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CONVERSATIONS WITH MIKE MILKEN

A Special Episode: Cancer 2020: Is The Cure In Sight?

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Mike Milken: Today our topic is focused on cancer 2020: Are new cures in sight? In February of this year, when PCF [Prostate Cancer Foundation] President and CEO Jonathan [Simons] and I returned from our medical conference in Johannesburg, I told him that I needed the PCF to look at everything that has occurred in cancer research in the last 30 to 40 years and see what would be usable to address the COVID-19 pandemic.

So let's talk a little bit about what's occurred in the last six or seven months, focusing on the potential collateral damage. The number of reported diagnoses of cancer, just in the United States this year, depending on which cancer you're focused on, is down 20% to 40%. Cancer has not gone to sleep, but we recognize that people who've had potential life-threatening diseases elected not to go into medical centers and be diagnosed. And so we will be seeing these issues. There's also other issues that have

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come up and they are, what's happened with medical research and the shutting of physical research laboratories during this period of time.

But at the same time throughout the world, research has gone on, and once again will change the face of cancer treatment and cancer outcomes for the better. I'd like to give you just a few statistics here before I turn it over to Jonathan: the first sequencing of the human genome took more than 10 years, led by Francis Collins, and cost of billions of dollars. Now the cost of sequencing your genome costs about $200 wholesale; one fifth of an MRI and less than certain chest X-rays. So the cost of sequencing and determining what your issues are is now available for almost every single person. This has dramatically changed research as we know it: computers a million times faster; data storage costs down one billionth, have allowed us to do things we could have never achieved before.

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Once we sequence, and once we understand the mutations, we discovered that cancer, a particular mutation is not related to any one area in your body. And more than 70 cancers have mutations similar to prostate cancer, and Jonathan will talk about it. The solutions that we are finding in prostate cancer have already shown to have applications in other forms of cancer.

But one of the things I want to stress on this panel is that many of the potential solutions and success that we are going to have in treating COVID-19 lie in work that we have done over the years at the Prostate Cancer Foundation to try to reduce the death rate and ultimately eliminate cancer as a cause of death.

Jonathan will set the stage for a discovery and analysis that was done at the Prostate Cancer Foundation in 1999 [based] on the fact that using ADT drugs – androgen deprivation therapy – results quite possibly in the prevention of the [COVID-19] virus from going to your lungs. A clinical trial was begun a couple of months ago on this issue to try to identify these drugs, which are often generic, and ones that are fast-acting, but only lasts for 30 to 40 days and can quite possibly prevent the movement to your lungs of this virus and serious side effects.

There are more than 70 anti-viral anti-cytokine vaccine and other products out of the more than 500 that are on the tracker at FasterCures, the sister organization of the

Prostate Cancer Foundation. And with that, Jonathan, I'd like to turn it over to you. Thank you for joining us today.

Jonathan Simons: Thanks Mike. As Mike said so elegantly, the road to curing COVID runs right through lots and lots of cancer research. Debra Scher will talk a little bit about the VA [Veterans Administration] as probably the least-publicized and most-important platform right now for ending COVID-19 deaths in terms of clinical trials, built on a platform of how Dr. [Himisha] Beltran and Dr. [Felix] Fang and I take care of cancer patients. Literally the new precision care models for oncology are being applied in real time to treating COVID. The revolution that the human genome has brought and the momentum that it's brought to cancer care and understanding how genes work and then how the software, called epigenomics, works from Dr. Fang, who's the vice chair of radiation oncology at the University of California, San Francisco as a physician scientist. Understanding genes has gotten deep into the software of what makes them actually active or quiet or turns them up or turns them down.

And Chris Haiman has taken the human genome and human genetics and done work in polygenics, or polygenic risk. Himisha Beltran from the Dana Farber and Harvard Medical School, a PCF Young Investigator, has gone on to become a world leader in precision oncology. So Misha, what's precision medicine for prostate cancer? Why is it the paradigm for taking care of over 73 other forms of human cancer? Why is it transformational? And by 2025 what will cancer care even look like with all of this?

Himisha Beltran: Thank you, Jonathan. Good morning, everyone. It's really wonderful to be here. This is really a topic that is near and dear to my heart because even in the last 10 years, doing research in this area and taking care of patients, this has really transformed the way we think about cancer and the way we care for individuals.

I thought it would be good to just kind of step back and think about the term precision medicine, because this is something we hear about a lot when we think about research, healthcare delivery. But it's really an approach that's quite simple. It's can we more precisely deliver healthcare or medicines to patients by matching the best therapy with the right patient? And by doing this, we can deliver better outcomes.

And this is extending beyond our traditional factors. Traditionally we think about things like the physical exam or laboratory values or radiology pathology. Now we're taking it one step further recognizing that every individual is different, and by studying the molecular features of people and their cancers, we can make more precise or personalized decisions when treating people diagnosed with cancer. A lot of precision medicine has focused on genetics, or alterations in the DNA, because we've known for many years that cancer is a genetic disease. And as Bert Vogelstein, one of our pioneers in cancer genomics said in the 90s, these DNA mutations or alterations can drive cellular

multiplication, increases in tumor size, disorganization and malignancy, the hallmarks of cancer. But despite knowing this for so many years, it really wasn't until recently that we had the capability to actually do this in real time for our patients that we're treating in the clinic.

And the reason for this is it's really complicated. There are billions of letters in the DNA. Each person, we're generating massive amounts of data. And then we expand this through hundreds or thousands of people, and we really required big-data scientists, computational tools to able to have clinicians like me interpret this. And we can now start to map out patterns of DNA alterations within a cancer cell by sequencing the genome. And then we can start to interpret this and figure out what is in the cancer. But also can we learn, where did that cancer come from? Where is it going similar, to a subway map?

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I was thinking about all the intersecting pathways that occur within a cancer cell, and this is allowing us to develop ways to intervene and treat cancer. And thanks to the Prostate Cancer Foundation, there have been over thousands of genomes already sequenced, and this has really led to changes in the way we practice and the way we treat individuals with prostate cancer with targeted drugs.

Recently the FDA approved a class of medication called PARP inhibitors – two drugs, Olaparib and Rucaparib – for a select subgroup of patients with prostate cancer based on the mutations found in the cancer DNA. And this has made precision oncology a reality for every prostate cancer patient.

Jonathan Simons: But a key thing is you have to have your biopsy sequenced, right, Misha? The precision comes in having the DNA read to match the right drug or the right treatment.

Himisha Beltran: Yeah. And I think one of the challenges is that as we're sequencing more genomes in cancer and especially in prostate cancer, we focus a lot on the common alterations because we think those are the ones that are driving the cancer, and can we develop drugs for that subset? But in prostate cancer, there are mutations that are present, not in everyone, but only a very small fraction of individuals – less than 5% of people, their tumors will harbor one of these mutations. And these have been particularly challenging to study because they're not frequent. But we do think that

they're important in conferring response to therapy and may help us develop novel therapies to target even rare subsets based on the genetic alterations in that cancer.

There are patients that achieve exceptional responses to therapy. A patient of mine, a 61-year-old with advanced prostate cancer who had developed progression after many lines of therapy, including androgen deprivation therapy, chemotherapy, abiraterone, anti-PSMA therapy on a trial, went on a clinical trial if be Ipilimumab, which you may know, is an immunotherapy, and that trial actually went to into Phase Three for prostate cancer. And the trial was not positive, meaning not everyone responded well to Ipilimumab. So this drug did not get approved for prostate cancer.

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But this patient, my patient had an extraordinary response. His PSA went from 72 to 1.4. His cancer nearly went away. He had a dramatic improvement in symptoms and he ended up coming off of the drug and did not require any additional treatment for almost two years. We sequenced his genome and we did not see anything that we would have expected to confer a response to this drug, things that have been associated with response to immunotherapy in other cancer types; but he did have a lot of these rare alterations.

And I think it's really our obligation as a field to really understand exceptional responders, because you can imagine that he's not the only person that would achieve a response like this to Ipilimumab. But what do we actually do with this? And this is a very early concept, but we've been, as a field, thinking about novel ways to bring treatments to patients based on not just their DNA alterations, but integrating things like RNA and other biomarkers and thinking about treating patients without requiring the numbers needed for a large trial, but being able to cast a broad net to identify these long tail alterations and allocate them to specific drugs in this sort of basket trial sense for prostate cancer specifically.

Jonathan Simons: So this one is interesting because if you're a patient and you have a mutation that we could put you in remission for, but only 2% of prostate cancer patients have that mutation. But 7% of ovarian cancer patients have that, and 9% of breast cancer patients have that, and 14% of colon cancer patients have that – all those patients that have the targetable mutation ought to be in a basket and get the drug, not based on the anatomic, but on the genomic. And that's going to require multicenter collaboration. Deborah Sher will talk about this in the VA as a model, but it's also going to require patients that are aware that they need to know what their mutations are and where they are. We're very excited about this because these other channels of looking

at these tumors also will be reduced to practice in the next five years. We need to make more drugs very specifically against these new targets, which we know are central to making the cancer tick.

Himisha Beltran: In five years, I do think that sequencing for every patient, not just with prostate cancer, but all cancers will be reality. We will be using these genomic reports to make better decisions and is not going to replace what we do today; when we see a patient, we do imaging and get pathology, but this will just help us take it again to a much more precise or informed level.

There will be broader accessibility to genomic expertise, drugs, and trials. And I think a lot of this will be due to the fact that telemedicine is really a reality now. This is something that I predict will stay. It happened overnight with COVID-19 with the pandemic, and I think will help bring people together and be able to help us match drugs with patients in a more effective way. And the next generation of precision assays are coming. We are moving beyond DNA, and technologies are moving fast. It's not just the genome now that we are going to be integrating into the clinical decisions in real time for patients beyond research.

Jonathan Simons: Data science is this huge and important area where we think cancer research and cancer care are going to lead. But the oncologist you want to take care of you is very familiar with how data for caring for a cancer patient works. Thanks Misha.

Felix Feng is a very special clinician-scientist and physician-scientist like Misha. He leads the UCSF Prostate Cancer Research Program in the laboratories there, but he's [also] a practicing radiation oncologist. I wonder Felix, if you could start with what's happened, let's say in the last 18 months, on how radiation therapy, which is used to treat 47 out of 100 cancer patients, can be curative in primary tumors or early cancer. But there's [also] a revolution going on using radiation therapy to treat metastatic disease. So I wonder if you could tell us a little bit about that and epigenomics and why, by 2025, this is going to be a central part as well in taking care of cancer patients?

Felix Feng: Absolutely. So the first part you've asked me to talk about is a revolution in radiation oncology across cancers. And, Jonathan, I think the word revolution is quite apt in the sense that if you look at the last two or three decades of radiation for cancer, we've always thought that radiation should be used only when cancer patients have what we call localized disease, and localized disease means confined to the area of origin; so in prostate cancer, it means prostate cancer confined to the prostate, but gone to the bones or someplace far away from the prostate.

But thanks to two very pivotal trials, what we now know is that patients who have a limited amount of metastatic disease, a subset of these can be cured with radiation therapy. And that's actually paradigm changing, not only in the field of prostate cancer,

but across all of cancer in the sense that now we can use a treatment modality – radiation – that is widely available, we can extend it into a disease space, metastatic disease, where it really hasn't been used. We can't cure all patients with limited metastatic disease, but a subset of them five, seven years after treatment are free of disease.

The question is, who are those patients? How do we identify them early? And potentially, how do we improve upon current cure rates with radiation for metastatic disease? Radiation has for a very long time been considered what we call a local treatment. It means that where you aim the radiation is where the radiation works; where you don't aim the radiation, the radiation doesn't work. But we might be changing that paradigm as well. There's something called the abscopal effect, and that basically means if you treat one spot of cancer with radiation, another spot that you didn't treat may actually respond.

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Now what we see is that, at least for a very small proportion of patients, radiation when given with immunotherapy might work where you don't aim it. These are very, very, interesting times, times of kind of great advancement in the context of radiation oncology for cancer. And I would say that thanks to the Prostate Cancer Foundation, research advances in prostate cancer are leading the way. Jonathan, do you want me to touch upon anything else regarding radiation before I switch?

Jonathan Simons: What is the new form of radiation that's used? It's very different and very scientific for, with curative intent for illegal metastatic disease. What is that Felix, and why did the cancer cells die?

Felix Feng: Sure. And so for, for many decades, when we gave radiation for any cancer, we would give a little bit of radiation every day over the period of many, many weeks. That was what I term "conventional radiation." But over the last few years, there's been the advent of newer forms of radiation that basically all revolve around the principle of giving very high-dose radiation to a very small area. And due to advances in how we give radiation and also advances in how we can keep patients in the exact same spot every day for a couple of days, we're able to give five to seven times the amount of radiation to a spot that we were able to do a decade ago. Back 10, 15 years ago, when we used to give a little bit of radiation every day for eight weeks or so, and what we hoped was that over the eight weeks together, there'll be enough damage from the radiation to the cancer to kill everything.

But now, within just five treatments we can give the same radiation dose we previously gave over eight weeks – and that has allowed us to treat metastatic disease. And more importantly, in the era of COVID, switching from a treatment that requires a patient to come to the hospital every day for eight weeks versus one that can be done in five treatments, not only is this improving convenience and quality of life for patients by reducing the number of treatments, but it's also reducing the risk that they catch COVID-19 from being in the hospital. And so all of these advances are benefiting patients in a multitude of different ways.

Jonathan Simons: Thanks, Felix. Teach us about epigenomics.

Felix Feng: Absolutely. I wear two hats. I'm a physician on one side like Misha, and also I'm a laboratory scientist on the other side, like Misha. And so, what I'm going to do is briefly talk about the word epigenome. The genome is, as Misha mentioned, focused on DNA, and much of the field of genomics focuses on changes in the sequence of DNA, or mutations, or changes in downstream levels of RNA. DNA is made in RNA. As Misha pointed out, there are multiple layers of data we can examine in a cancer cell. And one of these different layers is the epigenome.

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The epigenome was defined as chemical compounds and proteins that combined a DNA to turn genes on and off. And ultimately this may control cellular biology as much as the genome, the DNA sequence itself.

DNA and the architecture of DNA is quite complex, in the sense that there are chemical modifications to DNA called methylation that can make DNA more or less accessible, and therefore turn genes on and off. When you look at clinical trials for prostate cancer, precision medicine, much of that precision medicine is focused on changes in the sequence of the DNA. But I think if you look over the next five to 10 years, and you look at opportunities for precision medicine, those opportunities are going to arise as we understand additional layers of biology, including the epigenome in prostate cancer and other cancers, as we understand, architecture of DNA and chromosomes and accessibility of genes that leads to certain genes being on versus off.

Recently we did a large study where we profiled a component of the epigenome called methylation across 100 patients with metastatic prostate cancer, looking at the

metastases themselves. There are methyl groups, which are chemical compounds that are bound to DNA at different spots. And in the hyper-methylated subtype, they actually have more of these chemical compounds bound to more areas of the DNA. We don't actually quite know what it means yet in terms of all the biological processes; that's actually what we and others in the laboratory are studying. But what we do know is that patients with these hyper-methylated subtypes of prostate cancer have different outcomes. And again, all of these patients are patients with metastatic disease, and the hyper-methylated subtype, the patients with that actually do better. They have longer survival times once they're diagnosed with metastatic prostate cancer than the non-methylated patients. And so what we've identified through comprehensive methylation profiling of metastatic prostate cancer is that there are different subtypes, and it turns out now that these different subtypes, these hyper-methylated subtypes have been discovered in other cancers like brain cancers and leukemias and colon cancers as well. We know that the clinical characteristics or clinical outcomes of these hyper-methylated prostate cancers are different, and now we need to study them biologically. We need to understand how to target them therapeutically. And I just want to point out that all of this research was sponsored by the Prostate Cancer Foundation. Jonathan, I'll turn things back over to you.

Jonathan Simons: Thanks Felix. Understanding the activity of these genes is going to be essential and actually designing drugs because we have lots of ideas all of a sudden about making precision medicines just to stop one line in particular that's making a prostate cancer and a breast cancer cell divide. But also it may be very important to understand genes that are turned off that make you normal again. But this way of looking at a cancer patient's epigenome is going to be also how we care for every form of human cancer. And the work that Felix presented is actually given us really a framework for many other forms of cancer.

We've talked about genes in patients with advanced disease, but what if you could find cancers 10 years earlier? Or prevent heart disease? How would you do that? Well, it's also a big data problem that's been reduced to research practice rather amazingly. Chris Haiman is one of the world's leaders in population genetics, or how genes work in populations for diseases, and is also in an enormous entrepreneur scientific collaboration with over 100 collaborators around the world to get the numbers up to do this work. So, what's apologenic risk score? Why does it help explain the disproportionate burden of prostate cancer in African Americans? And what do you think is going to happen to apologenic risk scores in revolutionizing basically how doctors take care of us?

Christopher Haiman: Thank you to the Prostate Cancer Foundation for the invitation to be part of this panel. I think Jonathan and you and Mike have done a good job to set the stage for some of what I'm going to be talking about: the completion of the human genome sequence and human genome sequencing becoming affordable.

We've really learned a tremendous amount over the past decade, about the role our genes play in human disease. I'm talking about germline-inherited variants that had been passed on through generations. They've been found for many common diseases, including prostate cancer, and here I'm talking about user sequences, changes in our DNA sequences that exist throughout this, genome associated with risk of developing disease. But they're appreciably common. They're found in many individuals. And alone, they're not predictive of risk because they only are associated with small increases in risk. However, in aggregate, a composite of all of these variants is important and can tell you something very informative about someone's overall risk of disease. And when we aggregate them together, we put them together in the form of a polygenic risk score where the risk is determined for an individual based on the number of variants that exist and been discovered for a specific disease or create their frequency in the population, whether somebody carries it or not, and the associated relative risk for each of those variants, which I indicated is relatively small. However, in aggregate could be associated with an appreciable risk. And so this just highlights this distribution of risks that may be observed in a population, with some people having a score that places them at very high risk of disease, and others having scores that place them at much lower risk.

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Jonathan Simons: And Chris, this is looking at hundreds and hundreds of letters of the DNA code right, across the genome.

Chris Haiman: Exactly. And so this is a score that can now be calculated for any given disease or trait and used to assign people to different categories of risk based on their genetic makeup. And just one interesting thing that we have noticed when comparing these polygenic scores across different diseases, such as your heart disease, colorectal cancer, breast cancer, diabetes, and prostate cancer, is the polychronic score for prostate cancer is really quite unique and does a much better job of identifying men at very high risk of disease.

Something that is important to understand about polygenic risk scores: we've noticed when comparing them between populations, they're quite different. So for example, men of African ancestry have a substantially higher genetic risk of developing prostate cancer compared to men of European ancestry. I think an important question is now that we

have a better risk-prediction tool, that we have a stronger starting point for applying preventative and or screening measures to those at high risk, let's see if we can now begin to reduce disparities and both the incidence and mortality of prostate cancer between different populations.

Jonathan Simons: This is a very, very important part of the future of population science, for diseases, in terms of where your great, great, great grandparents came from in terms of disease risk

Chris Haiman: We can do a much better job of telling a man what his lifetime risk is based on his genetic information. If we incorporate family history, we incorporate other biomarkers, I think we'll get even further stratification of a man's risk of developing disease.

So I think the really important question, regarding polygenic risk and polygenic risk scores, is how are we going to use this information clinically to improve health? This is the big challenge in the field. By 2025 or so, I do expect that the polygenic risk scores will be generated and available for a whole host of diseases and will be used by physicians and patients to guide and inform either changes their lifestyle behaviors to prevent the disease if those are available or for risk stratified, screening. So, disease can be discovered earlier, before it becomes untreatable. And think this is conceptually very similar to what we've already been talking about, about how to treat cancer patient, moving away from this conventional one-size-fits-all approach for more personalized approaches to treatment. And I think now it's time to think about how this personalized approach can also be applied to prevention and screening that may be differentially based on one's level of risk, and may be important for some diseases or some cancers where the hard benefit ratio of a preventive measure, the hard benefit ratio of a screening tool is being questioned.

Jonathan Simons: We've talked about precision medicine, precision radiotherapy, genomics, epigenomics, and now we are talking about precision screening, big-data management. Polygenic risk score is going to be, we think, incredibly important in the future of human health and certainly in prostate cancer.

Debra Scher is the executive advisor to the secretary of the Veterans Administration, and Debra leads the Center for Strategic Partnerships and has been an extraordinary American citizen for improving the care of veterans. The VA is the nation's leader now in trying to bring all this precision care to every patient.

Deborah Scher: Thank you, Jonathan. What you have created is truly an extraordinary community here. If you were just funding researchers, that would be fantastic. But what you have also done in a very short period of time – this partnership was launched in 2016 – right now there are 12

centers of excellence. We think very quickly with your help, they're going to be 20 centers of excellence.

And why is that important? We have really built a community of researchers who are bringing best-in-class precision oncology to veterans across the country and are using that platform not just for prostate cancer innovation, but today for COVID innovation as well. There are nine million veterans served by the VA; a third of them live in rural communities; about 22% of veterans are minorities; about 10% of veterans are women and a tremendously growing population.

And before the Prostate Cancer Foundation launched this partnership, each of these centers were doing work on their own, and it wasn't until we had the momentum and the support and the guidance from the Prostate Cancer Foundation, that we could really build this nationwide network. It used to be, if you wanted to open a clinical trial at the VA, you had to go to every single center individually and go through every single center's IRB [Institutional Review Board], and then consent every single center's patients. Thanks to the incredible work of Dr. Rachel Ramoni, who leads the office of Research and Development at the VA, and in partnership with the Prostate Cancer Foundation, who has helped us knock down some barriers and put in some very important infrastructure, we now have essential IRB.

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We even have a relationship with a commercial IRB. And whereas it used to take over a year to open a clinical trial, we have a great story that one of the COVID clinical trials opened in five days. So you're really seeing tremendous transformation of a government organization that has the scale to truly make a difference in some of the most challenging medical problems.

There are 50,000 veterans who are diagnosed with cancer every year in the VA; 12,000 of those are prostate cancer, about 3,000 have already been sequenced. And this is starting almost from zero. And so it's really bringing state-of-the-art care to the VA. In many ways, the partnership has brought the VA more into the national conversation about where innovation is taking place and helped us stay current and helped the rest of the medical community stays current on the insights that the VA is generating.

So if you think about the evolution of philanthropy, you had Mike Milken thinking about partnering with a government agency in 2016, which many of you may have thought was a crazy idea, and now has built this incredibly powerful platform where we can enroll large numbers of veterans in clinical trials and develop insights that really transformed care across America. And now we have that same government agency committing its own funding to truly scale. So there's just been a tremendously exciting time at the VA.

Jonathan Simons: Debbie, you want to talk about how it's scaled also to lung cancer and breast cancer. The idea was always prostate cancer was the No. 1 cancer, so start there, but the model can be applied to every form of human cancer.

Deborah Scher: Right. So, building off of these centers of excellence, the VA has announced its own internal contest to stand 10 centers of excellence around lung cancer. And the VA has its first female deputy secretary this year, Pam Powers, and she has made women's oncology one of her priorities. And she keeps pointing to the prostate cancer center of excellence and says, "why can't we have one of these for women on women's oncology as well?" And there's no reason why we can't. And so there is a plan in formation to hire a director of women's oncology and to start to build that same platform for centers of excellence around that area as well.

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Jonathan Simons: One last thing Debra, the electronic health record has become a huge asset for COVID-19 research. It's a huge big-data asset for cancer research. Do you just want to talk for a moment about why the VA's electronic health record is so advantageous in fighting COVID?

Deborah Scher: The VA was actually the first health system in the country to have an electronic medical record. They created their own and built it up over many years. And so we follow veterans from the time they leave the service to the time they leave this earth. So you can really track patients across their lifetime [and] track comorbidities. They stay within the system and we pride ourselves on treating the whole person. So you have a very robust data platform to learn from and to make decisions around as we treat these challenging diseases. It's been a very exciting time for us and we've just been grateful for the partnership.

Jonathan Simons: Mike, I thought you wanted to make some observations as well.

Mike Milken: I just have a few takeaways I want to leave with everyone. Maybe 15% of all lung cancer patients who have advanced smoking-related induced lung cancer today are surviving substantially longer. And we might be using the word cured because of work done by the Prostate Cancer Foundation. Within a decade, we might not be talking about disease specific; we might be just talking about mutations. Work that we began in 1995 with Jim Allison, who won a Nobel Prize in medicine, 17 forms of human cancers today are now getting this checkpoint therapy. The outcome for cancer patients has improved dramatically. And the future is in using data that you saw today to analyze many of these issues.

In closing, I want to just comment very briefly on the importance of the VA. First, largest medical record [database] that we can access. Two, the idea that this program we launched in 2016, that if you serve the country of the United States, that you will have equal access to cancer care; the only place now that you have the same outcomes for African American men as you do for the general population is now in the VA. And so this program has enormous promise. And I want to just comment again how uniquely positioned this is and how our promise to veterans who have served, whether they're young or whether they're old, has come to bear here, which will affect and benefit all humans.

So thank you for joining us. All the best, and good health to you.
