were noted early with significant differences relative to placebo on ACR20 responses as early as one week after the start of therapy. All CP-690,550 dose groups demonstrated significant improvement relative to placebo on the HAQ disability index.

From a safety perspective, CP-690,550 was well tolerated. There were no serious infections or opportunistic infections, there were no significant decreases in hemoglobin. As we've noted previously there were dose related decreases in mean absolute neutrophil counts and dose dependent increases in LDL and HDL cholesterol. Similar to what we had seen in the study 1025 which was our background methotrexate Phase 2b study there were ALT and AST increases of greater than 3 times the upper limit of normal in the 5 and 10-milligram BID dose groups.

Tomorrow morning during this conference, we'll be presenting the update on our open label extension study which is Study 1024 and I'd like to review those results with you now. Study 1024 is designed as an open label long term multi-center safety study for patients who are enrolled in our Phase II and III efficacy studies. To date this has included patients enrolled in our monotherapy POC study 1019, the Phase 2b methotrexate background study, study 1025 and the Phase 2b monotherapy study, study 1035. Study 1019 ended in 2006 and thus patients were required to be rescreened prior to enrollment in Study 1024, whereas patients enrolled in Studies 1025 and 1035 were able to directly roll over into Study 1024. Patients in this study were allowed to have their background RA therapy modified to include traditional DMARDs, but they were not allowed to initiate biologic disease modifying therapies. Dose reductions and modifications were also allowed for adverse events in rescue medications where permitted.

At the time of this interim analysis which was as of December 1, 2008, 655 patients had been enrolled in Study 1024. As of the date of cutoff, 446 patients had completed six months and 125 patients had completed 12 months of treatment with a median duration of treatment of 161 days. Because patients from Study 1019 didn't immediately rollover into this study, their new baseline was defined as their disease activity and laboratory results measured within the 28 days prior to enrollment. For those patients who directly rolled over from Study 1025 and 1035, their baseline was considered the baseline prior to entering their index study.

The primary purpose of Study 1024 is to evaluate safety and as of the time of the interim data cutoff, most of the adverse events we have seen were mild or moderate in severity. There have been 10 severe adverse events, some of which qualify severe infections and these include a case of disseminated tuberculosis, diverticulitis, herpes zoster, pneumonia, staphylococcal infection



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and urinary tract infection. In terms of the patient with disseminated tuberculosis, this case was a patient who had unexplained fever, possible exposure to TB, and a gastric aspirate that had acid fat bacilli on smear. No micro bacteria were ever cultured, however. The patient was treated presumptively for disseminated tuberculosis and symptoms resolved. There have been 42 serious adverse events reported including nine serious infections in nine patients. For reference as of the time of the interim cutoff, we had accumulated approximately 255 patient years of exposure to CP-690,550. Given the nine serious infections we had reported to that point this translates to approximately 3.55 serious infections per 100 patient years of exposure. This is within the range of what has been previously reported with other DMARDs in the anti-TNF biologic therapies. There have been two deaths, both of which were sudden. One was a 57 year old man with a history rough multiple cardiovascular co-morbidity who died suddenly and one was a 70 year old woman with hypertension and epilepsy.

This slide depicts mean values at baseline month six and month 12 for patients who have been enrolled in study 1024. Similar to what we've reported in our control study, laboratory data at the time of the interim analysis reveal small reductions from baseline in hemoglobin and neutrophil count and increases in HDL and LDL. Although we only have data on less than 60 patients at one year, there does not appear to be a significant worsening in these laboratory values with longer term treatment. So in conclusion at the time of this interim analysis of Study 1024 we've found that CP-690,550 was generally well tolerated and had a similar safety profile compared to what has been observed in the randomized index studies.

Now I'd like to provide you with a summary and brief overview of the Phase III program. We're very pleased that all four of our Phase II RA studies have achieved statistical significance on the primary end point of ACR20 response. Our program has been extensive and has enrolled more than a thousand patients and evaluated CP-690,550 at doses ranging from 1 to 30 milligrams dosed BID. We've consistently seen that CP-690,550 dosed at three milligrams BID or greater was efficacious compared to placebo as measured by ACRs response rates, changes from baseline and DAF and DAF remission rates and improvement inpatient reported health outcome measures. Efficacy has been seen early within two weeks when CP-690,550 was dosed either as a monotherapy or on a background methotrexate.

In terms of the safety profile, the profile of CP-690,550 will continue to be monitored closely and further characterized throughout Phase III. In particular we'll be keeping a close eye on decreases in absolute neutrophil count and hemoglobin, increases in total LDL and HDL cholesterol and exploring the magnitude and mechanism of these changes. As with the 1039 study, we have seen increases in transaminasis particularly when CP-690,550 has been dosed on background methotrexate, thus transaminasis will also be monitored closely. With the potent immuno suppressive drug, an obvious concern is infections. When we look at our Phase II database in total, the current rate of serious infections is approximately 4.5 per 100 patient years exposure which is in line with rates reported with anti-TNF therapies in literature. We've not seen any opportunistic infections in our Phase II randomized study although one possible case of disseminated tuberculosis in study 1024. Based on the thorough dose ranging work conducted in Phase II, doses of 5 and 10 milligrams BID have been advanced into Phase III.

Earlier this year, we initiated Phase III and we now have four Phase III studies currently enrolling patients. These studies include treatment periods of six to 24 months in duration and patients enrolled in these studies will be eligible for long term open label extension. We expect to enroll more than 3000 patients in total across this pivotal program which provide a good foundation of data from which to provide a clinical profile. This comprehensive clinical program will include a valuation of CP-690,550 as a monotherapy in Study 1045 as well as on background methotrexate in studies 1044 and 1064, or in combination with conventional DMARD in study 1046. The studies are designed to evaluate the efficacy of CP-690,550 on signs and symptoms of RA as well as on the prevention of structural progression of disease.

So in conclusion we believe CP-690,550 is a potentially exciting new compound in the treatment of RA and could be the first new oral DMARD to reach patients in more than a decade. Based on the results we've seen to date from our Phase II program, we believe CP-690,550 may combine the ease and convenience of a small molecule therapeutic with rapid and clinically significant treatment effects seen with biologic therapies. Thank you for your interest and I'll turn the call back over to Jen for questions and answers. Jen?



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Jennifer Davis - Pfizer - IR

Thanks, Bernie. We'll now open it up for Q&A and we'll take questions from the phone lines and via the webcast. (Operator Instructions) We ask that you limit your questions to the CP-690,550 RA program to best take advantage of the teams expertise. Please start off by giving us your name and your firm. Operator? we'll take the first question, please?

QUESTIONS AND ANSWERS

Operator

Thank you. Our first question comes from David Risinger from Morgan Stanley.

David Risinger - Morgan Stanley - Analyst

Yes. Thanks very much and thank you for the presentation today. I have two questions that are pretty basic. First, could you please discuss your comfort with the impact of CP-690,550 on the liver? And second given the significant impact on cholesterol can you discuss the cardiovascular assessment you're planning for the NDA package and whether you've had specific discussions with the FDA about what the FDA will want to see from a cardiovascular assessment standpoint? Thank you.

Jennifer Davis - Pfizer - IR

Bernie, do you want to take that?

Bernie Zeiher - Pfizer - VP, Inflammation Disease

Yes. So first with regard to the liver. We have completed a extensive preclinical toxicology program and did not target the liver as a target organ for any toxicity. As you probably are aware, elevations are not uncommon in patients treated for RA and in fact methotrexate is known to increase transaminasis in some individuals and furthermore guidelines indicate that transaminasis should be monitored all patients while on methotrexate.

To date we have seen small numbers of elevations in ALT and AST, but the proportion of patients having elevation in monotherapy studies was no greater than background but as you seen in our methotrexate background study there was an increase in the proportion of patients having mild elevation in (inaudible). Importantly, we have not seen evidence of any other liver toxicity such as bilirubin elevation and we believe that going into Phase III, that there will be an appropriate risk benefit in patients treated with methotrexate or as a monotherapy so we are confident in that.

So now with respect to the lipids, the question was around what sort of cardiovascular assessment we'll be performing so throughout the Phase III programs, we will be adjudicating all cardiovascular events and we do anticipate having on the order of 3000 patient years of exposure at each dose and at least at that level of exposure. We have had discussions with the FDA as well as EMEA around our Phase III program in general, and there was no indication that we needed to perform any cardiovascular outcome studies at this time, so -- but we do believe it's going to be important to understand the mechanism of the lipid elevation, and we have been conducting and do plan additional studies both preclinically and clinically to understand the mechanism for this elevation.

Jennifer Davis - Pfizer - IR

Great. Thanks, Bernie. We'll take another question from the phone lines please, Operator?



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Operator

Thank you, our next question comes from Tim Anderson with Sanford Bernstein. Please proceed with your question.

Tim Anderson - *Sanford Bernstein - Analyst*

Thanks. I have a few questions. The first is, when I look at your registration studies almost all of those are placebo controlled. You have only in one trial which looks like maybe European registration trial, one arm of that that has an active comparator, and just given how crowded this field is and the fact this has been highlighted as one of those areas where there's going to be some comparative effectiveness research done and I'm wondering commercially how you feel about not doing anything but really placebo controlled trials. The second question is what is the dose limiting toxicity with this drug and the third question is when might we expect to see radiographic data? And do you have any evidence at this point that suggests you'll actually show favorable radiographic findings?

Bernie Zeiher - *Pfizer - VP, Inflammation Disease*

First, with regard to active comparator data, as you noted we are including adilumumab in the 1064 study which is a background methotrexate study that will include active doses to CP-690,550 as well as placebo. As you noted, it will be powered versus placebo but we will within the same study have an adilumumab treatment arm.

Whether there is addition all need for exit comparator data, we actually don't think we need that at this point from a regulatory standpoint and we also believe from the data that we are able to generate with the Phase III program we will provide a compelling story in terms of, first, we believe it's the oral mechanism of action and also with rapid onset of action as we've shown and importantly, it also will have a, because of its half life will have a more rapid wash out and we believe that there will be sufficient data to support its use as either monotherapy, background, or in a background methotrexate.

Whether there will be a need for additional studies after registration, I think that we have to further support the commercialization of the study is that the compound, we haven't determined that as of yet, but that may well be part of the program in the future.

So with regard to dose limiting toxicity as we've discussed this compound does inhibit JAK-3 which at high exposures can be immuno suppressive and we know from our transplant studies and again this is on background with MMF or (inaudible) as well as steroids and aisle 2 receptor induction therapy that there can be over immuno suppression and in fact have doses of 30 milligrams BID in an earlier study in transplantation we saw in excess of BK virus necropathy, so that dose was not advanced into -- transplant is not a dose that we have not been exploring in RA. Finally, you asked about radiographic data and when we would have that. Study 1044 is a study that will provide structure data. To date we do not have structure data in any of our Phase II studies, but we have confidence given the consistency of benefits, effects on CRP and just the magnitude of the fact that we will see structural benefits, but we do not have that data as of this time.

Jennifer Davis - *Pfizer - IR*

Thanks, Bernie, we'll now take a couple of questions from the web. I've got two from Chris Richard. We'll start with the first one. First, how are patients who dropped out of the study counted in the final analysis of the study? Was it LOCF or as treatment failures?

Bernie Zeiher - *Pfizer - VP, Inflammation Disease*

It's in the presentation. The primary analysis for Study 1035 was non-responder amputation. This is the FDA mandated approach to the sense that all patients who failed to complete the 12 week treatment period are considered non-responders regardless



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of whether they responded well prior to the 12 week treatment, completion of the 12 week treatment period so that was the primary analysis and the data that was reviewed today.

Jennifer Davis - Pfizer - IR

Okay, great and then the second question from Chris. Study 1035, were there any (inaudible) 28 scores of less than 2.6 and what were the (inaudible) scores on entry?

Bernie Zeiher - Pfizer - VP, Inflammation Disease

So again from Study 1035, baseline DAS scores using the DAS28.4 SED rate to ESR range from 6.3 to 6.6 at baseline. 9 to 17% of patients on CP-690,550 have achieved affirmation criteria, in other words having the DAS score of less than 2.6 versus 4% for adilimumab and placebo.

Jennifer Davis - Pfizer - IR

Okay, thank you. Operator? We'll take another question from the phone lines please?

Operator

Thank you. Our next question comes from (inaudible) with UBS. Please proceed with your question.

Unidentified Participant - Analyst

Hi, good evening. I actually had a few questions. First was can you give us some detail on what the (inaudible) used was in the Phase II studies? Specifically as it relates to 1035? Secondly, what do you think, what length of data will the FDA be looking for before they review this process? Will they need 24 month data or do you think the FDA will be happy to look at six or 12 month data? And thirdly, the Japanese study, there seemed to be a big difference across the different doses on hemoglobin. Can you explain what the mechanistic reactions for that might be?

Bernie Zeiher - Pfizer - VP, Inflammation Disease

Okay. So first with respect to the use of statins in Study 1035, let me first comment that statin use was not, the use of it was less of the investigator, to the extent that it was prescribed either it could be used at baseline or post baseline depending on the cardiovascular risk factors and other considerations at the individual patient level. The actual numbers of patients who were receiving statins though I wonder if I could ask Stan -- well, to answer.

Unidentified Corporate Representative - Pfizer

I don't have the numbers memorized but they were relatively small. They were statins were used in the course of the study but numbers were small.

Bernie Zeiher - Pfizer - VP, Inflammation Disease

Okay. Now with regard to the length of data needed for the FDA. So at this point we believe that 12 month data will be sufficient for regulatory review in terms of our data package, although we will be continuing studies such as 1044, the structure study



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out for two years. And finally, mechanism for the hemoglobin changes, so the hemoglobin changes are -- is a complex effect in the sense that there are a number of competing factors going on.

First is all of our patients enrolled in the studies do have regular phlebotomy as part of the actual study in terms of just collecting blood for safety analysis as well as for pharmacokinetics and pharmacodynamic measures so there is an effect of the phlebotomy. In addition, the disease itself, rheumatoid arthritis may as it does often cause a mild chronic anemia related to the inflammation that's present. Improvement in that inflammation often results in some improvement in the actual anemia. Why we have particular interest and have been watching hemoglobin closely is that erythropoietin signals via JAK-2 and actually, exclusively via JAK-2 and so inhibition of JAK-2 could result in anemia and as I mentioned earlier, we believe based on cell base data that our compound is functionally selected for JAK-2 and thus at the exposures we have been using, we have not seen significant anemia that requires intervention. We haven't had patients needing to discontinue for anemia or other interventions required for anemia.

Jennifer Davis - Pfizer - IR

Thanks, Bernie. We'll take our next question from the phone lines, please?

Operator

Thank you. Our next question comes from Seamus Fernandez with Leerink Swann.

Seamus Fernandez - Leerink Swann - Analyst

Thanks very much. Just to follow-up on some of (inaudible) questions there. The statin use, you said it's at low levels but I guess the question is my question would really be more rate of change, so was there an increase in statin use from baseline during the study? And then separately, do you have evidence that statins are actually impacting the LDL? I guess my only concern would be if you are altering the ability of the LDL receptor to operate properly over time, so it would be helpful to know if statins are impacting those LDL elevations when they are actually used? Thanks.

Bernie Zeiher - Pfizer - VP, Inflammation Disease

Well I'll take the second within first which is regarding whether statins do in fact still work on background or when CP-690,550. We have not studied this systematically at this time; however based on some of the transplant data that we have with patients on transplant, it's receiving 690550 for transplantation, we have noted that statins did appear to be effective at lowering lipid levels. We do plan additional studies to further understand the effect of statins in the rheumatoid arthritis population. Now, with regard to your second question about whether there was an increase in statin use in the context of our trial, again, ask Sam, the clinical lead if we have any information on that.

Unidentified Corporate Representative - Pfizer

Fundamentally we're still doing the analysis of what were the drivers of changes of lipids observed in the study including what and when the statins were intervened so can't really give you just a definitive answer, this percentage of this dose has statins and this would be a work in progress.



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Jennifer Davis - Pfizer - IR

Okay. Thank you. I will now take one of the questions from the webcast. Chris Schott at JPMorgan is asking can you remind us of the relative CP690 to JAK-1 versus JAK-2 and JAK-3 and also he knows it's early but can you update us on where enrollment stands at this point?

Bernie Zeiher - Pfizer - VP, Inflammation Disease

So in terms of the affinity, these are selected and potent for the JAK inhibitors in terms of its JAK-3 potency it was Monday manamollar, based on data predominantly in cell based assays, we saw it was equally potent versus JAK-1 and JAK-3 but functionally selective over JAK-2. So and then in terms of the question about enrollment, it is early. We do have four studies that we kicked off and are currently enrolling in Phase III. Currently enrollment is on target although it's too early to say much more about it because it is still very early. Operator we'll take another question from the phone lines, please?

Operator

Thank you. Our next question comes from [Edward Dulock] with Barclays Capital. Please proceed with your question.

Edward Dulock - Barclays Capital - Analyst

Yes, hi, good afternoon. I'm filling in for Tony Butler. Two questions please. First one with respect to the cholesterol effects of CP-690,550. If you could characterize the time course of that data as I seem to recall from last years ACR was Study 1025, we saw rather rapid increase in the cholesterol levels in general, kind of through the first couple weeks versus the first few weeks with a plateau around week 6 and it wasn't clear what the characterization of that effect was going beyond that week 6 period so if you can provide any commentary that would be helpful? And then secondly the risk of beating a dead horse here with respect to statin usage is there any data you have or just general commentary around those folks that were on statins at the initiation of JAK-3 therapy, the impact on their cholesterol levels and the need therefore to increase statin levels and perhaps some toxicity related to liver toxicity with increased statin usage as well as just with CP-690,550? Thank you.

Bernie Zeiher - Pfizer - VP, Inflammation Disease

Okay, so with regard to the first question and the time course of cholesterol elevation, the time course that we seen is consistent with what you described and what was reported previously was that on the order of between 6 to 12 weeks there's a plateau of the lipid effect and in fact what I reviewed today with you which is the study 1024, we had six-month data and 12 month data that didn't suggest there was any worsening out to 12 months when you look at it, again beyond six months, so we don't believe that there will be progressive worsening or progressive increases in lipids beyond what we've seen to date. With regard to your second question around statin use, we again have limited data on this and again as Sam mentioned, we are conducting additional analysis and we do plan additional clinical work to further understand it but we have not seen any evidence that there's any liver toxicity when you have combinations of statins plus methotrexate in our studies to date.

Jennifer Davis - Pfizer - IR

Thanks, Operator, we'll take our next question, please?

Operator

Thank you. Our next question comes from Jamie Rubin with Goldman Sachs. Please proceed with your question.



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Jamie Rubin - Goldman Sachs - Analyst

Thank you. My questions have been answered. Thanks.

Jennifer Davis - Pfizer - IR

Okay. Thank you. I'll take a couple from the webcast. first, Margo Eprick of Cardio Partners is asking. Why was the 3-milligram dose not considered for Phase III given potentially better safety profile and strong efficacy?

Bernie Zeiher - Pfizer - VP, Inflammation Disease

So we've mentioned the 3-milligram dose did appear to be effective in, particular on ACR20 but when you look at the higher levels of disease activity which are often sought by clinicians nowadays when you start looking at ACR50 and ACR70, 3-milligram did not show as consistent of efficacy in terms of its differentiation from placebo and overall the 5 and 10-milligrams appear to be the most appropriate doses to advance into Phase III based on the overall benefit and risk.

Jennifer Davis - Pfizer - IR

Thank you. Operator? Are there any other questions on the phone line, please?

Bert Hazlett - BMO Capital Markets - Analyst

Yes. our next question comes from Bert Hazlett with Oscar (sic) Capital Markets. please proceed with your question. Apparently we've renamed the firm. It's BMO Capital Markets today.

Jennifer Davis - Pfizer - IR

Sorry, Bert.

Bert Hazlett - BMO Capital Markets - Analyst

That's okay. Thank you for taking the question and thank you for holding the call as well. Maybe you mentioned this but could you give us a sense of the baseline levels of LDL of these patients and my apologies if you mentioned that before and I have a couple of other ones. Just in general terms.

Bernie Zeiher - Pfizer - VP, Inflammation Disease

How about I'll try to see if I can pull that up if you could provide me -- maybe go with your second question.

Bert Hazlett - BMO Capital Markets - Analyst

Okay, and then in Study 1024 the safety study, can you characterize any discontinuations due to adverse events in that study just in general terms what the magnitude, what types of AEs that might have been the source of discontinuation?



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Bernie Zeiher - Pfizer - VP, Inflammation Disease

So there were 23 patients who discontinued in Study 1024. 12 of those were serious adverse events. I don't have the exact break down of which of those might have been infection. We have obviously given--?

Unidentified Corporate Representative - Pfizer

Approximately nine of those were serious infection.

Bernie Zeiher - Pfizer - VP, Inflammation Disease

Okay, so as Sam had mentioned approximately nine of those were due to serious infection. We obviously had mentioned two deaths and both of those studies. There were, so those, that's the extent at least I have at my fingertips in terms of what the reasons for discontinuation. In terms of -- I was able to pull back up the baseline lipid levels and at baseline and again this now comes from Study 1024 which has a mixture of patients from 1019, 1035, and 1025 and so some of the lipid levels particularly those from, well the ones from Study 1025 and 1035 were their baseline before they even entered the double-blind studies. the baseline levels were LDLs of 112 and HDL of 56.

Jennifer Davis - Pfizer - IR

Thank you. Operator, we'll take our next question, please?

Operator

Thank you. Our next question comes from Thomas Wei with Piper Jaffray. Please proceed with your question.

Thomas Wei - Piper Jaffray - Analyst

Thanks for taking my question. Just a few here on the safety side. On both neutropenia and liver enzyme elevation, did any of those require dose interruptions or reductions and can you tell us were those patients who did have that or discontinued how quickly they resolved either the liver enzyme elevations or neutropenia and then is statin use mandated in the Phase III trials?

Bernie Zeiher - Pfizer - VP, Inflammation Disease

Okay so I'll take the second question first which is around the use of statins in Phase III, so in Phase III, we are not mandating the use of statins. it would be difficult to do because the use of statins should be based on a number of factors, not just the lipid levels, it should be based on individual cardiovascular risk factors, multiple treatment guidelines and so forth and so we are not mandating the use of statins; however, investigators will be provided with the lipid data as it's available. It's not blinded so they will have those results and they will be advised to follow guidelines around the treatment of these -- taking into account patients with individual risk factors. So that covers at least the lipid question.

Now with regards to neutropenia, we've had no patients discontinued for neutropenia and again when we're talking about we've had no patients who have gone to less than 500 which would be a marker of severe neutropenia and no patients who discontinued for that reason and I did mention we had one patient in the Japanese study who had discontinued for a low white blood cell count; however the total -- or the absolute neutrophil count in that patient was above 1500. It was at the investigators discretion to continue study of the drug. And with regard discontinues due to tansamynasis or holding a therapy I'm going to actually turn that over to [Stan Gwylick] again to see if he can answer that question.



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Unidentified Corporate Representative - Pfizer

It's a complex question that depends on the Study 1025, one dose reduction up to 5 milligrams in methotrexate was allowed and then persistent elevations above the upper limits required discontinuation. There were a handful who did think some of those patients had either potentially contributing drugs (inaudible) being one and in the 1035 Study there were a couple of patients who admitted to ethanol bridging before having their phlebotomy and in the 1024 dose reductions were allowed. That was the only study where dose reductions were allowed and there were only a handful of patients who did dose reduction and I don't believe any were for transaminasis, I don't recall exactly but the dose reductions were with 24 although allowed were extremely few. We're talking a dozen perhaps at the most over the entire course of the study.

Jennifer Davis - Pfizer - IR

Great. Thank you. Operator? We'll take another question from the phone please?

Operator

(Operator Instructions) Our next question comes from David Risinger with Morgan Stanley. Please proceed with your question.

David Risinger - Morgan Stanley - Analyst

Yes, thanks very much. A couple questions. First, with respect to Phase III, since you're not controlling for statin use and investigators are being provided the lipid data, is there any risk of unblinding from your perspective? And then second, going back to the cardiovascular discussion, could you just provide some more color on your discussions with the FDA about the cardiovascular risk assessment and did you specifically discuss the possible cardiovascular outcome study or did you just assume that it's not necessary based upon your general dialogue? Can you just help us understand that? Thank you.

Bernie Zeiher - Pfizer - VP, Inflammation Disease

Okay. So with regard to unblinding, we won't -- again, we will be providing the lipid data, we will not be providing things such as CRP which could definitely unblind individuals in the treatment effects and I guess in general, we do not expect that this would necessarily unblind people to treatment. So let me then talk about at least our interactions with the FDA. What we proposed to the FDA and the way the interactions go is they have seen the lipid data, they've seen all of the safety data that we have provided and that we've reviewed and what we provide to them are the proposed list of studies and we say is this adequate for a regulatory approval? And they agreed that -- and again with full knowledge of the lipid effect that we've seen that the package of Phase III studies were sufficient for regulatory approval.

Jennifer Davis - Pfizer - IR

Thanks. Operator, are there any other questions on the line please?

Operator

Yes, we have our next question is from Seamus Fernandez with Leerink Swann. Please proceed with your question.



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Seamus Fernandez - *Leerink Swann - Analyst*

Thanks again. So just a couple of other questions. One, just in terms of the data, so far we've only predominantly seen 12 week data and just wondering if we should read through to the sort of 75% ACR response that we're seeing at the ACR20 and in the longer term 1024 data, if we should anticipate that that will hold true in the data going forward. Obviously you've had the data from some of the earlier studies for some time but aren't presenting those until the ACR, so I'm just wondering if we should anticipate that the effect should remain strong? The second question is you showed a very strong effect versus Humera in its monotherapy study and I'm just wondering why you chose or was it specifically requested you do a study versus Humera and methotrexate. It seems to me that could have been a label advantage. And then the last question is commercially, how are you thinking about positioning the drug across all therapeutic areas, as a potential competitor to methotrexate over time or is basically everything on the table?

Bernie Zeiher - *Pfizer - VP, Inflammation Disease*

So with regard to your first question about whether we anticipate the results to be strong, so as I mentioned both studies 1025 and 1035 have completed what we presented to date were the 12 week interim analysis. We do plan to submit the 24 week interim analysis and unfortunately we can't present those at this time just because we would like to present them at an appropriate scientific forum and in general we believe the effects will be similar to what you've seen and we will be presenting those in more detail, submitting those at least at ACR and hopefully those will be accepted and we'll be able to present them at that forum.

Unidentified Corporate Representative - *Pfizer*

And what we have shown is that the 1024 patients have gone out to one year that the month six and the one year ACR response data have been quite stable, so one can draw the conclusions one wishes from the publicly disclosed long term data out to one year on those patients.

Bernie Zeiher - *Pfizer - VP, Inflammation Disease*

So with regard to inclusion of adilumumab in our Phase III program, that was part of our plan that we submitted and we do believe it's appropriate and it's important to have data within the same trial and think that it will be supportive of the, particularly for Europe where comparative effectiveness is especially important, although as I mentioned the study design itself, it is an active control. The study is not powered or designed to show superiority or non-inferiority to adilumumab.

Finally, with regard to the commercial positioning I'm going to turn it over to Mark White our commercial lead and let him address that question.

Mark White - *Pfizer - Senior Director, Information*

So with positioning obviously, it's still a little bit early given that we're just going into Phase III but we are very happy and excited with what we've seen in Phase II so far and we do think some of the key attributes that Bernie mentioned earlier will help us in the marketplace. Obviously a new novel mechanism of action with very strong efficacy is something that should be valued very strongly in the marketplace. There is still a lot of patients, in fact about three quarters of the patients today are still not in remission or not getting adequate response, so we think there's a strong need there. Obviously having something that's oral that has a rapid onset of action should be very valuable as well as the short half life where you'll have a rapid wash out. Also being a non-biologic we think will be some advantages there. So we do see that there will be a strong value proposition for the product and we will be able to position it within the marketplace as it evolves and a good position.



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Jennifer Davis - Pfizer - IR

Great. Thanks Mark and Bernie. With that, no more questions in the queue so we would like to thank you for your interest in CP-690,550 and for your time here this evening. Thanks.

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