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The following communications contain forward-looking statements within the meaning of the Safe Harbor Provisions of the Private Securities Litigation Reform Act of 1995 about Vertex Pharmaceuticals Incorporated and Aurora Biosciences Corporation. While the management of Vertex and Aurora make their best efforts to be accurate in making forward-looking statements, any such statements are subject to risks and uncertainties that could cause actual results to vary materially. The forward-looking statements herein address the following subjects: the belief that the field of kinase inhibitors is going to be a very fruitful field for drug discovery over the next 10 to 15 years and will be an important field in many pharmaceutical markets in the future; the belief that the partnership between Vertex and Novartis position Vertex and Novartis to hold a very significant position in the field of kinase inhibitors which will allow Vertex and Novartis to capture a portion of future markets creating drugs for patients, revenues, profits and shareholder value; the belief that Vertex is on track to deliver to Novartis; the belief that Vertex's chemogenomics and structure based platforms give Vertex a significant, competitive advantage in bringing drugs into the clinic; the belief that more than 10% of the 500 kinases in the humane genome will be viable drug targets; the belief that most developments regarding kinase inhibitors will be in connection with the treatment of cancer, which is the new way to think about the treatment of cancer; the belief that kinase inhibitors aimed at kinases other than those currently targeted will enter the clinic over the next few years for the treatment of cancer; the belief that multiple kinases inhibitors are required to treat various types of cancers; the belief that Vertex will have a number of drugs that will be effective Anti-angiogenic agents in the treatment of cancer; the belief that Vertex will name two development candidates by the end of this year for treatment of disease; the belief that Vertex will have additional partners developing kinase inhibitors that Vertex will develop over the next decade; the expected announcements of new drug candidates developed individually and with partners, which should expand to greater than two candidates per year; the belief that Vertex has a potential diabetic kinase which is the best, most favored diabetes target in any class; the belief that Vertex's drug development model and intellectual property protection strategies (i) are the right mechanisms relating to its drugs, (ii) are particularly suited for looking at large number of related proteins and (iii) will enable Vertex to be ready to mine the future kinase and protease universes; the belief that Vertex has major market opportunities in myocardial and neurological conditions; the belief that Caspase inhibitors have relevance in Sepsis, Liver distress and other things; the belief that Vertex has great potential in anti-biotic resistance, HCV and Alzheimer's disease; the belief that Vertex is developing strong chemistry and intellectual property foundations; the expectation of having 160+ scientists by the end of 2001; the belief that the merger of Aurora and Vertex will strengthen Vertex's ability to produce through-put in enzyme targets and other non-enzyme areas; and the belief that Vertex is in the process of rejuvenating big pharma companies' product pipelines creating opportunities for co-promotion.

The following factors, among others, could cause actual results to differ materially from those described in the forward-looking statements: the possibility of delays in the research and development necessary to select drug development candidates and delays in clinical trials, the risk that clinical trials may not result in marketable products, the risk that the combined company may be unable to successfully finance and secure regulatory approval of and market its drug candidates, the development of competing systems, the combined company's ability to protect its proprietary technologies, patent-infringement claims, risks of new, changing and competitive technologies and regulations in the U.S. and internationally, costs related to the merger, failure of Vertex's or Aurora's stockholders to approve the merger, Vertex's or Aurora's inability to satisfy the conditions of the merger, the risk that Vertex's and Aurora's businesses will not be integrated successfully, the termination of existing Aurora pharmaceutical and biotechnology collaborations, the combined company's inability to further identify, develop and achieve commercial success for new products and technologies, and risks associated with Aurora's new and uncertain technology, dependence upon pharmaceutical and biotechnology collaborations.

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THE FOLLOWING IS THE SCRIPT OF A PRESENTATION PRESENTED TO ANALYSTS, INVESTORS AND OTHERS ON MAY 31, 2001 AND POSTED ON VERTEX'S WEBSITE ON JUNE 1, 2001.

VERTEX INVESTOR DAY

"MEDICAL POTENTIAL OF KINASE INHIBITORS"
ROBERT MASHAL, M.D.

MASHAL: Hi. We'd ask you all to take your seats and I'll get started. My name's Robert Mashal. I'm not a familiar face to most of you. I'm a Program Executive for the MDR Program at Vertex. But one of the other things I do is serve on the Joint Reseach Committee for the Vertex/Novartis collaboration. And it's that hat that I'm wearing today.

And, what I'd like to do is to tell you all a little bit about the medical potential of kinase inhibitors. And show you some of the progress that we're making in bringing kinase inhibitors through the discovery process and into the clinic. And talk about, I think, how Vertex and Novartis are positioned to hold a very significant position. We think it is going to be a very fruitful field for drug discovery over the next 10 to 15 years.

So, I'm going to talk about kinases. What they do in terms of their role in cellular signaling and human disease. I'll give you some historical and new perspectives on kinase drug development. And then talk to you about the advantages that the Vertex/Novartis collaboration will have going forward. And I think that our partnership is going to lead to a very significant position in the field.

So, I'll talk a little bit about Why Kinases, Why Now, and Why Us? And I think, that there are several good reasons for this. It's been known for quite some time that kinases are important in many pathways that are relevant to human disease. That evidence first started coming out in the late '70's and early '80's. And people have always thought that these might be interesting drug targets. But, historically, there were lots of concerns about drug development efforts. So, as recently as the late '80's and early '90's -- drug companies had these small sort of nice efforts, aimed at targeting kinases. But they were viewed with sort of cautious skepticism -- in the sense that people thought it was very, very difficult to make in inhibitors that were specific to individual kinases. And, because kinases played a lot of roles -- and you were going to wind up making drugs that had a lot of different kinases, you are going to have a lot of toxicity problems. And those notions about drug specificity and toxicity have largely been dispelled, I think, over the last few years. And it's something that I'm sure that you've been reading about -- with the recent approval of Gleevec and CML. And, finally, why us? I think that Vertex and Novartis are uniquely positioned in the field. Our chemogenomics and structure-based platforms, I think, really do give us a significant, competitive advantage in bringing drugs into the clinic. And working with a partner who has proven expertise in kinase inhibitor development -- and now, sales and marketing. I think it's going to give a very, very significant competitive advantage -- all the way through commercialization to the Vertex/Novartis partnership.

So, let me just start by telling you what is a kinase? A kinase is an enzyme. And what that enzyme does is put a phosphate group on another biological molecule. And that other biological molecule is usually a protein. The phosphate group comes from ATP. So, what I've got here on the left is a protein. I've got here a picture of ATP. These three pink things are phosphate groups. And ATP gets converted into ADP. And one of the phosphate groups from ATP gets stuck on this biological molecule. And what a kinase does is catalyze that reaction of transferring the phosphate group from ATP to the other biological molecule. Now, when a phosphate group gets stuck on another biological molecule -- it usually modifies the activity of that target molecule. Usually, that modification is an activation of function -- although not always. Sometimes it is an inactivation of function. But, generally what this is -- is when a kinase sticks a phosphate group in another biologic molecule, that biological molecule

has an altered function -- usually becoming activated. And what inhibitors do is block that function in 99.99% of the time. And the way they do that is by occupying the ATP binding side of the kinase.

So that's pictured here. Here's ATP out here. This big, purple glob is a kinase enzyme. That deep pocket that you see in there is the ATP pocket, where normally ATP would go. Most of the kinase inhibitors that people are looking at, actually occupy that kinase binding side. They sit in there so that ATP can't get in there -- and the kinase enzyme can't perform its function.

So, Kinase Cascades -- they operate in cascades -- are important in lots and lots of signalling processes. So, what Kinases do is that they help cells mediate signals. Those signals generated usually on the outside of the cell - --and transmitted into the nucleus -- telling the signal what to do. And, kinases are generally the intermediary, messenger model. So they're involved in signallings for growth factors. For blood vessel growth. For telling the cell to divide -- which all provide targets for cancer. There are kinases in T Cells which are important to transplantation and autoimmune disease. Kinase is the insulin receptor. In fact, a kinase of the signal downstream -- most of the signals downstream are the insulin receptor are mediated through kinases. So, there are multiple points of attachment for that pathway. For Diabetes. Adrenaline signals through kinases. That's important in heart failure and so on, and so forth. What I really want to make a point is -- these are all big diseases.

So if you look at the sort of world-wide drug market -- you know, some of the diseases I've mentioned -- cardiovascular diseases, anti-infectives, cancer agents -- these are all multi-billion dollar type markets. And the real point to take away from this is that kinases are going to be important in many major pharmaceutical markets over the next few years, I think.

Now, kinases are a particularly rich family for drug discovery. There are about 500 kinases in the human genome. And if you just go historically looking at families like this one - -something around 10% of these are going to turn out to be viable drug targets. We know that many of these kinases, play a central role in major diseases. And a common feature of all of these enzymes is the kinase domain which includes that ATP binding pocket. And what that means is that this entire family is amenable to the sort of parallel chemogenomic discovery approach that we use here at Vertex. And I'm going to show you some ways in which the structural insights that we gain from using that approach allow us to develop very specific kinase inhibitors.

So, this is a rather a complicated slide. And what I want to do is to make a couple of points. All of these things in purple are kinases. And I'll use that color scheme throughout the talk. The things in orange are what are called "transcription factors". These are the things that receive signals from kinases. And then bind from DNA -- telling the cell to make certain genes which result in changes in cellular function. And, these other molecules marked in blue are molecules that are involved in kinase function - -- sorry, in cell signaling --but are not necessarily themselves kinases. The take-aways from this slide are: 1) there's a lot of cross talk between these pathways. Most of what I'm about to show you, going forward, is a little bit of an over-simplification. And, in fact, kinase biology is rather complex. The second is, that most of these kinases operate in Cascade. So, here it's a receptor kinase, will activate another kinase, which will activate another kinase, which will activate another kinase. If it's not sort of linear process as shown on this slide -- but it is really more of a chain reaction. And, if you remember -- sort of from your early Physics classes -- where you're modeling a nuclear chain reaction. And one marble hits two marbles which hit four marbles, which hit eight marbles -- and that kind of amplification. That's exactly what happens when kinases are involved. So, a single molecule like insulin might bind to its insulin receptor. And that single insulin molecule -- that signal from the single insulin molecule, gets amplified many

times before it actually gets down here to the nucleus and tells itself to start making genes that are involved in glucous metabolism. So, I'm going to show you a little bit of a movie now in terms of the way that kinases work. Again the things in purple are the kinases. I have here a growth factor. This is the epidermal growth factor. This is the epidermal growth factor receptor. And I'm going to turn on the movie and show you what happens. So the epidermal growth factor -- will bind to its cognate receptor. That will activate the receptor, which will in fact phosphoralate itself. That phosphoralation process activates another molecule called Ras. And when Ras gets activated through a complex pattern -- it recruits Raf, the nucleous, and Raf gets phosphoralated. Phosphoralated Raf is now active. It goes and activates MEK. When MEK gets activated, it activates ERK. When ERK gets activated, it goes and activates ELK by sticking phosphate groups on it. And when ELK -- this transcription factor --gets activated -- it binds to the DNA - -- activating the genes. So, normally all of these molecules are sitting in the cell -- inactive. And they only become activated when this growth factor binds to its receptor. And that kinase Cascade is the way the EGR tells the cell to start growing and dividing.

So, most of what you've heard about kinase and kinase inhibitors are in Cancer. And I think that's going to be true for the next few years. Most of the kinase inhibitors that you see -- going into the clinic will be developed in cancer. And that's because kinases play key role in processes central to tumor growth. I showed you a little bit about a growth factor signalling cascade. And, as you know, there are a number of drugs out there now which target the EGF receptor kinase. Kinases, as I'll show you, are involved in cell cycle regulation, or the process of cell division -- where cells double their DNA -- and split to two daughter cells. Kinases are also key to the process of Angiogenesis. And in cancer, we are already noticing a number of successful drugs -- which go after kinases. Herceptin goes after a growth factor receptor called Her-2. That's, in fact, a kinase. Gleevec targets the ABL -- ABL kinase and CML and the C-KIT receptors amd GI stromal tumors was developed by our partner, Novartis. And is, in fact, the first small molecule kinase inhibitor. Now, there are a number of other ones following -- Iressa and OSI-774. Small molecules Im-clone,C25, an antibody. All of which target another kinase -- the one that I showed you earlier -- the epidermal growth factor receptor.

This brings up the concept of molecularly targeted therapy in cancer. I think that this is really a new way to think about the treatment of cancer. Historically, cancer therapy has been relatively non-specific. It's aimed at any sort of any cells that are dividing or are around. It's comprised mostly of surgery, radiation, and chemotherapy. Or, if you're a patient -- slash, burn and poison. Thse are just non-specific ways to kill cells and to kill any cell that's sitting there dividing. In terms of molecularly targeted therapy -- we know that many proteins are subject to genetic alteration in cancer. And so by targeting these proteins that we know cancers alter to confer a growth advantage -- we know -- we think that we can target tumors more successfully in terms of being specific for tumor cells, and having fewer side effects on normal cells. So now with the examples of Herceptin, Gleevec, IRessa/OSI-774 -- we know that hitting these targets will shrink tumors, in many cases, more effectively than we can do with radiation and chemotherapy. And we can do that with fewer side effects than we see with conventional chemotherapy or radiation therapy. And I think that these drugs are really just the tip of the iceberg. You know, we're talking about targeting just a few kinases. There are lots and lots of kinases that fit this profile of being subject to genetic alteration in cancer. And I think, over the next few years, you are going to see lots and lots of kinase inhibitors, aimed at kinases other than these ones here -- enter the clinic, and start working in cancer patients. And so, I think most people have been really quite impressed with the effects of Gleevec.

And I think that most people are starting to think like Richard Klausner does -- that this is the way that we're going to target cancer in the future -- not with non-specific methods of killing

cancer cells. But but by using drugs which are more specific for alterations that we find in the cancer cells themselves.

So, let me show a little bit about how this works. And what's going on in CML. CML involves sort of a gene re-arrangement. So a piece of Chromosome 9, gets moves to Chromosome 22 -- and a piece of Chromosome 22 gets moved to Chromosome 9. So you have this unusual Chromosome called "the Philadelphia Chromosome". Because that's a city in which it was discovered. Now, it turns out that the breakpoint on Chromosome 9 -- or the place where Chromosome 9 always brings -- is in the middle of the gene that includes the ABL gene -- which-- abl is a tryosine kinase. Now, normally this tyrosine kinase is off. And it needs to be activated by other kinases. But, when it is translaticated to Chromosome 9 -- you make an infusion protein -- this BCR abl protein. And the BCR abl protein is an activated form of the gene. So, sticking that piece of BCR gene in front of the abl gene - -makes it an activated kinase. So, it has natural kinase activity, and no longer needs to be activated. , And, when cell-- get this. When blood cells get that molecular event happening, they turn from what you see here - -- a picture of normal blood into what is, in fact CML. And the major difference between these two slides is that there is only a couple of cells with purple centers here. And what's abnormal in the CML patients -- is that you get lots and lots more of these normal cells. So white counts, normally in the range of about 10,000 -- will go to the range of 100,000 or 150,000. So, what's happening to CML is that Novartis developed Gleevec or STI571 -- which is a specific inhibitor of the abl tyrosine kinase. And they gave it to patients with CML. And I think they really saw the most dramatic results we've seen in cancer chemotherapy for a really long time. 98% of the patients treated with this drug - -- in their initial Phase II study, had their blood picture go from something like this -- to something completely normal. In something like half of the patients had most, if not all of the cells-- with this abnormal chromosome completely disappear from the blood and the bone marrow - suggesting that you're killing all of the cancer cells -- by specifically targeting the abl kinase.

Now, in many ways, CML is a unique disease. 100% of people with CML have this molecular alternation. That's not going to be quite as true in many of the solid tumors. There is not a singular molecular alteration. But it does, I think, point out the potential of targeting specific genetic abnormalities in cancer. And I think that for breast cancer, which is a much more molecularly heterogeneous disease -- there may be three or four or five kinase inhibitors that are going to be required, in order to fully combat the full spectrum of patients with breast cancer -- or lung cancer, or any of the more major solid tumors with heterogeneous disease. Now, there are multiple kinase pathways that represent potential points of attack. We've talked a little bit about growth factor pathways, in terms of the Ras pathway. There's another one called the PI3 Kinase pathway. As I mentioned before, there are kinases that are involved in cell cycle regulation. Or the process of cell division. And I'll talk to you a little bit more about that. There are also kinases involved in the ability -- the metastatic capability of cancer cells. Or the ability of cancer cells to grow and move from their original point, and spread to other points of the body. And, then, another thing that I'll spend a little more time on is -- the process of Angiogenesis, which kinases play a critical role in the development of new blood vessels required for tumor growth.

So this is just the picture of the Cell Cycle. Cells are sitting here, normally in a resting stage. They will then go through this S-phase, where the DNA divides. They will then go into G2-M where the broader cells will pull apart. As you can see here, all of these things in purple are kinases that are critical to this process of cell division. Many of these kinases are involved with the same sort of either gene amplification as one sees for EGFR. Or rearrangement like one might see with BCR-abl. And all of these things in purple, represent potential points of attack for targeting cancer cells.

And so, just to show you some of the progress we've made -- we've developed inhibitors of one of those cell cycle kinases. What you see here on the left is the normal process of cell division. Normally the two daughters that you'll form - -- these tubulin sort of strings that hold onto the DNA. And those strings will pull apart as you form two daughter cells. And when you target one of the kinases involved in that process -- with one of our kinase inhibitors -- at least in the cell, you see that you completely disrupt that normal process of cell division. And if you treat cells for 24 to 48 hours -- at least in a test tube of one of those types of compounds - -you completely kill the cells. And so, we think that's a very exciting results. And are moving those sorts of compounds now into animal studies.

As I mentioned before, I'll talk a little bit to you about the process of Angiogenesis, or new blood vessel formation. This is a hot topic out there. And lots of people are looking for ways to target Angiogenesis. What I'm top point out to you is how that process works. So, normally, there's these cells that are called Endothelial cells, which form the inner lining of blood vessels.. Those will come together to form a tube. But that tube, in itself, it not enough to function as a blood vessel. And it has to be the recruitment of all of these other additional cells -- smooth muscle cells. Some Stromo cells and other things around that endothelial tube, which allow the formulation of a fully-formed blood vessel. And these blood vessels are required to deliver blood to the tumor as it grows. So, what tumors do is they send out signals, telling blood vessel cells to go through this process - -and make no blood vessels to feed the tumor growth.

Now there are multiple kinases that are involved in this process. One that you might have heard about are the VEGF receptors. There's a couple of VEGF receptors out there in the clinic. But what you may not know is that there are least five or six other kinases that are involved in the process of Angiogensis - -- at least as shown from multiple kinase knockouts. And in building on that biology capability that Dr. Boger mentioned in his talk - John Thomson will tell you a little bit about some additional biology work that we've done here at Vertex. But to validate additional, proprietary kinase targets that are involved in this process of Angiogenesis. And we think that by targeting some of these kinases as well as the kinase that we've identified through our own biology efforts -- we are going to have a number of drugs that will be effective Anti-angiogenic agents in the treatment of cancer.

So, moving now from Cancer to some of the other indications where one might be able to use a kinase inhibitor -- I'll talk to you a little bit about Restenosis. As you know, coronary artery disease is probably the most common cause of morbidity and mortality -- at least in developed countries. And in the United States there are some 700,000 procedures each year, to try and improve blood flow from blocked coronoary vessels. Now, even after those procedures, what will happen is that you get Restenosis. So, normally, the edge of this blood vessel is out here. And after that vessel has been re-opened with a ballon catheter -- using angioplasty, those vessels will clog up again. And so, instead of having this big huge looming for blood flow, or this large tube through which blood can flow -- what happens is, is that it narrows and constricts -- so that you only get that tiny little hole there. And if you take a look at what's all this stuff that's clogging up the blood vessel -- it's these things. It's smooth muscle cells. So, what causes Restenosis is migration of smooth muscles into the blood vessel, where they then divide and grow and clog up the blood vessel. Now, in about 20% to 40% of patients, this Restenosis will be significant enough, that the patients have to be re-vascularized. And, each year in the United States alone, we spend about $2 billion dollars trying to fix this problem of re-opening blood vessels that got clogged, even after the first time they were angioplastied. So what people are trying to do is --is there a way to prevent the migration and growth of these smooth muscle cells? And one way to do that is using kinases.

So, one of the factors that causes growth and proliferation of these smooth muscle cells is PDGF. There are a couple of others. And PDGF signals in a very similar way that EGF

signals, as I showed you on that first slide -- so that you can target any one of these receptors here, or any one of these purple things here -- these kinases - -- to try and prevent smooth muscle growth and proliferation. And, if you do that, you sort of get the results that we see here. So, here are some smooth muscle cells. And when you treat them with PDGF you find they start to migrate in this assay. And when you treat those smooth muscle cells with increasing doses of a Vertex Kinase Inhibitor -- that migrration is blocked. Similarly, if you treat those cells with PDGF, not only will they start to move, they'll also start to divide. And, again, by treating these cells with a Vertex kinase inhibitor -- you can completely block the growth and proliferation of smooth muscle cells. And so now we're starting to advance compounds like this one in animal models of Restenosis.

Now, here's another example of types of kinase signaling. This is sort of death signalling. So the process of cell death, also known as apoptosis - and there are a number of signals that came come from outside of the cell-- which are then transmitted through a kinase pathway, which tell the cells -- now, will you please start making genes that tell the cell to die. So, sometimes these death signals are not necessarily ones that you want to have. So, in the case of stroke -- which is depicted by this MRI scan -- Or in the case of Myocardial infarction or heart attack -- which is depicted by this EKG scan -- the death signal is lack of oxygen. And that lack of oxygen signal gets transmitted through kinases, and the cells wind up dying. So, if you could block some of these death pathway kinases, you might be able to block the process of cell death.

And so, we have some inhibitors of those cell death kinases. Whereas you can see from this slide, when the cells were given a death -- increasing amounts of a death signal -- you get increasing amounts of cell death. And that process is almost completely blocked when a Vertex kinase inhibitor is present in those cells. And so, these are the types of compounds we're advancing into animal models of myocardial infarction or heart attack -- and to animal models stroke. So, that's just a few examples. John Thomson is going to show you some examples of kinase inhibitors in diabetes. But I think we're making lots of progress across lots of therapeutic areas with our kinase inhibitors. And, as has been mentioned before -- we're really on track to name two development candidates by the end of this year for the treatment of a couple of diseases.

Now, I want to talk to you a little bit about the way in which the specificity problem can be addressed using Vertex's technology platform. As I mentioned to you earlier, the field of Kinase inhibitor development was really held up -- as recently as a decade ago. Because people didn't think it was possible to make a specific inhibitor. And if you couldn't make a specific inhibitor, you were going to get too much toxicity.

So, I want to show you the way in which we, at Vertex, use some of the tools that you heard about on the lab tour, in order to help us design a drug. And this is just some data that relates to VX-745, which is a compound that you heard about from John Alam. Now, what we'll do is we'll go to these proprietary databases. And some of you probably saw in the lab tour this morning. And you'll say -- gee there are three kinases that look like p38 -- but they're also members of the MAP kinase family. They are the JNK kinase and the erk kinase. So, ifwe want to make a specific inhibitor, how do we do that? So, the guys at Vertex who make these tools recognize that one day, doctors might be looking at this. So we need to make it so they understand it. So we make the important amino acids. We'll color them in green. So that the doctors will know which ones to go after. And if you take a look at say, Amino Acid 110 - -that's different across all three of those kinases. So, if you can make a drug which targets Amino Acid 110 or targets this GMp38 -- it's probably going to be specific for p38 -- relative to some of these other kinases -- because that gene is not present in these other twos. But if you made it, say for a -- if a targeted amino acid went away to 109

that wouldn't help you because those amino acids are identical, across all three kinases. So, we'll run some screens, and we little get some lead compounds, and then, we'll create structures od those lead compounds bound to the enzyme.

And this is a picture of that. So, this is the enzyme back here. And this is one of our early leads depicted here. There are some important pieces of information that we can get from that. One is -- it's this back half of the molecule depicted here, which is really stuck deep in that ATP pocket. So, if what we want to do is to make the drug more specific, and more potent -- that's the part of the molecule that we need to change. And we can get information from pictures like this one, telling us exactly what kind of changes we need to make. The second thing we need to do is -- or we can learn is -- this part of the molecule is really out here in the open air. So if we need to do things like alter the metabolism of the drug, or add groups which make it more bio-available, for example, we can put some of those chemical groups on this part of the molecule, and add those qualities -- without really affecting the potentcy or specificity of the drug. And what you'll notice here as we'd mentioned earlier - this early lead didn't really target amino acid 110. It did attack Number 109. But that wasn't one of the ones that really got us to specificity. So what we did was play with that molecule a bit, and made it so that it formed a hydrogen bond -- not only with amino acid 109,but also with 110. And, if what we thought about -- if what our database tools were telling us was correct -- that kind of a molecule should be relatively specific for p38 MAP kinase, relative to some of the other MAP kinases. And that was exactly what we foundwhen we tested VX-745, and compared and showed it is a very, very specific sort of about a ten-amino over p38 alpha. But, really doesn't touch R-2, or JNK 1 or JNK 2. Despite the fact that these are very, very highly related kinases. So that's some matter of fact the way in which we use our tools to make kinase inhibitors that are specific for individual kinases.

So,what is it that the discovery part brings? And how will Vertex and Novartis be successful in the Kinase world? You know, what Vertex brings to the table is a leading edge discovery technology found in structure-based design in the chemogenomic approach. So the people who, I think, to a large extent, had been leaders in kinase drug discovery -- took a look at the way we go out kinase drug discovery. Took a look at the way we go about discovering drugs as kinase inhibitors, and said -- you know, that's really a pretty good way to do this. And we're going to have you guys start working on all the kinase inhibitors that we're going to be developing over the next decade. Both companies, I think, bring to the table a strong record in kinase drug discovery. And, what we're very excited about is working with a committed partner, with proven expertise in assets, which are going to compliment our discovery technology. They were the team that brought the first small kinase inhibitor to market. They know how to develop these drugs. They're learning how to market them. They had critical development and marketing insfrastructure for the development of these drugs. And, in addition to all of that -- they can help us on the biology side, as they have made large investments in proteomics and target validation. And where that is applicable to kinases, that's information that they're sharing with us, so that we can apply these tools to the development of kinase inhibitors.

One final point that I'd like to make is that there's still a lot of opportunity out there. So, if this pie were to represent the 50 kinases that are potential, viable drug targets -- over the next decade or so -- people are really playing in a very small space right now. There's really only five or six kinases that are targeted by all of the drugs out there. One of them is our p38 MAP kinase inhibitor. You know, there are other companies with kinase inhibitors in the clinics -- some of which I mentioned. But really, most of this pie -- most of this space is up for grabs. And, it's this part of the space, that the Vertex/Novartis collaboration is aimed at capturing. And I think that when I show you this slide again -- hopefully in 2010 -- you'll

see the Vertex/Novartis chunk of this pie. And we'll have captured a fairly significant portion of that space.

So what I hope I've shared with you today is that kinases are excellent targets. They're involved in many human diseases. And those are large human diseases, and large pharmaceutical markets. But the next decade I think we'll see the introduction of multiple compounds that are kinase inhibitors. And many of these compounds are going to have blockbuster potential. And the Vertex/Novartis collaboration is really positioned to become a dominant player in this space. And what we hope that means, is that new drugs for patients. And revenues,profits, and shareholder value -- for all of you who are attending here today. Thanks so much for your time. And I'll be happy to take any questions.

END OF PRESENTATION

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Investors and security holders are advised to read the joint proxy statement/prospectus regarding the proposed merger when it becomes available, because it will contain important information. Such joint proxy statement/prospectus will be filed with the Securities and Exchange Commission by Vertex and Aurora. Investors and security holders may obtain a free copy of the joint proxy statement/prospectus (when available) and other documents filed by Vertex and Aurora at the Securities and Exchange Commission's web site at www.sec.gov. The joint proxy statement/prospectus and such other documents may also be obtained from Vertex by directing such request to Vertex Pharmaceuticals, 130 Waverly Street, Cambridge, MA 02139, Attn: Investor Relations, tel: (617) 577-6000; e-mail: InvestorInfo@vpharm.com. The joint proxy statement/prospectus and such other documents may also be obtained from Aurora by directing such request to Aurora Biosciences, 11010 Torreyana Road, San Diego, CA 92121, Attn: Investor Relations, tel: 858-404-6600; e-mail: ir@aurorabio.com.

Vertex and Aurora and their respective directors, executive officers and certain members of management and employees may be soliciting proxies from Vertex and Aurora stockholders in favor of the adoption of the merger agreement and the transactions associated with the merger. A description of any interests that Vertex and Aurora directors and executive officers have in the merger will be available in the Joint Proxy Statement/Prospectus.

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THE FOLLOWING IS THE SCRIPT OF A PRESENTATION PRESENTED TO ANALYSTS, INVESTORS AND OTHERS ON MAY 31, 2001 AND POSTED ON VERTEX'S WEBSITE ON JUNE 1, 2001.

VERTEX INVESTOR DAY

"CHEMOGENMICS: ACCELERATING VERTEX RESEARCH PRODUCTIVITY"

JOHN THOMSON

I'm John Thomson. I've just celebrated my 12th anniversary at Vertex. And that makes me (laughter) the longest serving member, except for Joshua. And, for that whole time, I get enormous pride and a feeling of privilege when I talk about the research engine at Vertex. I see other people laugh, talking about technologies and strategies for failing fast. Yet, we have a research organization that is more pre-occupied with succeeding fast. And what I want to do today is discuss with you some brief sampling of science. And some of the energy from Vertex. When I get into this, I get a bit energetic myself, so I've got to try to corral my thinking by way of a structure of this talk. Three basic areas.

I'd like to talk about the pipeline, and, as Lynne pointed out earlier -- we are hoping to announce five new drug candidates this year. A little bit on the Kinase Family as a prototype for gene families. Because it has important information regarding how we're on track to deliver to Novartis. But, as well, we're learning important lessons along the way, that will bode as well for the next gene families. And then, new directions. Adding new tools, and how we're going to move into new gene families.

So, to being with the Pipeline. It's impossible to guarantee any specific drug candidate in a particular year for many of the programs. So this is a list, if you like, of the current crop that we think is most likely to generate the five or more new drug candidates that we feel are likely to emerge in 2001. As you can see, there are multiple possibilities from our Novartis collaboration -- after only one year. They're in major indications. We also have a second family-oriented collaboration on Caspases that might yield one or two Visit candidates or drug development candidates during the course of this year. And then our crop, or class of individual targets. Mostly coming from Vertex 1.0 in the first decade. But, not all of them. Bacterial Gyrase are a very new program. Doing particularly well in terms of progress. And, currently unpartnered. All of the other programs are being partnered. So, this is where we expect those five or so to come from.

I'd like to now quickly sort of hop over some of the most likely candidates, and not go through that laundry list rigorously. And as we just heard from Rob Mashal -- Kinases are important in cell communication everywhere. They permeate all sorts of biological processes, and they offer opportunities for medical intervention and innumerable important disease areas and medical markets. This is just a cover story from one of the countless reviews on Kinases in Cellular Communication.

And to summarize the progress in the first year of our Kinase Family-oriented, or chemogenomic approach to kinase. We're doing very well on the drug discovery side of things. Developing component, drug-like compounds, in multiple models that, as you can see here, are major market potential, and major indications. So we feel that we're doing well towards delivering to Novartis. And we're also doing very well on the biology. Stuff that is driving the target validation to make sure that we're using all of this sort of drug design stuff on targets. As Joshua alluded to earlier -- we although we haven't touted this in the past, we are also proud of our biology at Vertex.

Now, to go into some of the specific examples. And, this elaborates a little more a set of data that Rob Mashal started to discuss. We have a Kinase target here that we believe is going to be important to cancer. And, we are investigating the Vertex Kinase Inhibitors in various cancer cell lines in vitro. And then, we're looking at a couple of biological markers. Histone phosphorylation, which really indicates condensation of chromosomes prior to cell separation. And then, tubulin assembly. The infrastructure of the cell that then separates to pull the two daughter cells apart. And we believe that these are two good representatives of cell division. And, as we know, in cancer cells -- cell division is what we want to stop. It's uncontrolled cell growth and division. So, here we see, uncontrolled cells in vitro. Here we see effective Vertex Kinase inhibitor in vitro. And you can see that it is almost completely inhibiting the chromosome condensation. And it is really perturbing the tubulin assembly. An effect that is very reminiscent of what's seen in Taxol. So, we see here, a Taxol-like effect. Its similar potency, and a non-Taxol-like affect. But, both effects-- being very relevant to halting the untethered proliferation of these cancer cells. So, in effect, these block mitosis - -they lead to cell death. And we've shown that basically the same pattern emerges in multiple cell cancer cell lines. The extra panel down here is just a different sort of photograph which shows that these cells are actually dying at last.

This is an example of coming about the science in a slightly different manner. Working with multiple kinases at the same times -- sometimes you find that you have a drug. And the target that you think is pretty interesting, and you'd really like to explore the drug opportunity, but you're not too sure of the target. So, we set out with our biology infrastructure -- to investigate this kinase with a knock-out experiment. Now, in a normal mouse, during early embryogenesis, the yolk sac of the mass embryo -- you can see regular vascularization down here in a different sort of staining. And this is reminiscent of angiogenesis. In Rob's talk. And prior to that you have all heard of the importance of angiogenesis. So, we thought that this particular target that we were going to develop a drug for, could be important to angiogenesis. So, we knocked it out. And well, the mice were early stage embryonic lethal. Now, some people would walk away and say, OK, that's a bad target to go and inhibit. But no, careful analysis of this shows that probably the reason that these mice weren't surviving, was because they're lacking this mechanism for angiogenesis. And it's not surprising that building blood vessels in an early-stage embryo is an important thing. But, in some indications, angiogenesis, or blood vessel formation is not such a good thing. Cancer, of course -- where want to limit the blood supply to the cancerous tissue. Or diabetic retinopathy --two of the examples where we might want to control angiogenesis. So, we think that this series of experiments, starting from a chemical interest and starting point, led to an exploration of an interesting knock-out, and established a novel kinase target in a validated kinase pathway associated with angiogenesis. You can see that there is patent protection being sought for this.

And, I'm sorry, I can't share with you the identity of that particular kinase.

This is another example. This is one that I can tell you what the target is. It's a Diabetic Kinase. We believe it is one of the best, most favored diabetes targets in any class. And we recently solved its crystal structure. Now, traditionally, we've been very good at solving crystal structures.

...this whole process of -- really this picture describes structure-based drug design. Made crystals. Self-structured. Take that information to build molecules - do it again, and then make the molecules better. So, we've always done the central part well, and given us a strategic advantage - but it's always been a tool to us. Other companies have been founded on the principle that this is the whole company. We're miniaturizing, automating and industrializing this process. That's why the companies are doing these pure-plays. But that's not where all the magic is in this process. The magic is over here where we're making drugs. That's where the value creation occurs. So, all of this -- it's complicated stuff to get right. And these are very sophisticated tools to develop. But they are tools. As I said, this is where the drugs emerge, and the valuation creation occurs. So, we are constantly refining good tools on this occasion. We accelerated the right to determine the process. The crystal deflection quality crystals, solve the structure quickly -- and then went on to multiple inhibitor structures that guided our drug design. And what are these magical part of the process? Well, here's some biological experiment from some of the inhibitors from that.

And this is in a knock-out of a knock-out mouse that is a well-respected diabetic mouse model. And what we do here -- it's an acute model. We introduced a slug of glucose to the mouse. Then, looked at the effect of Vertex compound dosed orally. And if we look at the heterogenous mouse, which really behaves normally, after the glucose does, it really handles the disposal very well. But the knock-out mouse, where the phenotype is expressed, doesn't dispose of the glucose well. It rises rapidly, and then maintains. When that mouse is treated only with the Vertex compound - -it disposes of the glucose in a much more healthy fashion. So, these results -- or these effects showed those responsiveness. So, we believed that they're following the right mechanism relating to our drugs. And the magnitude of this effect is quite significant. This can be compared to a horrible model -- not an exact replica of this model -- but a comparable model in which traglitazone was tested. And in that model, the effects were very similar. In fact, if you tried to normalize between comparison of apples and oranges, one would argue that this is, in fact, a bigger effect -- because it is the acute model. So, we believe this is the reflection of the magic in a drug-designed part of the process.

So, to change gears a little bit, and move to Caspases. Caspases have been implicated in two fundamentally important cellular pathways. Inflammation, and cell death. Programmed cell death or apoptosis. Both of these are extraordinarily complicated. But they can be roughly divided into inflammation - dominated if you will by Caspase I or Interlukin I converting enzyme, and a few others. But mostly Caspase I. And then, some other Caspases. But, generally programmed caspases contribute in sort of an integrated fashion, a cascade fashion to this process of programmed intended cell death. And it is this intended or programmed cell death avenue that we are now focusing on with our research team in the U.K.

And we've identified many human diseases - -important human diseases that have apoptosis underlying their mechanisms. Now, that is not to say that all of these are equally reliant on Caspases for this programmed cell death. Probably those that are most reliant on caspases, are those indications in here that are triggered by some sort of obvious stress scenario. The ones that we think are probably most relevant to caspase inhibition are here in red. And the starting points for our therapeutic interest. To summarize we have major market opportunities in myocardial and neurological conditions -- both big markets. We've done the regular Vertex style chemistry. Lots of scaffolds. Lots of patents around chemistry. And lots of crystal structures, and the structure-based drug design process behind it. And now, this has given us multiple chemical processes that we're exploring in many disease models. Therapeutic models to show the full potential of these processes of molecules. There's where we're up to in the Caspase program. And we believe that we have some possibilities coming to fruition. Now, this is the analysis of one of the Caspase program. And we believe that we have possibilities coming to fruition.

Now, this is the analysis of one of the Caspase inhibitors in a model in which what we do is to try to trigger an avalanche of the Caspase cascade in mice. We put an antibody signal here. This is a very heavy-handed sort of a model. But it really says, systemically -- OK, Caspase, go crazy and just avalanche. And what happens is that most of these mice die within 7 days, unless protected. And you can see by the start's response curve, that in an IV bolus, our Vertex Caspase inhibitor protects in that same period of time - -almost to 90 percent. Now, this shows a number of things. Firstly, our compounds not only protect cells. We're way beyond that. We can show you other data showing that these compounds protect cells. But they protect animals in a profoundly important way in a heavy-handed model of organ failure that has relevance to some very serious human situations. Sepsis. Liver distress, and other things.

Off the Caspases -- and onto the Bacterial Gyrases. As I said, one of our newer programs. And the underpinnings of this are that antibiotics is a huge market. But we're all concerned that the medical profession and industry is going to be experiencing worsening anti-biotic resistance. There is great potential here for new classes of antibiotics. The gyrase enzyme is a complicated enzyme. And it's a well-validated enzyme; but targeting in a different way than we are doing. Roughly $4 billion dollars annually is made by Gyrase inhibitors. But they're targeting the A-sub unit. For complicated technical reasons, we believe that targeting the B-sub units shown here is a better way to avoid resistance, and definitely a better way to get into new classes of antibiotics. So, that's our general strategy. It's a new program, but already we're experiencing very promising results.

This is just a token result showing a typical anti-proliferation, antibacterial, I would say -- using E.Coli. When, e.coli are allowed to divide and grow they stay well-separated and the division process goes on. When that is inhibited, by a mechanism that interferes with DNA replication they stay - -they fail to stay free-swimming and happy. But they form these long filaments and stop dividing. Eventually, those filaments degrade and die. We see that with a non-potent inhibitor of Gyrase B -- we see the sort of filamentation and cell death. We see the same sort of effect with one of our Vertex compounds, or a number of our Vertex compounds. We're doing well on the structural biology of this particular program with, I think, upwards of 20 structures solved already. And this is driving novel, patentable scaffolds. We have multiple compounds already. Equivalent potency to Novobiocin. And positive results in E.Coli, and some clinical S. Aureau. Now the challenge is to make it evenly broad across all clinically-relevant strains. Cell permeant and cell active -- in all clinical strains. So - we believe that we're making remarkable progress in that program.

I think this one takes it to another level yet again. HCV is a medical problem of enormous importance. It causes debilitating liver disease in three to four million Americans. And huge numbers world-wide. Current therapies are improving all the time. But they still are not as effective as they might be. Not clearly virus permanently in more than 50 percent or 60 percent of the population. And, having significant toxic side effects. And these reasons are partially related to the fact that both of the therapies -- the Ribaviron and the Interferon, target us -- the host of the viral infection. Not the virus itself. So they give rise to side effects, and uncertainty. Undesirable effects, and lack of efficacy. We believe that there is a major opportunity by targeting the virus directly. The world is acknowledging this. We've seen the power of this approach.

What happened when we started targeting the HIV Protease. It revolutionized the care in therapy for HIV sufferers. And we believe the same profound effect in medical treatment would be experienced by targeting this virus directly. But it is a very great challenge. Because unlike HIV Protease, it has a very flat surface area in the active side, where we have to do the business of our design. As our head of crystallography likened this to -- it's like trying to climb a rock-face. If there are footholds, fine. But if there are no footholds,you have a tough time. That's sort of what this problem is like. But we believe, and many of the competitors are experiencing a tough time, and announcing the departure from this particular approach. We feel very good about our program. And again, we've solved many, many structures. We have multiple proprietary lead compound classes, and they look like drugs at this point in time. Good cellular potency, followed by oral bioavailability. And favorable liver and plasma PK. Now, this is important because it's an infection that is centralized in the liver. But it's distributed also in compartments throughout the body. So, it's a unique PK targeting kind of requirement that we need here. Our compounds show very favorable PK profiles. Preclinical toxicology has begun. And, thus far, they look very promising.

Along the way we did some pretty clever biology here as well. This was a program that we got into, knowing that there was no in vitro, viral replication assay. And along the way, we built a surrogate for that. This shows a surrogate viral replication assay developed at Vertex - -where we're measuring a titration of a Vertex compound, showing that it dose-responsive. And that it is potent in these cells. That it is mechanism-based on the protease target. Now, taking this sort of cellular potency, and putting it together with the promising PK data - we could have multiple chemical classes. We can actually design several options going into the clinic. We're intending to do that so that we may not only achieve one drug development candidate in the near future -- but two, or even more -- to enable us to adapt in the clinic. To walk the real requirement that -- PK requirements -- that drug delivery are going to represent. So,that's the pipeline. Now moving onto back to the sort of general technology. The concept of Vertex 1.0 and 2.0.

This was essentially the first decade of history at Vertex. Vertex 1.0. We focused on one target at a time. We took a little while to get started --- from an inception of 1989. Only because this is a clinical analysis -- you know, entry into the clinic. But, thereafter, we had a nice ramp-up of targets at about the rate of one per year in our first decade. We're very proud of this track record. All of these compounds still have real potential as important drugs. We're very proud of this record. And we believe it is already unprecedented in the pharmaceutical industry. But, with the discovery of the -- the unearthing of the genomic information, we have enormous new opportunities. Roughly, all of the drugs in all of the time, by all of the pharmaceutical companies - -came from about 500 targets. The new genomic information is unearthing thousands of perhaps attractive targets. We have to have a way to mine that effectively. We believe we've already developed it with our efficiency of our research discovery engine. It relates to certain proprietary tools. Certain trade secrets -- just the way we do things. And relating to the structural biology. Computational chemistry and bioinformatics, and then an integrated platform of science. And it's already resulted in better -- we think, better drug candidates faster. And we believe that it is particularly suitable for looking at large numbers of related proteins in parallel. And that's concept 2.0. And we define it as our key genomic strategy. I believe we were the first to use the term. It's the first place that I've heard the term.

In concept, it is all possible drugs against all possible drug targets. And that's to some degree -- is an impossible, hypothetical goal. It's a concept. But, in practice, it's a highly efficient, highly parallel, ambitious drug design on hundreds of targets on one set or family of gene targets at once. Now, you can understand that analyzing targets when they're related, gives you a certain degree of scientific efficiency. I think that can be understood by everyone. Even without knowing what the efficiencies are. But it also relates in a certain synergies related to the establishment of intellectual property regarding products. And I don't mean protein products. Target products. I mean drug products.

The real value creation stuff in this enterprise. And you've seen that in our Version 1.0 -- we had a very healthy rate of new drug candidate production. Right now, we're in this phase. Last year, five new drug candidates. This year, hoping for -- expecting five or more. And we're looking very soon towards 8 to 12. So, I want to walk through with you now what we've learned from the Novartis collaboration.

It's a different model for a collaboration. And one of the things that's different is that it is focusing on eight NCE's-- this is why it's attracted to Novartis in my opinion. They get eight drug candidates. Pretty good stuff. Vertex certainly gets some financial security and a lot of attractive stuff on the financial side -- but what's really made me pleased about this deal -- is all these other drug targets -- and they belong to us. Once we're doing with the eight that are going to be developed by Novartis and Vertex - -then, we're into the Vertex only drug targets and drug possibilities.

So, in the first year of that Novartis collaboration, there are a few things happening. We're transforming the research organization. We're mapping the kinase universe-- learning all that kinases and developing new tools. And I believe that we're establishing strong chemistry, and intellectual property foundations. And I'd like to quickly touch on each of those areas.

We have hired aggressively. People have said from the beginning that it was going to be one of our biggest challenges. No problem. We have done very well in this regard. We're on target to meet the 160+ or so scientists needed in 2001. But, of course, this requires new organizational models as well. Not just sort of bringing in lots of people. And we've introduced successful new organizational models. And all, without an interruption of the process. And without any dilution of the flavor or the culture of innovation at Vertex.

We're very proud of Vertex 1.0. And how much we work on the balance of these different core competencies or basic technologies. And this has been evolving platform for Vertex in that first decade. It still remains at the heart of what we're doing in Vertex 2.0. But at this scale, working on many targets at once -- and at this scale -- there are the synergies that can be captured by arranging around teams. Teams that are supposed to be focusing on a specific part of the overall process to use the knowledge-base of the integrated platform -- what works well for one target. But to do things in a way that organizationally are more specific to many targets at once. So it is this team of different parts of the overall process -- around our core technology that is sort of different organizational model that we are finding more appropriate for chemogenomics. Also on the science side. We knew very early that we need to think about the problem of kinase inhibitor designed a little differently. You've got to define kinase chemical space. Normally people think about kinase's pathways, file the genetic trees, or sequence analysis. These are all important. But they are also important to introduce concepts of structure, and what's going on with what you're trying to do -- the drug design path. And it's knitting that altogether with sometimes quantitative analyses of the active sites of kinases that's important. And when you do that, you use various informatics tools to build rigorous scientific representations of kinase space. Multidimensional matrices as a various physical parameters of the kinase active sites.

And when we do this, we build up a kinase universe. And what happens with this is that you find related kinases culture, generally -- often. And, the kinases shown in red here, in this complete universe- the medically important ones --sometimes end up in different clusters and roll away from one another. And sometimes not the way you've worked before in the green -- these green targets are where we've worked previously. Perhaps medically important, perhaps not. And what we need is a way to sort of get to these red zones -- the therapeutically validated targets. This is easy to represent by something of a cartoon. Sort of a galaxy cartoon. And sort of two-dimensional cartoon that you can see -- if we're starting here, and we want to get, however, here to this kinase of interest - -there are connectivities between this clusters in this kinase universe that might be difficult to traverse if you don't know anything about these kinases in-between. But if you've got stepping stones to carry you across there, you are making short jumps to get across the kinase universe. And this enables you to reuse scaffolds and chemical classes to navigate the Kinase universe.

This is a static picture what we have here -- a cartoon. But, in reality this picture is constantly evolving. When every new piece of information that we put into it, it evolves. And we've built complicated tools -- sophisticated tools in our bioinformatics group to sort of describe this and keep track of it. And I think this will start on its own. This is one of those tools that's showing sort of a representation of Kinase space. The Vertex logo in this case is really hung by its - or it's ap-crop. Where we've worked on Kinase. And one, we've invested time, and it's just a period. And, as we learn about all that kinase, and learn more about the other kinases, we build the knowledge base. We expand the Kinase -- the non-Kinase universe. And it keeps expanding by our own information, and by information being gained externally and being pumped into our bioinformatics tool and database. And, eventually, we have outposts through that kinase space. And whenever a new kinase becomes implicated it is medically relevant. We're only a short distance to hop over and take a chemical class to be a relevant -- we believe biologically relevant kinase inhibitor to that target. So that's some of the technical lessons that we're learning in the kinase exploration.

One of the things that we're learning is that there are some pretty innovative things that you can do on the biology side, and on the intellectual property side. Now, the question just came up about what do we do about other people's target of intellectual property? This is one of the things that you can do, by understanding all kinds of kinases together. We genetically engineered a particular kinase that we liked using. But we used that as a template to build in the characteristics of the active site of other kinases that we wanted to target. We're never using the intellectual property,or the clone of that other kinase.

We're using our own clone that is protected by our patent. This approach is applicable, we believe to all kinase space - -and it will be one of the innovative things that comes out of this broad family-oriented exploration. But, of course, it's the drugs that come from it that is the most important thing. True to form. And we've already determined over 200 kinase inhibitor structures filed. Patent filings covering more than 100 distinct scaffold classes. And the structures and chemical classes explored in greater than 80 percent of its kinase universe. So, we believe that we're in the process of conquering kinase space.

What about new universes? What's next? Well Protease is a parallel universe that we feel very interested in. Already with non-drug sales of about $9 billion -- it's to some degree -- a market validated family -- but, this is only in two classes of targets. HIV Protease inhibitors dominate completely the protease drug market. So, with 400 other human protease genes-- many of which we believe to be essential and crucial biological events, and important to different therapeutic areas - -we believe that this is a very rich family.

And this is supported by what's going on in pharmaceutical research across the board. With more than 300 research programs under way in the industry. Just a sample of what's going on. These are some of the more interesting or favored protease targets. And you can see a number of things. They cover many important indications, and medical needs. Big markets. Big numbers in terms of prevalence. And the targets are -- importantly -- they're scattered across different sub-classes of proteases -- of different part clusters of protease universe. We believe we have an extremely impressive track record in protease research. We have studied proteases individually, or in families. In every case, we've been well-respected by collaborators with action with their checkbooks -- to the tune of these numbers.

We've also produced a drug. We've produced advanced clinical candidates. We've got middle stage clinical candidates, and early stage drug candidates. We feel very good about the productivity gone in the past in this area. And we've also covered much of protease space - covering Cysteine proteases. Aspartyl proteases, and Serine proteases. We've already invested three of the four most important protease classes. So, we're starting to develop the tools.

Mapping the Protease space. And we intend to put posts throughout the Protease universe -- in a fashion reminiscent of what I just showed was going on in kinases. Re-use the scaffolds to design from one end of the universe to the other. The global picture here is the same with proteases and with many gene families. The tools that we require will actually be different. This is not something that you can clone what we did with kinase, and make it work for proteases. No. This scale of enterprise requires hands-on care for each gene family. And we're going to have to add new tools.

But so far, we've also made a good start in terms of other targets here. One exciting program that we're making fast progress in now -- Alzheimer's disease is a complicated process. And there are multiple mechanisms going there. But, basically, in the gumming up of the brain, and the lack of performance that follows, Beta Secretase, and its processing of the Beta Amaloyd Protein, is regarded as perhaps the most compelling target,overall, -- causative of Alzheimer's disease. We've solved this structure. We have launched a chemistry effort. We feel very good about the status of this chemistry effort. I can't tell you too much about the status of this program. But this is one of the flagships emerging from that protease universe quest. So, we're underway with kinase, Caspases, spreading out to proteases. The broad family of proteases.

What's next? There are many other drug-rich target families. Some of them are not enzymes though. So those classes, in particular, we are going to have to add new tools.

Or, as Joshua pointed out earlier -- we are going to get, we believe some of those tools by a collaboration -- or the acquisition of Aurora Biosciences. Aurora also, had been from the beginning a visionary bio-tech company. They had been technologically advanced and sophisticated. They don't have undue overlap with Vertex. The offer great synergies. They're going to strengthen our ability to produce through-put in enzyme targets. More or less along the lines of what we've done in the past - and to expand our ability there. But they're going to offer us abilities in other non-enzyme areas. Because they're already world leaders in some of these targets that we've only been amateurs in. So, together this forms a very exciting amalgamation of what Aurora brings to Vertex. You've already heard a little bit about the vision. We think that the Novartis deal was a good example here.

We're in the process there of rejuvenating a big pharma companies own product pipeline. And at the end of it, we get to co-promote those drugs with the big pharmas rejuvenated pipeline. And if we really do it well, we're also going to finish up adding to our own independent drug pipeline. I took out the next step of this vision that I was going to ask you to imagine for yourselves -- but Joshua's already shown it. That is -- the next gene family. And the next and the next -- each establishing its own new class of drug targets. We believe the vision is exciting, and I think I'm going to leave it there for the moment, and leave it for Vicki Sato, shortly to try to come and help us out understand this overall vision a little more. And savor it a little more. So, with that I'd again like to point out that we've got a great scientific team at Vertex who deserve all the credit to enable me to go on the road to show -- on the road with a show that focuses on success like this. Thanks a lot. Any questions?

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Investors and security holders are advised to read the joint proxy statement/prospectus regarding the proposed merger when it becomes available, because it will contain important information. Such joint proxy statement/prospectus will be filed with the Securities and Exchange Commission by Vertex and Aurora. Investors and security holders may obtain a free copy of the joint proxy statement/prospectus (when available) and other documents filed by Vertex and Aurora at the Securities and Exchange Commission's web site at www.sec.gov. The joint proxy statement/prospectus and such other documents may also be obtained from Vertex by directing such request to Vertex Pharmaceuticals, 130 Waverly Street, Cambridge, MA 02139, Attn: Investor Relations, tel: (617) 577-6000; e-mail: InvestorInfo@vpharm.com. The joint proxy statement/prospectus and such other documents may also be obtained from Aurora by directing such request to Aurora Biosciences, 11010 Torreyana Road, San Diego, CA 92121, Attn: Investor Relations, tel: 858-404-6600; e-mail: ir@aurorabio.com.

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