Please be seated. Distinguished guests, dear audience, Professor Kemar, Hannah, family and friends, many here, some abroad in Australia and Aruba, welcome.

It is an honor and a pleasure to open the ceremony and to see so many of you here today in Omnia.

Ged Kemar, Professor of Phytopathology, is about to present his farewell address.

I'm looking forward to what you're going to share with us, Ged.

Let me start with citing some headlines from articles about your work. The banana is going extinct.

Can the banana still be saved?

Latin America is facing a banana disaster. These are quite alarming headlines, worrisome, perhaps even depressing.

But we all know that in all your years of researching plant pathology, you were not discouraged.

Ladies and gentlemen, Ged Kemar has been fascinated by plant breeding and plant pathology since his early student days. In 1984, he started his master's degree in plant breeding right here in Wageningen.

Not everyone may know this, but he spent most of his career studying wheat, not bananas.

One of his first projects involved airborne fungal diseases of wheat crops.

In total, Professor Kemar spent 35 years studying this topic.

During this time, he made one of his most important scientific contributions.

He discovered the mating system behind the causal agent of the blotch disease in wheat.

This was one of the most devastating diseases in Western Europe and North Africa. Professor Kemar's discovery opened up new research into the genetics of the fungus. And one of the strains he selected is now the reference strain for the global scientific community.

In 2004, Professor Kemar's career took an unexpected turn.

And this was because through his colleague, Professor Yetze Storfo, he was approached by the National Banana Corporation of Costa Rica.

They needed help on a fungus that was destroying their crops.

A specific fungus, Black Siga Toca.

It had been spreading rapidly through Latin America and it destroyed the banana trees.

In those years, the research into banana diseases received little attention in the Wageningen.

But clearly Professor Kemar changed this. The banana is now a pet crop in Wageningen and these are his own words.

Professor Kemar and his team performed excellent research into pathogens, genetics, resistance, fungicides, and more. He has published more than 140 peer-refued articles during his impressive academic career. And under his supervision, PhD candidates have also delivered excellent science on these topics. Moreover, Professor Kemar has raised a lot of awareness on the importance of bananas as a crop.

He has frequently appeared in the media, including in the children's TV show, Hit Clock House, where he showed the difference between a healthy and a diseased banana plant.

In 2018 Wageningen University was celebrating its 100 years anniversary and on that occasion Professor Kemar was all over the news. He had successfully grown the first native banana in a Dutch greenhouse.

As a result of all this publicity, many people now know how diseases are affecting the livelihoods of growers.

They also know about the consequences for food security in Latin America and elsewhere.

The societal impact of banana diseases has been the driving force behind Ged Kemar's research.

Over the years, he has given more than 100 seminars in 24 countries, speaking with fellow academics, corporations, and with growers from whom he wanted to help.

Professor Kemar also co-founded various companies that breed new varieties in order to move away from vulnerable monocultures. Ladies and gentlemen, I started this introduction with some alarming headlines, but there are some other headlines from the Dutch media that I think describe Ged Kemar's work much better, building a better banana and Ged Kemar's mission to save the banana. Dear audience, I don't think I need to emphasize that Professor Kemar is a true representative of the Wageningen approach of science as he combines excellent science with striving for impact. I'm very much looking forward to his farewell address entitled Beauty, Blame, and Battle, Fungi and Footcrops.

The floor is yours. Madame Rector, Hana, Geds, Grand Geds, family, friends, and colleagues.

This abbreviation may look familiar in these days of elections, but no political party, neither political preference, but BBB stands today for Beauty, Blame, and Battle, Fungi and Footcrops. So let's start with the beauty part first. So when I was a teenager, I had a girlfriend and she introduced me to this book, Do the Lentil or Silent Spring, written by Rachel Carson. And that opened my eyes for the danger of using chemicals and pesticides for disease control or weeds.

It was not necessarily an inspiration to go to agricultural college, but nevertheless, I took the book and I took it on a solo trip to the UK and Scotland.

While she said, I'm going with a group of youngsters from church to holiday, maybe I find better boys there. All right, that was a nice farewell.

What I can disclose now, that girlfriend became later my wife and she is here in the audience.

But the right part of this slide is the fungal tree of life, which is incredible diversity.

Fungi that can affect plants, but most of us probably know these fungi simply as mushrooms, right?

And the founding mother of the science of fungi on mycology is Johanna Westerdijk, the first female professor in the Netherlands.

And when she founded the central bureau for chemical theory, she started with a collection of about 80 fungi. And when she retired, the collection grew to 11,000.

And nowadays the Westerdijk fungal biodiversity institute contains 100,000 different species, but that's only a fraction of the close to four to five million species that are around here on the globe.

And only about 150,000 have been described.

8,000 of those are detrimental to plants, and about 300 can also infect humans. So let me give you one example here.

This is the Armillaria ostoriae, the largest living thing on earth in Malur national forest in Oregon in the US. It spans about 965 hectares.

That's about 1400 soccer fields, grows one meter under the soil, has a weight of maybe up to 30 million kilograms and has an age between three and eight thousand years.

To give you an impression. Or this one, termitomyces titanicus, the largest edible mushroom in the Guinness book of records.

So yeah, fungi and mushrooms in particular have inspired artists like what you see here in this window of the Cathedral of Châte in France, or Modern Art by Kasten Hüller, the giant triple mushroom that was placed on the place Place van Dom in Paris in 2024.

Now my first introduction into plant pathogenic fungi was the research institute for plant protection.

And I went there for an internship during my interview, and those of you who are not aware of this institute is IPO. And IPO will come back later in the talk as well.

Now, honestly, I never thought about plant pathogens. And during the day, somebody mentioned plant pathology was absolutely boring when it was taught to us in high school. Well, I had a very similar experience.

It was literally a book where you learn life cycles, difficult names, and that was it. So for me, that was absolutely not very intriguing. But the moment I stepped into that institute, and I met the late Ron Stubbs, who I later succeeded as a researcher.

And he told me that he was living or working his entire life on the striperous fungus of wheat, Poxynia 3-formis formis specialis 3TC.

And I really thought, can you really work your entire life on one fungus?

You know, that absolutely blew my mind, but it also grabbed me and never let me go since. So it was super fascinating.

I still remember the scene. It was about this time of the year, yellow lice in his

old greenhouse, the noise of the greenhouse, foggy weather outside, and then this compelling story that really grabbed me. So another person in the institute, Corphon Silphout, who is in the audience, I hope. Hey, Cor, there you are.

Thanks for coming. Really inspired me, too, because he introduced me to the origin of wheat.

And he studied the resistance of two striperous in, for instance, wild ancestors of wheat.

And that also has driven really my interest into plant breeding.

So I wrote a thesis, got a prize for that thesis, and eventually that also returned a job to me. So I started working for the Dutch wheat breeders to explore resistance to striperous in their germ plasm. That was a two-year job, but it really launched my interest in plant breeding. And when I was hired as a technician to work with Cor, I decided in 1984 to study plant breeding. While I had a job, because we had a family, and I'm very happy for my family to be here. Now, one of the slides that I used during my PhD defense was this, which says, in a nutshell, if you want to increase food production, you need better yielding varieties because they have limited land. So you have to make sure that your varieties simply produce more.

Of course, now 30 years later, we know, well, that's part of the story.

For instance, we also can decide to eat less meat that will free up land that we can use for arable crops.

Now, the next slide is important for you to see because you have to remember a few terms here. So what you see here is a scanning electron micrograph of a cereal leaf.

You look at the epidermis, and the orange parts are the stomates.

That's where the gas exchange happens between plants and the environment, carbon dioxide, oxygen, et cetera.

Right now, some fungi use these openings to enter the plant and start colonizing the tissue.

Others just use brute force to penetrate cells and just start growing from there.

And for instance, the fingers where I've been working on, that's just coming back later in the talk, xymosoptera triticide is just sneaky going into the opening of the stomate and just sneaks in in a stealthy way. Now, if you look into the cross section of such a leaf, you see that under those stomates, there are open cavities.

There are open cavities, right? So that can be used, and that is also the structure of the leaf. And what you will see is that fungi use that space. Now, so let me give you one example. This is the stripe rust fungus.

It just entered through the stomate, and it's recognizing the stomate by just sensing a very slight inclination of the leaf at the opening of the stomate. And then the fungus knows, boom, here I have to produce an upper thorium, or I have to go down. So that's what it did. And then in this cavity under the stomate is producing these white balls that you see, which we call stomatal vesicles.

And from there, it starts really colonizing the tissue, penetrating other cells, feeding itself until there is enough biomass to produce spores and to multiply. And

that's what you see here. So this is the symptom of stripe rust in weeds.

So those spores or those pestules, they just burst through the surface.

And this was the first plant pathogenic fungus that I saw and I was introduced to in the IPO. So it was a fascinating world. And if you zoom into such a pestule, this is what you see.

Isn't this just fantastic, right? You see all those individual spores that are blown by the wind over long distances, hundreds of kilometers, and sometimes even further.

So this is how these fungi spread, right?

This particular fungus. Now this is an asexual, this is an asexual stage of the fungus growing there and disseminating. And this fungus was considered to be completely asexual, whereas other rest species like stem rust and leaf rust, they have sexual cycles. But it's very complicated because you have to go to another host.

There they complete a sexual cycle. And sex has the advantage that you have recombination, right?

So this fungus is clonal. Each and every spore is theoretically identical. So the only way to change is by mutations, right?

Now later on, it was found that this fungus, yes, has also a sexual cycle. So from there, I think we should learn that probably never say this fungus is asexual, you just haven't looked hard enough.

You can find somewhere a sexual phase. And I got intrigued by this fungus and CORE gave me all the freedom to explore things. So I started to study spelt weeds. Because spelt weeds, you know, those are the spelt weeds that we now eat, right, which are very popular, is nothing special. It's just a hexaploid weed, like regular bread weed.

But it was once the major weed crop in Europe. And what I found is that those spelt weeds were resistant to all the striper's races that we used, surprisingly. So I wanted to know what is now the genetic basis of that resistance. So I had shown that are diverse. And to make a long story short, I found out that all those diverse spelt weeds carried one in the same gene, wire five, very surprisingly.

And the spelt weeds have been taken by the Romans down the river Rhine and Danube across Europe.

So and if you see villages with these names, like this village in Luxembourg, where I saw a spelt crop all of a sudden during an holiday break, you know, this really refers to that crop, which is still being grown in the Benelux, but also elsewhere. Right.

So that was stripe rust. But then once I finished my master's, we had been considering to continue for a PhD. And we thought, you know, stripe has been a sexual, pretty limited for further studies.

And then I was introduced through core by professor, the late professors, I hear y'all on the left side. He was a professor of plant pathology in the University of Tel Aviv. And he worked already for years on Xyma septoria treaticide, then it was called septoria treaticide.

And we knew there was also a sexual phase, which was called microcephalogramonicola.

Now we have merged this all into one species, just a species, Xyma septoria treaticide.

And Professor Zardok was my promoter for that studies.

And during actually during my defense of my PhD, Steve Gutten is also in the audience. And Steve, thanks again very much for coming over and contributing today.

He came and from there, we started to collaborate. So we know each other already for 30 years and have collaborated for 30 years.

That's this exceptional and also fascinating. And down in the right part is Michael Seidel and Eva Stukenbrock, Professor Eva Stukenbrock, with whom I started to collaborate to collaborate later on. Now let's go to the symptom of this disease septoria treaticide leave blotch. So you see that necrotic parts of the leaf, right? And some green part with the majority is necrotic and they're all small black dots. I hope you see those. Now, if you zoom into such a dot, this is what you see. So this is an asexual footing body of that fungus. And it right away fills that space under the stalemate, right? So there is space and it's producing there that pignetium.

And there is an microscopic matrix where the spores are sitting. So when it's humid, it's pressing the spores out of the stalemate on the leaf surface.

And there they are splash dispersed.

And if you then stain that fungus, for instance, here with a GFP label, a green fluorescent protein, this is what you see. So this is how it's getting out of those fruiting bodies.

Now, just a small calculation exercise. If you go to the average plant density of wheat in Europe, that's about 250 plants per square meter.

Let's assume that every plant has two stems. Usually it's more. But that's just for the sake of the example, take two stems. And each stem has three leaves, which is also very, very small, right?

And each leaf would have, let's say, a hundred pignetia.

And that's also very on the low side.

How many spores do you think that would translate per hectare?

Is there anybody who would accept you?

Is there anybody in the audience who has an impression?

15,000. That's one number. Anybody else? 7.5 trillion spores. 5 to the power 12.

So 7.5 to the power 12. And that's all clonal reproduction, right? So that's so beautiful in nature, the abundance of numbers, right? And also the opportunity for mutations. Now, one of the key questions when I started studying Satoria was, is there specificity?

And specificity is what you see here on the left part of the slide. So you have two isolates, you have two varieties, and one of those combinations you see that the

variety is infected and sometimes it's resistant. But you see this pattern, right? So that's what we call specificity, which is very common in the rusts.

That's why, you know, when you grow a resistant variety, you have so many spores, there is always a mutant that can circumvent that resistance, and all of a sudden that variety becomes susceptible, and you have to come up with a new variety to deal with the disease, right? Now, for this fungus, that was actually denied. Nobody believed that it occurred in this particular fungus.

And so that's where we started to work on. So this is the high plains in Ethiopia, where they grew wheat. My first visit to Africa, I still remember it very well, and we went to the hotspot for Satoria screening by CIMET, the Centro de Investigación de and the Mejaramiento de Maisi Triva, Center for the Improvement of Maize and Wheat. International Institute and IPO had a very strong collaboration with that institute.

So we went to that site, and I hope you can see in this slide, on the left part, there is a row of weeds that is actually almost defoliated by the disease.

It's very small, but if you look in the right part, there are rows that are just absolutely green and healthy.

And what we found out is that that population in Holeta was really adapted to breadweeds. So it killed all the breadweeds, but the green ones are durum weeds. And durum weeds are those weeds that we use for pasta production. They are tetraploids. So there are two genomes in diploid, so AABB, and hexaploid breadweeds is AABBDD, right?

So that was a fascinating observation. And I have to tell you, after 35 years of studying Xymosheptoria 3D Sci, including the entire community, we still don't know what the reason is for this differentiation. So there is still a task for those of you who still work on Xymosheptoria 3D Sci. Now, here I am sitting with a visitor, Juan Anona from Argentina, and he spent a year with us, and we had been screening numerous varieties with numerous isolates to see, do we see this type of interactions?

And yeah, we have seen them, but usually they were at a quantitative level. So not 1, 0, 0, 1, with 10, 20, 10, 20, 20, 10, or 30, 60, 60, 30, right?

And that really required these type of growth rooms fully seedlings.

After 10 days, you had to clip each and every seedling to get rid of the abundance of material that was growing there, because you just inoculate the phones, no laptops, so everything was written on paper by pencil. And then you brought this all up to the office, feed all those data and computer, and eventually start analyzing. So for those students who are here, remember, this was common sense 30 years ago, right?

So we saw those interactions, but of course, to make it relevant for practice, we were also wondering, do we now also see this in adult plants in the field? So you see Elsa Stappen, Elsa's in the audience, and she has been with me for decades as a collaborator in this research.

Thank you, Elsa, for being there all those years. And here we are inoculating an adult field experiment in Groningen.

And interestingly, indeed, later we saw that yes, if you use different isolates on different varieties in the field, we see those interactions as well.

And one of my largest criticasters in those days was James Brown from the UK, still active in wheat research. He said, I don't believe it. He said, well, if you don't believe it, come over, come to the trial and let's walk in the trial and see it. Well, I hope you can see it. We are standing in front of a healthy plot, but then the plot behind us is absolutely affected.

So fortunately, he was only five minutes in the field, and he was convinced. And since then, we started to collaborate. And through him, we also met the first resistance genes.

So that was very nice. So I confirmed what others had seen before.

But of course, repeating this over and over again, that's not interesting, right? So then you just repeat the things that you've done before. And then I was once visiting Simit in Mexico.

And there was a scientist from Chile, Ricardo Madarriaga. And he was the one who showed me for the first time how I can discharge spores from the sexual phase, which was then called Mycophallogramicola, how I could discharge those spores to an agar plate. It's not very complicated, but somebody has simply to show you how to do it. So you take a leaf that really looks very bad, like Eva said this morning, right? It's deteriorated. There is soil. But there are also fruiting bodies. There are tiny black spots. So we put them in water, blot the excess of water away, put it in a petri plate.

And 15 minutes later, those asco spores would absolutely smash to the agar. So the agar was absolutely covered with those asco spores. But there were cases like what you see here, there were only seven or eight apart from the rest. So I consider these might originate from one and the same ascos, right? So if my hypothesis is just correct, now I have the option to try it. So this is the spore if you see the two-celled asco spores, and on the sides you see two thick germ tubes growing, and they grow over the surface and eventually penetrate a stomache.

Now, so what I consider to do is that, okay, if I want to identify what is now the underlying mating system of this fungus, probably let's do a trial and error experiment. I take one spore, number one, I combine it with number two, one and three, one and four, one and five, et cetera, and inoculate those pairs on individual plants. That's what you see here in his growth cabinet. Put it in a plastic bag, incubate them, and then just put it outside. I've tried many, many things in the lab.

Nothing worked, but I knew the sexual phase was very active in the field. So I said, okay, put those inoculated plants with pairs of isolates, put them outside and see what happens.

And fortunately that worked. Now, to make a long story short, because of this genetic window that opened, we had an entire new field of research. That was something that never nobody had seen before. You know, that's the beauty of science. You discover things that nobody had seen before, right? It's a true discovery route. And then I could study the inheritance of virulence in that fungus, and it was considered to be very complex, multigenic, et cetera, no folks, single gene.

Very simple, classical Mendelian genetics, one to one.

So that helped us really eventually to clone the gene. But I have to tell you, that took us 18 years. And Harold is in the audience. I see him laughing. When he entered the lab, he looked at the data. We just had new data sets. And that Friday afternoon, we knew this should be the gene, and that was the gene. So, yeah, we

cloned the gene, and that was the first a virulence effector from this fungus. And that was really in the legacy of our department, because Pierre de Witte and his co-workers, Jan van Kaar and Mathieu Joester, cloned the very, very first fungal effector in Cladisporium fulvan in the lab. So we stayed within that legacy of the lab, right? But we also found another very intriguing biological fact.

And now we have to go to the cartoons that you see here, A, B, and C. So in A, what you have to remember with this heterothetic bipolar mating system, that sounds complex, but the simple reality is you have two mating types, one and two. And you have to bring one and two together, otherwise there is no sex, right? So you combine one and two all the time. So in the A cartoon, there are two isoliths, mating type one and mating type two. It's a susceptible plant. Both isoliths colonize the tissue and have sexual cycle. And those spores are released and you can collect them. In the B cartoon, there is one of those strains is a virulence on that host.

So it senses the stomade, but it cannot colonize. So only one parent's colonizing and building a biomass, right?

But yet there was sexual reproduction. And in C, there is a strain that is sensitive to a fungicide. So we first sprayed a tissue with a fungicide. The sensitive strain has difficulty, of course.

The resistance strain can easily colonize and build a biomass. But also in that case, there was sexual reproduction. So what is now the story?

We can control a disease by resistance.

We can control the disease by fungicides, but we never, ever can control sexual reproduction.

And that has really consequences for the population genetics. So if you translate that into a mathematical model, and I cannot do that. This is all collaborative work.

And so let's be very clear about this. But what we saw is that yes, contrary to the dogma, that a virulent islets participate in sex.

They were considered to be just out of the population because they can't do anything, right? But yes, they have an escape. They have sexual reproduction. They, of course, maintain those genes in the population.

And thereby resistance is slowing. The breakdown of resistance is really slowed down, right? So the durability of resistance is increased.

And I think this is true for this particular pathogen. But it belongs to a class of deltyrymices that, for instance, also contains the black cicatoca pathogen of banana. That's the which we'll discuss later on.

And the other thing that we noticed is a gene is encoded by the mitochondrion that is theoretically only inherited through the females.

Then the invasion of that gene is much faster in the population than a nuclear inherited gene. And that was really shown by the resistance to a new class of fungicides that were called the strobilurines. And they were considered to be the absolute replacements of the azole fungicides that were used already for decades.

But to the big surprise of many of us, that resistance was overcome by the fungus in just a couple of years and was absolutely booming. So the entire population

turned very quickly in complete resistance. And that was because the target is on a mitochondria, right? And that's also what we have seen in the black cicatoca fungus in banana.

Well, through all this work, we had one strain, IPO323, which is a very peculiar strain because this is a strain that shows these very clear switches or in pathogenicity on wheat varieties.

It was really 010101. So very qualitative, not quantitative.

And we started to use that gene, that isolate also for genome sequencing.

And of course, for me, it is a pleasure or a satisfaction to see that this strain has been adopted by the global community, the Xamarin Sartori community, as their ultimate reference strain. So it's a death strain from a farmer's field in West Brabant. And it was collected, the sample by my late colleague, Richard Darmon.

So IPO323, very, very well-known isolate. Now, we sequenced this strain and published that in 2011.

And only 12 years later, we were part of a consortium where we had sequenced thousands strains of that fungus. Well, I can tell you that's absolutely exceptional.

The technology has driven so far that we can now easily sequence an isolate for maybe 50 bucks, but sequencing IPO323 must have cost millions, really. And so in 10 years' time, the cost of sequencing went tremendously down and gives us a fantastic opportunity for a very high resolution to generate this type of data.

And because of this, we also identified genes, resistance genes, and also cloned them. So just to summarize in the Xamarin Sartori work, the worldwide third most important weed disease annually, 350 to 700 million euro costs just in France.

Market size 2 billion US dollars.

of fungicides, 1. And I had very, very effective collaborations with weed breeders outside the Netherlands, because I started with the Dutch weed breeders, but over time, all the Dutch weed breeders are gone. They have been bought by others. There's, I think, only one breeding company left, and all the others are in France, Germany, and the UK.

So I had to collaborate with farmers, with breeders in France, which was, by the way, fantastic. Always very nice lunches, not just a glass of milk and a roll, right?

So really, really very nice.

And of course, I'd like to really underscore, as I said already before, this is not a one-man show here, this is what you do with the team, right? And I already commemorated Els, and I highlight her here as well, because through all those years, I have worked with Els, very intentionally with all these PhD students here, and some of them are also here. So let's switch gears and now go to the blaming part.

You may wonder, blaming, where is that about, right?

Well, what I noticed, what I found out very quickly, that blaming is intrinsically linked to banana cultivation.

And I will explain you why. So what you see here is the late Romano Orlice. He was the president of the Costa Rica Banana Corporation, and he came to Wageningen University to visit Jesus Torfold, who conducted part of his PhD studies in Costa Rica on his farms.

He was him, you know, is there anybody here in Wageningen who knows something about Michael Srella? And of course, my fungus, or the fungus I worked on, was also called Michael Srella Gramonicula. So I said, oh, well, there was no internet, so we had a lot of phone calls, this and that. Eventually, they came to us, and I went to Pete Bonacombe.

Thanks for being here, Pete.

We had a meeting and said, hey, Pete, there is a banana corporation. They want to know something about Michael Srella. What do you think? Shall we just make time and discuss it with them? I said, yeah, sure, you know, do it. You know, freedom to operate. So that's what we did. And after that talk, Romano said, you know, you've got to come to Costa Rica, and I will show you all the ins and outs of the banana production. And that's how it started. So he invited me to Costa Rica, and for the first time, I was in a banana plantation. Now, I wasn't walking in the crop. I was walking under the crop, you know, those huge plants everywhere shadow.

But these symptoms, you know, so much necrosis, loads of spores, of course, and this fungus is very sexual, almost just sexual reproduction, actually.

But I was very much impressed by the effect of this disease on death crop. Also, knowing that this is such an important crop for, for instance, Costa Rica is an export crop. And what I didn't know then, I have to admit it, what I didn't know then is that it is also a super important food crop.

That's only what I learned later, right? East Africa, millions of people depend on cooking bananas for their daily food.

It's a staple. So this summer, Hana and I went to our sons.

One son lives in Aruba, and the other one lives in Australia. And on the way, we stopped in the Fiji Islands because I thought, pseudoscopes by phygiensis, I worked on this fungus for so many years, I have to be on that island and see it, right? So here you see in the middle photograph, that is Sigatoka Valley. And on the right side, you see the Sigatoka River. So that's where the name comes from, Black Sigatoka.

But I had expected a very wild environment, you know, rushing rivers and all that, forget it. This is an agricultural salad bowl of the Fiji Islands. It's just an agricultural area.

But that's where the name comes from. Maybe not necessarily originating from those islands. There is a debate ongoing, but the name is derived from the Fiji Islands. Now to control that disease, you have to watch this.

Can you still hear me when I speak? Good.

So this is a movie of a farm in Costa Rica. And the bananas that are now cultivated, I tell you a little bit more about later, is Cavendish variety. Those are the varieties that you all buy in a retail store. Super susceptible to this one.

So the only way to get it into the store is to make sure that you spray and not just once or twice, no, 60 times to give. That's what you have to remember, right?

So each and every banana that you eat in the future, remember one third of the cost is disease control.

That's the actual fact, right? And that's where the blaming starts, right? So recently there was a film on NOS, for instance, showing, you know, banana plantations, spraying and all that. Oh, bad, bad. Yes, really bad. But you know, we are part of that story, right? We want to eat bananas. We want them fast. We want them healthy. They should be green. This should be clean, right?

So there's two sides of that metal, right?

Of course, with such an important disease, why is there not anybody who had ever bred a variety that is better than Cavendish? Well, that's what I thought, but it's not that simple. But of course, by spraying all these fungicides, you put a tremendous selection pressure on those populations. And what I mean with it is that the more you spray, the more you push such a population to actually insensitivity to those fungicides. So you are arriving at a tipping point where you just don't know what to do anymore, right? Either stop banana growing or come up with new varieties or new compounds.

But yeah, everybody agrees that we have to reduce pesticide inputs, particularly also in these regions.

But remember, there is another side, and that is us as consumers. Now, we studied that fungus really in detail.

We also sequenced many ice lids, and here you see a cartoon of what we call a pan-genome that is being developed.

So the details are not important here, but what you see is gray bullets.

Those represent gromosomes that are always there, and green bullets are gromosomes that can be missed. All of a sudden, they are gone without any consequence for the lifestyle of the fungus, without any consequence for sexual reproduction or pathogenicity. So we call those dispensable gromosomes or accessory gromosomes. It has been discussed also earlier this morning.

Now, one influential book that I read when I started banana research, that required, of course, a lot of reading, is this book. And this book really describes the banana culture in Honduras, how bananas were exported to the states, how Panama disease, I have to introduce that to you, but believe me, that's a disease in bananas, was affecting that cultivation, and also later on, the introduction of black tikitoka and how that was controlled.

So I once found a movie on the internet, and that is a movie from the 50s in Rotterdam. That's the city where I was born, and where we lived after we married for a couple of years. And I saw this truck. I said, hey, that's interesting. Bananas on the truck and fives bananas.

And Mr. Andrew Biles, the former CEO of Chiquita, who was also in the audience, told us this afternoon that Chiquita bananas came only to the market in the Netherlands, or to Europe in the 66, right?

1966. So this is 50. So these were most likely, well, these are fives bananas, they are still in the market, but these are also most likely the iconic Gros Michel bananas.

You have to remember that name, Gros Michel.

That was then the favorite banana.

But in Central America, that was really absolutely slammed down by that disease that is called Fusarian Wills of Banana or Panama disease.

And eventually, the only way to control that disease was to inundate the land. So that's what you see here. Just put it on the water for a couple of weeks, kills all the fungus, kills the fungus, because it's a soil-borne fungus, sits in the soil, kills it. And then later on, they just plant it again, the same variety.

But some of those varieties or some of those plants were infected. And in those days, you couldn't see that, because there was no tissue culture. It was all reproduced by suckers in Dutch Stecker, right? So you just planted it. And that, of course, boomed the epidemic. So eventually, they had to give up banana production. They had a huge societal impact, unemployment, etc. So here you see the devastating effects of Fusarian Wills.

So the foliage is wilting, because it's a vascular pathogen, enters through the roots, occludes the vascular system, and makes it really to wilt. And then during this process, it's producing billions of spores. And if you look at a cross-section in the middle figure, you see that stem is entirely colonized. Fortunately, the industry ran into one variety that barely survived botanical gardens in the UK, which was collected in Southeast Asia.

And it got the name Cavendish.

So it's important to remember, Cavendish bananas is not a product of a breeding program. It's a wild clone. All of us are eating a wild clone, right?

And that is absolutely resistant to the strains that caused the previous epidemic. You could grow it on the most infested soils, still, without any problem. So remember, if you go to the retail store, you buy bananas, whether that is a ciquita label, demont or dole fives, whatever, it's all Cavendish. Nothing is different, right?

This is one single clone.

So you can imagine how risky that is, right? There's asking, essentially, for problems. So here you see then in a color image the impact of the disease, right? So you see the wilting, you see the cross-section.

And since I started with bananas and on the black cicatoga pathogen, I was really resisting to start working on fusarium, frankly speaking. Like what Ronnie said, I will never work on fusarium. Too complex. But I had two colleagues, and you see them here with the green circles, Teo van der Ley and Kees Wahlweg.

And they introduced me into fusarium. So here we were in a meeting in China. I think we suffered from some jet lag here. But there's also another story to it, but I will keep that case, don't worry. But we started working on fusarium, so they really introduced me into fusarium. I was not part of that community, didn't know too much about it, but very quickly afterwards we started collaborating with Embrapa from Brazil.

And Manuel Sosa, he was leading that group, and they were for five years in our institute. That was before I entered the university. And we had a very, very fruitful collaboration.

So we had many visitors coming, and Miguel Dita, who you see here on the slide, he was the one who was absolutely insisting, I just want to work on fusarium. He said,

okay, then I have to give in. But then let's do something very strategic, because there was now a new fusarium that was killing Cavendish.

So Cavendish used to be the solution, grown over millions of hectares, dominating the export by 95 percent, and the global production by 50 percent, but now all of a sudden being affected by a new fusarium strain that was called Tropic Erase IV. He said, okay, then if we want to be strategic, let's develop a molecular diagnostic for Tropic Erase IV. So instead of months identifying whether the strain is Tropic Erase IV or not, we could do it now, do it in hours. So that really boomed our work. And then together with Keis Jansse, Sizze Velama, Jätze Storovorro, we wrote a research program for what was then called INREF, which is now the Wageningen Global Sustainability Program, and as well as programs for bilateral collaboration with Indonesia. And in these programs, we could really hire quite a number of PhD students, and we really had a fantastic team to really engage on fusarium and banana.

And of course, these students were supervised by the blinking posters that you see. Harold Meyer in the audience, Carolina is back in Colombia, and Michael is in the audience, I guess, as well. Yes, thank you. And now my work is being continued in banana by underscore that this is all teamwork, right? You can't do anything on your own. Sometimes you have a good idea, but you discuss that also with your team. So yeah, and then the students that have red circles are those that have not yet finished. So I will continue to be their promoter, despite the fact that I'm stepping down. And the blue circles, those are PhD students working on bringing them over the finish line.

So these projects also included quite some field work. So here we were on a sampling trip in Indonesia, in Java, where we really talk to growers, understand their social structure.

They don't have big plantations. They just grow bananas in their backyard, small plantations, very diverse.

How do they deal with diseases? What diseases do they have? Where do they sell their bananas? All those questions. So if there's one thing that I learned in banana research, that is interdisciplinarity.

And indirect really gave us the foundation to do that.

So kind of stepping down from a very narrow focus on a particular problem to really seeing the entire picture, and also consider folks, we have a complex structure here. There's people involved, there's crops involved, there's soil, there's weather, all kinds of different pathogens. So I don't know whether I really learned to work interdisciplinary, but it was absolutely a lesson, a road to listen to others, to also realize that, yeah, you can be an expert yourself, but you need other colleagues that are experts in other areas to eventually have a solution. So thanks for that.

It was very important. And I think we have a fantastic foundation for that type of work here at Wageningen University, which in my opinion is still under explored.

So despite the fact, we have so many colleagues around us.

Let me give you one detail of those studies that we did. So Einar Martinez, a PhD student from Cuba, he collected fusarium strains all across Cuba, and we looked at a genetic diversity of those isolates, but then also in a context of Latin America.

And what he discovered is that those strains that killed the growth Bichell bananas that I talked about, the first big Panama disease epidemic in the 1950s, those

strains were actually very diverse, up to a level that we actually considered they are different species, different fusarium species.

And then with another PhD student, Anup van Vesterhofer, and the other team, I underscore that again, she put that collection in a global context. So now we have a global collection of about 500 isolates, and we saw exactly the same.

Raise one strains that caused the previous epidemic, very diverse, but the one that is now killing Cavendish is a single claw. And it is not a mutant from those raise one strains. That's what you expect, right? You have a resistant variety, you put selection pressure, you will have mutants and that will overcome the resistance. No way. This is not the way it works in bananas. TR4 is absolutely a new genetic lineage that is originating from Indonesia and spreads around the world, really.

And how does it spread? Not by natural means. There's another part of the blaming story.

We are spreading it. It's us. We are the vectors. We take plants, we take soil under our shoes.

We really show that I took DNA from the soil of my shoes and found TR4 in that soil.

Of course, it didn't take them in the plane, don't worry, but that's what many people do. So we are part of this whole blaming story. We spread this disease. So it was first found in 1967 already in Taiwan. We found it for the first time in Jordan in 2013. Remember, in 2010, we had a molecular diagnostic. So we had the tools to trace that fungus whenever it showed up.

In 12 years, 17 new countries in all major banana producing regions. And the last one just a few weeks ago in Ecuador, the largest banana exporter of the world. Fortunately, Fuzraim is a slow mover, but it makes these internationally intercontinental jumps, right?

So it's an absolute threat. And if that, for instance, would ever be introduced into Cuba, which is not a pipe dream, you know, it would eradicate 60 to 70% of the varieties that are absolutely indispensable for food security in Cuba. And the photographs you see in the background, and that tells us another story. And there we are to blame. Apparently, we don't tell the story well enough because that is an entrepreneur starting a plantation in the Dominican Republic that should grow to 9,000 hectares.

He'd have never heard of TF4.

They're traveling, they read English, they fly around, and yet they haven't heard of TF4. So we do something wrong, right? We can publish, but we have to do more than that, in my opinion. Now, is this not the end of banana?

Of course not. So we accumulated lots of knowledge and understanding. And when I called Mr.

Andrew Biles, then the CEO of Shakira, and it was taken, that was also what he told us, it was taken from the stock exchange in 2015 and became a family-owned company with an entirely different perspective with patience and also potential funding.

We said, you know, can we organize a fusarium satellite of the International Congress of Plant Pathology in Boston?

And he readily agreed. He said, yeah, let's do it. So Andre Drent from the

university, Professor Andre Drent from the University of Queensland in Australia in Brisbane, and I organized that fusarium satellite in Boston in 2018, and we repeated it in 2023 in New York.

And that was very functional to bring people together working on this same problem. So that is community building, very important.

So thank you, Andrew, for always supporting these initiatives. And Andre and I also said, you know, we were called by publishers and said, can you write a book about bananas? Well, one book, let's write three. So we decided we write a book on cultivation techniques, on germ pleasant diversity and improvement, and pests and diseases.

So that's will be anyway, you know, hopefully in banana offices in the future.

So that was then a little bit about the blaming, right? Let me just finish with one other blame that I was confronted with, which is not always easy. When I showed you this picture, where is it?

This one, we once were also blamed that we were actually bringing TR4 to those other countries, despite the fact that we'd never been in those places, right? And so sometimes you have really have to develop the kind of a thick leather hide to ensure that you can resist this type of accusations. And so blaming, that's what I really noticed in banana production is very, very common. For instance, small growers blame big growers all the time, and also vice versa. You know, you're not containing the disease, or you're not spraying, etc, etc.

So we even had a PhD thesis that primarily focus on the blaming issue, to overcome that social difficulties between small growers and big growers. All right, now let's go to the battle part.

Enough about blaming. So we were very fortunate eventually to be members of the BBB program.

So that's another, that's the same abbreviation, but with a different meaning, breeding better bananas.

And that was an initiative that was written by Professor Ronis Fennon, who is in the audience, who is the expert, I would say, in banana breeding.

He worked his entire life in banana research, and particularly in breeding. He was a professor at University of Leuven, now retired and continues to have the banana breeding program at ITA in Nigeria and Tanzania.

So we were fortunate to be members of the breeding better bananas program. This gave us also opportunity to hire more PhD students. And this program is really focused on the East African Highland bananas.

So that, those are the bananas that are absolutely a staple crop for millions of people in Africa. Yeah.

All right. So then over the years, as the director already mentioned, bananas became what I call the pet crop of Wageningen University. You know, when we started this banana, everyone was kind of raising their eyebrows, say, why, why, why did he actually start working on bananas, right? But, you know, once we started growing them in the greenhouse and saw all those big plants, many, many visitors came over to see those experiments.

And it is such an attractive crop for students. It has an appealing story that also

provides us with enormous podium to showcase what fighter pathology really means in practice, right?

And for the general public too. And still, after all these years and studies and papers, I really had a feeling we have to make a change.

We need to do something more than this. We've been lobbying for banana breeding with all the major companies and just found closed doors all the time.

So that was discouraging, I have to say.

But then in 2018, that was the centennial year of Wageningen University.

And together with uniform and a couple of partners, we decided, let's now grow bananas and let's just continue growing to flowering and to bunch production.

So that's what you see here in this greenhouse. So we had those tall plants reaching the roof of the greenhouse, but we had fantastic branches. And these banana plants were just now substrate. Cocoa peat or rockwool.

And you see rockwool here, you know, that's grown, that's used for tomatoes if you come with, but not for bananas. But yeah, these bananas produce fantastic fruits, right? So that was an absolute success story. And it really made us aware that, hey, if we can grow bananas to flowering in a greenhouse, we probably can also cause them in a greenhouse. So we don't have to go to the tropics to make crosses, we can just do it down here in the Netherlands.

So in 2014, we organized here in Wageningen, Wageningen Banana Day.

That really brought many stakeholders to campus, international and national collaborators.

And Orlando de Ponti, my previous director at IPO, was also there. And we sat on a table having a coffee, and he said to me, you know, Fred, stop talking to all these companies, you just have to start breeding yourself. Just start, right?

Thank you, Orlando, for that final push, because that's what we then eventually did.

So I met with Anker Serenson, who first started to do it himself, and found out, hmm, not a good idea, why don't we collaborate? So we teamed up, and then in 2018, Yellow Way was born, and we started to collaborate. We did lots of studies together, sequencing many, many eyes, etc.

And I have to say this, yeah, is a Dutch banana breeding company.

And we are also very Dutch.

You know, we consider, you know, we can do it better, we can do it faster, let's just do it, right? So we should be the ones who can come up with a replacement of Cavendish.

And in 2021, Chiquita, which was now this family-owned company, came on board and is now the major shareholder in Yellow Way.

And what we do there is knowledge-based breeding, as Anker explained this afternoon. So we sequence everything that is necessary to understand. So we sequence the over 100 banana diploids. We sequence two panels of isolates from the fusarium fungus, causing Panama disease of fusarium wilt, as well as many strains

from pseudosacrospora fijensis.

And bringing all that information together really helped us to kick off a knowledge-based breeding program.

So we phenotype all those accessions. So now we knew which accessions are resistant, which are required to come close to the composition of Cavendish, because we sequenced that as well. So it's a kind of a big, very, very big puzzle, right? So that's what we did. And yeah, as I said, nobody so far has been able to breed a variety that is outcompeting Cavendish, but that is our ambition. But more than that, we just don't want to replace Cavendish by another clone. That's another monoculture. That's not what we want. We want to actually get rid of monoculture. So you open a breeding pipeline, and that continuously delivers new, innovative, genetically different varieties. That's what breeders do. And that's what we have to do in bananas too, right? So that is the aim. So we really want to transform the industry. Huge ambition, but I'm sure we can do it. That may be very Dutch, but we have a fantastic team.

And this is what you see.

We cross accessions in the greenhouse, in Bemel, just a half an hour drive from here. We grow them into flowering.

Anker explained how difficult it is, but yet we do it, and it's super efficient.

So we do it all very close, harvest the seeds, take them into tissue culture, develop populations, map genes, develop markers so that we can get rid of the phenotyping. So it's becoming very, very efficient.

Now, through all these years, I have had a privilege, really, and I'd like to, you know, that's the start of my, toward the end of my farewell lecture. Throughout these years, I had the possibility to develop a fantastic network, working with many, many international colleagues. And that has been a true blessing, really. We gained a lot in know-how and understanding.

And as I said, banana research really taught me that we had to do something more. So that's why Yellow Wave was born. We have to translate all the know-how we have into action, develop new products, and engage, of course, with industry, as well as with smallholders.

So deliver varieties that they can grow. And there are companies, breeding companies, that do a fantastic job and reaching 65 million small growers in Southeast Asia. And as you know, the banana is a top food crop in Southeast Asia. That's the aim, right?

So start companies fast-track the know-how. And as I said, I've been very thankful for that opportunity and for all those collaborators throughout the years. Of course, also our sponsors and collaborators that I really would like to thank.

So I hope you have now got the impression that, yes, I'm not just a banana guy, right? Because there's the label that's always in my back somehow. But, you know, if you look at what we have done and what I could do, I started as a young guy with stripe rust under the guidance of Corphonsel, Falta, and Ron Stokes, then moved to Zymus, Satori, Tritisi, which was maybe the fungus where I learned most things of doing scientific research. It was my PhD thesis. But I could apply that know-how when we moved to banana. Fusarium, thank you, Case and Tao, for that introduction.

And, of course, Judas Koss by Phrygensis, which is related to the microfella fungus or the Zymus Satori fungus in wheat.

So that was in a nutshell 43 years of phytopathology. And what you also have to remember, I only became a professor during the last 25% of that stretch.

So 75% I was working at plant research, right?

That's where my origin is and maybe also, frankly, deep down my heart probably too. But I was appointed as a special professor in tropical phytopathology because of the banana work and very soon afterwards I became an interim chair and later the full chair of the laboratory phytopathology. And I'm very thankful that I had that opportunity as well.

So with first of all, as you said, as we noted, right, I didn't spend my entire life on a single fungus. Fortunately, there were a few. But, yeah, we could continue for really a couple of years, frankly speaking. But first of all, I really would like to thank the business unit by interactions in plant health. That's where my roots lie. We've been working for many, many years and thank you for being with me and being together and for all the things we have done throughout the years. Of course, I also owe a deep gratitude to the laboratory phytopathology for accepting me as their chair for the lessons that we learned and the path for the future that we developed together.

Again, underscoring together, right? So going beyond the only fundamental aspects of plant pathology, placing phytopathology in a more complex real world context with a new focus on major and important problems.

But of course, without dropping the necessary fundamental aspects of what we still need to discover. And we have a very collaborative team. So it was a blessing to work with you guys. Thank you for that opportunity. We celebrated 100 years of phytopathology two years ago together with the business unit, which was fascinating. We have still major problems to solve. But try this in lilies, for instance. If you read the papers, you see that many citizens complain about fungicide applications to keep those lilies clean. But we buy them, right?

So again, you also see the societal context of the work we do. A new program that we just launched in oil palm with very, very important diseases in collaboration with other science group at plant production systems and across the other science group problems in Latin America and Southeast Asia.

Very nice that you could start it.

And under the leadership of that and putting pathology in a more complex context. I'm also looking at dazzling, you know, looking at a microbial world on the foliage in the plant, in the soil.

Absolute fascinating. Finally, of course, I would like to thank my wife, Anna, for the love, despite fix first abandoning me, right, but accepting me as your husband. For all those years, you have supported me and enabled me to build relations, to travel. We were already married and I was still working for my bachelor's. You're still here. And I would say without you, all this would have been totally impossible. So thank you very much.

You can clap your hands. As you notice, it is moving, right? But that's fine.

I would like to thank my children as well for their patience, for me being out there traveling whatsoever, for your love and supporting me. None of you has chosen plant science or something related to biology, which is also fine.

So follow your own interest. That's important, right? So thank you for being there

all these years. And of course, also all the grandkids, even the smallest here among us. And to finish, I have another question to you.

Who knows what this is?

Let me disclose it to you. This is the first page of the printed 95 propositions of Martin Luther. So today, the 31st of October, that is the day where we commemorate the Reformation that was later called the Reformation, the movement that he started. Now he had 95 propositions.

When I did my PhD, I had 17. And I know some of you had even more. Now we are down to six.

Fortunately, we still have them, but just six. Two about your book, two about science in general, and two about society.

But you know, these propositions by Luther, besides reforming the church, they contributed immensely to society and to culture. For instance, Luther used the latest invention to spread his ideas, which was book printing, literally.

That's what he did. So, you know, it swept through Europe. And he was preceded actually by Erasmus.

And I'm proud of Erasmus because he came from Rotterdam, Erasmus Rotterdamus, right?

Yeah, sure. So he was actually the first one who built a Greek translation of the New Testament.

And he wanted to have a low-price copy to spread it as much as possible to convince people to start reading the Bible.

Now, that was perfect Greek. And that translation of the New Testament was the foundation for Luther's translation of the Bible in German. And that was fundamental for the development of German language, like it was for the Dutch language, the translation that we have, that was called the Stadt of Tali. They all go down to this translation by Erasmus in Greek.

And as I said, it also influenced culture immensely. And one of those exponents of culture that really touches my heart is the composer Johann Sebastian Bach.

And what Bach did under each and every composition that he completed, he wrote a three-letter abbreviation, SDG, which says, Soli Deo, Gloria. So I'd like to finish today with that claim. Praise to God alone. Thank you very much.