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## **Conference Call Transcript**

**PFE - Pfizer Inc at American Society of Clinical Oncology (ASCO) 2011 Annual Meeting**

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## PRESENTATION

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**Chuck Triano - Pfizer Inc. - SVP, Investor Relations**

Okay, I think we will get started now. Thanks, everybody, for coming to our ASCO review and I will introduce the folks here. To my right, we have Mace Rothenberg, who is the SVP of Clinical Development and Medical Affairs for Pfizer Oncology. To his right, Denise Bruns, who is the Vice President of Late Phase Development within the Pfizer Oncology group and [Glenn Andrews], who is the axitinib team leader. Mace will run through some slides and then we will all take some Q&A.

And before we start the presentation, I just wanted to introduce Geno Germano, who is the President and General Manager of our Specialty and Oncology business unit. So with that, I will turn it to Geno and then we will get going. Thanks.

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**Geno Germano - Pfizer Inc. - President & General Manager, Specialty Care & Oncology**

Thanks, Chuck. I really just wanted to take this opportunity to welcome you all here, thank you for taking the time out of your busy day to meet with us. This is a particularly exciting time as I am sure you can imagine for Pfizer and Pfizer Oncology. This is an area that we made a deliberate decision to get involved in and invest in and to focus on and I think that what you have seen this week and what you will see tonight through the

presentation is that we are pretty encouraged with a lot of the progress that we have made. You are going to see some exciting data from our late-stage compounds and maybe a glimpse of some of the earlier compounds that you may not know as much about.

This is part of Pfizer's innovative core and it is an area that we want to put more attention on as we focus our resources on medicines that can meet significant unmet medical needs and create a platform for a strong future for our Company. So with that, I am going to turn it over to Mace and have him take you through the exciting stuff. Thank you.

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

Thank you, Geno and thank you for the kind introduction and also thanks to all of you for being here. I know there are a lot of demands on your time and I appreciate you choosing to spend some of your time at ASCO with us. What I would like to do over the next 20 or 25 minutes is really give you an overview of why I am very excited about this portfolio and I think we have opportunities not only to see the impact of this in the near term, but in the long term as well. And so with that, I will start.

So we will start here with the forward-looking statements and you know that the statements that we will make here, some of them are anticipating certain event that may or may not happen. So please take that into account.

So when you take the title of this slide, you can put this on any company's logo and everyone will try and convince you they are going to be the leader in oncology. So what I would like to do is to really share with you a sense of why we believe that is possible for Pfizer to become a real leader in oncology.

We have a long history of oncology; we are not new to the field. It dates back to the 1980s when Pfizer and its legacy companies had epirubicin and subsequently brought in compounds like Camptosar and like Sutent and like Aromasin, but what we are seeing now is an inflection point in the growth of that oncology portfolio. And that this year, we have committed to bringing three new drugs to submission before registration -- crizotinib, axitinib and bosutinib. That has never been done before in oncology by any company. Already in 2011, we have gotten supplemental approval for Sutent in pancreatic neuroendocrine tumors. So we think we could actually back up many of the statements that we will be sharing with you this evening.

Personalized medicine, right drug, right target, right patient, incorporation of molecular profiling and whenever I attend ASCO, I try and step back and ask myself what was the one or two main themes of this year's meeting. And I think one of the things that I come away with is the idea that molecular characterization of tumors is really going from theory and something that is done only in major academic medical centers to something that is actually going to be reduced to practice and made accessible to physicians who treat cancer patients. It is going to become integrated into their practice, not in 5 or 10 years, but in the next one, two and three years.

Why? Because we have compounds now that actually can be administered based on the information you get from that molecular characterization. And this is not something that is new or foreign to oncologists. They have been doing it for decades with characterization of hormone receptors in breast cancer or HER2 or EGFR mutation more recently. This is an evolution, but I think you're really seeing a tipping point here when it comes to that becoming a reality.

This slide I think really steps you through the advantages of personalized medicine, the fact that, in clinical development, instead of looking for a small difference in a large unselected group of patients, you are going to be able to look for a larger difference because you are enriching the patients who enroll in that trial to those who have expression of the target. So you will see -- you will have an expectation for a bigger difference. What that means is that you don't have to do as large a trial to detect a bigger difference and you can complete that trial earlier.

In terms of the commercial benefits, taking a heterogeneous group of patients, some of whom won't respond or respond for a brief period of time and those whose tumors do express the target and are likely to benefit, now you are able to segment those out and say that those patients who are receiving that treatment are on for a longer period of time and more likely to benefit. So I consider the scenario a win-win-win situation. Certainly the patients win because they are more likely to receive a drug that is going to help them and less likely to receive a drug that is just going to provide toxicity.

The earlier submission and earlier launch is a win for the sponsor and for the public who will benefit from that drug. And then, lastly, it is going to be a win for the payers because they are not going to be asked to pay for drugs in patients who are not likely to benefit. In fact, in one of the sessions that I attended, they were actually showing that EGFR testing is actually, even though the cost adds an expense upfront, focusing that

treatment on patients with EGFR mutant tumors saves the healthcare system money. This is something that everyone is really concerned about and this is a step in that direction.

So let's look at what we have presented at ASCO 2011 and then later on give you a glimpse into the portfolio. Our late-stage portfolio now can be divided into three main categories -- renal cell cancer where we have two marketed compounds already, Sutent and Torisel, and at this meeting today for the very first time, the results of our Phase III trial of axitinib in second-line treatment of advanced renal cell cancer were presented. The second category is lung cancer. The third category is hematologic malignancies. So you can see we have a presence in each of these three areas.

So let's turn first to renal cell cancer. Even though we have a number of drugs and in fact, even the discussion today of Dr. Rini's presentation asks the rhetorical question, do we need another drug in this disease? Well, when you look and see that 13,000 Americans will die from renal cell cancer, there is your answer. As good as we are, we are not as good as we can be. So the answer is clearly, yes, we do need additional drugs for this.

Well, the drug that we are excited about is axitinib. These are all described as VEGF inhibitors. But each of the VEGF inhibitors have different characteristics. Sutent is more of a multi-kinase inhibitor hitting other targets as well like KIT and like RET. Axitinib is one of the most selective inhibitors that has been in development targeting just VEGF R1, 2 and 3. And it is obviously a very important pathway to hit in renal cell carcinoma.

This is a summary of the data that was presented by Dr. Rini from the AXIS trial. In the Kaplan-Meier curve, you can see an early separation of these curves showing that median survival for the axitinib was improved over -- median survival for the axitinib is improved over sorafenib and rather than the actual midpoints, the medians, I'd really draw your attention to the hazard ratio of 0.665. And I think that that is a more meaningful figure in understanding this because what that means is that the risk for progression is reduced by a third in patients who receive axitinib compared to those who receive sorafenib.

And as you actually turn that, the inverse of that is you actually prolong progression-free survival by 50%. So when one asks is this meaningful, absolutely meaningful. When you break it down into the subsets of patients, those who are previously treated with cytokines and then placed on this trial, the median progression-free survival for axitinib was more than a year, second line. I mean this is what we were looking at first line in previous trials, but we are seeing an almost doubling of that progression-free survival.

And even in those individuals who were previously treated with another VEGF R inhibitor, Sutent, you still see a meaningful progression-free survival with axitinib of 4.8 months compared to sorafenib 3.4 months, a hazard ratio here of 0.74. So I think that these data are clear, are consistent and are clinically meaningful and we are very excited about the possibility of this eventually reaching the market and helping patients.

Let's turn to lung cancer. 85% of patients are non-small cell lung cancer and we are actually moving very rapidly from an era of just characterizing patients by histology into two categories -- small cell and non-small cell and then by different histologies within non-small cell -- squamous, adeno, large cell -- to now look at molecular characterization.

And one of these that was discovered just a few years ago was ALK and people say, well, gee, it is only 5% of patients. What is that going to mean for Pfizer? When you actually look at unfortunately the high rate of incidents for lung cancer, we are looking at around 8000 Americans who will be diagnosed with ALK-positive lung cancer this year and around 40,000 worldwide.

So when you put that in context, this exceeds the number of patients each year who are diagnosed with Hodgkin's disease or testicular cancer or CML. No one would characterize those as rare tumors or tumors that are too insignificant for us to develop new therapies in.

So this is what I have talked to you about and actually this slide was prepared recently, but not so recently that we don't need to update it because those of you who heard Mark Kris' presentation of the molecular characterization of more than 1000 patient tumor samples through the lung cancer molecular consortium saw that when they selected 10 what they considered driver mutations, which included EGFR, included ALK, they were able to identify and drive mutation in more than half the patients' tumors.

So what was previously a black box of what was driving lung cancer is now opening up and we are shedding more light, gaining more insight and now being able to take action on that because we have got drugs that can hit some of these pathways and hopefully more in the future.

This, in one slide, depicts some of the excitement that we have about crizotinib. By the top, you see the timeline. This entered clinical trial in 2006 and it has now been submitted for registration in 2011, a period of five years, and for those of you who know about oncology drug

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development, you know that this is about half the time that the typical cancer drug takes in clinical development. So that I think is a real testament to the efforts of the team, to the efforts of collaboration between industry and academia and government to bring this important drug forward.

What makes it so important? Well, the fact is you see that approximately 90% of the patients whose lines there are below the median or below the baseline, 90% of tumors shrank on this therapy. And in about 60% of those, it met the criteria for objective response.

What is not shown on this slide is what does that mean to patients in terms of symptoms. What I can tell you is that many of these patients, and I will show you in the next slide actually before I get to that, in the slide to the right is the data that was presented yesterday for the first time and that was the survival and you can see that, at one year, the survival rate, the updated survival rate was 70% at one year and 55% at two years.

And you say, well, is this just a good subset of patients who would have survived long if you hadn't given them chemotherapy or no therapy. And what Dr. Shaw was able to do is to say, well, let's take a look at ALK-positive patients who were seen and treated before we had crizotinib and match them for age, non-smoking status, etc. and see how they did.

And what they found was, at the one-year mark, the survival in that group was 44% compared to 70% of patients with ALK-positive tumors who got crizotinib. At two years, the difference was 55% for patients who received crizotinib, 12% for those who didn't. Now this is not from a randomized trial, but this drug has moved forward so quickly that this at least is able to put these results into context with a carefully matched control group for important known prognostic factors. We think of that as very meaningful and it's going to help us in our negotiations with both regulatory agencies and payers.

This is what I was alluding to earlier. This is the data on quality of life. Is this meaningful to patients? Well, certainly survival is meaningful. What about symptoms? These are highly symptomatic patients. They have shortness of breath, they have chest pain, they have cough, they have fatigue and what you can see here is that all these base lines start at zero and it really is extrapolated back to zero, but a meaningful change in this measurement is a decrease of 10 points or more and you can see that all those points that have stars next to them mean that this is a clinically and statistically meaningful improvement in fatigue, in pain, in shortness of breath and in cough.

And in hearing stories from physicians who see these patients, these improvements begin weeks or even days after starting treatment. It really is a remarkable experience for those not only patients and families, but for the physicians and healthcare providers who observe this transformation over a short period of time. So this is really quite an impactful drug.

Let's now turn to the third category and that is hematologic malignancies. As David Roth, my colleague, who is a hematologist reminded me, that when you lump all hematologic malignancies together, you don't separate out lymphomas and leukemias or one kind from another, you take them all together, hematologic malignancies are the second-leading cause of cancer death behind lung cancer, not a trivial area for a pharmaceutical company interested in hematology oncology to go after.

CML, another area where we have a number of useful drugs. Do we need another one? Well, we have brought forward data that we think the answer to that question is yes with a drug called bosutinib. And then non-Hodgkin's lymphoma patients, about half of them relapse following treatment with first-line therapy. And many of those patients are elderly or infirm and are ineligible for the one salvage therapy that may harbor hopes of long-term survival and that is bone marrow transplantation. So these patients have very few options and we have a drug called inotuzumab, which may give these patients new hope and we are very excited about the preliminary data that we have demonstrated from this.

This is inotuzumab. We actually have Phase II data as well that has provided the background for a Phase III trial that we have combining inotuzumab with Rituxan in patients with relapsed or refractory aggressive non-Hodgkin's lymphoma who are not eligible for transplant. That trial has just begun, but the report that came at this year's ASCO was actually in acute lymphocytic leukemia and this was generated at MD Anderson through an investigator-initiated research trial.

We realized that all the good ideas about how to develop these drugs don't come from within the Company, so we worked very hard to establish relationships with people who are really expert in this disease area who understand the biology. And when they do make proposals to us and they are meritorious, we will provide drug and support for them to do a trial independently.

One of these trials was presented today by Elias Jabbour out of Hagop Kantarjian's group at MD Anderson where they said this target for inotuzumab, called CD22, is present on acute lymphocytic leukemia. So we have got a lot of these patients, they've got a terrible outlook after they relapse, so they did a trial and they actually saw more than a 50% objective response in these patients.

And in Dr. Kantarjian's words, this is the most active single agent therapy ever for ALL. Now that is a very bold statement. We couldn't make this. These are not our words. These are the principal investigator's words. Clearly this experience is going to have to be replicated and expanded, but this shows you the collaboration that we must have between academia and industry for us really to move forward in thoughtful ways.

For bosutinib, this is a SRC-Abl [atarecine] kinase inhibitor that has shown in this ASCO with updated results that in patients with relapsed and refractory CML who have received imatinib and some of whom have received dasatinib and/or nilotinib, this drug has activity and although it is too small for you to see, you can see that this breaks down into subgroups. The really take-home message is that there are a substantial number of patients who have prolonged progression-free survival and overall survival even in the setting where they could not tolerate or did not respond any longer to the existing agents. So we feel that this drug from these data have -- this drug may have a place in patients with relapsed and progressive CML.

In the frontline setting, a head-to-head trial was done called the BELA study. This was updated with 18-month data at this meeting and it shows you that, in the green bars, you can see that both the cytogenetic responses in green and the molecular responses in the green here occurred faster with bosutinib than it did with imatinib. And actually when you look at any of the second-generation SRC-Abl kinase inhibitors, you will see that, over time, over a long follow-up, imatinib does catch up with these, but those events occur later on. And so there is more of a period of time for bad things to happen before imatinib's effects are seen. So we think this is a beneficial effect.

Importantly, in this data, are disease-progression events and when you look at treatment failure, which includes transformation from chronic phase, which may last for months or years, into accelerated phase or a blast phase CML, whose disease course is measured in weeks or months, you see a lower transformation rate, as well as fewer deaths in those individuals who were treated with bosutinib. So we think that both in the first-line setting, as well as in the relapsed setting, we have data here that are compelling and we are interacting now with regulatory agencies to understand what information they will need for their review.

So is this it? Do we just have these drugs that are going to be emerging this year into registration and then a gap? The answer is no. We actually have one of the more robust pipelines. And what you can see here is the fact we have multiple agents that we are very excited about and these agents come from different categories. We are not just in one therapeutic category. So we have SMO and hedgehog signaling inhibitors that can target what we feel is a very important pathway. We have PI3K and mTOR inhibitors, which can distinguish themselves from others in that they are a combined inhibitor of both, which we think is important and we have an oral preparation and an IV preparation, which is more than just a convenience factor. It may actually confer a biological difference.

We have an irreversible Pan-HER inhibitor that we are developing in lung cancer and I will talk about that in a moment. We have a drug that is an active [end] receptor-like kinase 1 inhibitor that was presented yesterday that showed very significant activity in the early phase trials. That is very exciting. This is a new kind of angiogenesis inhibitor and we have a CDK 4/6 inhibitor, which inhibits cell cycle progression.

So you see we've really covered a wide therapeutic area and we think that all these are tied together by being first in class, best in class and potential for having a diagnostic to go along with it to be able to understand which patients might benefit from this the most.

This is a summary slide for PF-299. This is our small molecule irreversible inhibitor of HER1, HER2, HER4. HER1 is EGFR, HER2 you know about and HER4 is sort of a silent partner in all this. So this is actually sometimes referred to as an EGFR inhibitor, but it really distinguishes itself from Tarceva, not only biologically in that it hits other receptors in this family and is an irreversible inhibitor rather than the reversible inhibitor of Tarceva, but also clinically that we demonstrated in previous ASCO showing that for progression-free survival in patients who have already received chemotherapy that you have a significant improvement hazard ratio of 0.704 improvement in progression-free survival for patients with non-small cell lung cancer treated with PF-299 versus Tarceva.

So this is has actually prompted initiation of two Phase III trials, the ARCHER trial that seeks to replicate this in a larger group of patients, Tarceva versus PF-299 in second or third-line therapy and BR.26 trial being conducted jointly with the NCI Canada that is looking at patients who already received chemotherapy and an EGFR inhibitor like Iressa or Tarceva and still have progressed and this is randomization between best supportive care and best supportive care with PF-299. The first study is about to be initiated; the second study is already underway.

This is a CDK 4/6 inhibitor. This is interesting in that it is cell cycle inhibition that has a dysregulation in cancers, in certain cancers and you could actually characterize that when you actually get the tumor sample and look at certain characteristics to see whether this might be a driving process within that tumor. So we have a trial underway now looking at this drug in breast cancer and multiple myeloma.

The first phase of the trial was to take unselected patients and find what the best and most tolerated dose is for PF-299 1 with letrozole and then collect that information. And then in the second phase take only those patients whose tumors have these certain molecular characteristics that we

think will enrich for sensitive tumors to inhibition of CDK4. So we are in the process of analyzing the first set of unselected patients to see if those markers might work. If they work, we are well on our way to confirming that in the ongoing trial. If they don't work, we have a chance to revise our theory and adjust that into the ongoing trial.

So we think that this is a rational way of developing this drug. It also shows that our commitment to personalized medicine is not limited to just crizotinib and ALK, but actually we are using this as a first step to bring other drugs forward in a more targeted, more rational fashion.

So in closing, what are the key takeaways for this presentation? Well, the first is we are delivering on the promise of personalized medicine and becoming a leader in oncology. And the demonstration of that is the rapid discovery and integration of biological insights in the development of ALK with a diagnostic biomarker.

Number two, we are making steady progress in accelerating science in areas of unmet need, including renal cell carcinoma with axitinib. We are laying the foundation for the future with a rich and deep portfolio of novel compounds like PF-299, like inotuzumab, like CDK4 and ALK-1 and we are a committed partner in maximizing the understanding of our portfolio in the interest of cancer patients worldwide by collaborating with academic medical centers and researchers, corporate groups, patient advocacy groups in order to make sure we are bringing in the best ideas and using those to inform the design of our clinical trials.

So with that, I would like to stop, thank you for your attention and open it up to questions. And when you do have a question, please let me know where you are from and what your name is and wait for the microphone.

## QUESTION AND ANSWER

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### **Dave Risinger - Morgan Stanley - Analyst**

Dave Risinger from Morgan Stanley. I have three questions. The first is a high-level one. If you could just help us understand, Mace, what is changing under the new head of R&D, Mikael Dolsten, at Pfizer?

Second, with respect to the ALK inhibitor, it is not quite clear to me how patients are going to be identified. Meaning you mentioned that 5% of patients could be candidates for crizotinib. Other data suggests 3% or 4%, but will all patients, once it is launched, get lung biopsies, extra lung biopsies to identify the small percentage that would be a candidate? And then would those biopsies occur when other biopsies are taken? And do they need to be sent to a special diagnostics provider? So I am just trying to understand the actual process for identifying these patients since lung biopsies are required.

And then third, is there an opportunity to price bosutinib very high given that it will be for patients that are resistant to other therapies? Thank you.

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### **Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

Well, thanks. Great questions. I didn't want to forget any of them, so I was writing them down as you asked them. So first was the R&D under Mikael Dolsten. Mikael has done a very thoughtful job in reviewing all the therapeutic areas. And as you have heard earlier in the year, the Company has made a decision to exit some therapeutic areas where we felt we could not be leaders in the field. Allergy is one and pulmonary is another.

So he has gone through a very thoughtful process and being able to focus our research efforts both by category as well as by site. So the Sandwich site has been closed. So I think that the idea is a responsible review, identifying areas where we can innovate and win. So I think he has had a very tangible impact on this process and we look for wonderful things to come from his R&D organization.

In terms of identifying ALK-positive patients, actually this could be done on the specimens that were obtained previously for diagnosis. So you have the formal and fixed paraffin-embedded tissue. So they don't need a new biopsy to, as some diagnostics require, they don't. We can get this information off of existing samples. Even the site doesn't necessarily have to be the lung. We have seen biopsies that might be from a lymph node or some more easily accessible area be just as helpful in diagnosing this.

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And the last part of that question was do these have to be sent to a specialized lab. With the test that is being used right now in the diagnostic development, it is a FISH assay. This does require some specialized expertise, but it can be done in many academic medical centers and major medical centers, as well as regional laboratories that have been set up by Abbott.

We are also exploring other platforms that could be used, reverse phase PCR, as well as immunohistochemistry and we are looking to try and reduce as many barriers as we can to allow as many patients who have lung cancer or a biopsy with suspicion of lung cancer to have it sent for testing because we feel that the worst thing would be to miss a patient who has ALK-positive lung cancer and would not get crizotinib.

So we think that this is something, just like it has been integrated into breast biopsy, those are automatically sent for receptor testing and HER2 testing just like bone marrow biopsies are sent for cytogenetic tests. This is not a new concept to oncologists. In fact now more and more physicians who treat lung cancer are sending specimens for molecular testing, for EGFR mutational analysis.

So we think that this is something, and actually if we step back and think about what is the major theme of this year's ASCO, one that I take away is that molecular testing, which is more of a hypothesis than theory, is now actually moving into real-time practice. And I think we will see that in the near term.

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**Catherine Arnold - Credit Suisse - Analyst**

Catherine Arnold from Credit Suisse. Could you update us on your plans for crizotinib in regards to ALK-resistant programs that you might have in terms of a follow-up ALK in the case of patients that have resistance and/or combinations that might help overcome the resistance?

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

Great question. So as we see with other targeted therapies, and as we are seeing with crizotinib, some patients develop resistance. This is not surprising. It doesn't necessarily undermine the value of a target agent, but it really challenges us to get smarter. So with the original publication of the crizotinib lung cancer data in the October New England Journal of Medicine, what we saw also was the first report of resistance and that resistance was due to kinase mutations that prevented the ALK from inhibiting -- prevent crizotinib from inhibiting ALK signaling.

But what we are also recognizing is that, yes, we and others are developing backup compounds that still have activity in those resistant tumors, but that may not be the whole story and in some cases, it may be not through the mutation of the kinase and ALK, but through the activation of complementary pathways that can also drive lung cancer, like EGFR, like IGF-1, like a myriad of others like MET. And so it is really becoming important for us to be able to now get biopsies in those patients with progressive tumors to understand what they represent so we can then match them to the right treatment.

So it is something that I think that simply saying we have a drug now that is active in tumors that have kinase mutations that are resistant to crizotinib may end up being a relatively small piece of the pie of crizotinib resistance. But the gratifying thing is to see how many patients are still progression free on crizotinib.

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**Alison Yang - Barclays Capital - Analyst**

This is Alison Yang from Barclays Capital. I wanted to ask one more follow-up question on crizotinib. In the profile studies, what is experienced with patients who are not as heavily treated. Are those patients enrolled in this study and how active is the agent?

Secondly, some of the physician feedback we saw is that lung cancer patients go through this process where you start with EGFR, testing KRAS and finally crizotinib, by which time often there is not enough tissue left and you have to go back through a second biopsy. And they really would prefer the non-FISH assay and how far along on that process?

And finally, can you discuss, now having three potential agents in renal cell, where do these three agents place in first, second and third-line, clear cell/non-clear cell, how do you -- would you come axitinib with Torisel? Can you just describe your commercial strategy there?

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

Great. So in terms of crizotinib, not in the profile studies, which is a Phase III trial, there actually we do have two ongoing trials right now in lung cancer. One is in first-line randomized trial of crizotinib versus standard chemotherapy. The other is in second line where we have just single agent crizotinib for all patients. So we will be getting information, more information on crizotinib in patients without prior chemotherapy, but we don't have that data yet.

We do have information from our Phase I trial, the 1001 trial, where as more experience was gained with the drug, investigators were really lobbying us to allow patients with newly diagnosed ALK-positive lung cancer onto the trial. And so we amended the trial to allow that and there have been a handful of patients who have had crizotinib first line and what we can say is that the drug does have activity.

The number is very small, so the actual response rates could be misleading, but I think this has been presented at previous meetings. We see about 80% objective response rate in that first line, but it is very -- the confidence intervals around that are very wide and that needs to be confirmed in the Phase III trial.

So we think that if this is truly a driver could it have activity as first line and replace chemotherapy. A forward-looking statement for sure, but that is why we are doing the Phase III trial. But actually if EGFR is a precedent for that, the answer appears to be yes, at least for progression-free survival and we are seeing the practice standard of care change in that direction.

In terms of Asian patients, we are seeing some trends in that direction, but because we are not doing -- at least we don't have the results yet on specific trials in these groups, they do seem to have a bit of a higher response rate, slightly higher plasma concentrations of the drug. What that means overall we are not sure, but this is one of the prices we pay for the very rapid development of a drug. We know that there are going to be gaps in our knowledge. And we will have to go back and fill those as the months and years pass, but we still think we have enough information to move forward and allow this to be marketed to help many patients.

The issue of tissue adequacy and alternative to FISH testing, we think that that is an important issue and you raised two aspects. One is can we use a non-FISH platform and we are now working with a number of medical centers to actually -- for them to collect ALK-positive samples and to actually do, in a blinded fashion, a FISH assay and a non-FISH assay, RGPCR or immunohistochemistry, to ask the question what is the concordance between those and how well do those predict for sensitivity to crizotinib.

Ideally, you are right that there is limited amount of tissue that we have and if we have to send it off to one laboratory for EGFR testing, another for FISH testing, another for KRAS testing, we are going to run out of tissue. So this is one of the challenges we face is can we develop technologies that will allow us to multiplex this on a single platform and say do a molecular characterization of lung cancer and we will get maybe those 10 driver mutations on a single sample. And we know that that is possible given the consortium for molecular characterization of lung cancer results that were shared at this ASCO. So this is a rapidly evolving area, but I think it is within our grasp.

And your last question had to do with RCC. How many drugs is enough? Well, we feel that there may be a role for each of these drugs and as we learn more, we will be able to understand how best to utilize these [innovations]. So we now have -- each of the drugs, even though again they are all lumped together as VEGF R inhibitors, they have somewhat different characteristics. They have different side effects and we have a different amount of data in each one.

So for physicians who say I really want to know that this is a well-tolerated drug, that this is a drug I can give safely to my patients, that we have got long-term safety, we've got long-term efficacy. Well, we have more than 100,000 patients who have been treated worldwide with Sutent since it was launched. We have more than 10,000 patients who have been treated on clinical trials of Sutent and physicians have five years of experience with the drug. So there is that element for a familiarity and activity.

We now have Torisel, which we know is very active in patients with high -- whose tumors have high-risk characteristics. So we have information on that drug. It was the basis for its registration originally that we really haven't probed as carefully with the other drugs. So there, again, a physician might say I know you have got renal cell and there are a lot of choices, but you have got some really bad characteristics here. I feel like the best body of data exists for Torisel.

And then we have axitinib, which has the data generated in the second-line setting both after cytokines and after Sutent. So we think that we have actually a family of compounds and a RCC franchise covering both of the active categories of drugs, VEGF R inhibitors, as well as mTOR inhibitors. So we see that we can actually integrate these and really provide the right drug for whatever the physician's preference is based on the patient characteristics.

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Chris Schott at JPMorgan. Just coming back to crizotinib, I have a number of questions here about ALK testing. Can you just help us, from your perspective, from a commercial standpoint, how long do you think it is going to take before we can get broad enough ALK testing, enough tissue, etc. to really make this drug a success? And is this something that's going to happen quickly or is this going to be a couple years down the road?

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

Well, we are working closely with Abbott on molecular diagnostics where we also recognize that awareness is half the battle. At these meetings, we talk to each other, we talk to other oncologists. But who are the people who make the diagnosis originally and get the tissue? Pulmonologists or surgeons. So we have to make sure that we reach out and educate them.

What about general practitioners or internists who may look at a nodule and say I afraid that's lung cancer, it looks metastatic. This is a person who is going to die and maybe I will just manage him symptomatically, so I don't subject him or her to the toxicities and ravages of chemotherapy, which may not be effective. It is really an education, so for people to say wait a minute, but it has these characteristics, there is a very different future for them and we really need to pursue that line of investigation.

So I think that what we are going to try our best to do is remove as many obstacles as we can as quickly as we can to make sure physicians are aware of this as a distinct subset of lung cancer, that does have a very effective therapy and that this is something that can be done on archival tissue.

So it removes that perception that I have got to subject this person to another test and that has costs and morbidity associated with it. So I think we can do that and we should be able to do that sooner rather than later.

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**Chris Schott - JPMorgan Chase - Analyst**

And a second question is with crizotinib. Are there other cancer types that you are pursuing this in and what would those be and how do you see the opportunity beyond non-small cell lung cancer?

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

Remember, ALK stands for anaplastic lymphoma kinase. It was first described in 1992 in a rare kind of lymphoma and then was subsequently identified in a neuroblastoma. And so we know -- in a rare soft tissue sarcoma called IMT. So we know this is activated in each of those cases through a translocation. But when you look in gene expression arrays where it may be upregulated or amplified, you now have other diseases, things like gastroesophageal tumors, breast cancer, colorectal cancer, a myriad of other cancers.

But there, the mechanism is not as clear. So it is upregulated, what else is upregulated and is this something that is going to have a benefit in those patients and will the benefit be as marked as it is in tumors that carry translocations. So we have now opened the trial to address just that issue, looking at other tumors and to be able to efficiently evaluate the role of crizotinib in other tumors besides lung cancer.

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**Chris Schott - JPMorgan Chase - Analyst**

And then the final question was just this personalized medicine approach that was kind of the theme throughout the presentation. When we think about the R&D budget in oncology as you are pursuing more and more of these opportunities relative to what you maybe pursued with Sutent and kind of these broad programs before, should we be thinking about the R&D budget in oncology going down as this kind of plays through or is it simply allowing you to pursue more targets and we can keep up more of a static R&D budget as we go through the next two years I guess?

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

I hope it doesn't go down because I think that what we are seeing here is a real shift to be able to have the drugs that we develop -- we have more insight into why they work and why they may not work, not pursue many of those dead ends and be able to move these forward in a more efficient manner and bring those forward in a more targeted group of individuals who can benefit from that.

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But what we shouldn't lose sight of is the fact that whereas before we were developing drugs for a certain organ, lung cancer, breast cancer, now we are saying this is a molecular pathway or target that may be present in multiple cancers. And when you add all those other indications to this, and we don't know what that is going to be for crizotinib, you actually may aggregate even more and more beyond where we started from.

So this is a challenge in terms of precision or personalized medicine. Where is this going to take us? And I think that we have -- but this is clearly the right direction to go. I think this is what the patients expect, payers expect and we expect.

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**Seamus Fernandez - Leerink Swann - Analyst**

Thanks. This is Seamus Fernandez from Leerink Swann. So just a few questions. Maybe you can just update us on any pursuit of axitinib in the front-line setting and how those trials are designed. Second, my question is really kind of, as we think about crizotinib, maybe this is too simplistic of a question, but is this, A, Gleevec; B, Tarceva; or C, vemurafenib in terms of the kind of progression-free survival? There are three I think very distinctly different curves there that you can think about in terms of survival.

And then the last thing is, as we look at all of these companies pursuing the same targets over and over and over again, it begs the question of overt industry inefficiency. I don't know if this is a question more for Geno as you kind of think about the pursuit of targets like this, but I am just thinking about partnering across different companies as you pursue targets and if that is something that Pfizer will consider realistically and starting to pursue.

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

Great, thank you. I will turn the first question over to Glenn Andrews, our asset team leader, for axitinib to address the question of what the ongoing efforts are in first-line evaluation.

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**Glenn Andrews - Pfizer Inc. - Team Leader, Axitinib**

We have one study, Phase III study comparing sorafenib with axitinib in the first-line setting, 250 patients in first-line and another 200 in second line. That study has recruited and the readout will be some time in 2012. We have one he other study, which is looking at axitinib also in first line, which compares uptitrating or increasing the dose versus not in axitinib patients looking for the benefit there and that will read out also in 2012.

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

And your second question was is crizotinib more like A, Gleevec; B, Iressa; or C, vemurafenib. I would say D, none of the above. I think that when you think about Gleevec in its original efforts, the molecular abnormality that drives CML, the Philadelphia chromosome was identified in 1960. Gleevec was marketed 41 years later in 2001. The outtranslocation that is driving in lung cancer was identified in 2007. Four years later, we have a drug that is registered or submitted for registration.

Is it like Iressa? That was identified -- it was developed almost purposefully without a marker and failed originally and has been around kind of bouncing around for a while until the science caught up with the drug and now we know how to specifically use it. And then the Raf kinase inhibitor, they are looking at a specific translocation that occurs in the tumor and has actually also rapidly moved forward. We think that there are certain characteristics that are different in terms of the way the drugs have been developed, but it is probably most similar with that.

And then lastly, are these small segments going to lead to industry inefficiency? So I don't know, Geno, do you want to answer that or maybe we could ask Denise who is our late stage development lead or even David Roth who is our early phase development lead to take a crack at that. Any of you have any insights that you want to share about that?

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**Geno Germano - Pfizer Inc. - President & General Manager, Specialty Care & Oncology**

I was going to pass on the question actually. I mean I think that we are going to let the science lead us. And I think that we have a robust library of compounds and we will continue to learn from the science as we go along. And if we feel that development within in-house is the right way to go, then that is the way we will go. If we think that partnering with someone else makes sense, then we will be willing to explore that. And I think

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in terms of will there be an abundance of too many compounds with similar pathways, there is still a challenge of figuring out how to -- where in therapy, where in the course of treating the patient is the right tool at the right time for the right patient. And I am not sure it is going to be so simple in the long run that there will be an abundance of compounds that all are competing for the same patient at the same time.

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**Denise Bruns - Pfizer Inc. - VP, Late Phase Development, Oncology**

Yes, I think part of the -- part of your question was on the partnering and with respect to I guess inefficiencies. And I would look at it in terms of a bit like our retirement portfolio. So at any given time, there may be a gap in one area or another and I think that, as we then would have more successes or more trials that may not read out positive that I think we would be looking to fill to the optimal level.

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

David, any comments?

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**David Roth - Pfizer Inc. - VP, Early Phase Development, Oncology**

I think the (technical difficulty) opportunities that come along with collaboration. And over the past several years, there has been an increasing interest in different types of projects to encourage across company collaborations in particular for combining different targeted agents. So I think we are going to see more and more of this as we move forward.

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

Another aspect to that response could be what action we took this week to outlicense our PARP inhibitor. So here is an area where we actually had the first PARP inhibitor in clinical testing and then it didn't have the kind of pace or forward progress that we would have preferred and BiPar and [Sassy] compound made a big splash at ASCO I think two years ago. And then [Kudos] and AZ came along and so now we felt we were going for the leader, we are kind of trailing them, how do we accelerate the development here.

And then as more information has evolved that BiPar and Sanofi large unselected trial that failed and now they are trying to look through for subgroups that may benefit from that. We, at the same time, we, as an organization, made a decision to really focus our research and development budget, as you all know.

So we looked at this and we said this is a compound that has been around for a while. We believe that this is an excellent compound, first in class and maybe best in class. Its development hasn't proceeded as we would have preferred, so now we are in not a prime position and we also have limited resources where is it we can make the impact and what does this drug need to really move forward and be a differentiated compound.

What we realized was, almost like considering whether you could be a good parent for your child and realizing maybe you should put the child up for adoption and find it a good home. And I think that is what happened with Clovis, that we were able to find a company with experienced people in drug development who understood the biology and understood the clinical development pathway. We actually interviewed several potential parents to see which one would be the best one and we finally found the good home for it.

I think Clovis will be able to provide the focus and the resources that are needed to move this drug forward to the market. I think many of us have great confidence this is a very important compound and a tractable target that will have clinical impact. The key is what trials can be done efficiently to bring that forward. And I think that will happen.

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**Seamus Fernandez - Leerink Swann - Analyst**

On crizotinib, since this is a rolling submission, can you just tell us like has the FDA seen everything that they need to see? Are they waiting for additional data? Just give us an update on that, what is going on there. Thanks.

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

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So it was a rolling submission and the submission is now complete so they have all the data that they will need to review and act on this. Certainly the process of approval includes site visits of the investigational sites, site visits of the sponsor, site visits of the manufacturing facilities. All these are being scheduled to try and provide the FDA with all the information it needs to make a final decision.

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**Seamus Fernandez - Leerink Swann - Analyst**

But those additional studies that you mentioned, they are not waiting on those at all?

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

No.

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**Seamus Fernandez - Leerink Swann - Analyst**

There's an agreement already in place?

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

Correct. So we have worked with them and the understanding is that we have submitted this for accelerated approval with acknowledgment that Phase III trials are ongoing now, that we will be able to provide additional data to really complement the information that we have currently. And so that was something that was acceptable to the regulators.

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**Seamus Fernandez - Leerink Swann - Analyst**

And then just one additional, on this slide of the next wave of products, five or six of them there. Any data that we should be looking for in this calendar year?

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

Yes, I don't know how much I can tell you what to look for. What I can say is that with our -- and really credit goes to David Roth for really carefully looking at our Phase I portfolio and recognizing compounds that really didn't have a path forward. So we dropped things like a backup compound for Sutent and a backup compound for axitinib. We didn't think that we needed additional compounds with these moving forward as they did, compounds that really were kind of meandering around.

What we have left include drugs like a gamma-secretase inhibitor that was presented at ASCO that we are seeing early signals of activity, seeing responses in some tumors that are very refractory to treating them like desmoid tumors. If you ask a metabolic oncologist about desmoid tumor, he will roll his eyes and say that is really not cancer. It doesn't metastasize.

So it doesn't really kill the person, but if you talk to a surgeon about desmoid tumor, they will just leap out of their seat and say this is really a challenge for us because if it affects a joint, I try and remove it and I do my best and it comes back and I send it to the medical oncologist, they have got nothing for them. I send it to the radiation oncologist, radiation isn't effective and I have to whittle away and eventually lose the finger and then we may lose the hand and so it is a very different kind. It is an early signal. We are also looking at it for other tumors as well.

We have a smoothened inhibitor that we are excited about. We think that some of the characteristics that it has may have certain attractive features that may allow it to be developed effectively. We have the CDK 4/6 where we have a molecular -- a potential molecular signature there. And so we are very excited about what we are seeing now in the early portfolio and now the issue is how do we take that information and efficiently move that forward and again try to target it most effectively in patients who are most likely to benefit.

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**Kyle Rasbach - Cowen & Co. - Analyst**

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Kyle Rasbach from Cowen & Co. I guess one of the other themes from this ASCO was potentially immuno oncology and I had a question relating to tremelimumab. And it has been dropped in melanoma. But I was curious if it is still being pursued in other tumor types or if it has maybe received a fate worse than adoption?

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

We are looking for a good home for treme as well. This is a child that is still in the orphanage, but I think it is tantalizing. It came this close to actually having a result, but yet it missed its target and it was a negative Phase III trial and we are very happy for the melanoma patients who will be able to benefit from Yervoy. But when I think about immunotherapy and actually when I have spoken to people in academic medical centers who are really the experts in immuno oncology, the consensus is that affecting one arm of the immune system with one drug isn't going to be enough. It is not optimal.

So what we are doing with treme and other CTLA-4 antagonists is we are taking the foot off the brake and allowing the immune system that has been suppressed to actually be revved up and recognized and destroy the tumor cells.

But it also has some nonspecific effects. If you rev up the immune system, it is going to affect other parts of your body like the GI tract. And so what we really need to do is say what else can we add to that that is going to be able to direct the immune system more towards the tumor more effectively.

So I think what we are seeing now is really a very early phase in immuno oncology. And hopefully with compounds that Pfizer, as well as other companies develop, we will be able to actually allow that next big step to be taken.

So I think that immuno oncology with Provenge, with Yervoy, we are seeing that this is no longer a hypothesis, this is reality, but it is just the first step of what we hope will be a much longer and more successful journey.

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**Chuck Triano - Pfizer Inc. - SVP, Investor Relations**

We have one question from the webcast, which is I think a bit more of a clarification question. In the slides, we referenced that there are about 40,000 ALK-positive patients a year in non-small cell lung cancer. The question is is this a developed world figure and do we view this as an addressable patient pool?

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

It is a developed world figure and we think being a drug that is given orally twice a day that has a very moderate toxicity profile that we think that this does lend itself to widespread use.

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**Chuck Triano - Pfizer Inc. - SVP, Investor Relations**

All right, we have exhausted the questions.

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

We still have 15 minutes. It's been a long day.

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**Chuck Triano - Pfizer Inc. - SVP, Investor Relations**

One more over to your left.

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**Alex Arfaei - BMO Capital Markets - Analyst**

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Alex Arfaei with BMO Capital Markets. A follow-up on your immunotherapy comments. We saw some early-stage data in RCC with some vaccines in combination with Sutent. I believe two of them are going to Phase III very shortly. Any thoughts on that?

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

I think that it is a very interesting and promising concept. So this is now saying we have a pathway. The HIF-1, alpha, VEGFR pathway we know is important. We also know immunotherapy is important in renal cell cancer because, 20 years ago, we have seen the activity of interleukin-2 in this disease and the adjuvant activity of interferon in this disease. So it is tantalizing and we've sort of forgotten about those as more convenient agents have come along.

But the question here is, well, what if you combine both. What if you are able to actually target both the immune system and unleash its effect, inhibit the angiogenesis impact? Can you say one and one is going to equal three if you combine those? So I think it is a very rational approach and we are supportive of further evaluation of Sutent with immunotherapies in this setting.

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**Alex Arfaei - BMO Capital Markets - Analyst**

And a follow-up on crizotinib. If I recall correctly, it is also a potent MET inhibitor. Is that correct? Any thoughts about I guess exploring that further to expand the market potential for the drug?

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

Right. And actually crizotinib entered clinical testing as a MET inhibitor. And so when we began observing the activity, the expansion cohorts that were created were those patients with ALK translocated non-small cell lung cancer and another one was those patients who have c-MET amplified or overexpressing tumors. And so that trial is still going on, but we are seeing responses there as well.

So we think that this is an aspect of a compound that we can't lose sight of. It is very easy when you have got something so exciting over here, but we think this is also a very important element as we have seen with other compounds that have been presented here that are pure MET inhibitors. And the frequency of MET as an escape pathway and whether a single agent MET inhibition is going to be sufficient and if so, in which patients are all questions that we are all grappling with. But I think that this is something that we are actually currently exploring.

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**Alex Arfaei - BMO Capital Markets - Analyst**

Are those two types of agents mutually exclusive, the ALK overexpressed and the c-MET (inaudible) patients?

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

In Mark Kris' presentation, it was very interesting, when they looked at more than 1000 lung cancer specimens, to their surprise, they found the vast majority had only one of these that was mutated or activated. So very often, they are mutually exclusive, not 100% of the time, but most of the time they are.

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**Seamus Fernandez - Leerink Swann - Analyst**

Thanks. Since you guys want to have a couple more questions, my question is really on maybe you can give us some insights into figitumumab and what is happening with that compound, should we see a move forward and can you just give us some thoughts on -- there is an awful lot of IGF-1 antibodies where there were posters all over ASCO. There are IGF-1 pathway targets. So give us your rundown and your thoughts on the pathway and what has happened with figitumumab and how you're going after the pathway.

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

Thanks very much because I think that that's a story that was so hot a couple of years ago and now no one talks about it. I think that that is a perfect example of where the compounds ran ahead of the science. We had great information from laboratories showing that the activation of

IGF-1R was very important in a number of tumors in breast cancer, in lung cancer. We had a randomized Phase II trial of adding figitumumab to chemotherapy that showed very promising response rates in progression-free survival.

We had everything we needed to launch into an aggressive Phase III program as we did with two Phase III trials. So you can imagine that there is no one more surprised on the planet than me when I got a call from the independent data monitoring committee saying we are recommending that the trial be discontinued. It was a total shock. Now looking back, and we did a postmortem on these trials what happened.

So one of the things we realized was that our Phase II experience was in a limited number of medical centers, each of which had a lot of experience with the drug. When we did the Phase III, it was a worldwide effort with a lot of centers, some of which had very little experience. We were combining this with chemotherapy, carboplatin and paclitaxel, people were familiar with. Just add IGF-1 inhibitor and you'll see the patient back in a few weeks.

What we didn't realize was there were some side effects that were subtle that centers that had experience with the drug could pick up, but centers that didn't have experience didn't pick up and so we saw a signal of toxicity from that.

In addition, the activity that we have seen, and I am convinced that this is an active drug in this disease and probably other diseases, but those were isolated cases and try as we might to identify a biomarker for those, for IGF-1 levels, IGF-1R expression on the tumor, we couldn't make a strong compelling story for this being a useful biomarker for this compound.

And so it was a difficult decision because when I have spoken to physicians all over the world about their experience with figi in lung cancer, and they tell me stories about individual patients who had remarkable responses, responses that they have never seen before with chemotherapy alone. And yet we weren't smart enough -- we are still not smart enough to know who those patients are.

So we had the team get together, look at the future of this and ask the question -- are we ready to move this forward now? And the answer was no. When would we be ready to move that forward? We don't know. And in the current environment, if those are the answers we get, we had to say this is something that we can no longer invest in. So we kind of put it on the side and are now looking for a partner for this compound as well.

I can almost guarantee that one day, probably at this meeting, we will hear about the diagnostic for IGF-1 sensitive tumors and we will hit ourselves on the forehead and say why didn't we think of that. But that I think is going to happen and when it does then we will be able to have this drug used in the patients who are most likely to benefit from it and most likely to tolerate it and then see it really realize its potential, but that date is not today. Other questions? On the webcast?

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**Chuck Triano - Pfizer Inc. - SVP, Investor Relations**

We are all clear.

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**Mace Rothenberg - Pfizer Inc. - SVP, Clinical Development & Medical Affairs, Oncology**

Okay. I would like to thank you for spending part of your evening with us. If you have any other questions, Chuck Triano is your point person. We will be staying around afterwards if you have any additional questions. Thanks so much and have a great day.

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